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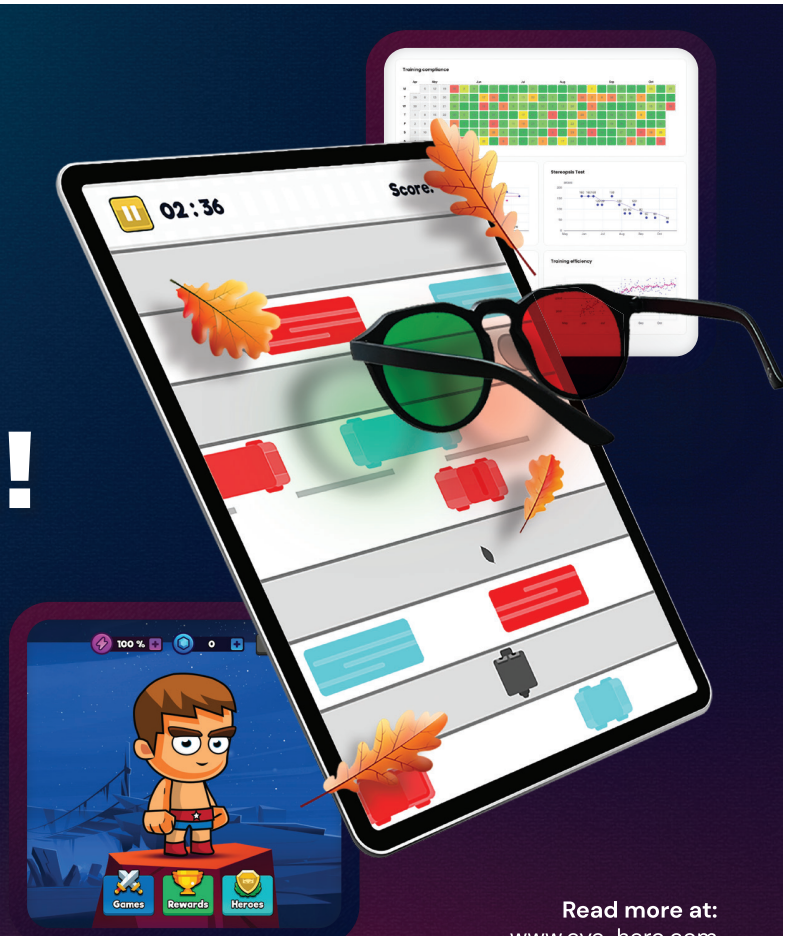

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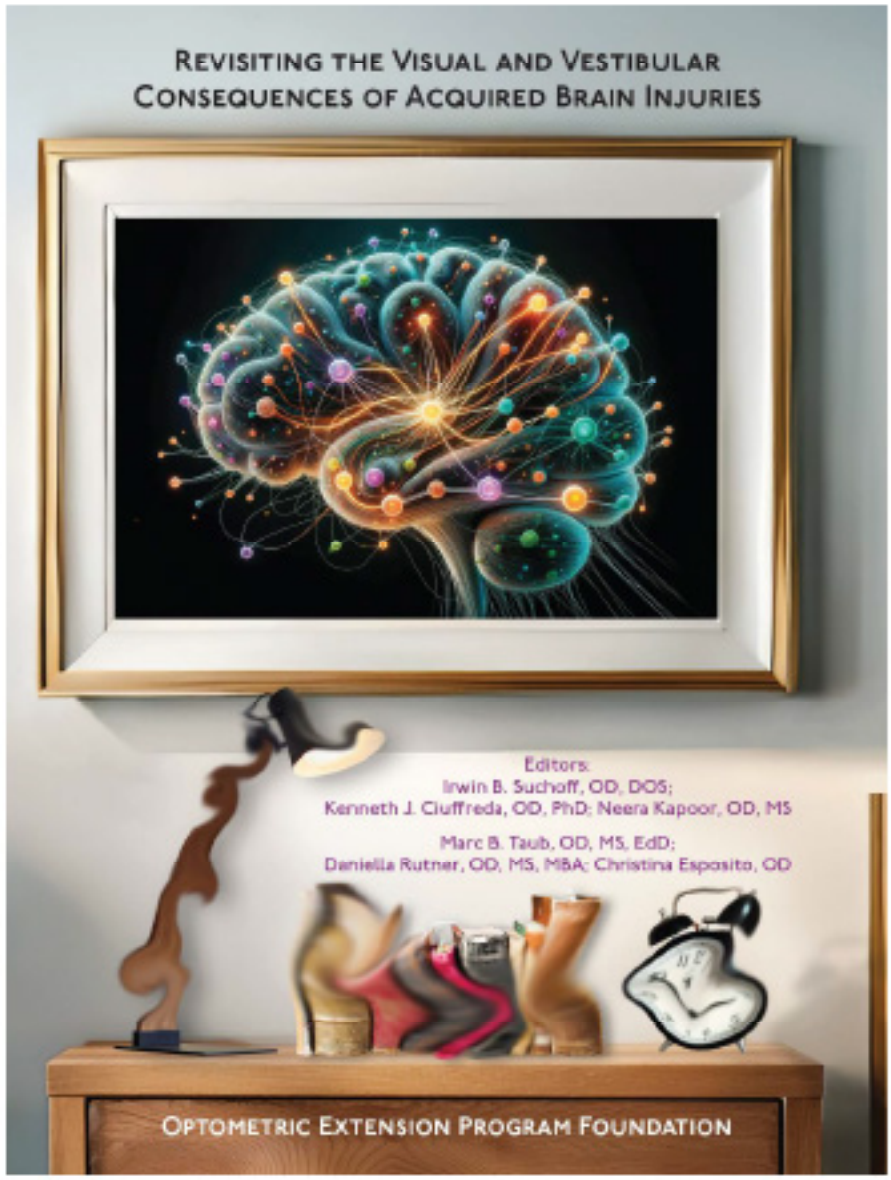
Understanding vision and balance after brain injury requires more than one lens.



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Editorial • An Interview with OEPF Board Chair, Dr. Pamela Schnell

What is your educational background?

UNC Chapel Hill, BS Biol, 1996

SCO, OD, 2001

SUNY, Pediatric Optometry Residency, 2001

What was your motivation to become an optometrist?

Lifelong interest & glasses as a young kid

Tell us about your optometry school experience and how it shaped your desire to do a residency.

Prior to optometry school, I enjoyed several opportunities to work with children, including working as an Assistant Leader for a local Girl Scout troop and teaching young children at church. I became interested in pediatric care during my 3rd year of optometry school, when we began seeing older kids come through our primary care clinics. I had enjoyed participating in school vision screenings during our 2nd year as well, and early clinical experience solidified that interest.

Tell us about the residency program.

