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Swan Syndrome Following Complicated RGP Lens Removal: a Teaching Case Report

Jenna D. Koskey, OD, FAAO, Dipl. ABO, ABCMO, and Emily M. Ulry, OD | Optometric Education: Volume 49 Number 1 (Fall 2023)

Background

Swan syndrome is a rare condition first discovered in the 1970s. It occurs in patients with history of cataract extraction via scleral incision. Patients present with hyphema, vitreous hemorrhage and potentially angle neovascularization. Swan syndrome is often a diagnosis of exclusion, requiring careful case history including review of past surgeries, injuries and systemic disorders, and lab testing. Treatment and management of patients with Swan syndrome are highly dependent on severity of findings as well as recurrences. Patients should be managed closely to ensure proper treatment is initiated if necessary.

Case Description

History

An 84-year-old Caucasian male presented to clinic with decreased vision in a red, painful left eye (OS). The patient reported that symptoms began 4 days prior following difficult removal of a rigid gas permeable (RGP) contact lens OS. After unsuccessfully trying to remove the lens, the patient reported sleeping with the lens in the eye for 2 nights. His symptoms began on the third day after he finally succeeded in removing the lens. His symptoms of pain and redness persisted after he removed the contact lens, which prompted his visit to the eye clinic. He denied any additional ocular or head trauma.

Most notably, the patient's ocular history OS included penetrating wire trauma requiring lens extraction and cryotherapy in 1980, which resulted in aphakia, chorioretinal atrophy and pannus and corneal edema secondary to RGP contact lens overwear. The patient's ocular history included pseudophakia in the right eye (OD). He had controlled type 2 diabetes mellitus since 1998 with no history of diabetic retinopathy or clinically significant macular edema in either eye.

In addition to type 2 diabetes mellitus, the patient's medical history was positive for hypertension, hyperlipidemia, chronic kidney disease, hypothyroidism, atrial fibrillation, stroke, nocturnal aortic stenosis, vitamin B12 deficiency, age-associated memory impairment, dysphasia, seizure disorder, obesity, insomnia, osteoarthritis, remission for alcohol dependence and tremor. Medication use included insulin, amlodipine besylate, clonidine, hydrochlorothiazide, metoprolol, rosuvastatin, ezetimibe, levothyroxine, apixaban, omeprazole, aspirin, cyanocobalamin, thiamine, iron, folic acid, levetiracetam and sertraline.

Examination findings

The patient presented with 20/25 visual acuity OD and light perception OS (no improvement with pinhole). His vision was previously correctable to 20/25-2 OS. Consistent with records from the patient's last eye exam, the pupil OD was round and reactive to light, and the pupil OS was fixed (due to history of trauma). No afferent pupillary defect was present. Confrontational visual field testing results were full to finger counting OD and full to light perception OS. Extraocular motility was full in both eyes (OU), and tonometry (iCare) showed intraocular pressure (IOP) of 14 mmHg OD and 22 mmHg OS.

Slit lamp examination OD was remarkable for 2+ meibomian gland disease. Slit lamp examination OS was remarkable for 2+ meibomian gland disease, trace diffuse bulbar conjunctival injection, and trace bulbar conjunctival edema. The cornea had 3+ diffuse edema with guttata and 3 clock hours of pannus and neovascularization **(Figure 1)**. The anterior chamber had a 1-mm tall hyphema **(Figure 2)** and anterior chamber reaction of 4+ cells and 1-2 flare. Clear and complete visualization of the iris was challenging due to the hyphema obstructing the inferior iris, 3+ corneal edema and 4+ cells and 1-2 flare anterior chamber reaction, making it difficult to completely rule out iris neovascularization. Posterior structures were also difficult to assess due to the corneal edema, hyphema and anterior chamber reaction. Therefore, a B-scan ultrasound was performed. No masses or retinal detachments were detected **(Figure 3)**. Fundus examination and gonioscopy were performed only OD due to the presence of the hyphema OS. No abnormal vessels in the angles, angle neovascularization or other abnormalities were noted OD.
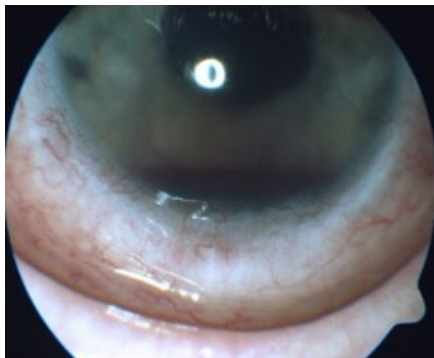
Figure 1. *Hyphema, 3+ corneal edema, pannus and corneal neovascularization of the left eye at initial visit.* *Click to enlarge*
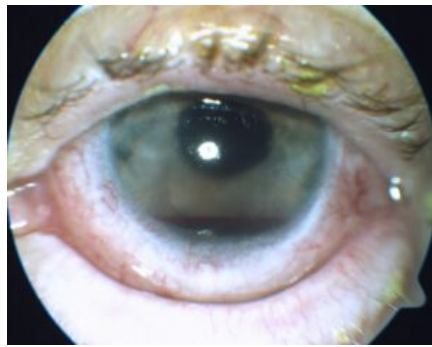

Figure 2. *Hyphema in the left eye present at initial visit.* *Click to enlarge*
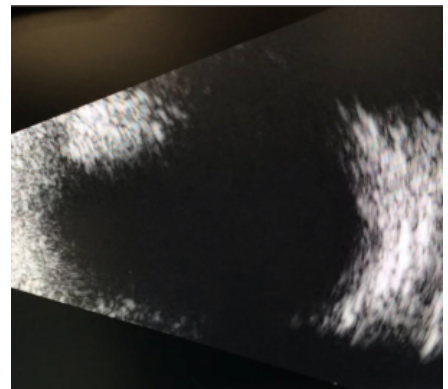

Figure 3. *B-scan ultrasound of the left eye on the day of patient's initial presentation to clinic. Note the absence of retinal detachment or masses.* *Click to enlarge*

Initial treatment

To quell the anterior chamber reaction, the patient was prescribed 1% prednisolone acetate to be used every hour OS and 1% atropine to be used 3 times per day OS. The patient was instructed to stay on bed rest with head elevation of at least 30 degrees at all times, avoid any type of straining, and wear a protective eye shield full-time OS to promote resolution and avoid re-bleeding of the hyphema. Contact lens wear OS was to be discontinued until further notice.

Out of an abundance of caution, a cornea culture was obtained OS. The patient's recent blood test results were reviewed. Additional blood tests — for protein C and protein S, herpes simplex virus (HSV) and sickle cell anemia — were ordered to rule out systemic diseases with known ocular complications and to gain a better sense of the patient's systemic health.

Follow-up

The patient was monitored closely during the following weeks, and the hyphema, anterior chamber reaction and corneal edema gradually resolved **(Figures 4 and 5)**. The hyphema cleared 6 days after initial presentation, while the anterior chamber reaction cleared 10 days after initial presentation. When the anterior chamber was free of cells and flare, the topical steroids were tapered to prevent rebound inflammation. The patient went from one drop 1% prednisolone acetate every hour OS (~ 12x/day) to 8x/day for 1 week, 4x/day for 1 week, 3x/day for 1 week, 2x/day for 1 week, 1x/day for 1 week and then discontinuation. The patient returned to clinic at each 1-week interval to be evaluated for rebound inflammation before continuing taper of the steroid. Atropine 1% OS was also tapered when the anterior chamber reaction resolved, decreasing from one drop 3x/day to 2x/day for 1 week to 1x/day for 1 week and then discontinuation. Toward the end of the prednisolone and atropine tapering, approximately 6 weeks after symptom onset, the patient's best-corrected visual acuity (BCVA) improved to 20/25-2 OS.
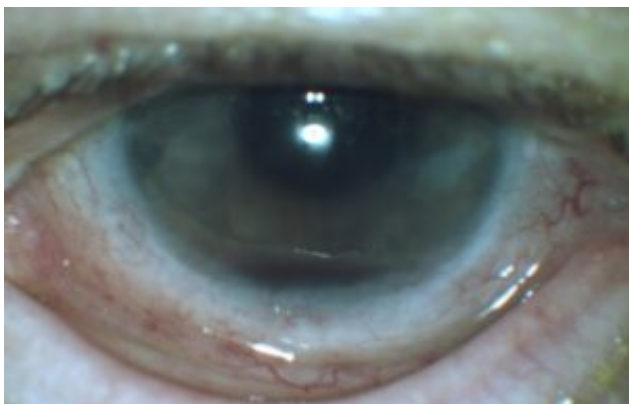

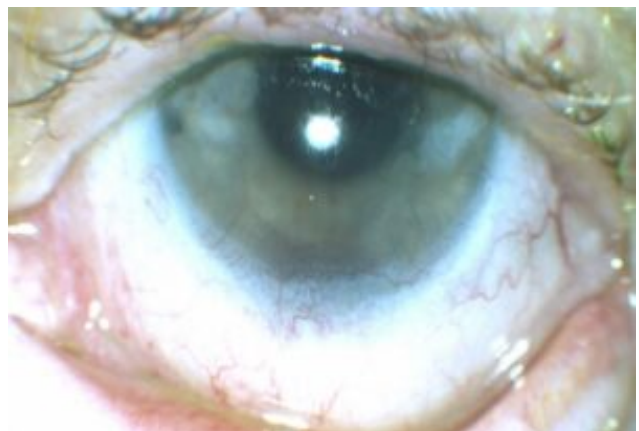***Figure 4.*** *Hyphema in the left eye at the 2-day follow-up visit.* *Click to enlarge*

Figure 5. *Hyphema in the left eye at the day-3 follow-up visit. Note the steady resolution of the hyphema.* *Click to enlarge*

Gross fundoscopic view of the posterior chamber was possible approximately 1 week from presentation and revealed a vitreous hemorrhage OS. The hyphema completely resolved 6 days after initial presentation; however, gonioscopy was not performed until 1 month after resolution of the hyphema to prevent triggering a re-bleed. Gonioscopy was performed OS and was open to ciliary body band in all four quadrants. No abnormal vessels in the angles, no angle neovascularization, or other abnormalities were noted.

Upon discovery of the vitreous hemorrhage, the differential diagnosis list expanded to include the following: Swan syndrome, ischemic proliferative diabetic retinopathy, ocular ischemic syndrome, central retinal vein occlusion, traumatic vitreous hemorrhage and spillover hyphema due to RGP lens removal, sickle cell retinopathy, herpetic eye disease and leukemic retinopathy. Although diabetic, sickle cell, herpetic and leukemic retinopathy may present with asymmetry, these conditions most often manifest bilaterally. As a result, these conditions moved lower on the differentials list. A clear view of the retina OS was possible 1 month after initial presentation, and no retinopathy was noted.

Laboratory test results

The cornea culture came back negative for white blood cells, organisms or growth in 72 hours. No abnormal hemoglobin or sickle cell traits were detected. Normal protein S and protein C levels and normal partial thromboplastin time (PTT) suggested a normal coagulation system. HSV-1 and HSV-2 were not detected.

Prothrombin time (PT) and international normalized ratio (INR) were elevated, indicating the patient's blood takes longer than normal to clot, but this was confounded by his use of anticoagulants aspirin and apixaban. Elevated vitamin B12 ruled out pernicious anemia and was explained by the patient taking vitamin B12 supplements. Interestingly, the patient's HbA1c rose from 6.3% to 8.7% in 6 months, which suggests poor control of type 2 diabetes.

Treatment and management

Swan syndrome surpassed diabetic retinopathy as a potential diagnosis when a clear view of the fundus was possible (~ 1 month after initial presentation) and no retinal hemorrhages, cotton wool spots or exudates were noted in either eye. The negative cornea culture and lab findings further supported the diagnosis by ruling out the remaining differentials. The diagnosis of Swan syndrome was confirmed after review of the lab results, the patient's history of ocular trauma and aphakia, and the findings of vitreous hemorrhage and hyphema.

The patient's hyphema and vitreous hemorrhage both completely resolved in 1 month. This was the first occurrence of hyphema and vitreous hemorrhage in the patient's history. The plan was to continue to monitor him for re-bleeds or recurrences on a 3- 6-month follow-up schedule. The patient was thoroughly educated regarding cessation of contact lens wear and to report to clinic immediately if any signs or symptoms suggested re-bleeding. If recurrences do occur in this patient, referral for surgical intervention would be considered.

Refractive correction

Once the hyphema, anterior chamber reaction, corneal edema and vitreous hemorrhage resolved and was stable, approximately a month and a half after initial presentation, refraction was performed again. The updated refraction was OD: +1.00-0.25×090 with BCVA 20/25, and OS: +12.25-0.50×108 with BCVA 20/25-2.

The patient had been aphakic for more than 30 years and had a long history of both soft and RGP contact lens wear. In the past, he had worn a single-vision soft contact lens OS but switched to an RGP lens for better clarity and easier handling. His most recent correction consisted of bifocal glasses to correct distance vision OD and near vision OU, worn over an RGP contact lens for distance correction OS.

Due to the patient's history of soft and RGP lens overwear, poor lid hygiene and concurrent ocular pathology, continued contact lens correction was avoided. Scleral lenses were considered but not a great option due to the diagnosis of Swan syndrome and the possibility of a pinched anastomosed vessel causing a re-bleed. Spectacle correction was therefore the only option, but it presented a unique challenge due to the marked anisometropia resulting from pseudophakia OD and aphakia OS. The patient also expressed a strong preference for a single pair of glasses as opposed to separate pairs for distance and near.

To minimize aniseikonia when placing the spectacle order for this patient, it was important to specify equal base curves and slab-off prism along with 1.74 high-index lens material. A request was made to the lab to place the bevel forward on the higher plus left lens and further back on the less plus right lens in order to manipulate vertex distance and reduce disparity in power factor magnification between the two lenses. In addition, the patient was encouraged to select smaller frames to minimize weight and lens edge thickness. The adjustments to lens, frame and material should mitigate negative effects of aniseikonia for

the patient.

Education Guidelines

Learning objectives

At the end of the case discussion, participants should be able to:

1. Recognize the key features of Swan syndrome
2. List differential diagnoses for hyphema
3. List differential diagnoses for vitreous hemorrhage
4. Describe the epidemiology, key signs and risk factors associated with Swan syndrome
5. Describe the treatment options for Swan syndrome
6. Describe the treatment options for patients with aniseikonia due to aphakia
7. Properly educate patients on prognosis and long-term management of Swan syndrome

Key concepts

1. Identifying Swan syndrome in patients with recurrent hyphemas and/or vitreous hemorrhages
2. Differential diagnoses for hyphema
3. Differential diagnosis for vitreous hemorrhage
4. Treatment options for patients with aniseikonia secondary to aphakia post trauma
5. Delivering clear education to the patient regarding diagnosis, treatment and long-term management of Swan syndrome

Discussion points

1. Knowledge, facts and concepts required for critical review of this case

 a. Describe the epidemiology, pathophysiology and risk factors associated with Swan syndrome
 b. Describe the key clinical findings in patients with Swan syndrome

2. Differential diagnosis

 a. Know characteristic signs of Swan syndrome
 b. List appropriate clinical diagnoses for hyphema
 c. List appropriate clinical diagnoses for vitreous hemorrhage

3. Disease treatment and management

 a. What are the benefits of closely monitoring this patient?
 b. What is the standard of care for treating a patient with Swan syndrome?
 c. Discuss the most likely prognosis and possible complications following treatment of a patient with Swan syndrome
 d. What is the appropriate long-term management plan for a patient with Swan syndrome?

4. Patient education and communication

 a. How would you educate the patient regarding the suspected diagnosis of Swan syndrome?
 b. How would you educate the patient regarding the hyphema?
 c. How would you educate the patient regarding the vitreous hemorrhage?
 d. How would you educate the patient regarding long-term follow-up care and management?

5. Critical thinking

 a. How would you have managed this case? Justify your answer based on the findings
 b. What would have been a sign of poor prognosis?

Discussion

Teaching instructions

This case report is intended for optometry students, residents and clinicians with an intermediate knowledge base of ocular

anatomy, anterior and posterior segment ocular health, ophthalmic optics, pathophysiology and pharmacology. The case report can be taught in a variety of settings including formal academic classrooms, virtual education platforms or small-group clinical settings. The learning objectives above can be assessed through multiple modalities, including multiple-choice TurningPoint or formal exam questions in an academic setting, multiple-choice pop-up questions in a virtual format, or open-ended discussion questions in small groups.

In a formal academic classroom setting, the case can be presented in full as a traditional PowerPoint lecture with detailed explanations of differential diagnoses, epidemiology, pathophysiology, signs/symptoms, risk factors, treatment and management options. Understanding can be evaluated through open-ended questions to the class or multiple-choice questions posed electronically via TurningPoint or written as an exam. Students can be encouraged to practice critical-thinking skills by suggesting alternative treatment or management of the case.

In a virtual education setting, the case can be presented in full as a traditional PowerPoint presentation with audience participation multiple-choice questions incorporated throughout to gauge comprehension and understanding. Questions would include key concepts such as risk factors, clinical findings, differential diagnoses, testing interpretation and treatment and management options. Students would be evaluated on ability to determine appropriate ancillary testing, testing and laboratory results, treatment and follow-up. Feedback would be provided immediately as detailed explanations of why the correct answers were correct and other answers were incorrect.

In a small-group clinical setting, the case can be discussed in sections to promote independent thinking and decision-making. For example, presenting the case as though the patient just walked into the student's clinic with a red eye, decreased vision and hyphema, what are the appropriate tests to perform? Students can be encouraged to have an open discussion on how they would have handled the exam and determined appropriate testing, lab work and management of the patient. In addition, discussions could involve a management plan not addressed in the case but supported by other clinical testing and ideas.

Background

The term Swan syndrome refers to intraocular bleeding from abnormal limbal wound vessels secondary to crystalline lens or cataract extraction involving a scleral incision resulting in hyphema and/or vitreous hemorrhage. First described in the literature by Dr. Kenneth Swan in the 1970s, these instances of often-recurrent bleeding occur later in the postoperative period, months to years after surgery.

Diagnosis and clinical findings

Patients with Swan syndrome present with acute onset blurred or blocked vision, pain and blood in the front of the eye. Patients typically report no recent history of conspicuous ocular or head trauma. Clinical signs include blood in the anterior chamber, vitreous or anterior chamber angle.

The key clinical findings in patients with Swan syndrome include aphakia or history of cataract surgery involving a scleral incision, hyphema and vitreous hemorrhage. Gonioscopic examination of multiple patients with shared clinical history and presentation often exhibited focal areas of abnormal, leaky blood vessels near surgical incision sites, but these abnormal vessels may not be seen on gonioscopic examination of every patient. Postmortem histological analysis of eyes has revealed episcleral vessels extending deep into stromal wounds and terminating as fragile, leaky capillaries.[1]

A variety of triggers of bleeding episodes in cases of Swan syndrome have been reported, such as riding on a boat[2] or vomiting.[3] In some cases, there has been no apparent trigger.[2,4] In the case of the patient in this case report, bleeding likely occurred due to the minor trauma of a difficult RGP lens removal due to poor dexterity.

Differential diagnosis

Swan syndrome is a diagnosis of exclusion; therefore, more common known causes of hyphema and vitreous hemorrhage must be ruled out with thorough patient history, clinical examination and blood testing.

A positive history of crystalline lens extraction is a key element in making the diagnosis of Swan syndrome. It is also important to ensure that the patient has not undergone a recent significant major trauma, such as a blow to the head or globe. Careful fundus examination is necessary to confirm the absence of retinal breaks, retinal neovascularization and other significant retinopathy. Laboratory blood tests should be ordered to rule out the various hematologic disorders mentioned below.