I spent a fantastic year in pediatric optometry at the State University of New York College of Optometry. Working with Dr. David FitzGerald (always a capital G!) as my supervisor was a privilege; I learned a tremendous amount from him about infant care. I am honored to say that he quickly became a close friend and mentor. During the year, I also had the opportunity to learn from a number of other notable optometrists in the areas of pediatrics and vision therapy, including. Harold Solan, Dr. Israel Greenwald, Dr. Ira Krumholtz, Dr. Sid Groffman, Dr. Neera Kapoor, Dr. Ida Chung, and Dr. Marilyn Vricella. All of these (and many more) showed me the wide variety of philosophies and treatment protocols available to us in pediatric care. Fitz (he goes by no other name) also inspired in me a love of teaching that provided me with a completely new direction for my future, and I'm so grateful!

What was your path to becoming an educator?

See above

What led you to come back to SCO as an educator?

Throughout my years at SUNY (I stayed on faculty there for a total of ten years to the day from the start of my residency) I always imagined one day being able to have a lifestyle more similar to what I had grown up with: a house outside the city and (finally!) a dog! I loved the people I worked with at SUNY, and I enjoyed life in NYC, but living in the Big Apple carries a level of background energy that can get to be a bit much as one gets older! I had stayed in touch with several friends back at SCO, and when one mentioned at an alumni event that they were looking for peds people—and my husband and I realized that the timing was right—we were off to Tennessee.

When did you join OEPF and why?

I joined OEPF in 2018 when I became a Director/Trustee for the organization. I had been introduced to the behavioral philosophy when I returned to SCO, and I quickly fell in love with the holistic nature of the diagnostic and treatment ideas contained in it. To have an opportunity to serve the organization as a Board member seemed like something that I might do at some point, but the need arose to fill a position, and I was thrilled to accept!

What are your responsibilities are Board Chairperson?

As Board Chair for OEPF, my responsibilities largely involve working closely with our President

and CEO, Line Vreven, to ensure that the Foundation continues to run smoothly day-to-day. Through the internationally known Clinical Curriculum, our newly established Wednesday night lecture series, several chat groups for both students and practitioners, and a wide variety of publications, our mission of providing up-to-date, inspiring education in behavioral vision is well supported. We also sponsor the International Congress of Behavioral Vision (ICBO) once every four years, which brings together vision therapy organizations from all over the world in one location to learn from, collaborate with, and enjoy growing friendship with doctors and therapists who all love VT! Our next ICBO will be March 1-4, 2028, in Las Vegas, Nevada. We will also celebrate OEPF's Centennial, so it's going to be a fantastic celebration—we'd LOVE to see you there!

You also serve as the Managing Editor for OVP, how did that happen and what does that entail?

I began working with OVP's forerunner, the Journal of Behavioral Optometry, in 2011, serving as their Associate Editor. When that journal's run came to an end and we founded OVP, it was natural for me to move into the same role at the new publication. After a year or so, I was promoted to Managing Editor and have thoroughly enjoyed working with Dr Marc Taub and the Associate Editors to bring you this amazing international collaboration!

How do you think OEPF can serve the next generation of optometrists?

As we learn more and more about the links between neuroscience and vision rehabilitation, it has become more and more obvious to me that the behavioral philosophy—a holistic approach to vision therapy that pays close attention to the patient's underlying reasons for the visual adaptations that they have made—is the best way to help guide patients to their best visual potential. Moving into the future, OEPF can not only help established providers become more experienced with their VT, but it can also inspire whole new groups of ODs who want to expand their practices into new and exciting areas of patient care. We're here for you, come check us out!

What are your hobbies and how do you relax in your non-optometry time?

I'm a bit of a homebody, so I enjoy spending time there with my husband and our dogs! I love to read and crochet and be able to relax doing "nothing" on occasion, but I also love seeing new places and being able to travel when the opportunity comes along. I have had several opportunities to lecture in locations across the world, including Israel, South Africa, Mexico, Canada, and most recently Poland.

A bit more than a hobby now, I am also a semi-professional musician. I have played a number of instruments since childhood and still perform on flute fairly regularly. I started singing as a young child in church choirs and picked up that again when I moved to New York; I met my husband David in the Marble Collegiate Church choir in the city, a classical tenor who inspired me to keep learning. We've been singing together in various choirs and organizations ever since! I am currently a member of the choir at First Baptist Church in Memphis (my home congregation), and I do gig work for a number of others, most notably St. John's Episcopal Church, with whom I have had the honor of traveling to both Ireland and the UK to participate in choral residencies at St Patrick's Cathedral in Dublin and Liverpool Cathedral in England. I am also a member of the Memphis Symphony Chorus and the Memphis Symphony Chamber Choir, so my weeks are kept busy! I honestly wouldn't know how to live a life without music and am so grateful for the opportunities I've had.

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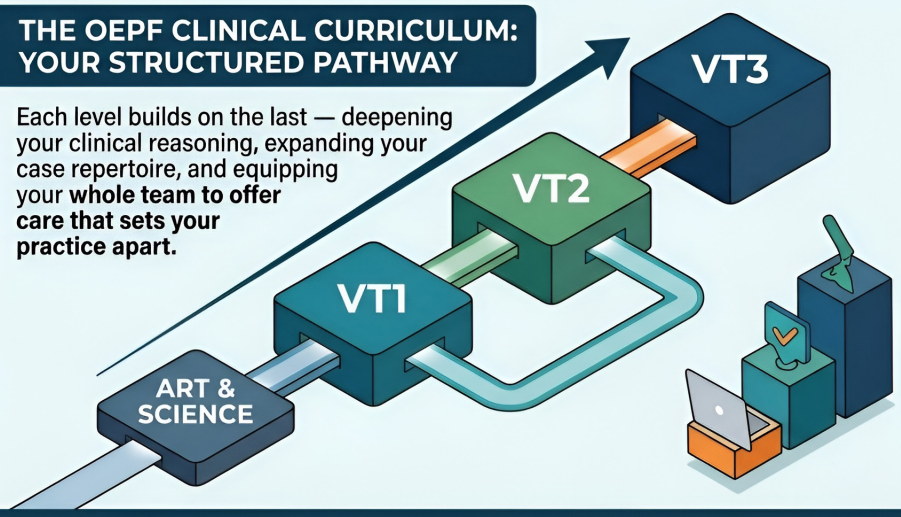

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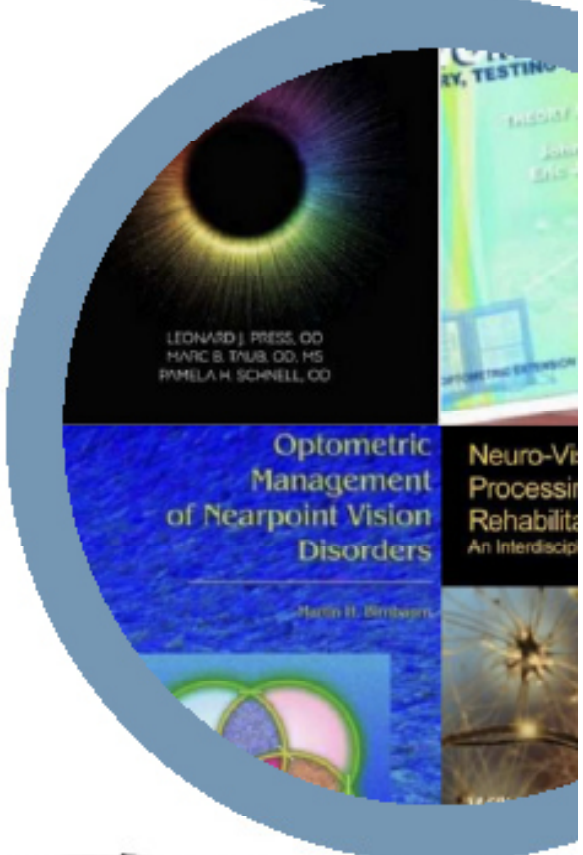

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Viewpoint • Assessment of Fixation

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The ability to fixate accurately and stably is critical for optimal viewing of the world. However, many of our patients do not fixate well.[1] This would include those with nystagmus, macular degeneration, optic atrophy, albinism, stroke, traumatic brain injury, and more.[1-3] Thus, it is incumbent upon the optometrist and others (e.g., a vision therapist or neurologist) to assess a patient's fixational ability appropriately. In this paper, various methods for assessing fixation, both traditional clinical and objective approaches, will be considered.

Table 1 presents a wide array of the more traditional clinical approaches to assess fixation.[1,4,5] Gross visual observation is perhaps the most common and simplest approach, although it requires excellent observational skills and knowledge of potential abnormalities on the part of the clinician (Figure 1). The doctor has the patient fixate upon a small target, such as a pen tip or Wolff wand, for 10 seconds (or more), monocularly and binocularly, and integrates by visual memory what is observed, such as the number of fixational losses and their estimated magnitudes, differences noted between each eye and binocularly, the presence of large saccadic intrusions or drift, subtle nystagmus, and more.[1] Then it is repeated across different diagnostic gaze positions (e.g., downgaze in reading). This is an adequate screening tool for detecting gross abnormalities. It can also be performed using the slit-lamp for higher magnification and hence better detectional ability, especially for abnormalities such as subtle, small, intermittent nystagmus.[1] The next four approaches listed in Table 1 mainly apply to the assessment of eccentric fixation (EF) in amblyopic eyes. The Haidinger brush technique is very useful, as it is easy for the patient to visualize, readily provides both direction and magnitude, and can be performed in a young child. The others listed are rarely used in the clinic.

Table 1. Traditional Clinical Approaches to Assess Fixation

Gross visual observation
Angle kappa determination
Blind spot comparison
Maxwell's spot
Haidinger's brush
Visuoscopy

The last approach, however, namely 30-second, low-light visuoscopy developed by Selenow and Ciuffreda,[4] is extremely useful and very informative. It not only provides the EF location but also allows the clinician to observe directly on the retina the actual fixational pattern and the underlying types of eye movements (e.g., increased amblyopic drift). It was based in part on an earlier experiment by Lawwill in 1966,[4] in which he observed that excessive light intensity exacerbated, or even "created" (i.e., iatrogenically induced), any fixational abnormalities. Hence, what Selenow and Ciuffreda developed and tested was a low-light technique. Thus, the light intensity of the visuoscope is reduced until the doctor can "just" detect the projected calibrated grid and central target on the patient's fundus. Of course, the patient readily perceives it. The patient is then instructed to fixate carefully on the central target. Since EF is a time-average, perceptual-motor phenomenon,[4] the foveal/macular region is assessed for 30 seconds (or more) to build up a sufficient mental image of the EF's direction, magnitude, variability, and overall pattern. The observed details are then fully described and recorded. The fellow eye is likewise tested for comparative purposes. It is repeated during the course of therapy to assess for likely reduction and hopefully

Figure 1. Test of fixation by visual observation

Table 2. Objective Approaches to Assess Fixation

Infrared limbal sensing
Video recording
Microperimetry

the attainment of consistent and accurate foveation.

Table 2 presents some of the objective approaches to assess fixation. This allows for a more detailed, quantitative, and unbiased appraisal of fixational ability, including the overall pattern and types of abnormal eye movements (e.g., increased amblyopic drift, large saccadic intrusions).

An early approach was the infrared, horizontal, limbal technique (Figure 2).[1] Basically, the eyes are bathed with safe levels of infrared light, and the two detectors for each eye are aimed at the horizontal nasal and temporal regions to assess the differential light reflection on the sensors: the white sclera reflects more light than the darker iris. This type of system has been used in many laboratories over the past 75 years, as well as in the clinical domain (e.g., ReadAlyzer; https://www.compevousa.com/). Again, the patient is instructed to fixate carefully on a small target on a screen, usually 40-57 cm away along the midline in the primary position, for several seconds. The resultant horizontal fixational pattern can be viewed as a function of time, printed out for the record, and even quantified if a prior calibration was performed.

Newer technology uses a video-based approach (Figure 3).[1] Here, small video cameras are aimed at each eye, and both the horizontal and vertical eye movements are recorded during fixation. Thus, this provides a more complete picture of fixation. These systems are easy for the clinician to use and provide a wide linear range of movement for accurate assessment. This includes various laboratory devices (e.g., Tobii; https://gaming.tobii.com/) as well as clinical devices (e.g., RightEye; https://righteye.com/). However, the latter system does not allow for testing of fixation under monocular conditions, which is critical for both

Figure 2. Infrared limbal eye movement system

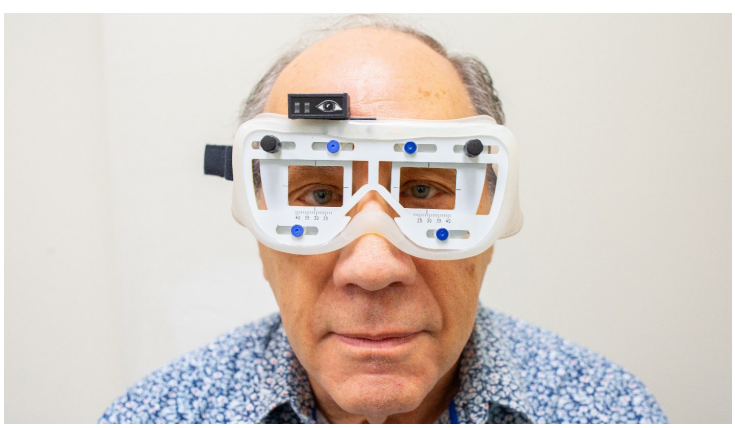

Figure 3. Tobii eye tracking video system assessing reading eye movements

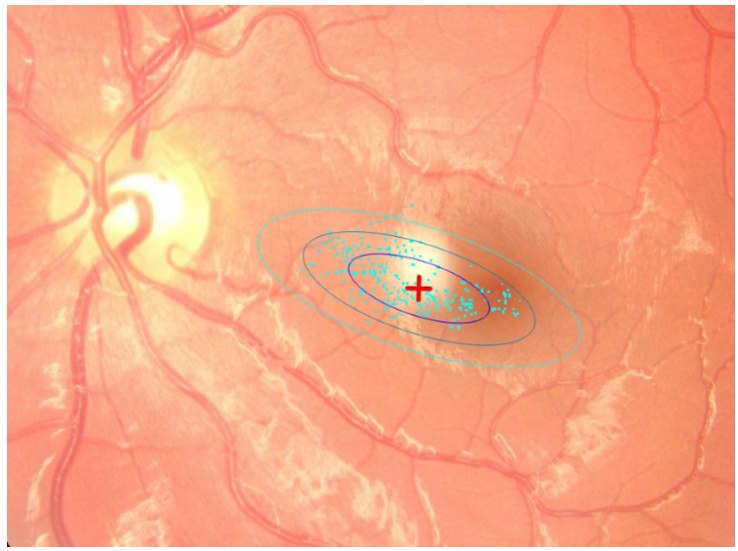

Figure 4. Microperimetry showing eccentric and unsteady fixation in an amblyope

EF assessment in amblyopic eyes, as well as eccentric viewing (EV) assessment in ocular disease/visual field loss situations; furthermore, it does not present the eye movements as a function of time like most instruments do. Rather, it presents each fixational sample in x,y Cartesian coordinate space with disregard for time.