The differential diagnoses of hyphema include trauma, uveitis-glaucoma-hyphema syndrome, juvenile xanthogranuloma, leukemia, child abuse, lens-extraction surgery sequela, Fuch's heterochromic iridocyclitis, sickle cell disease and rubeosis

irides.[5,6]

The differential diagnoses of vitreous hemorrhage include retinal break, trauma, retinal neovascularization, posterior vitreous detachment, ruptured microaneurysm, retinopathy of blood disorders, Valsalva retinopathy and Terson syndrome.[5]

Management and treatment

Patients with hyphema and vitreous hemorrhage, especially those with recurrences, must be made aware that it is critical to determine underlying etiology so appropriate follow-up care and treatment can be recommended. They should also be educated about side effects and potential risk factors for recurrence. Patients should expect to have thorough ocular health examinations, lab tests and frequent follow-up visits initially to monitor for changes and improvement of hyphema and vitreous hemorrhage.

Once a diagnosis of Swan syndrome is made, patients must be thoroughly educated about the potential for recurrences of hyphema and/or vitreous hemorrhage, which can be exacerbated by trauma or other factors, including those that contributed to the initial episode. Patients should know to seek care promptly for eye pain, redness or acute vision loss. Each patient should be made aware of the causative abnormal limbal wound vascularization, as well as treatment options for both acute and recurrent episodes of hyphema and vitreous hemorrhage.

A variety of treatments have been applied to patients with Swan syndrome. Goniophotocoagulation with argon laser has been performed in some cases with varying degrees of success. For example, some patients did not experience a re-bleed during follow-up periods ranging from 36 months to 5 years following a single photocoagulation treatment, but others required goniophotocoagulation re-treatments when vitreous hemorrhage or hyphema recurred.[2,3] Other approaches, including re-opening of the surgical incision to perform cryopexy or diathermy on the leaky blood vessels or direct surgical excision of wound blood vessels, have been reported in the literature as well.[1,7] Efforts to suture across the limbal incision site to close any remaining wound gap have also been attempted, but may introduce a large amount of astigmatism.[4]

Ultimately, multiple treatment methods may be required on a case-by-case basis. For instance, Chen and Kwon described a case of Swan syndrome in which the patient did not show a reduction in bleeding episodes after gonioscopic laser photocoagulation. Their next approach was to re-open the surgical incision so that limbal wound vessels could be cauterized. The patient continued to have frequent hemorrhages, however, and sutures were placed across the incision site to increase wound apposition. This reduced the frequency of the patient's hemorrhages enough to warrant careful observation without further surgical intervention.[4]

Aside from direct treatment of wound neovascularization, hyphema and vitreous hemorrhage must also be managed in Swan syndrome patients. Patients with hyphema should be instructed to wear a protective eye shield, sleep with their head elevated at least 30 degrees and, if possible, avoid blood thinners until the hyphema resolves. Topical steroids and cycloplegic agents should be used to eliminate any accompanying anterior chamber reaction and stabilize the blood-aqueous barrier. More invasive clinical examination techniques such as gonioscopy and scleral depression should be avoided until at least 1 month after resolution to prevent a re-bleed.

If IOP is elevated due to hyphema or topical steroid use, IOP-lowering agents may be used. Pilocarpine and prostaglandin analogues are contraindicated due to their pro-inflammatory nature and destabilization of the blood-aqueous barrier.[8] Additionally, patients with underlying sickle cell disease should not be treated with acetazolamide or other carbonic anhydrase inhibitors to lower IOP. These agents may promote sickling of red blood cells in the anterior chamber, potentially obstructing trabecular aqueous humor outflow, because they increase ascorbate concentration and lower the pH of the aqueous humor.[6]

The prognosis for vision recovery following hyphema is generally good with approximately 76% of patients achieving recovery of 20/50 or better (if not a total hyphema).[9] However, patients must be monitored for future angle recession (if traumatic etiology) and red-cell/ghost-cell or uveitic glaucoma.[5]

As in hyphema management, instructing the patient to maintain head elevation, rest in bed and avoid anticoagulants when possible may promote settling and resolution of the vitreous hemorrhage. Pars plana vitrectomy should be considered for idiopathic vitreous hemorrhage persisting more than 6 months, a non-clearing hemorrhage in a diabetic patient lasting longer than 1 month, and in other situations involving an intractable elevation of IOP, reduced visual acuity in the fellow eye, or when a retinal break or detachment is suspected.[5]

Finally, protective eyewear with durable frames and impact-resistant lens material, such as polycarbonate, should be strongly recommended to patients with Swan syndrome to protect their eyes from trauma that may trigger re-bleeding.

Significant anisometropia with resultant aniseikonia complicates quality of vision in any patient. Patients with sufficient ocular surface health and gentle dexterity will likely get better reduction in aniseikonia symptoms with contact lens correction than with spectacle correction. This is because contact lenses have a small center thickness and vertex distance, which reduce differences in shape and power factors of magnification, respectively. However, for patients with symptomatic aniseikonia for whom contact lenses are not an appropriate option, strategic manipulation of the base curve, center thickness and vertex distance of each spectacle lens can help reduce the difference in magnification between the two lenses. This principle is known as iseikonic lens design.[10] Additional treatment options for aniseikonia include refractive surgery, clear lens exchange, secondary intraocular lens placement or occlusion.[11]

Fortunately, surgical incision wound vascularization with subsequent hyphema and/or vitreous hemorrhage has not been reported in eyes that have undergone more modern cataract surgical techniques involving clear corneal incisions.[3]

Conclusion

The patient in this case report was diagnosed with Swan syndrome based on his ocular history of trauma resulting in aphakia, and exam findings of hyphema and vitreous hemorrhage with no other significant concurrent pathology. The case not only demonstrates a rare condition, but it also highlights the importance of pursuing multiple differential diagnoses to arrive at a clear diagnosis. Patients with suspected Swan syndrome should be monitored closely not only during initial diagnosis but routinely after resolution of hyphema and/or vitreous hemorrhage to determine whether additional treatment is necessary.

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Theme Edition to Focus on Global Optometric Education

We are pleased to announce a theme edition of the journal that will be dedicated to global optometric education. We welcome manuscript submissions that highlight research, curricula, pedagogy, public health initiatives and other projects that align with the theme edition's mission of sharing ongoing efforts to advance the profession of optometry worldwide.

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The submission deadline for this theme edition is January 2024. Send your cover letter with an intact and blind copy of your manuscript to submissions@opted.org. Email *Optometric Education* Editor Keshia Elder, OD, MS, MS, FAAO, if you have any questions about the theme edition.

Using an Expert Simulated Patient Strategy to Teach in the Low Vision Optometry Clinic

Sharon Oberstein, PhD, SFHEA, and Elizabeth A. Beckmann, PhD, PFHEA | Optometric Education: Volume 49 Number 1 (Fall 2023)

Introduction

Clinical experience is important in healthcare teaching, including optometry, to ensure future practitioners have professional competencies.[1-4] Face-to-face patient consultations enable optometry students to consolidate their theoretical knowledge and show how they can apply it to authentic experiences with patients.[5-7] Opportunities for clinical experience are provided through external placements in private practices, hospitals and organizations, and internal placements at the University of New South Wales Optometry Clinic (UNSW-OC). In the latter, qualified experienced optometrists supervise senior optometry students as they provide free optometric examinations to patients who are often referred with complex visual conditions. These authentic clinical interactions ensure high-quality learning experiences for students, while the one-on-one or similar ratios support high-quality patient care. In addition to primary care clinics, the UNSW-OC provides clinics in advanced care fields such as low vision (LV), pediatrics, ocular pathology, color vision and contact lenses. These clinics enable patients to be assessed by optometrists with knowledge, experience, research-based expertise and access to specialized equipment in distinct areas of optometry. The UNSW LV clinic (the main context for this paper) caters at an expert level to the specific needs of people with vision impairment. Patients are referred from across Australia and overseas.

Teaching clinics present notable management and pedagogical challenges to ensuring equitable and adequate learning opportunities for all students, especially in advanced care fields. The naturally uncontrollable aspects of this teaching context are exacerbated when patients are "no-shows," i.e., canceling with late notice or missing scheduled appointments. These unplanned shortfalls in the patient pool impact student learning opportunities, clinic staff costs, and satisfaction and long-term commitment among sessional clinic instructors. Since 2019, this complex situation has been addressed at UNSW-OC by the development and use of an expert simulated patient (ESP) strategy. This is a protocol whereby optometry educators use their expert knowledge of the field alongside access to detailed patient case histories to role-play a specific patient for a student in a clinical learning environment. The ESP strategy is an innovative extension of existing clinical teaching concepts. For example, "expert patient" or "patient as teacher" approaches use patients who are knowledgeable about their own diagnosis and treatment to provide feedback to students.[8,9] The "simulated/standardized patient" model uses volunteer or paid actors trained to act as real patients in simulating symptoms or problems in clinical education settings and to give professional feedback to students.[10-12] Though influenced by these models, the ESP strategy differs by training supervisors to role-play specific real patients, including those patients' relevant complexities. This provides highly authentic clinical learning and enables seamless continuity in teaching in the event of late cancellations or no-shows.

By early 2020, the ESP strategy was in place at UNSW-OC and already indicating successful outcomes. Students could be effectively assessed even in "no patient" situations, and students and staff were reporting satisfaction. Then, as happened across the world,[13] UNSW-OC had to be closed because of the COVID-19 pandemic, and the university shifted to online learning. For approximately 2 years, until mid-2022, the clinic experienced only short periods of "normal" functioning when personal protective equipment was used under strictly enforced social distancing.[14] As with other universities, the clinic's closure naturally put at risk optometry students' learning and graduation.[13,14] Students seemed unlikely to achieve the required clinical experience or receive effective assessment of their practical clinical knowledge, which would impact negatively on final course grades. Fortunately, the ESP strategy provided an outstanding mechanism with which to give students authentic clinical learning experiences even when all learning activities had to be mediated online. The ESP approach met the requirements for assessment of face-to-face skills and ensured students were ready to deliver face-to-face patient care as soon as the clinic re-opened. The ESP approach quickly became the clinic's default approach throughout the closures necessitated by Sydney's lockdowns in 2020 and 2021, enabling all students to have clinical experiences and assessments and graduate as planned.

This paper describes both the developmental thinking behind the design of the ESP strategy and some of its successes and challenges in the past 4 years, including in virtual settings during periods of online teaching. The outcomes suggest this approach could be used to great advantage in other clinical optometry teaching contexts.

The Constraints of Optometry Teaching Clinics for Advanced Care Fields

University optometry clinics worldwide share the need to balance at least 6 key components of effective functioning: safe

patient care, excellent education that meets national/international standards for accredited competencies, employment and development of professional optometrists working as sessional/contracted staff, purchase and use of specialized equipment, full compliance with all university policies and procedures relevant to student learning in professional clinical contexts, and maximizing student well-being. Ensuring that students experience advanced care clinics is essential from both educational and professional perspectives. A survey of optometrists providing gerontology and LV care found up to one-third of respondents provided such services after having experienced their teachers' specific knowledge, passion and empathy in these fields.[15] Conversely, optometrists who reported lacking the confidence to provide gerontology and LV services had not experienced any university learning with elderly or LV patients. It is thus in both the profession's and the community's best interests to facilitate student learning in diverse advanced care clinics.

As a teaching clinic, UNSW-OC is constantly dealing with management and pedagogical challenges. Ongoing effort ensures the availability of multiple highly skilled supervisors (instructors) who are appropriately expert in their specific field(s) of optometry and in teaching. These supervisors must be professional optometrists who can effectively guide, instruct, assess and inspire students to meet patients' specific needs and complexities.

The clinic's employment strategy must ensure maximum patient care and safety through low patient-to-supervisor and supervisor-to-student ratios. At UNSW-OC, the ratios in most clinics are one patient per student, two to three patients per supervisor, and two to four students per supervisor. This gold-standard teaching model is naturally expensive as it must constantly be adjusted based on the limited availability of optometry professionals, and it must ensure their professional development in advanced care clinical teaching skills.

In the UNSW-OC advanced care LV clinic, patients referred by their home optometrist usually have complex conditions and circumstances and have often traveled long distances. Having students work in pairs and focus on teamwork capitalizes on these special learning opportunities. Clinic managers pay careful attention to patient quotas and logs to create equitable access to a variety of cases, diagnoses and case difficulties for all students. Nevertheless, patient case histories, diagnoses and treatments are unpredictable, and many aspects of patient consultations remain unforeseeable, including final diagnosis, case complexity and outcome.

Patient attendance at LV clinic appointments is a key issue. The older age and comorbidities of many patients with LV and the added complications around COVID-19 since 2020 result in an increased likelihood that in each scheduled LV clinic at least one patient will cancel late or fail to arrive. As many patients rely on others to transport and accompany them, appointments canceled at the last minute can rarely be filled. In our LV clinic context, this can mean an annual no-show rate of up to 38% (n = 150) of scheduled patients. The logistical challenge of coordinating expert staff, specialty equipment and students makes rescheduling student-patient experiences difficult. Delivering additional tutorials can provide students more contact with supervisors but does not replace patient consultations. No-shows thus pose a serious problem in clinical teaching, with many students potentially missing authentic learning experiences and supervisors' formative feedback on clinical skills before summative examinations. Optometry students are likely to have anxiety in clinical environments, heightened by clinic schedules and fears of not meeting expectations or being unprepared.[6] Patient no-shows and consequent lost learning opportunities add to their stress burdens. No-shows also result in financial losses for the clinic because teaching supervisors already on site must still be paid.

Clinical Teaching: the Scope of Patient Role-plays

From the pedagogical perspective, an ongoing challenge in clinics is to ensure optometry supervisors can inspire by giving detailed feedback on students' clinical skills, techniques, use of specialty equipment and behavior as optometry professionals.[6] Given the importance of evidence-based practice and clinical-thinking and reasoning, supervisors must facilitate development of students' competence in clinical judgment within complex professional practice through conscious decision-making.[4,16,17] In this context, the success of the objective structured clinical examination (OSCE) in assessing clinical skills is well-known.[18] As many OSCEs require human "targets," optometry has adopted the use of both simulated and standardized patients common in other healthcare professions. These actors or volunteers are trained to present the signs and symptoms of real cases, give consistent verbal and behavioral responses to the person doing the examination/consultation, and provide feedback enabling standardized assessment.[12,19-21] The UNSW School of Optometry and Vision Science has long used simulated patients in OSCE-style examinations.

The conventional method of using simulated or standardized patients in clinical teaching and assessment is to identify paid or volunteer healthy individuals (often senior students in the same or related fields) and train them to act as patients. They learn scripts, lists of symptoms, particular case histories and ways to respond to students' questions.[21,22] However, this approach generally adds complexities for clinic managers. Honoraria or wages need to be paid to volunteers, actors or casual staff, and additional administration costs may be incurred. Also, the quality of training given to the simulated patients influences the

overall impact of the learning experience, and the well-being of the actors/volunteers must always be considered.[23]

Clinics can address some of these complexities by utilizing technology. Simulations have thus become more accessible worldwide in education contexts, even pre-pandemic, with digital interactive simulations, or virtual patients, being used to train clinical reasoning in medicine, nursing and optometry. Good design requires relevancy, support for a case's complex or difficult aspects, interactivity and effective assessment.[24] However, developing and maintaining technology-based virtual simulations remains costly, and their effectiveness as teaching tools is not fully proven. For example, one study of clinical decision-making found optometry students generally overestimated their skills in selecting the appropriate question or test, recognizing critical symptoms or signs, or appreciating referral urgency, but could not improve their self-assessment through experiences with virtual patients.[25]

Developing and Implementing the UNSW-OC Expert Simulated Patient Strategy

The ESP strategy is an approach whereby the simulated patient is role-played by an expert optometry professional who is also the supervisor (teacher, instructor) in the clinical optometry education context. The ESP acts physically as the patient, for example, allowing physical examinations of eyes and vision and responding to students' questions. The ESP uses an actual patient's record card (anonymously extracted from the clinic's comprehensive database) as the script to guide every part of the consultation authentically. As required by the specific teaching context and the individual student, the ESP strategy allows the instructor to shift in and out of the patient role as necessary, enabling the ESP to simultaneously provide and formatively assess the clinical experience.

Pilot testing of the ESP strategy in the LV clinic led to a decision to stay faithful to a single patient's case history. Initially the lead clinical supervisor constructed each case scenario for the relevant ESP by combining relevant aspects of different patients' case histories to create an ideal patient in terms of the clinical learning experience it would afford students. Students completed a LV record card on the constructed patient and were given feedback on their competency in the required tasks. However, students could not use these experiences as case reports because the artificial construction meant some patient data was mismatched, which could confuse students with limited clinical experience and no authentic explanations. While mismatching can happen with real patients, authentic explanations are possible. For example, a real patient with LV might be prescribed an inappropriate strength magnifier because they prefer its portable design or cheaper cost.

As the pilot testing showed constructed cases made the student-patient clinical encounter less authentic, we shifted the ESP protocol toward using a very close rendering of the history of an existing patient, based on that patient's record card, recently examined in the clinic. This approach was immediately found to be more effective for both the ESP/instructor and the student, and has become the norm. Role-playing a specific patient is therefore the crux of the ESP teaching strategy. The patient is not created based on the professional's experience, nor are different patient histories merged, nor are comorbidities simplified. Instead, the ESP sticks to the case history as recorded, including any noted behaviors, concerns, anxieties and medical and genetic history. The de-identified patient's record card becomes the script for the ESP during the clinical examination by the student. In response to the student's case history and examination questions, the ESP describes the real patient's vision, current life situation and clinical results. Key facts are never fabricated or constructed, keeping the simulation more authentic. For example, if the actual patient reported having left their medical history at home, the student would have to navigate the patient's history and examination without a list of conditions or medications, an authentic scenario. The privacy and confidentiality protocols required of staff and students for ESPs are the same as for real patients, and the patient whose case history is used always remains anonymous to the student.

In terms of clinical professional training, simulations must authentically replace all aspects of the live patient-optometrist interaction. For example, experienced optometrists know that real LV patients may forget what medication they are taking, may be confused about comorbidities (or fail to mention them until late in a consultation when they are more trusting of the optometrist), and may be highly anxious about the consultation's outcomes (for example, no longer meeting requirements for a driver's license).[26] The ESP protocol allows these attributes to be included authentically. However, while live patients aid objective training in technical skills, simulated patients cannot substitute some physical attributes. For example, clinical techniques such as trial frame refraction and pathology screening can be practiced in simulations, but little can replace the learning of refraction or viewing the posterior pole of an older eye with its media changes and smaller pupils. Where the real patient's signs, symptoms and data differ from the ESP's physical attributes (which is to be expected), the use of an authentic patient's records allows the ESP to provide verbal feedback and coaching in relation to the recorded results for the real patient. For example, students might indicate their intention to conduct ophthalmoscopy, and the ESP could ask the student to demonstrate the technique. If that skill is not required as part of the current assessment, the ESP could provide the actual result or a photograph from the authentic patient's notes to manage the next section of the consultation. Real patients also provide powerful learning through their direct feedback to practitioners and students on their professionalism, empathy and skills in physical examinations, which the ESP also provides.

The ESP strategy's primary contrast to a traditional simulated patient is that the expert role-plays the patient while remaining the instructor. The latter's expertise allows for high-level judgements that meet pedagogical goals, ensuring that the ESP strategy always puts teaching and the student's learning needs first. For example, sometimes a student makes a clear error of knowledge, judgement or behavior that could have a significant effect on the outcome of the consultation, on other students in the group, or on the clinic's reputation. In this situation, the ESP employs a "time-out" — moving openly back into instructor mode to intervene and provide professional support and guidance to the student — before returning to the patient role. This approach reduces student anxiety about delayed feedback and ensures ESP consultations are authentic, interactive and immediately valuable learning experiences for all students while remaining safely within the simulation context.