The third and newest approach has been the use of microperimetry for fixational testing (Figure 4).[7-9] This involves direct visualization of the fundus area of interest (similar to visuoscopy), typically the foveal/macular region of only one eye. The patient is asked to fixate the target carefully, and the calibrated system samples both the horizontal and vertical eye positions at 25 Hz (i.e., 25 samples per second) or higher for 30 seconds. Once the test is completed, an overlay of the area of fixation is presented as a series of calibrated bivariate ellipses, typically enclosing the retinal regions of fixation 25, 50, 75, and 100% of the time:[1] the less fixational scatter, the smaller the ellipse, and the better the fixation. From this, one can obtain the retinal point of maximal fixation and its variability, all objectively documented—that is, either the fovea, the EF point, or the EV point. This approach is so important because the

clinician can visualize the oculomotor activity and the visuomotor, spatial centrode directly on the patient's fundus in real time.

In conclusion, the clinical and laboratory assessment of fixation in our patients has expanded greatly over the past 20 years. This is mainly the result of improved technology and rapid automated analyses, with excellent graphical display for the clinical record. Such objective documentation is helpful for the parent to understand how the abnormal eye movements negatively impact their child (e.g., slowed reading), as well as for presentation as an expert witness in the courtroom.[10] Future technological advances are likely to include expanded use of virtual/augmented reality to create more naturalistic viewing/testing scenarios.

Acknowledgements: We thank Dr. Putnam for her discussions regarding microperimetry and Dr. Wang and Bhavatharini Ramakrishnan for providing the microperimeter fundus image.

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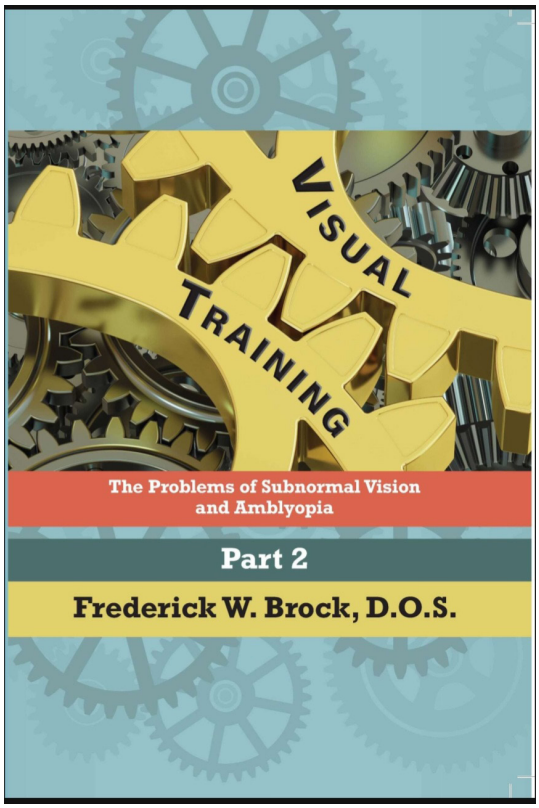

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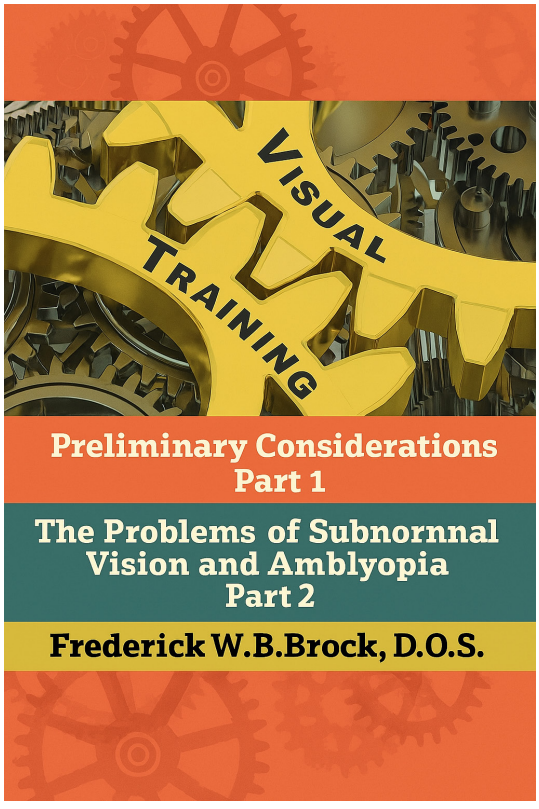

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Article • Objectively Measuring Acquired Ocular Torsion Secondary to Trochlear Nerve Palsy with Zeiss Cirrus Optical Coherence Tomography

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ABSTRACT

Background: Optical coherence tomography (OCT) allows the identification of key fundus landmarks (fovea and center of optic disc) to help calculate estimated ocular torsion amounts in acquired ocular torsion disorders. A case of chronic trochlear nerve (CN IV) palsy secondary to stroke, in which objective ocular torsion was measured with OCT software and basic trigonometric calculations, follows in this report.

Case Report: A 68-year-old female patient with a history of brainstem stroke from five years earlier showed a right-eye hyper deviation of 2$^{\Delta}$. Resultant Parks-Bielschowsky three-step testing, as well as ocular torsion testing with OCT software measurements, helped determine that the patient's hyper deviation was secondary to a right CN IV palsy. Her objective ocular torsion was calculated using measurement tools and trigonometric functions derived from her OCT scans.

Conclusions: OCT technology can provide objective methods for measuring ocular torsion in acquired ocular torsion disorders. Potential OCT software development and future study regarding the use of OCT in these ocular torsion disorders is needed.

Keywords: ocular torsion, optical coherence tomography, trochlear nerve palsy

Introduction

Abnormal cyclotorsion has been known to occur with acquired ocular torsion disorders such as trochlear nerve palsies and skew deviations.[1-5] Historically, the most common way to measure ocular torsion objectively has been with fundus photography.[2-5] Newer reports document OCT as another tool to measure objective ocular torsion in acquired cases.[1,6-8] This case expands on previous OCT ocular torsion reports using Spectralis OCT (Heidelberg Engineering, Heidelberg, Germany)[1,6,8] to include Cirrus OCT (Carl Zeiss Meditec, Dublin, California, USA) as a potential way to assess acquired ocular torsion disorders.

Case Report

A 68-year-old female presented for routine eye examination, with a chronic vertical diplopia complaint for five years since she suffered a brainstem stroke secondary to her history of hypertension and diabetes mellitus. Her diplopia disappeared when covering either eye, suggesting binocular vision dysfunction. She was able to alleviate her diplopia fully in primary gaze with a small left head tilt. Her medical history included hypertension and osteoporosis. Visual acuity was 20/20 OD and 20/25 OS. Pupils, extraocular motilities, and confrontation visual fields were normal OU. Cover test revealed a 2$^{\Delta}$ hyper deviation OD. With the Parks-Bielschowsky three-step test, the hyper deviation worsened with right head tilt and in left gaze, suggesting right superior oblique weakness.[1,2,4] External examination was unremarkable OU. Retinal examination was unremarkable except for mild macular drusen OU. Increased excyclotorsion (-11°) of the right eye and normal torsion (-4°) of the left eye were noted objectively with fundus photography, consistent with a mild trochlear nerve palsy OD (Figure 1). Cirrus OCT macular thickness scans were also performed on the patient OU. Using the linear measuring tool embedded within the Cirrus OCT software, two linear micron measurements were drawn: one from the center of the optic nerve to the fovea, and the second to estimate the horizontal meridian. Using these linear OCT measurements, the disc-to-fovea angle was calculated to estimate the amount of ocular torsion present in each eye (-11.4° OD, -6.6° OS; Figure 2). This shows a

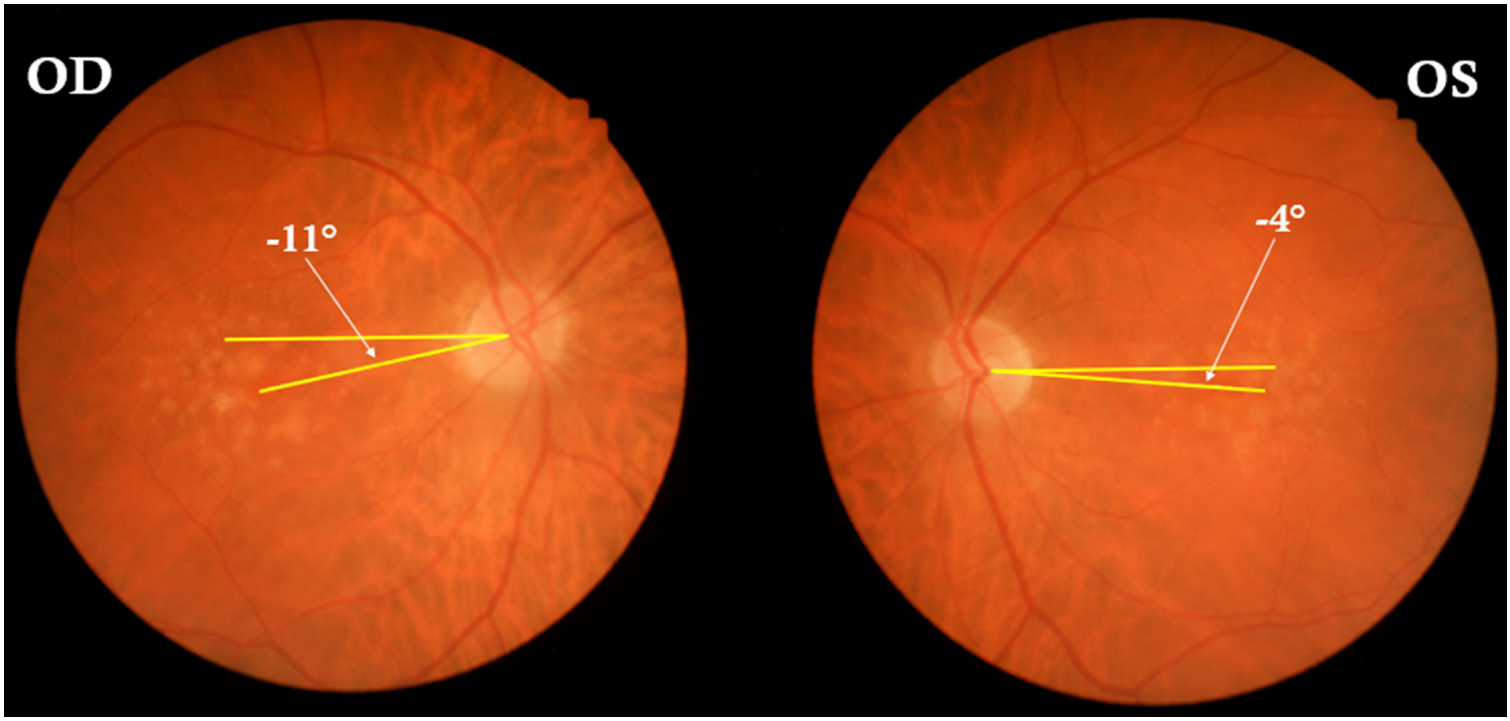

Figure 1. Standard fundus photography of the right eye (OD) and left eye (OS). Note the increased excyclotorsion OD (-11.0°) compared to the normal ocular torsion OS (-4.0°). This is consistent with a right trochlear nerve palsy.