The ESP approach helps students learn how to question and gently probe to discover details that may lead to appropriate diagnosis and treatment. Real patients may give answers that confuse students; whereas, while remaining true to the patient record, the ESP may choose to elaborate, quantify, clarify, or omit specific aspects of history (such as comorbidities) to maximize learning outcomes. For example, in one case the student asked the ESP role-playing a specific 80-year-old patient if the latter's adult child had been checked for macular degeneration. The ESP answered the question, showing the student the question was appropriate, even though that actual question had not been asked of the real patient on whom the role-play was based.

Importantly, as an ESP, the supervisor remains present in the consultation room with the student(s) throughout the consultation instead of moving between up to four patient consultation rooms at any given time. This ensures a much closer perspective on students' competencies. The ESP is constantly assessing the student throughout the consultation, including competency in examination techniques, and can provide comprehensive feedback immediately afterward. Setting a positive tone in establishing the case history is particularly important in LV examinations, and the ESP can give particularly valuable and highly specific feedback on a student's approach to the case history and clinical decision-making. From a first-person perspective as ESP, the instructor can encourage students to avoid a repetitive, interrogative approach ("Do you have cataracts? Do you have glaucoma? Do you have macular degeneration? Do you have high blood pressure?") and instead help them focus on more open and empathic questions ("Have you been diagnosed with any eye conditions — cataracts, glaucoma?"). Importantly, in instructor role, the ESP can also guide students directly in professional best practice in giving bad news to a patient, for example, by explaining how to ask if the patient understands the outcome or has any questions ("Is this what you expected to hear?") or specifically teaching the SPIKES protocol, a multi-step approach for delivering unwelcome diagnoses.[27]

As the ESP is experiencing the students' examinations first-hand rather than simply through observation, more opportunities appear for highly nuanced feedback on behavioral practice. For example, one ESP took a time-out to explain gently to a student how unpleasant it felt to have the student's face so very close during a visual field test. Although the student had already been observed performing this test by many instructors, no concerns had been raised previously. In instructor mode, the ESP explained that the extreme closeness may have been less obvious from a supervisor's usual side view. The student was very grateful to receive this feedback at a stage when changing the stance was still easy.

ESP consultations are fully logged and contribute to clinical logbooks in equivalence to a normal clinic session, with students' assessments addressed in the usual way. As the ESP activity is based on an authentic case, students may use that case to write their assignments and case reports. Evaluation using three independent markers showed that case reports on ESP cases provided outcomes and opportunities for learning to be demonstrated equivalent to cases based on real patients.

Training instructors in the ESP strategy has proved straightforward, and they report that teaching through ESP is rewarding and contributes to their development of advanced teaching capabilities and confidence. Given the premium rates paid to optometry professionals employed as sessional academics in face-to-face clinics, it also made economic sense to ensure these professionals could add significant value to students' learning even when patients did not arrive. Thus, administratively, the ESP strategy proved highly cost-effective and elegantly solved the clinic's problems with regard to no-show patients.

Shifting the ESP Strategy Online During the COVID-19 Pandemic

In March 2020, the COVID-19 pandemic forced full lockdowns in Sydney. On short notice, the UNSW-OC was closed as a face-to-face teaching clinic. This caused significant anxiety among students. They worried about the lack of clinical skills training and assessment and the possibility the course would be extended well beyond planned graduation dates, which would have significant implications for living/tuition costs and career prospects. Staff also faced multiple challenges. These included becoming competent themselves as telehealth practitioners,[28] transitioning their clinical teaching into fully online environments without access to patients, continuing to process patients' contact lens orders and mail prescribed spectacles, and communicating with anxious patients.

Fortunately, the ESP strategy was already well-developed at UNSW-OC by that time, and teaching staff were already comfortable with implementing it. As such, extending ESPs into the virtual world of online teaching proved viable and practicable. Just before leaving campus for lockdown, staff copied de-identified data from patient record cards to create a database of scripts for ESP consultations. Teaching timetables were re-organized, and students were allocated to virtual versions of clinics, tutorials and grand rounds. The virtual clinics were conducted with ESPs via the Blackboard Collaborate Ultra platform, which facilitated file- and video-sharing, immediate-response polls and group work "rooms."

In the usual face-to-face context, students and instructors would simply arrive at the clinic with the usual expectations of an on-campus clinical experience, whether or not ESP was needed. The preparatory logistics for the virtual ESP clinics were more complex. To help manage student well-being and anxiety alongside online/virtual clinic etiquette, preliminary communications were sent to all students. The messages explained how students would access the virtual clinic, including videoconferencing platform, internet, camera and microphone access. According to the university's equity and diversity policies, students were advised of the need for cameras, and that both their screen/room background and attire had to meet professional standards. For example, students who usually wear head coverings needed to treat the virtual clinics as external experiences, and those working from bedrooms needed to review their backgrounds. Special consideration, with no assessment penalties, was given to students who could not participate fully in the professional virtual settings.

Using a flipped class pedagogy,[29,30] the introductory emails also gave students pre-clinic research topics relevant to their upcoming ESP case. In the LV virtual clinic, the research topics often included Australian requirements for a Conditional Driver's License and Disability Support Pension (Blind). During the virtual LV clinic, students would be asked to confirm whether the ESP (as patient) met the requirements for this license or pension.

In all virtual contexts, the emphasis was on student involvement and student-led activities with an ESP strategy. Instructors collaborated to keep ratios at 2:1 per session, although occasional limited availability gave rise to ratios of up to six students per instructor. In this situation, supervisors used time-outs for more formal switches from patient to instructor mode so they could act as ESP and teach effectively in the online environment. Control of the virtual clinic was also rotated to different students, so every student learned how to run the consultation in an authentic telehealth format. This approach smoothed communication as students had to request a time-out if they needed to ask the supervisor (not the patient) a question.

Once these online approaches were established, supervisors and students reported having clear and consistent understanding of their roles. An unexpected benefit from the slightly larger groups was that students modeled professional behavior for one another. That peer learning compensated for any higher than usual student-to-instructor ratios. Interactive engagement was reinforced by enabling students to ask questions or comment using diverse options, including voice audio, Blackboard Collaborate Ultra tools (whiteboard, online chat, virtual hand-raise), and direct comment onto PowerPoint slides. Any complex or difficult aspects of an ESP case were supported in instructor mode to ensure learners' needs were addressed.

In virtual clinics, the ESP focus is primarily on developing students' problem-solving and analytical diagnostic skills. The online instructor often directs a focus on non-clinical techniques relevant to examining eyes with pathology or older eyes, such as case history and case analysis. Nevertheless, some clinical skills can be effectively assessed with an online ESP, including quality of instructions for visual acuity tests ("cover your left eye and read the chart") and visual field testing (such as Amsler grid scoring and confrontation testing for ESPs reporting a restricted visual field). Being familiar with how patients with specific pathology typically respond, the instructor can mimic this when role-playing the patient. For example, the ESP might stop reading letters and wait to see if the student encourages them to continue trying. If the patient script refers to someone with macular dystrophy or macular degeneration, the ESP might read the first and last letter of a line, omitting the central three, and see how the student records that outcome and proceeds. Shifting into instructor mode allows direct feedback to be given to the student. Relevant clinical procedures that cannot be simulated, such as retinoscopy or trial frame refractions, are reviewed in interactive online tutorials before virtual clinics.

Even virtually, the ESP role-play fully engages students in professional behavior. Shown a photo of the clinic, students are asked to visualize the patient sitting in the waiting room and decide exactly how they would invite the patient into the clinical room. In this visualization, the students are asked to notice any health attributes of the patient, such as swollen fingers or use of a walking stick. In patient mode, the instructor can assess in quite subtle ways the instructions students give for specific tests (such as the student's choice of language, clarity and tone of voice). This level of immersion into the simulation adds to the authenticity of the learning and alleviates students' concerns that they are missing out on their clinical learning during virtual learning. Students receive feedback after each virtual clinic, which reinforces key learning concepts. All students have a LV patient record card they must complete to the same standard and with the same assessment components as for a face-t--face clinic, which they submit online after the virtual clinic. Other assessable components online are the same as those for face-to-face clinics, and students are advised of these in advance.

Outcomes

Although the COVID-19 pandemic encouraged optometry educators everywhere to develop teaching approaches with some similar characteristics,[31] the ESP strategy had already been well-developed and trialed as a pedagogy before 2020. However, during the pandemic, what had been designed as a fall-back approach for patient no-shows suddenly became the clinic's main teaching strategy. With ESP already well-tested, the shift online became a much more positive experience than might have been expected or that was experienced by some other university clinics.[13]

Overall, in the COVID-19 context we found the ESP strategy was excellent in enabling students to continue their clinical training in virtual environments. It also supported future professional life using telehealth consultations. Some UNSW-OC staff and students wrote about their experience[32] for mivision, the key communication and news portal for ophthalmic professionals in Australasia, giving their two perspectives:

Instructors: *... our online clinics allowed us to give our students thought-provoking cases at every online experience ... Our students were able to practice finding their voice: having difficult conversations, giving clear instructions, and speaking appropriately to the patient and situation without jargon. ... all virtual patient experiences were real cases that have come through our clinic doors. The mix of patients was valuable, and reflected real life, where you never know who or what is going to sit in your chair. (Instructors)*

Final-year students: *... our supervisors ran sessions of 'virtual patients' where they played two roles – as themselves and as a patient. The online learning experience brought an array of unanticipated enjoyment while still allowing us to grow in clinical knowledge. ... real cases ... challenged our deductive skills to come up with a diagnosis and management plan. We worked as a team ... to come up with the best answer together. ... we could see a variety of cases and become more efficient at clinical decision making, before seeing our real-life patients. This was also an opportunity to get more 'one-on-one' time with supervisors and ... drown them with questions [which] is harder in a clinic setting where they are usually juggling several patients and students at the same time.*

When the campus clinic re-opened for COVID-safe face-to-face sessions, students reported that the ESP virtual clinics had prepared them well. Taking case histories online had prepared students effectively for optometry telehealth consultations, an approach given new prominence, scope and funding in Australia since the pandemic. Given the Australian government's new support and the significant constraints of travel to city-based teaching clinics for Australia's rural and remote patients, telehealth case histories were continued across all LV vision clinics in 2021 and have now been permanently introduced into all LV clinics.

The ESP strategy is clearly sustainable and applicable to diverse contexts. For example, in early 2022 a satellite LV clinic hosted by an industry stakeholder was re-opened after the COVID19 disruption and relocation. When this clinic had staff and students rostered for a usual clinic session (two appointments) but no patients were able to attend, the supervising optometrist was sent relevant anonymized patient records and instructed how to use the ESP strategy. Excellent outcomes were reported by both students and staff. The intangible benefits of having ESP available in this context were significant. Having the clinic remain open was a major strategic outcome, as it made excellent use of the sessional academics' professional expertise and funding; enabled the students to participate in a LV clinic, complete their assessments and have a case for later written assignments; and, crucially for ongoing relationships, allowed the industry stakeholder to bring optometry students onto its premises.

Conclusion

As an innovative extension of the simulated patient and "patient as teachers" models, the ESP strategy was specifically designed to support students in a university clinic's teaching context where no-show or canceling patients were negatively affecting learning and management outcomes. Although real patients are always the ideal, having an ESP strategy in place has removed the teaching clinic's dependency on scheduled patient attendance. Now when a patient is a no-show, the clinic's supervisors can immediately engage in an ESP role-play. Students do not miss their rostered clinical experience sessions and assessments, and they still receive deep-layered formative feedback to improve their clinical practice. The ESP strategy has proven to have distinct pedagogical and economic advantages for clinical teaching, the student experience and the wider impact of the university clinic.

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Latest Edition of *Clinical Procedures for Ocular Examination* is Thoughtfully Updated

The book: *Clinical Procedures for Ocular Examination* (fifth edition), McGraw Hill, 2023, 390 pp.

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Clinical Procedures for Ocular Examination, now in its fifth edition, continues to stand as a cornerstone reference for anyone involved in the fields of optometry and ophthalmology. The stated purpose of this new edition is to expand upon the previous foundation and incorporate new technologies and advanced procedures while staying true to the primary mission: to describe how to perform a wide variety of useful tests without a large body of theory. This edition does exactly that.

The fifth edition places significant emphasis on clinical relevance, offering valuable insights into the practical aspects of ocular examination. To model today's modern optometric practice, some procedures were added and others were removed or condensed. Flowcharts for the standard sequence of testing are found at the beginning of some chapters to give students and novice clinicians a sense of direction. Clinical "notes" are interspersed throughout the text, helping readers appreciate best clinical practice. The authors emphasize the importance of patient safety throughout the examination process, providing valuable insights on communication, patient education and the ethical aspects of eye care that include HIPAA considerations for patient privacy. Additionally, the inclusion of high-quality color illustrations and photographs enhances the learning experience and aids in the understanding of various examination procedures.

One of the standout features of this book is its user-friendly approach. Complex techniques are presented step-by-step, which makes it easy for readers to follow. The instructions to the clinical procedures are clear and concise. The text is thoughtfully organized, with each color-coded chapter covering a specific aspect of ocular examination, from patient communication and entrance testing to refraction and ocular health. There are also chapters on functional tests, contact lenses (including ortho-K), systemic health and cranial nerve testing. This structured approach — with a new numbering system for all procedures — facilitates easy navigation and reference, making the book an excellent choice for busy practitioners and students alike.

What sets this edition apart from its predecessors is its dedication to staying current with the latest advances in the field of optometry. The authors have expanded the content with two additional chapters to encompass instruction on the most recent advances in ocular imaging and advanced procedures that include small surgical procedures and lasers as new treatment modalities, ensuring that readers receive directive on the most relevant techniques available.

In conclusion, the fifth edition of *Clinical Procedures for Ocular Examination* is a must-have resource for anyone involved in eye care. Its comprehensive coverage, user-friendly approach and commitment to staying at the forefront of the field make it an invaluable tool for students, educators and practitioners. This edition has been thoughtfully updated to incorporate the latest advances in the field, ensuring its relevance in an ever-evolving medical landscape.

Feedback Changes Metacognitive Accuracy Over Different Time Scales

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Background

The ability to assess one's thought processes is known as metacognition.[1] Metacognitive accuracy, the degree to which someone "knows what they know," is often assessed through assigning probabilistic judgements to decisions.[1,2] This practice can be instantiated in classroom and/or laboratory settings by asking students to issue a confidence rating that expresses the probability that a "just-given" answer is correct.[3] For example, "You told me that hyperopic eyes have less power. How confident are you that this is correct?" The student's metacognitive accuracy is determined by comparing their judgement (confidence rating) and outcome (correctness). Students exhibit accurate metacognition when their judgement and the outcome are aligned (e.g., confident and correct), whereas erroneous metacognition is evidenced by a misalignment between their judgement and the outcome (e.g., confident and incorrect).

A student's overall metacognitive accuracy, assessed throughout a class session or throughout a course, can be quantified using several metacognitive indices that summarize multiple judgements and outcomes.[4] The first metric is error (E): a mismatch between a student's average confidence rating and their average correctness. Consider a student who is typically 75% confident and 75% correct: They have zero error because their average confidence level matches their average correctness. On the other hand, when one's confidence ratings either exceed (overconfidence, positive values) or fall below (under confidence, negative values) their average correctness, metacognitive error is present. A second metric, calibration (C), goes beyond the average levels of confidence and correctness by comparing a student's percent correct at each confidence level. A student who is well-calibrated is one whose confidence and correctness are closely aligned (e.g., always 10% correct when 10% confident and always 90% correct when 90% confident) whereas poor calibration is evidenced by misalignment across each level of confidence. The ideal value for calibration is also zero because this demonstrates strong alignment between average correctness and confidence at each confidence level. Resolution (R) is a third metric that generally describes the width of each student's "confidence spectrum": Students who always issue the same confidence judgement have poor resolution, whereas those who use many levels of confidence in their judgements have high resolution. The ideal value of one indicates the latter scenario (i.e., more is better).

The strength of computing and utilizing multiple metacognitive indices is that each index represents a different facet of a student's metacognitive accuracy. For example, it is possible for a student with minimal error to have poor calibration. Imagine a student whose average confidence and correctness are both 50% (i.e., no error), but they achieve this through being 50% correct when 0% confident and 50% correct when 100% confident. This student is poorly calibrated because the discrete confidence levels do not align with the associated outcomes. Similarly, students who are steadfast in their self-assessment can possess zero error and be well-calibrated, yet have poor resolution by always issuing incorrect responses with 0% confidence and by issuing correct responses with 100% confidence. Thus, because each metric describes a different yet important aspect of a student's metacognition, viewing all three indices together provides the most comprehensive picture of a student's self-awareness. Each metric has an ideal value; therefore, tracking them over time can provide instructors a means of providing tangible feedback regarding the veracity of a student's self-assessment. This feedback could then be used to explore how each individual student approaches a problem. For example, "I see you consistently underestimate your abilities. Why is that?" **Tables 1-3** elaborate further on these concepts and contain examples of ideal and non-ideal values for each index.

Table 1. *The error index ranges between -1 and +1 and is derived by comparing the overall confidence and % correct. The light gray shading indicates the ideal value of 0. Click to enlarge*

Table 2. *The calibration index ranges between 0 and +1 and is derived by summing the discrepancy between average confidence and average correctness at all confidence levels (i.e., 0% through 100%). The light gray shading indicates the ideal value of 0. Click to enlarge*

Table 3. *The resolution index ranges between 0 and +1 and is derived by summing the discrepancy between average correctness and overall correctness at all confidence levels (i.e., 0% through 100%). The light gray shading indicates the ideal value of +1. Click to enlarge*

We were interested in determining if and how optometry students incorporate such feedback to change their metacognitive accuracy. Optometry students must develop the ability to accurately assess their thought processes because this skill is at the heart of optometric decision-making. Specifically, knowing their assessment of themselves is accurate allows students and practicing optometrists to take proper action after diagnostic ("I am confident this is bacterial; therefore ...") and therapeutic ("I am unsure how to treat this; therefore ...") decisions are made. Inaccurate self-assessments have less desirable outcomes (e.g., incorrect diagnoses and/or unnecessary referrals). It is unclear whether optometry students typically possess accurate metacognition and, if so, whether it can be improved over time. Therefore, we designed a teaching intervention to answer these two questions with the ultimate goal of determining whether directly teaching metacognitive accuracy would be beneficial in optometric curricula.

Methods

Teaching intervention

The investigation was conducted as a teaching intervention within the Board Preparation course (OPT 7999) at Nova Southeastern University College of Optometry. Corresponding author KTW served as the instructor of record (IOR). The OPT 7999 course was held for 6 weeks during the beginning of the winter semester and was graded on a pass/fail basis. The primary goal of the course was to prepare students for Part I of the National Board of Examiners in Optometry (NBEO) examination. The class activities were designed to review content and practice self-assessment. Passing grades were given to all students who completed the class assignments described below in Procedures. This was the first time the course was taught with this structure.

Participants

Eighty-eight third-year students from the College of Optometry participated in the investigation. The sole inclusion criterion was enrollment in the course: All students in the third-year class were eligible to participate because OPT 7999 is mandatory. There were no exclusion criteria set forth; however, each student was required to take part in an informed consent discussion on the first day of class and then issue their written consent if interested and willing to participate in the teaching intervention. A total of 15 students chose not to participate or did not upload their workbooks at the conclusion of the semester. Three students were absent during the first week of class. We included them in all analyses except those that compared the first and last weeks of the course. We treated all 88 participating students as a single cohort in our subsequent analyses because each of them had taken the same courses and examinations throughout their time in the professional program.

Procedures

The following procedures and analyses are graphically and verbally summarized in **Figure 1** and **Table 4**, respectively. Each of the six class sessions lasted for approximately an hour and consisted of 20 sequential multiple-choice questions. The multiple-choice questions were derived from both optometric faculty members' courses and from Butterworth-Heinemann's Review Questions for the NBEO: Part One.[5] The questions spanned multiple optometric content areas including anatomy, biochemistry, immunology, pathology, optics and pharmacology. Each item had a total of four possible answers (i.e., A, B, C or D) with a time limit of 1.5 minutes imposed. The sequence of each session's questions was chosen by the IOR to equivalently cover each of the content areas during each session. Each session thus contained a mixture of optometric content and did not explicitly focus on one area from week to week.
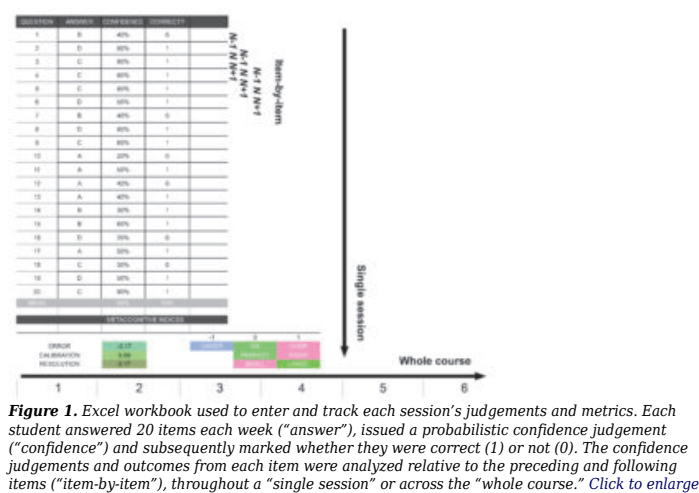

Figure 1. Excel workbook used to enter and track each session's judgements and metrics. Each student answered 20 items each week ("answer"), issued a probabilistic confidence judgement ("confidence") and subsequently marked whether they were correct (1) or not (0). The confidence judgements and outcomes from each item were analyzed relative to the preceding and following items ("item-by-item"), throughout a "single session" or across the "whole course." Click to enlarge

TABLE 4
Patterns of Data Amalgamation and Segregation Used for Each Temporal Scale

	Items	Sessions	Students	Metrics
Item-by-item	Sequence preserved: N_{-1}, N, N_{+1}	Number discarded	Identity discarded	Metacognitive accuracy categories
Single Session	Number preserved: 1, 2, 3 ... 20	Number discarded	Identity preserved: 1, 2, 3 ... 88	Metacognitive indices (i.e., E, C, R)
Whole Course	Number discarded	Number preserved: 1, 2, 3 ... 6	Identity preserved: 1, 2, 3 ... 88	Metacognitive indices (i.e., E, C, R)

E = error; C = calibration; R = resolution

Table 4. Each temporal scale requires a different pattern of data amalgamation. The item-by-item analysis (first row) preserved the relative sequence, but not absolute number, of each item. The single session (second row) and whole course (third row) analyses both preserved student identity while segregating responses by either the absolute number of each item (single session) or of each session (whole course).
Click to enlarge

An introductory lecture given before the course began introduced the structure of the course and related teaching intervention. The concept of metacognition and the indices to be used were introduced with the Excel workbook and lecture slides serving as demonstrative illustrations. Then, during the course, feedback regarding metacognitive accuracy was made available to each student over different time scales. First, after answering a question, each student was instructed to issue a confidence rating reflecting their probabilistic assessment (0% to 100% in 10% intervals) that their answer was correct. Then, upon the correct answer being revealed, each student marked whether their answer was incorrect or correct. This revelation of correctness, in conjunction with the associated confidence rating, served as an immediate form of feedback regarding the student's metacognitive accuracy on the given item. Second, the three metacognitive indices of error, calibration and resolution were calculated at the end of each session and made available for each student to view. This form of feedback was included to help each student evaluate their metacognitive accuracy for each class session. Last, because each student had access to each of the six sessions' metacognitive indices, they were also able to track how their metacognitive accuracy changed throughout the course.

The answers, confidence judgements and correctness were all recorded in an excel workbook provided to the students (Figure 1, left). Students entered each of the six sessions' data on dedicated weekly sheets labeled as 1, 2, 3, etc. Each student's average confidence, average correctness and the three metacognitive indices (i.e., E, C and R) were visible at the bottom of each week's sheet and computed "live" as the students continued to answer each of the session's questions. The average metrics were finalized once all 20 questions had been answered. Color scales were used to help students visualize the meaning of each metacognitive index: green indicated "ideal" metacognition, red indicated "poor" metacognition, and blue indicated under confidence for the error metric alone. These colors were based on the ideal values of each metric (i.e., zero, zero and one). Excel interpolated between each of these colors to generate a colored cell, which showed each student where they fell on the metacognitive accuracy spectrum (Figure 1, bottom). For example, pure blue, pure green and pure red represented values of -1.0, 0.0 and +1.0 for the error metric. In Figure 1, the student's actual error of -0.17 is illustrated by a unique color that fell between pure blue and pure green on the color scale. Similarly, colors that fell between pure green and pure red (C = 0.09) and pure red and pure green (R = 0.17) were used to illustrate their calibration and resolution.

Analyses

The confidence judgements and associated correctness outcomes from all items, sessions and students were analyzed on three different time scales related to the delivery of feedback: item-by-item, single session and whole course. This was done to determine whether providing feedback regarding metacognitive accuracy can change metacognitive accuracy itself over short, medium or long periods of time. A total of 10,500 items (88 students answering 20 items per each of the six sessions, with three students missing in the first week) were included in the subsequent analyses.

<u>Item-by-item</u>

The item-by-item time scale represents the ~ 1.5-minute interval between successive items. The question at the heart of this analysis is "does knowing my metacognitive accuracy on the current item influence my metacognitive accuracy on the next?" In other words, if I know my judgement and outcome were just aligned, will this lead me to be similarly accurate on the next item? We determined whether the metacognitive accuracy exhibited on the current question (N) was associated with the preceding (N_{-1}) or following (N_{+1}) metacognitive accuracies in the following manner. First, we labeled all items as "uninformed," "misinformed," "partially informed" or "well-informed" using binary classifications of both confidence level and correctness. The metacognitive accuracy categories were labeled as originally proposed by Hunt:[6]

- Uninformed if the item was incorrect and their confidence was < 50%
- Misinformed if the item was incorrect and their confidence was ≥ 50%
- Partially informed if the item was correct and their confidence was < 50%
- Well-informed if the item was correct and their confidence was ≥ 50%

Second, we computed the proportion of each category that tended to precede or follow each specific category for all possible items. For example, for items identified as uninformed, we checked the collection of categories for all preceding questions (N_{-1}) and computed the percentage of preceding items labeled as uninformed, misinformed, partially informed or well-informed. Then, continuing, we computed the proportion of categories present on all items following uninformed items (N_{+1}). This procedure was repeated for each of the remaining categories (i.e., the proportions of categories for items preceding or following items labeled as misinformed, partially informed and well-informed). This comparison of preceding and following proportions was done without respect to the actual item number, the session number or student identity (Table 1, first row). A total of 9,486 items were included in this analysis because the first and 20th items did not have any items preceding or following them, respectively.

The symmetry of the N_{-1} or N_{+1} distributions indicates whether metacognitive accuracy tended to fluctuate or stay the same across items. Symmetric distributions (i.e., the same proportion of each category) would suggest that metacognitive accuracy fluctuated because any of the four metacognitive accuracy categories was just as likely to occur before or after a given category (e.g., an equal chance of being uninformed, misinformed, partially informed or well-informed before uninformed items). On the other hand, asymmetric distributions would suggest that metacognitive accuracy tended to stay the same (e.g., uninformed items tend to precede and follow other uninformed items). We quantified this notion of symmetry by performing a Fisher's exact test on the proportion of categories both preceding and following uninformed, misinformed, partially informed and well-informed items. This statistical test determines whether one of the categories tended to occur more often than the others: An odds ratio significantly greater or less than one suggests the distribution of proportions is uneven.

Single session

This time scale represents the ~ 1-hour interval between the first and last questions in a session. Analysis at this level asks: "can receiving continual feedback throughout a session change my metacognitive accuracy?" We determined whether metacognitive accuracy tended to change throughout a session in the following manner. First, we pooled the responses from all sessions to create a distribution of six judgements and associated outcomes for each of the 20 items for each student. Thus, because student identity was preserved, there was a distribution of 88 values describing the error, calibration or resolution for each of the 20 items across all sessions (Table 1, second row). Error, calibration and resolution were computed using the formulas described by Fleming et al.: All compare different facets of confidence ratings and performance outcomes and are derived from the extant metacognitive literature.[1]

The difference between the indices derived from the 20th vs. first items was used to compute three additional variables (ΔE_I, ΔC_I and ΔR_I) representing the direction and magnitude of metacognitive change between the beginning and end of a session. For ΔE_I, a positive difference represents a trend toward either no error or overconfidence. Negative ΔC_I values indicate calibration has improved throughout a session, whereas positive ΔR_I values indicate resolution has improved during class. We planned a two-sided t-test to determine whether the collection of ΔE_I, ΔC_I or ΔR_I values were significantly different from zero. We chose a two-sided t-test because we did not know the direction in which metacognitive accuracy would shift for any of the metrics.

Whole course

This time scale represents the interval between the first and last sessions of the course. Analysis at this level asks: "can receiving continual feedback throughout a course change my metacognitive accuracy?" We addressed this question in the following manner. First, we pooled responses from all items to create a distribution of 20 judgements and associated outcomes for each of the six sessions for all students. This was also done for each student; therefore, there was a distribution of 85 of each of the metacognitive indices that summarized our cohort's metacognitive accuracy for each of the six sessions (Table 1, third row).

The difference between the indices derived from the sixth vs. first sessions was then used to compute three additional variables (ΔE_S, ΔC_S and ΔR_S) representing the direction and magnitude of metacognitive change between the beginning and end of the course. These variables have the same sign convention as the difference variables mentioned previously, and as for the single session analysis, we also planned a two-sided t-test to determine whether the collection of ΔE_S, ΔC_S or ΔR_S values were significantly different from zero.

Results

Item-by-item

Figure 2 displays two important findings revealed in our item-by-item analysis. First, the proportion of metacognitive accuracy categories was overall asymmetric: Most items were labeled as well-informed. There were a total of 3,345 (36%) items labeled as well-informed, 2,849 (30%) labeled as uninformed, 2,314 (24%) labeled as partially informed, and 996 (10%) labeled as misinformed. This suggests that most items were evaluated with metacognitive accuracy. Items answered with high confidence ended up being correct, and items issued with low confidence ended up being wrong.
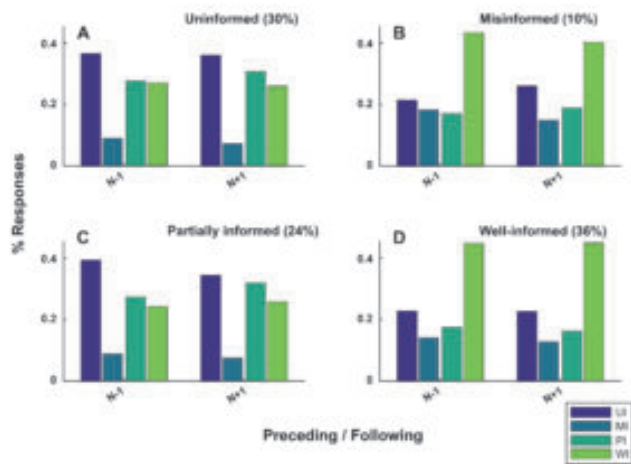
Figure 2. *Confidence remains static across short time scales. Low confidence responses (A, C) were most likely to be preceded and followed by low confidence responses, whereas high confidence responses (B, D) tended to be followed by high confidence responses. The gray text shows the metacognitive accuracy category for the current item analyzed (N) and the percentage shows the total proportion of items with that label. The colored bars in each panel show the proportion of each category preceding (N-1) and following (N+1) the current item. Click to enlarge*

TABLE 5
Odds Ratios Showing Likelihood that Preceding and Following Metacognitive Categories are Asymmetrically Distributed

	Preceding (N₋₁)	Following (N₊₁)
Uninformed	4.01 (3.38 - 4.75)	4.22 (3.53 - 5.05)
Misinformed	2.90 (2.22 - 3.78)	3.69 (2.83 - 4.82)
Partially Informed	3.92 (3.25 - 4.74)	3.66 (3.01 - 4.45)
Well-informed	4.13 (3.56 - 4.80)	5.30 (4.55 - 6.18)

Table 5. *The proportion of metacognitive accuracy categories preceding and following a particular category are asymmetrically distributed. Each row contains odds ratios describing the likelihood that a relationship between confidence and correctness exists for both the preceding and following proportions of categorizations. Click to enlarge*

Second, confidence ratings tended to remain similar across temporally adjacent item. Low confidence judgements tended to be preceded and followed by low confidence judgements, and high confidence judgements tended to be preceded and followed by high confidence judgements. For example, Figure 2A illustrates that items categorized as uninformed were most likely to be both preceded and followed by uninformed responses. Similarly, Figure 2C shows that partially informed items were most likely to be both preceded and followed by either uninformed or partially informed items. Both uninformed and partially informed responses are issued with low confidence; therefore, the predominance of these categorizations surrounding items with a similar category suggest a temporal stasis of confidence was sometimes operative. The same "hysteresis" of confidence was seen for high confidence items. Both misinformed and well-informed items tended to be followed by items categorized as well-informed. Fisher's exact test confirmed the presence of asymmetric distributions: The odds ratios computed for each of the eight collections of N_{-1} and N_{+1} proportions were significantly greater than one **(Table 5)**.

Single session

Figure 3 shows how our three metacognitive indices of error (E_I), calibration (C_I) and resolution (R_I) changed throughout a session. The value of each index tended to fluctuate as a session progressed. This is evidenced by the "jagged" nature apparent in each of the graphs. The mean error at the beginning of a session (i.e., for the first item) was -0.084 and changed to -0.167 by the end. The mean initial calibration for our cohort increased from 0.007 to 0.009 between the first and 20th items, whereas the mean initial and final resolutions were both approximately 0.01. The t-test confirmed that both the ΔE_I and ΔC_I distributions were significantly different from zero: ΔE_I values fell between -0.080 and -0.085, whereas ΔC_I values ranged between 0.016 and 0.018.

Whole course

The three metacognitive indices also appeared to fluctuate throughout the course **(Figure 4)**. The mean error at the beginning of the course (i.e., for the first session) was -0.090 and changed to -0.18 by the end. Calibration for our cohort remained constant between the beginning (0.105) and end (0.120) of the course, whereas resolution appeared to increase from a mean of 0.127 at the outset of the course to a mean of 0.147 at the end. The t-test confirmed that both the ΔE_S and ΔR_S distributions were significantly different from zero: ΔE_S values fell between -0.089 and -0.085, whereas ΔR_S values ranged between 0.017 and 0.019.
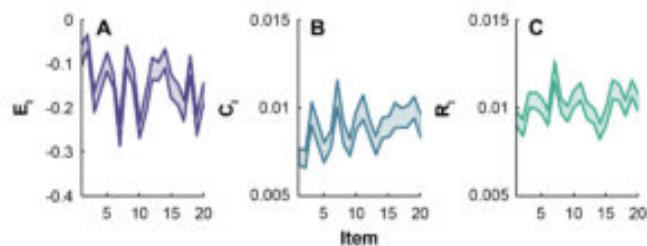
Figure 3. *Error and calibration change throughout single sessions. The average error value tended to decrease (A), calibration values tended to increase (B), and resolution values tended to stay the same (C) throughout a class session. Each shaded bar represents the mean +/- 1 standard error of the mean. Click to enlarge*
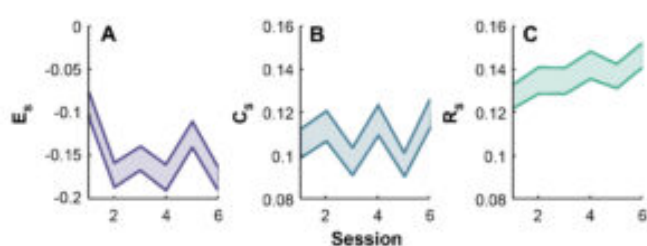

Figure 4. *Error and resolution change throughout the whole course. The average error value tended to decrease (A), calibration values tended to remain the same (B), and resolution values tended to increase (C) throughout the course. Each shaded bar represents the mean +/- 1 standard error of the mean. Click to enlarge*

Student subgroups

The above analyses treated our students as a single cohort, which appeared to show the same general changes in metacognitive accuracy across both single sessions and the whole course. We performed two exploratory analyses intended to examine whether this assumption was true.

We first performed a supplementary principal components analysis (PCA) using the entire collection of Δ values (i.e., ΔE_I, ΔE_s, ΔC_I, ΔC_s, ΔR_I and ΔR_s) to determine whether these general trends were exhibited by all students. This procedure determines whether a combination of factors or one factor alone best explains the variability within a set of data. PCA revealed that ΔE_I and ΔE_s values together could explain 96% of the variability within our cohort's shift in metacognitive indices. This shows that error was the primary way in which metacognitive accuracy changed both throughout class sessions and throughout the course as a whole. **Figure 5** shows how the direction in which the error metric changed over both medium and long time scales partitioned students into four distinct categories. We named the first group "receptors": Their error tended to become more positive throughout both class sessions and the course. The absolute error values (i.e., E_I and E_s) of this group were negative; therefore, this positive shift shows that they were able to use the provided feedback to shift their error values toward zero during class (an initial gain) and then maintain this shift throughout the course ("positive retention"). The opposite was true for a group we named "inverters": Despite being shown they had consistently underestimated their abilities, this group tended to underestimate themselves more as class sessions and the course progressed. The subgroups we named "doubters" and "reflectors" showed a combination of both patterns. Doubters displayed positive shifts during class but did not maintain them over time, whereas reflectors did not improve their error during class but eventually did over the course.

Second, to evaluate this further, we undertook an analysis of ΔE_s values to determine whether there was a relationship between improvements vs. decrements in metacognition and performance over the duration of the course. To do this, we created two distance metrics, which captured improving (positive values) and worsening (negative values) metacognition independent of a student's initial under- or over-estimation of their abilities. This is necessary because students with either initially positive or negative errors can improve their metacognitive accuracy. Most students appeared to initially and progressively underestimate their abilities; however, treating them as a single cohort may have obscured students who did not follow this general pattern. For example, a student with E_{S1} and E_{S6} values of -0.60 and +0.20 would have an ΔE_s value of +0.80 (positive retention) and "traveled" 0.60 toward zero and 0.20 away from zero. The shift toward zero is a metacognitive improvement, whereas the shift away from zero is a metacognitive decrement. A student with E_{S1} and E_{S6} values of +0.60 and -0.20 would instead have an ΔE_s value of -0.80 (negative retention) but "traveled" 0.60 toward the ideal zero value and 0.20 away from it. Thus, while the ΔE_s values shows whether a student shifted toward under- or over-estimation, partitioning of this shift into zero-referenced distances shows how much of that shift was an improvement and how much was a decrement. One can also imagine cases in which a student's error becomes progressively more negative (as in the majority of our cohort) or positive throughout the course. In this case, the student's metacognitive accuracy is continually worsening and the distance "traveled" is always away from zero.