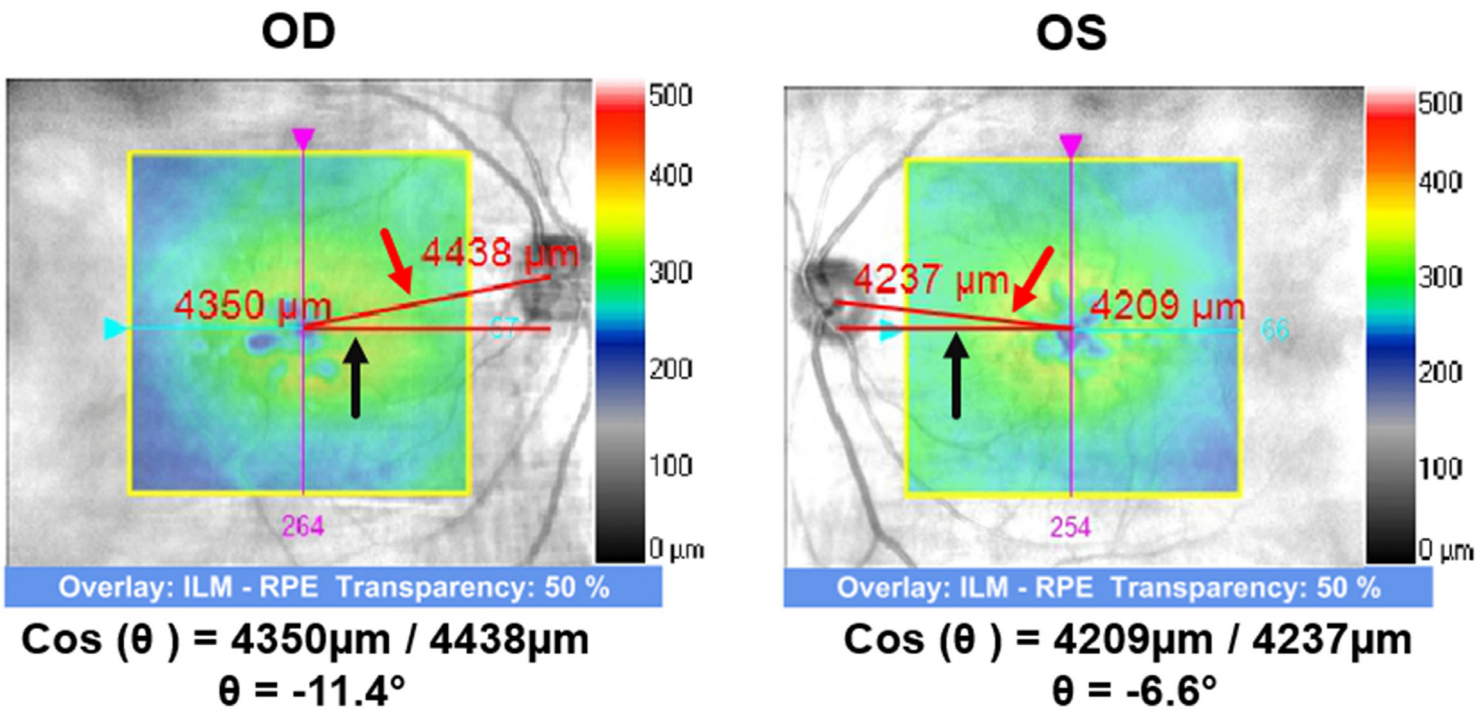

Figure 2. Optical coherence tomography (OCT) of the patient in this case using Cirrus OCT (Cirrus 5000, Carl Zeiss Meditec, Dublin, California, USA) macular thickness scans of the right eye (OD) and left eye (OS). The standard measuring tool on the Cirrus OCT was used to measure the linear distance (microns) from the center of the optic nerves to the fovea (red line with red arrow) and the horizontal meridian (red line with black arrow). These two distances were used to estimate the disc-to-fovea angle with the cosine trigonometric calculations below each respective photo (-11.4° OD, -6.6° OS). The excessive excyclotorsion of the OD is consistent with a right trochlear nerve palsy.

nice correlation between the objective measures of ocular torsion in this case, as obtained from fundus photography and OCT.

Discussion

The ability of OCT to measure ocular torsion is based upon its ability to estimate the angle between a line drawn from the optic nerve center to the fovea and the true horizontal plane, called the "disc-fovea angle."[5,9] Previously documented mean normal ranges of cyclotorsion with Spectralis OCT have been reported to be -6.6° ± 2.8°.[5] A recent study compared cyclotorsion with Spectralis OCT in normal versus trochlear nerve palsy patients.[9] This study reported average cyclotorsion in trochlear nerve palsies of -11.3° (range: -7.7° to 14.9°).[9] However, to the author's knowledge, studies of other OCT platforms to measure cyclotorsion specifically, such as the Cirrus OCT used in this case, do not exist. The Cirrus OCT has a measuring tool within its standard software capable of measuring linear distances (microns). Using this tool to measure the distance from the fovea to the center of the optic nerve, and then measuring a second distance from the fovea along the horizontal meridian to form a right triangle, a clinician could perform a simple trigonometric cosine calculation to estimate the disc-fovea angle. This would then allow the clinician to document the degree of ocular torsion, as in this case (Figure 2). Additionally, the true foveal center can be easily identified on standard OCT macular thickness scans, allowing more accurate ocular torsion measurements.

Since the patient was able to alleviate her vertical diplopia with a small head tilt, she declined further treatment beyond monitoring in this case. The patient is being followed on a yearly basis due to the chronicity and stability of her vertical deviation. The patient's systemic risk factors (i.e., hypertension and diabetes mellitus) account for her stroke history and subsequent trochlear nerve palsy in this case. Given the known anatomy of the trochlear nerves, the likely lesion location in this case is either the inferior dorsal midbrain (at the level of the inferior colliculus) or further along the trochlear nerve's course towards the superior oblique muscle, involving the nerve's vasa-nervorum blood supply.[10]

Conclusion

Cirrus OCT platforms, along with some basic trigonometric calculations, can provide an objective means to estimate degrees of cyclotorsion in acquired ocular torsion disorders, such as this case of trochlear nerve (CN IV) palsy. This is important since some patients are subjectively poor responders to classic tests that measure ocular torsion, such as the double Maddox rod. OCT has the potential to provide objective measures of ocular torsion that do not depend on patients' subjective responses. Potential OCT software development and future study regarding the use of OCT in these ocular torsion disorders is needed and should be encouraged.

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Borgman CJ. Objectively measuring acquired ocular torsion secondary to trochlear nerve palsy with Zeiss cirrus optical coherence tomography. Optom Vis Perf 2026;14(2):125-7.

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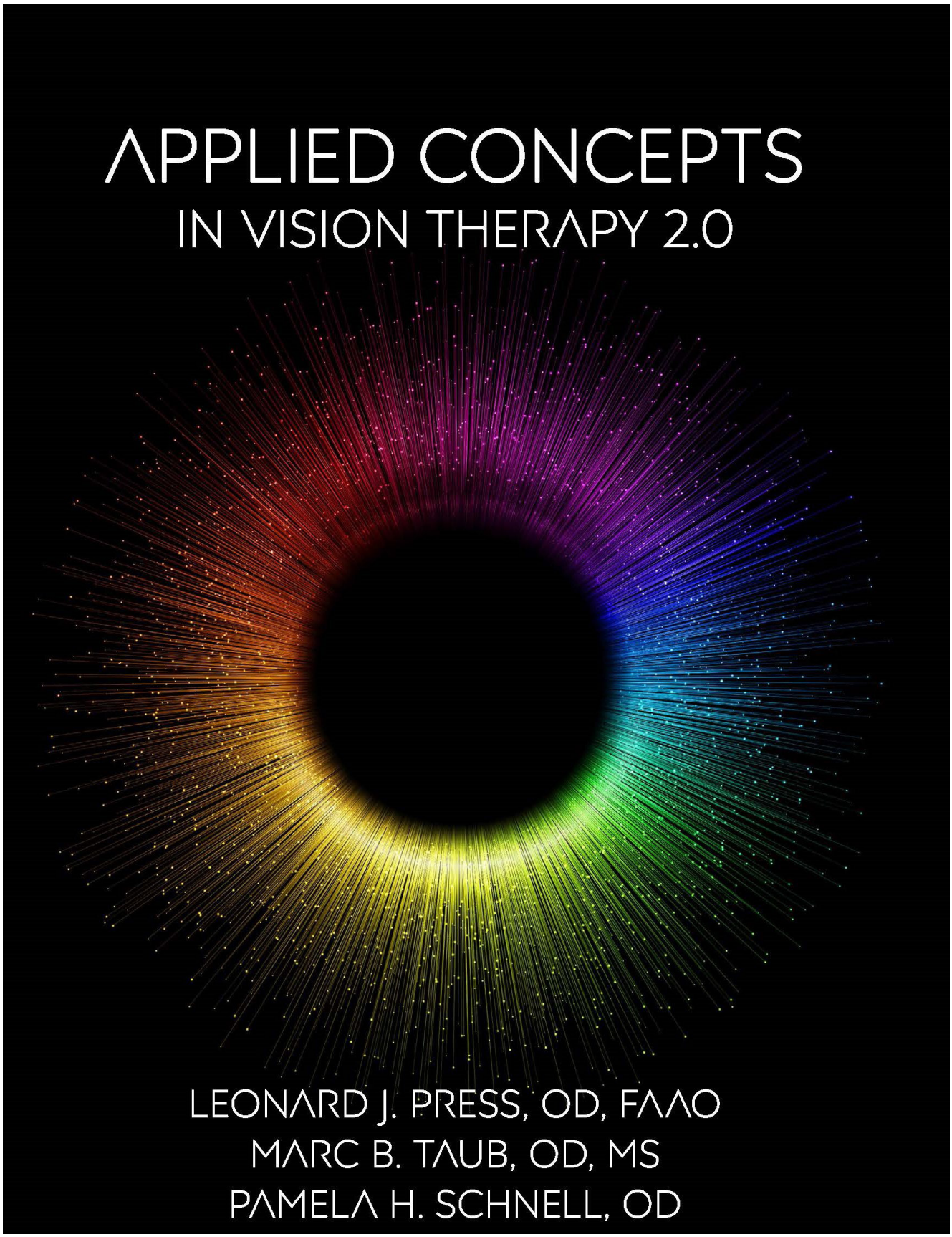