Figure 6 shows that there is a gradient of both improvements (toward zero, in green) and decrements (away from zero, in blue) for students with varying levels of initial and final error. Overall, approximately one quarter (26%) of our students' ΔE_s values contained only improvements, another quarter (23%) contained both improvements and decrements, half contained only decrements (48%), and a minority did not change (3%).
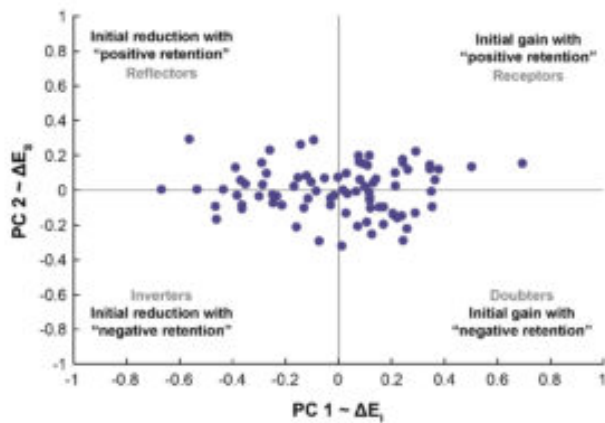
Figure 5. *Changes in metacognitive error occur in different directions and at different times. Metacognitive error became more positive or negative throughout a session (x-axis, an initial gain or reduction, respectively) or throughout the course (y-axis, "negative retention" or "positive retention"). There was an approximately equal number of students (one dot = one student) who fell into each of the four quadrants defined by these axes. Each quadrant is labeled with a proposed name, which encapsulates how metacognitive error changed. Click to enlarge*
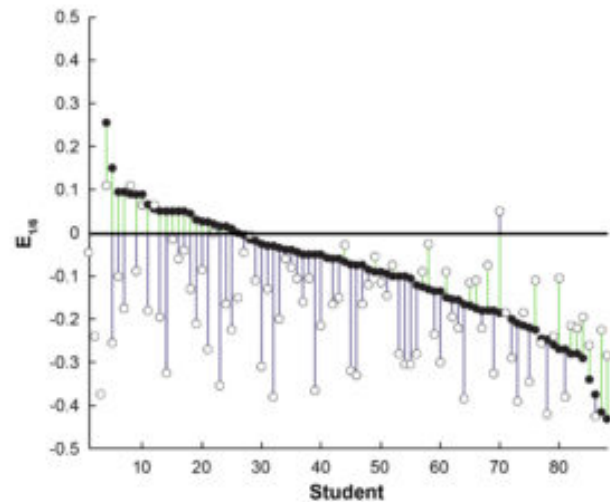
Figure 6. *Error shifts across the course consist of both improvements and decrements in metacognition. The difference between a student's initial error (E1, black circles) and final error (E6, white circles) represents the whole course shift in metacognitive accuracy (ΔES). This shift can be positive (black circles below white circles) or negative (black circles above white circles) and consists of metacognitive improvements (green lines), decrements (blue lines) or both. The reference line at zero indicates the ideal error value. The three open circles at the leftmost portion of the graph show E6 values for the students who were absent during the first week of class. Click to enlarge*

A correlation between our cohort's distance metrics and their change in performance throughout the course showed that students with the largest magnitude of metacognitive decrements tended to demonstrate improvements in performance (i.e., more correct answers in the final week of the course, $\rho = -0.46$, $p < 0.001$) whereas there was no relationship between the magnitude of metacognitive improvements and changes in performance. On the other hand, students who scored lowest on a 350-item mock national board examination given before the course began tended to show the greatest metacognitive improvements throughout the course ($\rho = -0.38$, $p = 0.015$). These findings reiterate the complexity inherent in determining relationships between metacognition and performance because all students' journeys consist of different locations (i.e., starting and ending points, direction of travel) and take a different amount of time (i.e., items, sessions or a whole course).

Discussion

This is the first investigation to characterize how feedback can change metacognitive accuracy over time. Our results showed that providing feedback over short, medium and longer time scales gave students the potential to improve their metacognitive accuracy, error, calibration and resolution. However, not all students utilized the feedback to this end. This is shown by the variety of ways in which the metacognitive indices changed from one item to the next, throughout a session and throughout the course.

Our item-by-item analysis revealed the presence of confidence "streaks." Low- and high-confidence responses both tended to be followed by responses with similar confidence judgements. This shows that students did not necessarily use the immediate feedback regarding question correctness to their advantage. For example, partially informed items, which occur when a student is not confident but correct, tended to be followed by uninformed or partially informed items. This suggests that instead of adjusting their confidence to match potential correctness on a following item, students instead tend to issue low confidence judgements independent of the eventual outcome. Similarly, the rare occurrence of misinformed responses, which occur when a student is confident but incorrect, tended to be followed by well-informed responses. This suggests that "wrong but confident" items were brief slip-ups surrounded by otherwise correct but confident items. The association between misinformed and well-informed responses supports results from a previous investigation by Metcalfe who found that errors committed with high confidence are more readily corrected.[7] Thus, students with high confidence may not always be correct, but instead continually possess the willingness to learn from their mistakes. The presence of our "item-by-item" temporal patterns showed that it is confidence, not metacognitive accuracy, that is maintained over short time scales. One would expect associations between either uninformed and well-informed or misinformed and partially informed items if the latter were true (i.e., one continually knows what they know and don't know).

Most of our students tended to underestimate their abilities at the beginning of a session and at the beginning of the course: The average value of the error metric was negative in both instances. This is a metacognitive inaccuracy: Negative values result when one's average confidence is less than their average correctness. Knowing they had underestimated their abilities (via visualization of the metacognitive indices) did not improve metacognitive accuracy in most students. Instead, the average error value tended to decrease further both throughout sessions and the course. This supports several observations made regarding the current generation of students who are sometimes broadly characterized as possessing high levels of anxiety, insecurity and risk aversion.[8,9] Each of these characteristics may lead a student to avoid committing errors and ultimately learning.

Our single session and whole course results also suggest that learning to modify calibration and resolution may take different amounts of time. For example, while calibration values tended to increase throughout a single session, resolution values tended to increase throughout the whole course. The increment in calibration values represents a reduction in metacognitive accuracy and shows that students learned to less closely match their confidence judgements with their eventual correctness during class. On the other hand, the increment in resolution values is an improvement in metacognitive accuracy because it suggests that students learned to utilize a greater gamut of confidence ratings by the end of the course. Thus, despite eventually possessing a greater spectrum of internal confidence judgements to choose from, students were not able to align this spectrum to the associated outcomes.

The persistence of confidence judgements across short time scales and the persistent pattern of underestimation (evidenced by reductions in error values and increments in calibration values) across both medium and longer time scales show that our cohort of students took different amounts of time to incorporate the feedback provided to them. This heterogeneity was highlighted by our supplementary PCA analysis, which identified four subgroups of students, each of whose medium- or long-term metacognitive error shifted toward under- or over-estimation. A partitioning of the whole course metacognitive error shifts into improvements and decrements further highlights the heterogeneity inherent within our cohort of students. For some, a negative shift in error took them from initially overestimating their abilities to correctly assessing them. In others, a negative shift in error compounded their initial inaccuracy despite demonstrating the largest improvement in course performance.

The different directions and period over which our metacognitive indices changed may be related to the notion that dedicated neural circuitry is responsible for handling learning over different time scales.[10] This is a critical observation to make when designing and implementing optometric curricula. For example, imagine giving feedback while teaching tonometry. You are teaching a student who is overconfident and immediately professes proficiency in the skill. They are humbled over the next couple of weeks and display a pattern similar to the student on the leftmost side of Figure 6: Their metacognitive error is initially positive but improves over time. We speculate this may involve learning to utilize a greater gamut of confidence ratings (improving resolution). On the other hand, imagine students who are instead intimidated by the procedure but also improve over time. It is a joy to witness students who also believe in this improvement (rightmost side of Figure 6) and sometimes perplexing to watch students who don't. How do you best teach each of these students? Our study showed that because students process feedback in different ways over different time scales, there is not a single answer to this question.

Our investigation possessed several weaknesses. First, the collection and sequence of items both within and across sessions was not standardized. This is likely what led to the apparent fluctuation of metacognitive indices. Second, the IOR did give some verbal encouragement and feedback to students during the course. This may have influenced the way students rated themselves. Third, the students were not given any practice issuing probability judgements before the course began. This is a possible explanation for why the majority of the change in error occurred after the first session of the course. Last, because the national board examinations became progressively closer in time as the course went on, each student's confidence and/or correctness could have changed as the date drew nearer. We look forward to addressing these weaknesses in a future investigation by:

- planning a practice session before the teaching intervention officially begins
- using items standardized for content familiarity and difficulty (This would enable "cleaner" tracking of metacognitive ability across items, sessions and the course.)
- using this investigation's exploratory analyses to formally examine the relationship between each of the metacognitive indices
- using a survey to gauge whether students felt the course improved their metacognition

Conclusion

The field of metacognitive accuracy and its trainability is ripe for examination in optometry and the medical professions as a whole. We are excited to use the current investigation as a platform to begin asking larger questions that can help improve the way we guide our students through professional optometric programs. Our results highlight that students truly do interpret our

messages in different ways and take varying amounts of time to incorporate them into their optometric schema.

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Stargardt Disease: a Teaching Case Series

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Introduction

Stargardt disease is a genetic condition affecting the retina and is one of the most commonly inherited retinal diseases.[3] The condition affects the macula and leads to the damage and eventual loss of photoreceptors and underlying retinal pigment epithelium (RPE), which causes decreased central vision and possible central/paracentral scotomas. Although age of onset can vary, Stargardt disease typically begins to manifest during early childhood. Onset during early or late adulthood is less common, but later onset is associated with a better prognosis.[2,4,5]

The disorder is an inherited retinal disease with the majority of cases inherited in an autosomal recessive pattern secondary to a genetic mutation(s) within the ABCA4 gene. The condition is referred to as Stargardt disease 1 (STGD1) if the ABCA4 gene is implicated. The ABCA4 gene encodes for the ABCA4 protein, which is responsible for removing toxic substances from the photoreceptors.[6-8] In a patient with this mutation, the ABCA4 protein is defective, leading to accumulation of a toxic substance known as lipofuscin within the retinal cells. This ultimately results in cell death.[9] Rarer autosomal dominant forms of inheritance also exist for Stargardt disease.

There is no treatment for STGD1 at this time. However, clinical trials are currently investigating gene replacement therapy, stem cell therapy and pharmacological interventions. This case report highlights two STGD1 patients with different phenotypic presentations. It also reviews pathophysiology, differential diagnosis, multimodal imaging results and potential treatment and rehabilitation options.

Case Description

Patient 1

A 21-year-old African American female presented for a comprehensive examination complaining of longstanding distance blur in both eyes. Her ocular history was remarkable for probable STGD1, which was diagnosed at an outside office 3 years prior. At that visit glasses were prescribed and no follow-up was scheduled. The patient's medical history was unremarkable. Her family's medical and ocular histories were unremarkable. She denied any medication use or any allergies. Best-corrected visual acuity was 20/400 in the right and left eye. The patient noted that her visual acuities were stable to the previous examination's findings. Entrance testing and slit lamp examination were normal. Intraocular pressure measured 15 mmHg in the right and left eye. A dilated fundus exam revealed a cup-to-disc ratio of 0.4/0.4 horizontally and vertically in each eye. Both optic nerves were pink with distinct margins. Macular atrophy was present with surrounding pisciform flecks in both eyes as evidenced by fundus photographs **(Figure 1)**. A spectral domain optical coherence tomography (SD-OCT) scan was performed and revealed RPE atrophy along with focal and hyper-reflective RPE thickening and disruption. Ellipsoid zone (EZ) atrophy with extension nasal and temporal to the fovea was evident in both eyes **(Figure 2)**. Fundus autofluorescence (FAF) showed large amounts of central hypo-autofluorescence corresponding to the RPE atrophy. Hyper-autofluorescence of the pisciform flecks was also observed in both eyes with a surrounding hyper-autofluorescent border. Peripapillary sparing on FAF was also visible **(Figure 3)**. Based on her early age at disease onset and fundus findings, the patient was diagnosed with STGD1. Unfortunately, genetic testing was not readily available at this time. The patient was referred for low vision rehabilitation (LVR) services but was lost to follow-up.
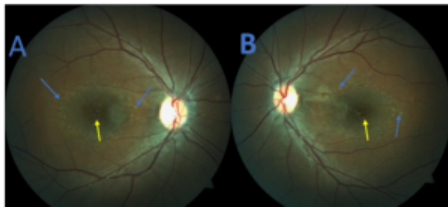
Figure 1. Fundus photographs of the right (A) and left (B) eye of patient 1 demonstrating macular atrophy (yellow arrows) with surrounding pisciform fleck accumulation (blue arrows).
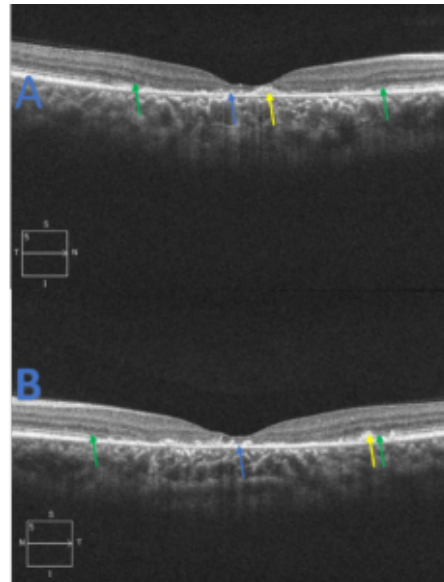Click to enlarge

Figure 2. Spectral domain optical coherence tomography raster scans of the macula, right eye (A) and left eye (B), for patient 1. The scans reveal central RPE atrophy (blue arrow) along with focal and hyper-reflective RPE thickening and disruption (yellow arrow). Foveal ellipsoid zone (EZ) atrophy is evident with extension nasal and temporal to the fovea in both eyes. The junction of the preserved and damaged EZ is indicated (green arrow). These findings correspond to the appearance of the fundus in Figure 1 and the central hypo-autofluorescence observed in Figure 3. Click to enlarge
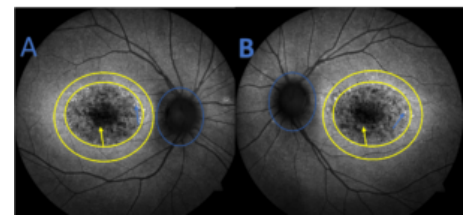
Figure 3. Fundus autofluorescence imaging of the right (A) and left (B) eye of patient 1 revealing hypo-autofluorescence at the macula (yellow arrows) corresponding to the macular/retinal pigment epithelium (RPE) atrophy observed in Figures 1 and 2. Scattered areas of hyper-autofluorescence (blue arrows), corresponding to RPE disruption and lipofuscin accumulation/pisciform flecks seen in Figure 1 and 2, are observed around the RPE atrophy. A hyper-autofluorescent border is also noted surrounding the central lesions (yellow circles) in addition to peripapillary sparing (blue circle).
Click to enlarge

Case Description

Patient 2

A 45-year-old African American female first presented with a chief complaint of longstanding blurry vision. Her medical history was unremarkable and she was not using medications. However, she reported a history of prior cigarette smoking (1/2 pack a day for 15 plus years). Her family's medical and ocular histories were unremarkable. Best-corrected visual acuity was 20/50 and 20/30 in the right and left eye, respectively. Entrance testing and slit lamp examination were unremarkable. Intraocular pressure measured with Goldmann tonometry was 17 mmHg in each eye. The dilated fundus examination showed a cup-to-disc ratio of 0.3/0.3 horizontally and vertically in each eye. Both optic nerves were pink with distinct margins. Fundus photographs of the macula in each eye showed RPE atrophy with adjacent RPE hyperplasia. Pisciform-like deposits were noted extending into the arcades and midperiphery in both eyes **(Figure 4)**. The periphery was unremarkable in both eyes. SD-OCT scans of both eyes showed larges areas of RPE atrophy nasal to the fovea with minimal foveal preservation. RPE and EZ disruption were also evident on the SD-OCT scans, involving and temporal to the fovea **(Figure 5)**. The macular findings and the patient's relatively good entering visual acuities were suggestive of an inherited retinal disease. A tentative diagnosis of fundus flavimaculatus (FF), a subtype of STGD1, was made. Unfortunately, genetic testing and FAF were not available in the clinic at that time. The patient declined a referral to a retina specialist as she had minimal complaints and relatively good visual acuity. She was therefore advised to return yearly to be monitored for disease progression.

The patient was lost to follow-up and returned 8 years later. Her medical and ocular histories were unchanged. Best-corrected visual acuity was 20/800 and 20/200 in the right and left eye, respectively. Entrance testing and slit lamp examination were unremarkable. Based on Goldmann tonometry, intraocular pressure was 16 mmHg in each eye. The dilated fundus examination showed a cup-to-disc ratio of 0.3/0.3 horizontally and vertically in each eye. Both optic nerves were pink with distinct margins. The macula in each eye showed extensive RPE atrophy, adjacent RPE hyperplasia and retinal flecks extending into the midperiphery as seen with ultra-widefield imaging **(Figure 6)**. FAF showed large areas of central macular hypo-autofluorescence **(Figure 7)** corresponding to the macular atrophy observed in Figure 6. Mixed hyper- and hypo-autofluorescence was noted surrounding the central macular hypo-autofluorescence and likely represented active lipofuscin accumulation (hyper) and reabsorbed lipofuscin/RPE atrophy (hypo). Peripapillary sparing was also visible in both eyes. Generalized RPE involvement was indicated by global hyper-autofluorescence extending into the arcades. Progression of the atrophy was noted when compared to the fundus photos from her initial visit **(Figure 8)**. Genetic testing confirmed STGD1,

subtype FF, as the results revealed mutations for ABCA4 c.570+1798A>G (intron variant) and ABCA4 c.5114G>A (missense mutation). The patient was advised to schedule an appointment for genetic counseling based on her test results and was strongly advised to seek LVR services due to her vision loss. The patient declined LVR services and was asked to return in 1 year to be monitored for progression and to reconsider LVR.
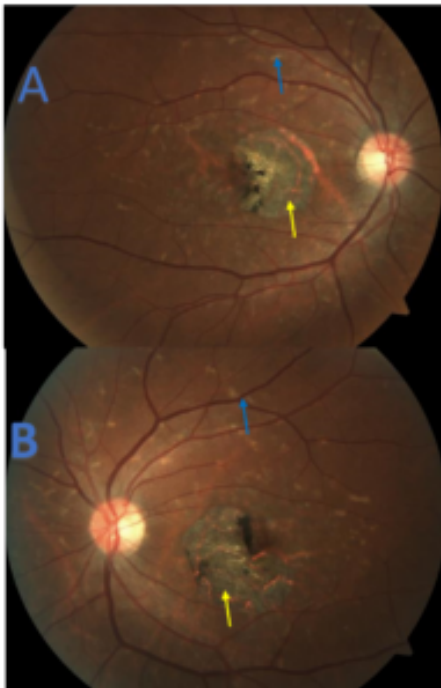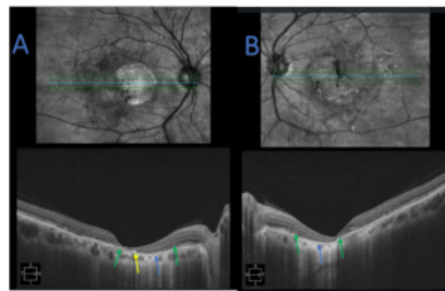

Figure 4. Fundus photos upon initial presentation of patient 2. Retinal pigment atrophy (yellow arrows) is evident in the right eye (A) and left eye (B) with partial foveal preservation in both eyes. Large amounts of pisciform fleck are observed extending into the arcades of both eyes (blue arrows). *Click to enlarge*

Figure 5. *Spectral domain optical coherence tomography raster scans of the macula from the right eye (A) and left eye (B) of patient 2. Scanning reveals retinal pigment epithelium atrophy, more so nasal to the fovea in both eyes (blue arrow), which corresponds to the fundus appearance in Figure 4. Retinal pigment disruption is noted as hyper-reflectivity (yellow arrow). Ellipsoid zone compromise, but not complete atrophy, is noted and corresponds with the patient's visual acuity. The junction of ellipsoid zone disruption and loss is noted (green arrows).* *Click to enlarge*
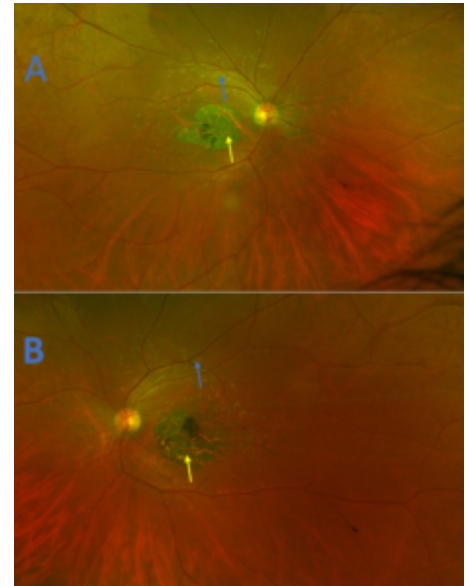
Figure 6. *Ultra-widefield imaging demonstrating retinal pigment atrophy (yellow arrows) in the right eye (A) and left eye (B) of patient 2 that has progressed temporally when compared to initial presentation (Figure 4). Large amounts of pisciform fleck are observed extending into the arcades of both eyes (blue arrows).* *Click to enlarge*
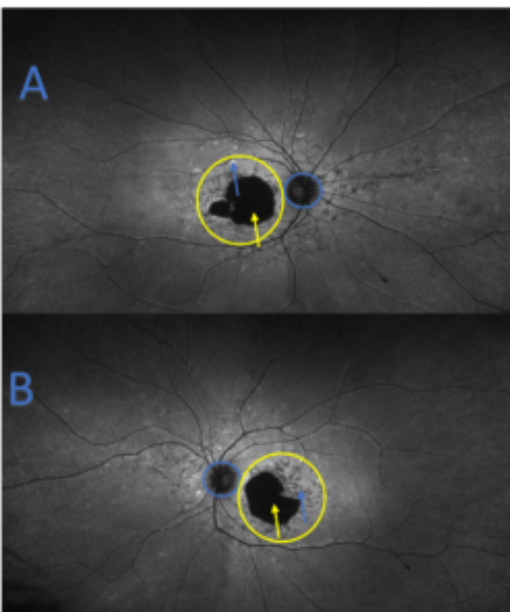
Figure 7. *Fundus autofluorescence of the right (A) and left eye (B) of patient 2 showing large areas of central macular hypo-autofluorescence (yellow arrows) corresponding to the macular atrophy observed in Figure 6. Mixed hyper- and hypo-autofluorescence (blue arrows) is noted surrounding the macular atrophy representing active lipofuscin accumulation (hyper) and reabsorbed lipofuscin/RPE atrophy (hypo). Peripapillary sparing is also visible in both eyes (blue circles). Generalized retinal pigment epithelium involvement as indicated by global hyper-autofluorescence is seen around the macular hypo-autofluorescence (yellow circles).* *Click to enlarge*
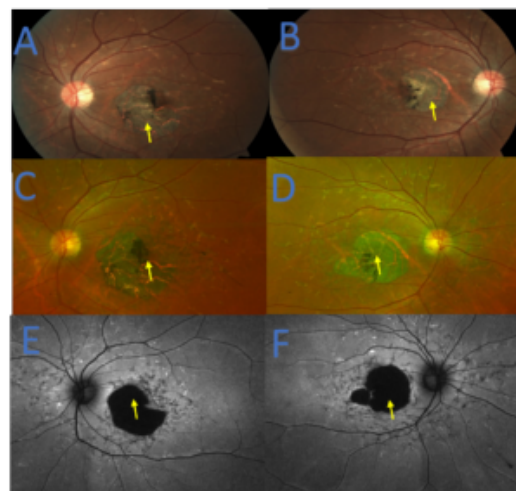
Figure 8. *Montage progression images of the right eye (B, D, F) and left eye (A, C, E) for patient 2. Visit one (baseline) fundus images (A, B) show macular atrophy with compromise of the fovea (yellow arrows). Visit two fundus imaging (C, D) shows expansion of the macular atrophy with adjacent retinal pigment epithelium hyperplasia (yellow arrows) that now involves the fovea. This corresponds to the decrease in the patient's visual acuity from visit one. Fundus autofluorescence (E, F) further highlights the macular atrophy from visit two as evident by the large amounts of hypo-autofluorescence (yellow arrows).* *Click to enlarge*

Education Guidelines

Key concepts

1. The pathophysiology and specific genetics of STGD1
2. Importance of multimodal imaging in STGD1
3. Current and future treatment options for patients with STGD1

Learning objectives

1. Define and recognize the varying clinical presentations of STGD1
2. Identify the possible signs and symptoms associated with STGD1
3. Discuss the possible differential diagnoses for STGD1
4. Recognize the importance of utilizing additional testing to aid in diagnosing and managing STGD1
5. Staying current and informed about novel research and potential treatment options

Discussion points

1. Knowledge, understanding and facts about the case and condition presentation

 a. Describe the typical appearance and presentation of early onset STGD1
 b. Describe the typical appearance and presentation of late onset STGD1
 c. Compare and contrast the pathogenesis and genetics of these two subtypes of STGD1

2. Differential diagnosis

 a. What other conditions present in a similar clinical manner to STGD1?
 b. How can a clinician differentiate between these similar conditions based on patient presentation (history, signs and symptoms) and multimodal imaging results?

3. Patient management and role of the optometrist

 a. What additional testing should be ordered for patients, and their family, who potentially have STGD1?
 b. Understand what referrals should be made for patients who present with visual acuity/visual field loss

4. Critical-thinking concepts

 a. Understand the varying phenotypes of STGD1 and how genotype influences this presentation
 b. Realize the potential of current research in regard to treatment options

Assessment of learning objectives

STGD1 is a complex disease owing to its complicated genotypic-phenotype interaction. Therefore, these teaching case reports are appropriate for third- and fourth-year students, as well as residents, who already have familiarity and knowledge of basic retinal anatomy, genetics and multimodal imaging interpretation. Suggested assessments include:

- Presenting the cases and discussion points as a component of a journal club at a student's optometry school, optometric rotation site or residency site. The students and/or residents could work in groups or independently to answer the discussion questions.
- In a classroom setting, students could be presented the cases with the goal of formulating a final diagnosis and proper management plan.
- Comprehension and knowledge could be further evaluated through open-ended questioning to the class or through formal testing with multiple-choice questions. Comments and further expansion on tested concepts can be provided if the question is answered correctly or incorrectly. This would allow students to build upon their current knowledge base.

Discussion

On a genetic level, STGD1 is caused by mutations to the ABCA4 gene, which encodes for the transmembrane ABCA4 protein, an ATP binding cassette transporter that is exclusively found in the rim of the rod and cone outer segment discs.[2] From the photoreceptors it transports retinoids to the RPE. More specifically, during the visual cycle, all-trans retinal is converted to 11-cis retinal. Light-activated rhodopsin and opsin release all-trans retinal, which combines with phosphatidylethanolamine to

form N-retinylidene-phosphatidylethanolamine (N-ret-PE). N-ret-PE is typically transported to the photoreceptor's disc surface for removal via the ABCA4 protein.[1,2] In STGD1, the malfunctioning ABCA4 protein causes the retinoid compounds to accrue in the outer segments of photoreceptors, leading to toxic levels of phosphatidylpyridinium bisretinoid (A2PE) in the photoreceptor membranes. A2PE is then hydrolyzed to A2E, an extremely toxic metabolite and component of lipofuscin in RPE cells. Toxic/excessive levels eventually amass, which results in RPE damage and atrophy along with adjacent photoreceptor damage and loss.[1,2,9]

Other variations of Stargardt disease, due to autosomal dominant mutations, exist but to a much lesser extent than STGD1. One such mutation is found in the ELOVL4 (Stargardt disease 3, STGD3) gene on chromosome 6q16. ELOVL4 is fundamental for the proper synthesis of very long chain polyunsaturated fatty acids (VLC-PUFA). VLC-PUFA constitute a large part of the phosphatidylcholine that is found in the outer segments of both rods and cones.[10,11] While the exact role of VLC-PUFA in the retina is unknown, it is believed to play an important role in photoreceptor membrane fluidity while also being potentially involved with phototransduction.[11] This mutation(s) in ELOVL4 results in a deficiency of VLC-PUFA that eventually results in retinal degeneration that damages the RPE cells.[11] Stargardt disease can also be inherited in an autosomal dominant fashion due to mutations in the PROM1 gene (Stargardt disease 4; STGD4). This gene has been implicated in the synthesis of photoreceptors with mutations leading to improper migration of PROM1 into photoreceptor outer segments with subsequent photoreceptor and RPE damage.[10] Discussion of STGD3 and STGD4 are beyond the scope of this article; therefore, STGD1 is the focus.

As mentioned previously, mutations in ABCA4 are implicated in STGD1. ABCA4 is considered a large and complex gene thus mutations can present with diverse phenotypic appearances, even among STGD1 patients.[1] Genetic and environmental factors are also thought to influence the phenotypic presentation as even family members with the same mutation can present differently.[2] Mutations in the gene have been implicated in cone-rod dystrophies, retinitis pigmentosa and age-related macular degeneration (AMD).[2,9,12] Genotype correlations to phenotypic presentation can be difficult owing to this heterogeneity. Overall, mutations can be classified into null mutations (which cause total loss of product encoded by gene) or missense mutations (alteration of the product encoded by the gene).[5] Patients with null mutations tend to present with early onset cases of STGD1. They correspondingly have more severe presentations and a poorer overall prognosis. In contrast, patients with missense mutations have symptoms that correlate to a later disease onset with milder clinical signs and a better visual prognosis.[13]

Early clinical signs of STGD1 may include a normal appearing fundus that progresses to bilateral loss of the foveal reflex and/or granularity or mild disruption of the RPE. This would correlate to normal or mild vision loss.[14] Fishtail or pisciform flecks can then develop within the posterior pole. These characteristic white-yellow lesions are composed of lipofuscin and are found at the level of the RPE.[2,12,15] They can vary overtime being that they may remain, resolve and eventually result in RPE atrophy and significant visual acuity loss depending on their location. As the condition continues to progress, RPE and outer retinal damage occur leading to RPE and sensory retinal atrophy, sometimes termed a "beaten bronze" appearance.[7] Further atrophy can in some cases result in a characteristic "bull's eye" maculopathy, which would correlate to severe vision loss and likely central scotomas. Severe cases can show retinal atrophy extending into the arcades.[7,16] Other findings can include a characteristic peripapillary sparing with STGD1. The reason for this is not fully understood. It is hypothesized that there may be an ideal ratio between the photoreceptors and RPE at this area and/or that the peripapillary area is less susceptible to photo-oxidative damage and lipofuscin accumulation owing to a thicker peripapillary retinal nerve fiber layer.[17-19] Late onset STGD1 can present similarly but with milder findings. The fovea tends to be spared or mildly involved leading to better visual outcomes.[20] It is unknown why the fovea is generally spared, but it is thought to be due to possible missense mutations.[5]

At times, the terms STGD1 and FF are used interchangeably; however, clinicians should note that they represent different phenotypic presentations with similar genotypes. FF can present in an equivalent manner to STGD1 but with more extensive retinal involvement with flecks extending into the midperiphery and a later disease onset.[1,21] The fovea tends to be less involved, which may allow for better visual acuity/outcomes.[21]

This phenotypic heterogeneity was evident with our two patients. Patient 1 likely had early onset STGD1, with findings isolated to the macula, resulting in significant macular atrophy and subsequent visual acuity loss. In contrast, patient 2 likely had FF as indicated by the extension of the pisciform flecks into the midperiphery. The preservation of fairly good visual acuity until middle age also supports this diagnosis. Also, the patient having one missense mutation and no null mutations correlates to her later disease onset and initially milder clinical signs.[13] It should be noted though, that patient 2 could have also used eccentric viewing to achieve her relatively good entering visual acuity. Eccentric viewing is a common technique used by patients with damage to the macula. By using this strategy patients can direct their eye so that the image falls on a non-foveal point or preferred retinal locus (PRL).[22] PRLs can be determined using microperimetry. Unfortunately, this test was not available to us.

Grading systems

Due to these heterogeneities, various attempts have been made to aid clinicians in grading and classifying Stargardt disease. The grading systems differ in that they are based on fundus signs, genetic testing and results of multimodal imaging such as FAF. The Fishman system was developed by Dr. Gerald Fishman and categorizes Stargardt disease based on funduscopic appearance and electrophysiological testing.[23] Stage 1 is characterized by macular pigmentary changes including mottling within 1 disc diameter of the fovea. Stage 2 shows pigmentary changes or flecks that extend into and beyond the arcades and/or nasally to the disc. Stage 3 shows RPE and choriocapillaris atrophy secondary to reabsorption of the flecks. Stage 4 is a continuation of stage 3 with worsening atrophy. Both patients in this case report would be classified as stage 4 based on this system. Genotype classification is based on the type and degree of underlying genetic mutations. Category A is for patients with two or more null mutations while category B includes patients with one severe null mutation and one or more missense mutations. Category C is patients with no null mutations but two or more missense mutations while category D is patients with one missense mutation.[24] While genetic testing was not performed on patient 1, patient 2 would be in category D based on her single missense mutation. Other grading systems also exist based on FAF and electroretinogram (ERG) findings as well as a classification system for early onset Stargardt disease.[16,24]

Multimodal imaging

Although cases of STGD 1 may be obvious on initial presentation, many cases are not so apparent. As mentioned previously, early onset cases present without obvious fundus signs. One study noted that a quarter of children with STGD1 present with no obvious retinal lesions.[25] Unfortunately, this can lead to delayed diagnosis. Research has revealed a median delay in diagnosis of 3 years.[25] Misdiagnosis is not limited to early onset cases. Late-stage findings in STGD1 include RPE atrophy, which may be mistaken for atrophy seen in late stages of AMD. This underscores the need for multimodal imaging to aid in timely and correct diagnosis, which may allow for prompt referral for LVR and possible genetic treatment trials. A multitude of testing options are available to clinicians to help aid in the diagnosis and monitoring of STGD1. This includes traditional fundus photography, ultra-widefield fundus photography, fluorescein angiography, FAF and electrophysiology testing.

Fundus photographs allow for documentation of retinal findings such as RPE atrophy and/or pisciform flecks, which were easily visible in both patients. However, it is lacking in that it can only detect more obvious or superficial lesions. Nonetheless, it allows for long-term monitoring of patients and can be used as a patient education tool. Clinicians should perform fundus photography on all patients but, if available to them, should specifically consider ultra-widefield imaging/photography. This allows for the detection of not only central presentations but also peripheral presentations of STGD1 such as the extension of flecks observed in patient 2.[5]

Fluorescein angiography has been used traditionally to detect the so-called dark choroid finding that is present in a majority (up to 80%) of STGD1 cases.[7] The dark choroid represents lipofuscin accumulation in the RPE, which results in the blockage of normal choroidal fluorescence.[7,26] Although fluorescein angiography has its purposes for early and mild cases of STGD1, it is not as valuable for advanced stages as testing will reveal large amounts of RPE atrophy/window defects correlating to the absence of the dark choroid finding.[2]

By far the most useful test available to clinicians is FAF. As explained previously, STGD1 is characterized by the accumulation of lipofuscin, which leads to classic findings such as pisciform flecks and RPE atrophy. Lipofuscin has autofluorescent properties and thus FAF can visualize/detect these findings despite the appearance of a normal fundus. Two characteristic findings are seen on FAF: hyper-autofluorescence and hypo-autofluorescence. Hypo-autofluorescence indicates a lack of RPE and thus a lack of lipofuscin, which correlates to RPE atrophy that can be seen in STGD1. Hyper-autofluorescence indicates an excess of lipofuscin in the RPE and thus a diseased or sick RPE. This also correlates to pisciform flecks of various sizes and shapes. Ultimately, the hyper-autofluorescence seen with pisciform flecks and within the RPE will transition to hypo-autofluorescence as the flecks and lipofuscin are reabsorbed leading to RPE atrophy. Other findings are also present with FAF. One finding that can be seen in patients is a hyper-autofluorescent border surrounding RPE atrophy/hypo-autofluorescence. This is thought to indicate possible disease progression in terms of RPE atrophy. FAF patterns have also been classified into subtypes based on the degree of atrophy. Subtype 1 is thought to represent the mildest form with slower rates of progression while the last subtype, 3, is associated with more rapid progression.[16] Lastly, peripapillary sparing is more evident and obvious with FAF when compared to fundus examination/photography.[2] These FAF findings were apparent in both patients as they presented with central hypo-autofluorescence, surrounding hyper-autofluorescent flecks, a hyper-autoflourescent border around the hypo-autofluorescence, and peripapillary sparing (Figure 7). Clinicians should perform baseline FAF in all patients that have or are suspected of having STGD1 based on the findings above. Baseline FAF should also be considered in family members if history or examination findings suggest possible familial involvement.

SD-OCT serves as a vital tool for assessing specific areas of retinal and choroidal involvement and for monitoring disease progression. The earliest known finding in children that has been imaged with OCT is external limiting membrane thickening.[27,28] However, these patients may be asymptomatic at such a young age making its detection more difficult. As

STGD1 is a disease of the outer retina, the inner retinal layers will be spared thus typical findings include damage and disruption to the macular outer retina layers including the EZ and the RPE. As the condition progresses, total atrophy of the layers will occur with preservation of the more peripheral macular tissue. These findings were seen in both patients as patient 1 had central EZ loss with accompanying RPE atrophy. Her peripheral retina was spared of any damage. Patient 2 had more mild initial findings as seen with foveal EZ and RPE disruption. However, this eventually transitioned to more EZ and RPE involvement and vision loss. This correlates to the typical bull's eye maculopathy appearance that is associated with STGD1. As almost all commercially available SD-OCT instruments have progression software, clinicians can use specific parameters such as total retinal thickness, macular volume and outer retinal thickness to monitor progression.[1] Although not needed to diagnose or monitor the condition, newer types of OCT, such as enhanced depth OCT and swept source OCT, can be used to visualize and image the choroid and its possible role in STGD1. Researchers have described four choroidal patterns that correlate with the loss of retinal structures and retinal integrity: stage 1 represents a normal choroid; stage 2 represents a reduced Sattler or Haller layer; stage 3 represents a reduced Sattler and Haller layer; stage 4 represents reduced Sattler and Haller layers with choroidal caverns.[4] Currently, investigators are unsure why these caverns form. Enhanced depth imaging was not performed in either patient involved in this case report. However, based on the appearance of the choroid on the available SD-OCT scans, patient 1 likely had stage 2 choroidal involvement with a reduced Sattler layer while patient 2 likely had stage 3 choroidal involvement.