Article • Case Series: Optometric Approach to Vision-Related Concussion Symptoms, Diagnosis, and Management

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ABSTRACT

Background: Sport-related concussions frequently result in persistent visual dysfunctions, including accommodative, binocular, oculomotor, and oculo-vestibular impairments. These deficits can significantly impact quality of life and are often overlooked in standard eye examinations. Optometric vision therapy offers targeted rehabilitation to address these dysfunctions. This case series highlights the benefit of vision therapy in three female patients with sport-related concussions referred for visual evaluation and treatment long after the concussion was sustained.

Case Reports: Three patients (ages 20, 27, and 34) with persistent visual symptoms years after one or more concussions were previously examined by eye care providers, yet visual complaints remained unresolved. Each underwent a comprehensive binocular vision assessment revealing various combinations of accommodative excess (patients 1, 2, 3) or insufficiency (patient 2), fusional vergence dysfunction (patients 2, 3), oculomotor deficits (patients 1, 2, 3), and vestibulo-ocular dysfunction (patients 1 ,2, 3). Patients completed 11–12 sessions of individualized vision therapy, supplemented by daily home exercises. Following therapy, all patients demonstrated clinically significant improvements in objective findings and self-reported symptoms. Brain Injury Vision Symptom Survey scores improved by 55–59% (34–35 points) across the three cases. Notable functional gains included reduced photophobia, improved reading, less fatigue, and return to academic or physical activities.

Conclusions: This case series underscores the essential role of optometrists in post-concussion care. Optometric vision therapy is an effective intervention, even when initiated long after the initial injury. Comprehensive visual function assessment and interdisciplinary collaboration are critical to identifying and managing post-concussion visual dysfunction, ultimately improving patient outcomes.

Keywords: concussion, optometric vision therapy, traumatic brain injury

Introduction

Concussion, often referred to as mild traumatic brain injury (mTBI), is defined by the Centers for Disease Control and Prevention (CDC) as injury to the brain resulting from abrupt acceleration and deceleration of the head during impact, causing the brain to collide or twist with the skull, thereby damaging brain cells and triggering chemical changes.[1,2]

Diagnosing concussion can be challenging due to rapid fluctuations in signs or symptoms[3] and potential delayed inflammatory responses.[4–6] A recent Delphi-based framework developed diagnostic criteria, which include the mechanism of injury, clinical signs, acute symptoms, clinical examination findings, and neuroimaging.[7]

Visual symptoms after concussion frequently include blurred vision, photosensitivity, dizziness, headaches, and difficulties with eye coordination or depth perception.[8] Convergence insufficiency (CI) and accommodative disorders are common in individuals after mTBI.[9,10] These visual disorders can significantly impair everyday tasks[11] and impede academic, occupational, and athletic performance. They may also limit attendance at school, work, or sports,[2] resulting in both an economic and mental burden.

Table 1. Summary of Studies Examining the Effect of Vision Therapy and Oculomotor Rehabilitation in Individuals Suffering from Head Injury

Author(s)	Study Design	Participant(s) Age (years)	Key Findings / Takeaways
Ciuffreda et al. [24]	Single case report	55	Vision therapy (20 sessions) + prism glasses improved fixations and quality of life in a patient 6 years post-blunt head trauma.
Kapoor et al. [25]	Two case reports	40 and 50	Oculomotor rehabilitation improved oculomotor function in two middle-aged men with acquired brain injury.
Simkhovich et al. [26]	Single case report	36	Seven sessions of oculomotor auditory feedback therapy improved convergence from 45 cm to 2 cm in a patient with central scotoma.
Gallaway et al. [27]	Retrospective cohort study (n=218)	mean age: 20.5 years	Majority of concussed patients with CI and AI showed improved visual outcomes following vision therapy.

Growing evidence demonstrates the efficacy of optometric interventions such as vision therapy in mitigating visual deficits and symptoms and enhancing functional vision and quality of life in individuals following head injury.[12] Table 1 summarizes key studies that illustrate the range of positive outcomes associated with these interventions, including oculomotor control, convergence ability, and patient-reported quality of life. The publications encompass single-case reports, small case series, and one large retrospective study, demonstrating clinical benefits across diverse age groups and injuries.

This case series presents the long-term follow-up of three patients recovering from one or two sport-related concussions with concurrent vision disorders (1, 2, and 5 years since the last injury), demonstrating how optometric evaluation and intervention can effectively manage post-concussion syndrome and support sustained improvements in visual function.

Case Reports

Three female patients were seen by an optometrist (ZR) in Reykjavík, Iceland in 2023. All patients sustained one or more sport-related concussions and were referred by their physiotherapist or cognitive therapist for a binocular visual evaluation due to their visual symptoms, despite having previously been seen by an eye care practitioner.

Case Report 1

A 27-year-old female sustained a concussion without loss of consciousness in December 2021 after falling backward while ice-skating. She immediately developed headaches and was advised to rest. During a seven-day follow-up phone consultation, she was prescribed citalopram, esomeprazole, Selexid, Norgesic, melatonin, Atarax, and Botox. In June 2022, she began physiotherapy, receiving acupuncture and cognitive therapy for persistent headaches, fatigue, reading difficulties, and photophobia, which worsened with computer use. She was referred to the optometry clinic by her cognitive therapist and was evaluated in September 2023.