Newer imaging technologies such as optical coherence tomography angiography (OCT-A) are emerging as possible imaging options for clinicians. Although FAF and SD-OCT are considered better imaging modalities, OCT-A has the benefit of allowing imaging of the retinal and choroidal microvasculature. Although they are a rare finding in STGD1, OCT-A could aid in diagnosing choroidal neovascular membranes that may not be visible with SD-OCT and/or FAF.[29] OCT-A has also allowed for a better understanding of the pathophysiology of STGD1. Research suggests that RPE damage on FAF is larger than choriocapillaris damage on OCT-A thus providing evidence for initial RPE involvement followed by choroidal damage.[4]

Functional testing including visual field testing can also be used in the monitoring of STGD1 patients. As the condition is mild in the early stages due to limited RPE and outer retinal damage, formal testing with static and kinetic perimetry is usually normal. As the condition progresses secondary to greater amounts of RPE involvement, relative central scotomas can be detected with either testing modality. Eventually, with greater amounts of RPE damage, patients transition to absolute central scotomas.[21,30] Exceptions can include patients with spared foveas, which correlates to ring scotoma-like field defects.[31] Although not traditionally associated with STGD, peripheral visual field loss can occur in patients whose disease has spread to the retinal periphery as evidenced by ultra-widefield imaging.[32] As noted previously, microperimetry can also be used in the management of STGD1 as its built-in fundus tracking and fixation monitoring enable PRL measurements. This allows clinicians to quantify a patient's remaining visual function.[2] Unfortunately, this technology is not readily available to all clinicians; the devices tend to be located at larger clinical research sites or LVR clinics. This underscores the need to refer patients to such sites when appropriate. Although visual field testing was not performed in either patient in this case report, the amount and degree of retinal atrophy seen in both patients would likely have resulted in absolute scotomas.

Next, electrophysiological testing can aid in the diagnosis of STGD1 but may be better suited for helping to predict patients' prognosis. Pattern ERG and focal ERG signals are typically not present or are noticeably reduced in patients with STGD1. This would correlate to macular involvement and damage; however, this might be also noted on OCT and FAF.[33] What may be of more importance to clinicians is the classification system based on electrophysiological findings created by Lois et al. They categorized subjects into 3 groups: group 1 – severe pattern ERG but normal scotopic and photopic ERG; group 2 – loss of photopic function; group 3 – additional photopic disfunction along with scotopic disfunction.[34] With this classification system in place, another study demonstrated that patients with ERG patterns in group 1 had the best visual prognosis while patients in groups 2 and 3 had intermediate and poor prognoses, respectively.[24]

Differential diagnosis

Different combinations of ABCA4 mutations are predicted to result in distinct phenotypes. Evidence indicates that ABCA4 is involved in the development of various other retinal diseases beyond STGD1, such as AMD, cone-rod dystrophies and rod-cone dystrophies such as retinitis pigmentosa. Therefore, these phenotypic variations should be considered as differentials before a final diagnosis is confirmed. Other common differentials are:

• **AMD.** AMD tends to present later in life and is characterized by the presence of drusen, which may be confused for lipofuscin flecks, in its dry form and the presence of choroidal neovascular membranes in its wet form. The flecks and drusen can be distinguished with FAF as the flecks will show intense hyper-autofluorescence while the drusen will show little to no autofluorescence.[12] Both conditions tend to present with RPE atrophy in their respective late stages; however, this presentation should be evident at an earlier age in STGD1 patients. AMD patients may also have a strong history of smoking and/or exposure to ultraviolet light.

- **Cone-rod dystrophies/rod-cone dystrophies.** Patients with a cone-rod dystrophy have an obvious lack of color vision but may also have foveal atrophy as seen in STGD1 patients. They lack the flecks (either on FAF or fundus examination) seen in STGD1 patients. The fundus presentation in rod-cone dystrophies shows bone spicules, vessel attenuation and optic disc pallor, features that are not seen in typical STGD1. Nyctalopia is also associated with these patients, a symptom that is rarely seen with STGD1 patients.
- **Pattern dystrophies.** These are a set of conditions that manifest at the macula in middle age and include adult onset vitelliform dystrophy, butterfly-shaped pigment dystrophy, reticular dystrophy, multifocal pattern dystrophy simulating Stargardt disease, and fundus pulverulentus. Most of the patients present with only mild vision loss with little or slow progression. The pattern dystrophies are due to mutations in the PRPH2 gene, which can be confirmed through genetic testing. The conditions also lack a dark choroid that can be seen in most STGD1 patients.[12]
- **Pentosan polysulfate sodium (Elmiron) toxicity.** Pentosan polysulfate is a medication commonly used in the treatment of interstitial cystitis but recently has been linked with a maculopathy that presents similarly to STGD1 based on fundus presentation and FAF patterns. A history of chronic pentosan polysulfate use can support the diagnosis.[35]

Treatment

As previously mentioned, no treatment for STGD1 is currently available.[10] However, gene replacement, stem cell therapy and pharmacological approaches, specifically vitamin A visual cycle modulators, are being investigated in clinical trials.

The objective of gene therapy is to introduce a properly functioning gene into the retina to account for the missing or mutated gene in question. This can be done either through intravitreal injections or through subretinal delivery of the gene.[2] This treatment option has been effective as evidenced by the many current clinical trials for inherited disease with one approved gene therapy on the market, voretigene neparvovec-rzyl (Luxturna).[36] Gene therapy could potentially be applied to STGD1; however, the size of the ABCA4 gene (6.4 kb) presents a problem. Most human gene therapy has used adeno-associated virus (AAV) vectors, which have a capacity of approximately 4.7 kb.[5,9] In hopes of overcoming this problem, researchers have shifted toward dual AAV vector delivery in which a gene is split and placed in two separate AAV vectors. Studies at this time have been limited to preclinical trials.[9] An alternative is lentiviral vectors which have a larger capacity (8 kb) to house the ABCA4 gene. Unfortunately, studies have shown little efficacy with this method.[2]

Stem cell therapy is an additional future treatment option. In a phase 1/2 clinical trial, STGD1 patients received RPE cells derived from human embryonic stem cells.[14,18] The study showed no safety concerns but was too small to determine efficacy.[37] A similar study was performed in which patients received escalating doses of human embryonic stem cells/RPE cells. Results were similar to the first study with no adverse effects but also no improvement in retinal function or vision.[38] Follow-up studies are ongoing.[9] Clinicians should also consider whether stem cell transplantation is a viable long-term option as it does not address the actual cause of the disease, the gene mutation. These stem cells are possibly a temporary option; however, they themselves can possibly still accumulate A2E and other toxic molecules.[39]

Other treatment possibilities under investigation are pharmacological agents or visual cycle modulators. These target different components of the visual cycle in hopes of preventing the accumulation of toxic molecules in the RPE.[2] One such agent is emixustat, which inhibits retinol isomerase RPE 65 leading to the reduced availability of 11-cis- and all-trans-retinal, and eventually toxic A2E.[2] Another option being studied is a deuterated vitamin A (ALK-001). In this form of vitamin A, hydrogen atoms are replaced with deuterium atoms. This substitution impedes vitamin A dimerization and thus reduces production of A2E.[2,9] Fenretinide is also being examined as a treatment option. It is a synthetic derivative of vitamin A that binds to and reduces free circulating retinol binding protein. This complex is excreted in the urine leading to decreased levels of vitamin A and thus A2E.[2,9]

While vitamin A alternatives are being studied, clinicians should also keep in mind the role of natural vitamin A supplementation in STGD1 patients. Excess intake of vitamin A is thought to lead to accelerated disease progression in some STGD1 patients as it leads to increased dimerization and thus lipofuscin formation.[40] However, an evidence-based review of vitamin A supplementation in STGD1 patients concluded that there are very few large studies from which to draw solid conclusions.[15] At this time, the National Eye Institute and the National Institutes of Health still advise patients to avoid supplements that contain more than the daily recommend allowance of vitamin A.[41]

RPE atrophy in STGD1 is due to A2E and other bisretinoids; however, evidence suggests that these compounds also activate the complement system leading to further atrophy.[2] Complement activation has also been implicated heavily in the development and progression of RPE atrophy in AMD patients.[2,42] Specifically, overactivation of the C5 and C3 components of the complement system leads to eventual activation of a membrane attack complex (MAC). MAC activation then leads to cell lysis and RPE and photoreceptor atrophy. Therefore, inhibition of C5 and C3 could lead to downstream inhibition of MAC and decreased RPE atrophy.[43] Due to the similar features of the conditions, therapies designed for AMD may be applicable to

STGD1. Two such recently FDA-approved medications are pegcetacoplan (Syfovre), a C3 inhibitor, and avacincaptad pegol (Izervay), a C5 inhibitor. Results from trials of both drugs have been promising in terms of preventing RPE progression in AMD.[44-46] However, indications for STGD1 are still under investigation. A phase 2b clinical study, the STAR trial, is ongoing for Izervay in the treatment of STGD1.[47]

Dietary supplementation for the treatment of STGD1 disease is also being studied. Omega-3 fatty acids including docosahexaenoic acid (DHA), eicosapentaenoic acid (EPA) and alpha-linolenic acid (ALA) are being investigated to determine whether they can improve macular function. A recent study showed improvement in objective and subjective vision in AMD and STGD1 patients using omega-3s.[48] Although promising, further research is needed as this study was limited to 21 patients.

With so many treatment options being investigated, the newest option may provide the best outcomes. Clustered regularly interspaced short palindrome repeat (CRISPR) is gene editing technology that can correct for gene errors.[49] The technology has been demonstrated to repair DNA in mice with genetic disorders and has the potential to treat varied inherited retinal disorders.[50] Currently, studies are lacking in human subjects.

Until a proven treatment option exists for STGD1 patients, clinicians should consider rehabilitative options, mainly LVR, to improve or maintain a patient's quality of life.[51] LVR can allow patients to resume and continue to perform activities of daily living such as reading, writing and driving with the assistance of hand magnifiers, telescopes and electronic devices.[52] Patients can be followed with multimodal imaging yearly to monitor for disease progression while waiting on potential treatment options. However, until that time, LVR services are vital to maintain quality of life. Although both patients in this case report were educated on the benefits of LVR, they ultimately did not receive these services as one patient was lost to follow-up and the other declined. The decision to seek LVR is ultimately the patient's; however, clinicians should continue to advocate for it even when patients think the services are not necessary.

It is also recommended that patients with STGD1 and patients who are suspected to have STGD1 undergo genetic testing and receive genetic counseling.[10,16] Genetic testing can help confirm and refine the diagnosis and allow patients to better understand how STGD1 can affect their vision throughout life.[16] Genetic testing can also help individuals qualify for certain clinical trials and future treatment options.[23]

Conclusion

STGD1 is a highly complex disease based on its phenotypic and genotypic heterogeneity.[2] It can manifest with variable signs and symptoms. Therefore, multimodal imaging and genetic testing play an important role in arriving at a proper diagnosis. Although no treatment exists currently, research into potential therapies is promising. Until an effective treatment emerges, clinicians should continue to monitor their patients for disease progression and make appropriate referrals for LVR.

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Assigning Fair and Defensible Clinic Grades: a Normative Statistical Approach

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Background

There are many ways to calculate a final grade in any course.[1] However, when it comes to individual evaluations of student work, there are only three types of grading: atomistic, holistic or analytical.[2] Atomistic grading strictly follows rubrics or grading keys, such as in static multiple-choice examinations. This method of grading is rigid and may not be well-suited for activities such as clinical patient care that require higher-level thinking.[3] Holistic grading uses an overall impression of the student performance and views grading keys as guides. This method embodies the idea that all professionals, regardless of discipline, approach any academic work with a set of expectations. Nilson refers to these expectations or criteria as dimensions and describes how most graders of written scholarly work can rely on as many as 20 dimensions to arrive at a single grade.[3] Sample analogies of these dimensions in graded clinical care may be the use of appropriate terminology, accuracy of clinical findings and proper analysis of these findings. However, it would be unfair to grade novice clinicians based on multiple dimensions gained from years of clinical experience. In addition, those dimensions may be different among graders. Thus, we turned to an analytical approach at our institution. In analytic grading, rubrics define expected performance levels for specified key dimensions of the overall graded task. Graders are then required to assign a separate score for each dimension.[4] Analytical grading remains challenging, even for very experienced faculty.[5] Despite supplied external criteria, some graders (knowingly or unknowingly) have a difficult time turning their private opinions or judgments into public ratings.[6]

Those of us who assign grades in clinical courses know some of the challenges very well. First, there is no consensus regarding the value of even assigning numerical or letter grades.[3,7-14] Assigning grades can keep students accountable and can be motivating, and students also care about grades.[3,8] However, at least one author has suggested that grading is largely based on cultural assumptions.[10] Others have also challenged grading as a reward, suggesting it is a demeaning practice.[11] At the very least, grading can inhibit learning.[12,13] For example, teamwork and cooperation is important in providing quality healthcare, but both can be discouraged by sorting students into merit levels.[3,14] The second issue is that, if grades are assigned, students want them to be a fair and valid representation of their work, but clinic academic managers mostly want grades to represent whether students will (or will not) succeed at the next level.[15,16] The third issue, which is the subject of this communication, is how evaluations from multiple clinic preceptors in various settings should be aggregated into a final course grade.

Previous authors have examined overall practices and imprecision of grading in medical clerkships, and at least one study has examined the effects of training on clinical performance evaluations.[17-19] While the studies are few, they all reveal that grades (which are often simple arithmetic averages of performance evaluations) are often inflated. We have also observed grade inflation and deflation and believe they occur when the responsibility of assigning the grade — rather than just evaluating clinical performance — is placed on clinic preceptors.

Clinic preceptors at our institution had previously expressed surprise or disagreement with the final clinic grade of certain interns even though their submitted clinic grades are often generous and uniform such that nearly the same grade is given to all interns across most patient encounters. This was perhaps the biggest motivator of the current work, where we simply describe our technique of transforming analytical evaluations from multiple clinic preceptors into what we consider fair and defensible grades. The technique is criterion-based, allowing students to understand how their clinical performance is tied to posted dimensions of clinical care. It is also normative-based, transforming raw numerical grades into standardized (i.e., z-) scores for each grader. To our knowledge, no published statistical approaches address the disparity in clinical performance evaluations submitted by various clinic preceptors. The purpose of this paper is to present a fair and defensible statistical method that allows preceptors to evaluate clinic performance as they see fit and enables clinic academic managers to assign grades using these evaluations in a way that corrects for between-preceptor differences in evaluating student performance.

Methods

Individual evaluations

Both third- and fourth-year optometry interns at our in-house clinics are given a single grade per day from each clinic preceptor, regardless of how many patient encounters they share. Interns may then receive as many as four different grades per day depending upon how many different preceptors they are assigned that day. This is administratively unavoidable due to scheduling and clinic productivity pressures. We have observed that some preceptors are unwilling to grade students differently, limiting the spread of their grades. In addition, preceptors may be biased toward grading leniently or stringently. To combat this, all interns and supervising preceptors are supplied with scoring criteria. We use five grading dimensions: efficiency (speed), accuracy of findings (skills), knowledge/analysis, communication, and charting/billing **(Table 1)**. Preceptors assign either exceeds, meets, or below expected for each dimension and enter the grades into a customized commercial medical education system (Meditrek, HSoft Corporation, Morrisville, PA). If an intern meets expected levels for all dimensions, their grade is 85%. For each individual dimension, intern grades are adjusted up (if exceeds) or down (if below) for that dimension. These adjustments are shown in the headers in Table 1. Preceptors are also encouraged to provide written feedback to interns, elaborating on either the overall performance or on specific dimensions. When entering the grade, preceptors are shown a draft (including the calculated numerical grade) before final submission. Interns never see the numerical grade. Rather, they only see written comments and whether they met, exceeded, or were below expected on the five dimensions.

Table 1. *Click to enlarge*

Arithmetic adjustments

To achieve the goal of improving fairness in grading, we had two sub-goals. The first goal is that mean grades for each given preceptor would be equal to the grand mean (GM, i.e., the average grade for all graders combined). This controls for inter-grader variability or directional (positive and negative) grader biases. The next goal is to control the intra-grader variability. That is, by example, we want a grade that is one standard deviation above the mean grade for a given preceptor to transform into a grade that is one overall standard deviation (OSD) above the GM of all grades.

Table 2. *Click to enlarge*

The best way to describe our adjustment technique is by a sample of hypothetical grades. All the presented data are hypothetical, and all statistics, tables and figures were produced using Microsoft Excel (2021). **Table 2** shows hypothetical data for five graders and five students. These hypothetical graders are lenient, average or stringent and give either uniform or varied grades. Obviously, there may be other types of clinic graders, but these are sufficient to demonstrate the technique. The third column of the table shows raw grades given by the grader to each student for each day in clinic. The raw grades are transformed into standardized (z-) scores by $z = (\text{raw grade} - M)/SD$, where M = preceptor mean and SD = preceptor standard deviation. For example, a raw grade of 98 from grader 1 (mean = 98.40, SD = 0.89) would be: $z_1(98) = (98 - 98.4)/0.89 = -0.447$. The z-scores are subsequently transformed back into adjusted grades by: Adjusted grade = GM + z-score x OSD, where GM = grand mean of all grades (88.32) and OSD = 6.33 for all raw grades. Using the same example of a raw grade of 98 from grader 1 (i.e., z-score = -0.447), the adjusted grade = -0.447 x 6.33 + 88.32 = 85.49. So, a grade that was approximately 0.5 SD below the preceptor's mean adjusted to a grade approximately 0.5 OSDs below the grand mean.

Results

To determine the relationship between raw and adjusted grades, we calculated the correlation coefficient (Pearson's r

reported), r = .318. This correlation was not statistically significant (p = .121) and may indicate for this small hypothetical sample that raw and adjusted grades are different representations of clinical performance. A scatter plot of raw vs. adjusted grades shows this limited relationship in **Figure 1**.
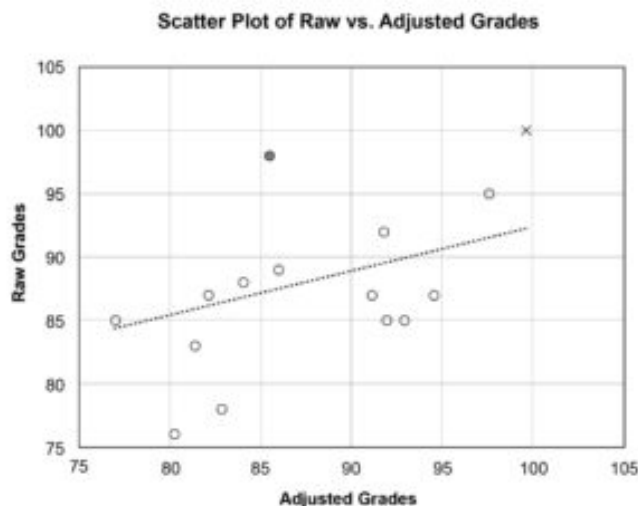

Figure 1. The filled circle and 'x' represent grades given by a lenient grader with a limited spread in grades (Grader 1). The raw grade of 98 represented by the filled circle is a below average grade for Grader 1; therefore, it adjusts to a below average grade overall (i.e., 85.49). The raw grade of 100 represented by 'x' is nearly two standard deviations above Grader 1's mean (z = 1.789) and adjusts to a grade 1.789 overall standard deviations above the average grade overall (i.e., 99.64). *Click to enlarge*
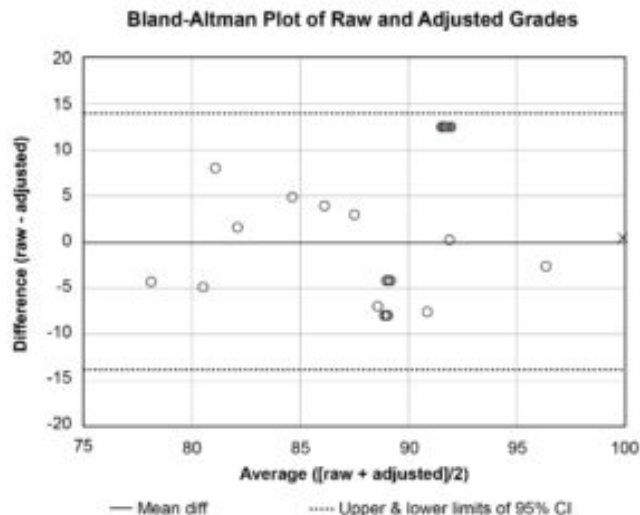
Figure 2. The data appear equally scattered above and below the mean difference line, which indicates that neither raw nor adjusted methods are biased toward high or low grades. The filled circles and 'x' represent the same points as in Figure 1. *Click to enlarge*

TABLE 3
Hypothetical Grade Data Sorted by Student

Grader	Student	Raw Grade	Adjusted Grade	Raw Mean	Adjusted Mean	Diff (raw - adjusted)	Average (raw, adjusted)
1 - Lenient, Equal	Student - 1	98	85.48	93.60	87.75	12.51	91.74
1 - Lenient, Equal	Student - 1	98	85.48			12.51	91.74
1 - Lenient, Equal	Student - 1	98	85.48			12.51	91.74
3 - Average, Equal	Student - 1	87	91.15			-4.15	89.08
3 - Average, Equal	Student - 1	87	91.15			-4.15	89.08
2 - Lenient, Varied	Student - 2	88	84.06	83.60	83.06	3.94	86.03
2 - Lenient, Varied	Student - 2	87	82.13			4.87	84.56
2 - Lenient, Varied	Student - 2	89	86.00			3.00	87.50
4 - Stringent, Varied	Student - 2	78	82.86			-4.86	80.43
4 - Stringent, Varied	Student - 2	76	80.28			-4.28	78.13
1 - Lenient, Equal	Student - 3	100	99.64	91.00	92.72	0.36	99.82
1 - Lenient, Equal	Student - 3	98	85.48			12.51	91.74
4 - Stringent, Varied	Student - 3	85	91.96			-6.96	88.48
4 - Stringent, Varied	Student - 3	87	94.56			-7.56	90.78
4 - Stringent, Varied	Student - 3	85	91.96			-6.96	88.48
2 - Lenient, Varied	Student - 4	95	97.61	88.40	93.65	-2.61	96.30
2 - Lenient, Varied	Student - 4	92	91.80			0.20	91.90
5 - Stringent, Equal	Student - 4	85	92.94			-7.94	88.97
5 - Stringent, Equal	Student - 4	85	92.94			-7.94	88.97
5 - Stringent, Equal	Student - 4	85	92.94			-7.94	88.97
3 - Average, Equal	Student - 5	85	77.00	85.00	84.41	8.00	81.00
3 - Average, Equal	Student - 5	87	91.15			-4.15	89.08
3 - Average, Equal	Student - 5	87	91.15			-4.15	89.08
5 - Stringent, Equal	Student - 5	83	81.39			1.61	82.19
5 - Stringent, Equal	Student - 5	83	81.39			1.61	82.19

Note: The last two columns contain the ordinate and abscissa data for the Bland-Altman plot

Table 3. *Click to enlarge*

Table 3 is sorted by student and shows all raw and adjusted mean grades. The last two columns of Table 3 are the paired grade differences (i.e., raw – adjusted) and averages (i.e., [raw + adjusted]/2), respectively, which are plotted against each other in **Figure 2** according to a technique described by Bland and Altman.[20] In Bland-Altman (B-A) plots, lines representing the mean difference — as well as upper and lower bounds of acceptable differences — are also plotted. Any group of standardized scores will always have a mean z-score equal to 0, and the final step of our adjustment technique is to multiply z-scores by the OSD then add the grand mean. Therefore, in our technique, the mean of all adjusted grades will be equal to the grand mean of the raw grades (see Table 2 for sample). The mean difference line will then always be along the x-axis (i.e., mean differences = 0). While Bland-Altman does not prescribe the upper and lower bounds, it is suggested that all points should lie within the 95% confidence interval (CI) of the mean difference. All our points lie within these bounds. The B-A plot can also highlight whether raw or adjusted grades are systematically too high or low. If so, most points would be above (if raw > adjusted) or below (if raw < adjusted) the zero line. Because the points on the sample B-A plot in Figure 2 are scattered equally above and below the x-axis, it suggests no consistent bias of one method vs. the other.

Discussion

Clinical interns are faced with managing many different conditions of variable complexities, and it makes grading their efforts challenging. Much of the difficulty in assigning fair grades in clinic courses arises from the use of multiple graders who want to run their own "classroom" and assign their own grades. We believe our technique fairly adjusts for most differences in preceptor grading.

The hypothetical grade data presented here helps us demonstrate the technique, but extreme cases could arise that are worth mentioning. Most of these cases would involve preceptors who give a limited spread of mediocre (i.e., "your work is adequate") or generous grades. The first (and easy) example to handle is a grader who awards the same grade to every student. In these cases, all z-scores are 0.0, and all their grades are adjusted to the grand mean for all grades. However, there are, in our experience, graders who award mostly the same grade but a limited number of generous or below average grades. For example, assume a preceptor awards 50 grades of 85, one grade of 70, and one grade of 100. The mean ± SD = 85.00 ± 2.29, and z-scores for the two "exceptional" grades are $z(70) = (70 – 85.00)/2.29 = -6.56$ and $z(100) = (100 – 85)/2.29 = 6.56$. Assuming, by example, our GM of 88.32 and OSD of 6.33, these grades adjust to adjusted(70) = $z(70)$ x OSD + GM = (-6.56 x 6.33) + 88.32 = 46.80 and adjusted(100) = (6.56 x 5) + 88 = 129.84. We believe these are fair adjustments, but they may create a perception of norm-referenced grading if the criteria are not properly and/or regularly applied. Barriers to doing so remain the subject of future studies.

We believe our technique corrects for two further, but related, challenges of clinic grading: bias and training. We have observed that there are extreme graders who are biased toward assigning either lenient or stringent grades. What are some causes of this? Leniency may be influenced by how "private" clinic grading is or how much they delay grading. One study found that individual graders were more likely to forget details than groups, but groups were more lenient graders after that same delay.[21] However, in our clinical settings, students work with an individual grader, so this source of leniency is unavoidable. One alterable source of leniency may be lack of training, as Ogden et al. demonstrate that training shifts preceptors toward more stringent, criterion-based grading.[19] They also suggest another likely cause of grade inflation: the tension between the mentor and grader roles of clinic faculty. That is, clinical faculty struggle with the shift from advocate or mentor during patient care encounters to judge during grading intern performance during these encounters. However, we believe this is one of the strengths of our technique. It allows clinic faculty to provide honest, legitimate, useful feedback to interns but puts the responsibility of calculating the final grade on the course coordinators.

Regarding training, there is documented intra-school inter-grader variation in medical school clerkships, and one known barrier to consistency between graders is lack of grader training.[18] Training matters, and it has been shown that face-to-face training changes grading practices more than computer-based tutorials.[19] However, there are administrative challenges to delivering face-to-face training with multiple preceptors practicing at multiple clinic sites. Regardless of the delivery mode, we encourage institutions to critically examine their training practices. Regarding non-administrative barriers to giving graders feedback about their biases toward stringency or leniency, it is possible that informing extreme graders of their practices may just create more "average" graders. In fact, our method may not always be wise, as arithmetically handicapping certain graders has also been shown to create more extreme grading.[6] Further investigations of the effect of training on preceptor grading is warranted.

There are also issues dealing with multiple clinical encounters in a single day. Some preceptors are known to adopt a more sensitive "parallel" paradigm. That is, single clinical mistakes result in a low averaged grade for the multiple encounters. Others use a more forgiving and specific "series" approach. These graders give low grades only if the student makes mistakes on more than one or all the encounters. In addition, is it a limitation to have the grade displayed as a draft before submitting? It is certainly possible that it encourages preceptors to reject the analytical grade in favor of a more holistic "this is the grade I think they deserve" approach. Another limit to our approach is that most interns switch preceptors approximately halfway through the semester. If a preceptor shifts toward more stringent or lenient grading within a semester, the within-grader norm-referenced nature of the technique would cause an unfair shift toward lower or higher grades, respectively. However, this is no different than if the grades were not adjusted. We have no data on how many series or parallel graders we have or whether our preceptors shift in leniency or toward holistic approaches. These would be interesting extensions of our observations.

Student perspectives should not dictate final grades, but their opinions are valuable.[22] There was concern that students would not like having the numerical grade withheld. They can calculate individual grades from the rubric, but they are reminded that their final grade is adjusted and are made aware of this technique. We never solicited opinions of students prior to launching this technique, but there is an informal global student acceptance based on post-course instructor evaluations.

We did not focus any of the current effort on the written feedback provided to the students by their preceptors. The software allows free text space to provide written feedback, but this is not required of preceptors. Written feedback has been shown to improve performance on follow-up, but our anecdotal experience is equivocal.[23] Some interns tell us that they appreciate the feedback while others do not use or even read it. The latter behavior seems unusual but compares with previous reports.[24] Formal analysis of intern and faculty acceptance of adjustment techniques is a worthwhile area of investigation.

Conclusion

Grading students is challenging. The variability of clinical encounters makes grading student clinicians even more so. While we cannot draw any inferences about clinic grading at other institutions or training settings, we do present a reproducible method of converting clinic performance evaluations into fair and defensible grades. The technique adjusts for between- and within-grader differences in leniency and uniformity, while interns still receive criterion-based preceptor feedback they can use to improve their clinical performance. We challenge fellow clinic academic managers to honestly examine their grading practices and consider a similar technique.

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Alcon

Meet the Journal's New Editor

| Optometric Education: Volume 49 Number 1 (Fall 2023)

Keshia Elder, OD, MS, MS, FAAO, discusses milestones and making her way to optometry

Keshia Elder, OD, MS, MS, FAAO, talked with Kimberly O'Sullivan, ASCO's Director of Communications, about becoming editor of the Association's peer-reviewed online journal, *Optometric Education* (OE):

ASCO: Dr. Elder, congratulations on becoming the editor of *Optometric Education*. Before we talk about the journal, people may not know that you are the new(ish) dean of the University of Missouri at St. Louis College of Optometry and the first Black/African American woman to be appointed dean of an ASCO institution. Congratulations and what an historic achievement.

Dr. Elder: Thank you! Thank you! Thank you! Whenever someone makes the comment that I'm the first Black/African American female dean of an optometry institution, it catches me by surprise. Surprised because it took until 2022 for this milestone to be reached. Of course, I am grateful to be in this place and able to continue my journey in optometric education. As I have said before, I may be the first, but I will not be the last.

ASCO: Congratulations again on becoming dean. Can you explain your journey to becoming the editor of OE? What interested you in the journal?

Dr. Elder: I think this journey has been a long time coming. I served on the Editorial Review Board for years when then-editor Aurora Denial, OD, FAAO, DAAO (OE), sent out a call for an associate editor. I was very interested in this opportunity and reached out to her. I come from a family of educators. My mom and dad were elementary school teachers. My undergraduate degree is in education. I taught high school math for a semester before I started optometry school. I consider myself a clinician-educator. The journal, which as you mentioned is peer-reviewed, is the only journal solely focused on optometric education, and it is very much aligned with my interests. I was thrilled to become an associate editor and I learned so much that becoming the editor has been less daunting. I couldn't be more grateful for the opportunity.

Keshia Elder, OD, MS, MS, FAAO

ASCO: You mentioned that OE is the only journal dedicated to optometric education or academia. What do you think people should know about academic optometry that they may not know?

Dr. Elder: The first thing that comes to mind is that most optometry professors are not trained teachers. We are mainly clinicians and subject-matter experts. Just because you are a great clinician does not necessarily mean you are a great teacher. Most of us must work at it. People should understand that when you are in the optometric education space you need to maintain clinical skills, but you also need to devote time to becoming a better educator. The pedagogy of teaching is very important and OE addresses that.

ASCO: What is the process of getting an article published in OE?

Dr. Elder: *Optometric Education* is an open-access, peer-reviewed journal that is available online free of charge. I recommend people review the Publication Guidelines online if they are interested in submitting an article. If a submitted manuscript is appropriate for the journal's audience, the editor assigns it to two or more members of the Editorial Review Board, who are subject-matter experts on the manuscript topic, and the peer-review process begins. Initial reviews are usually completed approximately a month after submission, and when a paper is approved it is published within the next 12 months. Authors should submit two copies of their manuscript, one blind and one unblind. The blind copy omits the name(s) of the author(s), their institution(s) and any other information that could reveal their identity.

ASCO: What would you say to someone who is interested in becoming a peer-reviewer and/or submitting an article for

consideration?

Dr. Elder: For people who are considering submitting – I say do it. Take a look at the journal homepage to see the types of articles that are published. Some go through the peer-review process, and others do not. We publish original research, Teaching Case Reports, Educator's Podiums, Guest Editorials and special themed issues such as the one we're working on now regarding global optometric education. I would also suggest potential submitters consider what their peers at other institutions are researching and don't be afraid to ask questions.

For those interested in becoming a peer-reviewer, I recommend they publish a peer-reviewed paper first. I have found that a good peer-reviewer is typically also a good researcher. Feel free to reach out to the journal staff because we periodically update our Editorial Review Board. Of course, also talk to the dean/president at your institution and ask for any insight and suggestions they may have.

ASCO: Tell me more about you and your journey to optometry. I remember you saying once that you "found your people" in optometry. I love that. Can you expand on that a bit?

Dr. Elder: I find that Doctors of Optometry are a dichotomy. Many of us are personally introverted but we talk to people all the time. I know many of us were drawn to optometry because we are caregivers who are very comfortable in one-on-one settings but may be less comfortable in larger settings. We are intellectuals who care for the greater good. When I reflect on myself, my personality and other Doctors of Optometry, I see how we do a lot of great things to help patients quickly and I see how we are directly and profoundly impacting the lives of people.

I truly do believe I have found my people.

ASCO: Thank you. As we wrap up this interview, we must say a huge thank-you to Dr. Denial who was editor of OE for 14 years! Her accomplishments and dedication to the journal cannot be overstated.

Dr. Elder: I absolutely agree. Dr. Denial has been an outstanding editor, leader and support system to me as we were navigating the outgoing and onboarding. When I think of her and all she has done for optometric education and for me personally, I don't have the correct words to fully describe all the gratitude I have.

ASCO: Thank you, Dr. Elder. We look forward to reading the future editions of *Optometric Education*.

Getting to Know ChatGPT – Notes from Our Initial Conversations

Narayanan Rajeev, PhD, Zixuan Qiu, PhD, Dylan Eng, MOptom, and Nagaraju Konda, PhD | Optometric Education: Volume 49 Number 1 (Fall 2023)

Generative artificial intelligence (AI) is the term used for algorithms that can generate new content including text, audio, images and video.[1] As you likely know, a recent breakthrough in generative AI is the chatbot ChatGPT (Chat Generative Pre-trained Transformer). ChatGPT (https://chat.openai.com) is able to answer a wide range of complex questions and perform many advanced tasks. It is based on an enhanced version of GPT-3, a speech-processing AI model developed by OpenAI. GPT-3 is equipped with a staggering 175 billion parameters, making it the largest and most complex language model ever.[2]

It has been widely reported that within 2 months of becoming available to the public, ChatGPT attracted more than 100 million users. Curious as to whether this powerful new tool could, or should, be used by optometry educators and students, and inspired by scholarly contributions from health care,[3,4] we used multiple prompts to test the bot's capabilities in February 2023.

Opportunities and Caveats

Table 1 lists inputs/prompts we used to explore ChatGPT's capabilities along with our general assessments of the resulting outputs/responses. We found that the technology can serve as a useful virtual assistant to optometry educators for certain routine day-to-day tasks, potentially freeing up time for higher-level tasks. In addition, because ChatGPT converses naturally on knowledge and comprehension, it may enable educators to focus more on facilitating student learning, for example, by empowering students to tackle certain basic learning activities on their own. Similarly, using ChatGPT as a self-directed learning tool is a potential way students can take ownership of their learning, which is key to their success in optometry school and their lifelong success as optometrists. Prompted with custom queries, ChatGPT can provide personalized interactive support, real-time feedback, and assistance with self-assessment and revision.[5]

However, if educators and students are to contemplate uses such as learning facilitation and self-directed learning, they must be aware of ChatGPT's current shortcomings. For example, while the bot is able to predict the next possible word in a sentence, it does not seem to understand whether the information is true. Some of its output may be false, vague, biased and devoid of depth.[6] Also, the output is not always the same even if the same prompt is repeated, and responses seem to be more accurate in the recall of facts and concepts (i.e., lower-order Bloom's taxonomy) than for higher-level, more cognitive questions.[4]

Table 1. *Click to enlarge*

Given the current shortcomings of ChatGPT, some education institutions have partially or completely banned students from using it on school servers.[7] Institutions and educators who are encouraging use of the tool to improve learning[8] should actively guide students with regard to its potential benefits[8] and limitations. For example, much like case discussions and team-based projects, ChatGPT could be used to promote critical-thinking skills, but only with educator coaching on the need for critical analysis of the authenticity of the information it provides. Additionally, because ChatGPT has been shown to write essays[9] and answer questions well enough to pass medical exams, now is a good time for institutions to reinforce the dire consequences associated with plagiarism and cheating.

Conclusion

The functionality and authenticity of ChatGPT will undoubtedly change rapidly. As time goes on, it will likely impact the field in many ways. Already, it has put educators on alert for ChatGPT responses in students' written work. Furthermore, assessments of student competencies that require them to reproduce knowledge without interpretation or application are likely to have a limited role in the future. In the meantime, based on our initial exploration of its utility as summarized here, this AI tool can be cautiously embraced in optometry education.

Acknowledgements

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Student Mental Wellness and Optometric Education

Keshia S. Elder, OD, MS, MS, FAAO | Optometric Education: Volume 49 Number 1 (Fall 2023)

During my 17 years in academic optometry, much has changed. I think about one change in particular — students' increasing struggles with mental wellness — exponentially more today than I did a decade ago. Today's students are more open with discussing mental wellness, and my colleagues and I frequently discuss the anxiety and stress they manifest.

What we see in our schools and colleges matches what is being documented in the literature, especially since the COVID-19 pandemic. An analysis of data from the long-term national Healthy Minds Study showed that in 2020-2021, more than 60% of college students met criteria for one or more mental health problems, a nearly 50% increase from 2013.[1,2] Results from the analysis also indicated that while mental health has been worsening among all undergraduate and graduate students, racial and ethnic minority students face the additional burden of what the study authors called a mental health "treatment gap."[1] In general, current mental health needs are outpacing the resources on most college campuses.[3]

It is clear that student mental wellness is an issue that needs our attention. Not so clear at this time is exactly what we should be doing.

How Do We Best Support our Students?

Often, student mental health support is focused on wellness education and programs, improving access to care and reducing the stigma of seeking help.[4] At least one university school of medicine tested a different approach and explored altering aspects of the curriculum, such as scheduling and grading, known to be student stressors.[4] Strategies that have been implemented in various settings (often through student services departments), including at schools and colleges of optometry include:

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- educating faculty and staff about the resources available at their institution
- training faculty and staff on how to identify a student in distress and the protocol to follow
- wellness activities such as meditation and yoga
- providing access to on- or off-campus virtual or in-person counseling (This can be achieved through campuswide resources or by contracting with a local counseling group.)
- hosting a wellness group with associated programs and activities, driven by either students or faculty
- regularly scheduled mental health check-ins

Time to Dig Deeper to Find the Best Solutions

While any of these strategies can be a step in the right direction, they leave us with multiple unanswered questions we will need to address. Although current supports are likely helpful, are they enough? What do students really need? What actions would go beyond supportive and reactive to be preventive? What should be the day-to-day role and responsibility of faculty and staff? How do our institutions adequately meet the mental wellness needs of our students with limited resources and limited mental health expertise?

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