The patient was a compound myope who reported wearing spectacles for the past seven years. Her most recent eye examination was in 2022, with a prescription of -1.00-0.50x170 OD and -1.00 -0.25x010 OS. Examination findings are presented in Table 2. Unaided visual acuity was 0.52 logMAR OD and 0.40 logMAR OS, improving to 0.00 logMAR in both eyes with subjective refraction of -1.00 OD and -0.25-0.25x030 OS. Cycloplegic refraction with 1% cyclopentolate yielded plano-0.75x175 OD and -0.25 -0.50x030 OS, both achieving 0.00^{-} logMAR acuity. At follow-up six days later, non-cycloplegic refraction was -0.50-0.25x165 OD and -0.25-0.50x010 OS, with 0.00^{-} logMAR acuity in both eyes. This prescription was dispensed.

As described in Table 2, cover testing revealed distance orthophoria and 2^{Δ} esophoria at near. She was able to converge to the nose without reporting double vision but was unable to clear the +2.00 D lens during monocular and binocular accommodative facility (MAF/BAF). Her dynamic monocular estimation method (MEM) retinoscopy was plano. Her negative relative accommodation (NRA) was +1.00, often indicating accommodative excess. Due to the reported reading difficulties, the King Devick test was administered, and her score was equivalent to 10 years old. During the vestibulo-ocular motor screening (VOMS) test, she reported an increase in headaches. Her brain injury symptom survey (BIVSS) score was 75 (predictive score is > 31).[13] The Maples oculomotor test findings included head movements, undershooting on saccades, and

Table 2. Summary of the Visual Evaluation Findings Before and After Vision Therapy (Case 1)

	Pre-treatment findings		Post-treatment findings	
	Right eye	Left eye	Right eye	Left eye
First visit since the last concussion	2 years (2021)			
Previous treatment	glasses, PT, CT, medical treatment			
Uncorrected VA LogMAR	0.52^{-2}	0.4^{-2}	0.5^{-}	0.10
Habitual prescription (D)	-1.00 -0.50x170	-1.00 -0.25x010	-0.25 -0.75x175	-0.25 -0.50x010
Cycloplegic refraction (D)	pl-0.75x175	-0.25 -0.50x030		
Corrected VA	0.00	0.00	0.00	0.00
New Rx for distance (D)	-0.50 -0.25x165	-0.25 -0.50x010		
CT (dist, Δ)	ortho		ortho	
CT (near, Δ)	2 esophoria		2 exophoria	
Associated phoria (distance, Δ)	No prism			
Associated phoria (near, Δ)	No prism			
AC/A	4/1			
NPC (cm)	TTN		TTN	
AOA (D)	8	8	8	8
BAF (cycles/minute)	failed +		11	
MAF (cycles/minute)	failed +	failed +	15	14
MEM (D)	plano	plano	+0.25	+0.25
NRA (D)	+1.00		+2.25	
PRA (D)	-2.75		-2.50	
Vergences (distance, Δ)	BI: x/8/6	BO: x/20/18	BI: x/8/6	BO: x/20/18
Vergences (near, Δ)	BI: x/10/8	BO: x/35/30	BI: x/10/8	BO: x/35/30
VF (cycles/minute)	13		13	
KD (years of age)	10		14	
BIVSS	75		31	
VOMS increase in symptoms	VOR & visual motion		No increase in symptoms reported	
Saccades (Maples)	Head movement: 3 Ability: 4 Accuracy: 2		Head movement: 5 Ability: 5 Accuracy: 4	
Pursuits (Maples)	Head movement: 2 Ability: 4 Accuracy: 2		Head movement: 5 Ability:5 Accuracy: 5	
OVT sessions	12 recommended		10 sessions completed	

jerky pursuit movements. External and internal ocular surface examination were unremarkable.

Based on the poor MAF and BAF, low NRA, low esophoria at near, and low MEM results, the patient was diagnosed with accommodative excess.[14,15] Based on poor eye movements, she was diagnosed with oculomotor dysfunction.[14] Based on the increased symptoms during VOMS, she was diagnosed with vestibulo-ocular dysfunction.[14] Optometric vision therapy (OVT), which included accommodative therapy (lens sorting, lens rock, Hart chart, binocular accommodative rock), oculomotor therapy (Brock string, tranaglyphs, vectograms, wall saccades, computer HTS program), and vestibulo-oculo reflex exercises, was recommended to help relax the accommodative excess; improve her fixation, saccadic, and pursuit ability and accuracy; and reduce symptoms. She completed 10 weeks of in-office optometric vision therapy with 30 minutes of daily home exercises.

Vision therapy protocols, based on Scheiman and Wick[13] and Scheiman and Rouse,[14] consisted of 10 in-office sessions (40 minutes each) and 30 minutes of daily home exercises. The patient reported improved visual quality after seven sessions and greater comfort with reduced fatigue upon completion. Final outcomes are presented in Table 2. Notably, the monocular and binocular accommodative facilities were improved to 15 and 14 cpm in the OD and OS, respectively. Additionally, the NRA increased to +2.25 D.

She used spectacles primarily for driving and prescription sunglasses outdoors. Although she did not return to skating, she successfully enrolled in two academic courses, reported easier reading, and her BIVSS score improved to 31 (59%), with only occasional mild headaches.

Case Report 2

A 20-year-old female basketball player sustained a concussion with a concurrent neck injury in 2017 after falling backward. She remained conscious, was hospitalized briefly, and was discharged the same day. In 2018, she reported persistent severe headaches, and MRI revealed gray matter changes. Following physiotherapy, she was cleared to resume sports but experienced a second concussion during a game, leading to disorientation and withdrawal from athletic activity. Post-injury, she was diagnosed with Ehlers-Danlos syndrome, attention-deficit/hyperactivity disorder, and postural tachycardia

syndrome. Her treatment regimen included medications for cardiovascular, neurological, and pain management, including: amitriptyline, sumatriptan, Volidax, bisoprolol, pregabalin, Ajovy, midodrine, Cerazette, Sertral, Xeomin, Decortin, esomeprazole, magnesium, Procoralan, Telfast, Ovixan, and Elvanse. She also received bi-monthly Botox injections for migraines.

In January 2019, she reported visual decline and visited an optometrist, where she was prescribed glasses for the first time. Her prescription was -0.75-0.50x015 OD and -0.75-0.50x180 OD. She was prescribed home-based Hart chart exercises, which she reported as mildly beneficial. In May 2023, the patient was referred by her physiotherapist for a binocular vision evaluation due to complaints of visual focusing difficulties at all distances, visual and general fatigue, reading difficulties, photophobia, clumsiness, headaches, migraines, and chronic pain. Examination findings are summarized in Table 3.

Cycloplegic refraction (1% cyclopentolate) was +0.25-0.25x010 OD and +0.50-0.75x180 OS. She was prescribed her dry refraction (plano-0.25x180 OD, plano-0.50x010 OS; VA: 0.00 LogMAR) with a +0.75 D near addition due to reduced accommodative amplitude (6.00 D OD; 4.00 D OS; expected: 11.8 D).

The patient reported reduced ocular fatigue, improved reading, and decreased clumsiness with the prescription, but she continued to experience unstable distance fixation, photophobia, and headaches. Near point of convergence was to the nose; however, recovery was delayed, with difficulty in regaining fixation. Pursuits were poor and jerky. Cover test revealed orthophoria at distance and 2$^{\Delta}$ esophoria at near; associated phoria was unstable on the cross test. Vergence ranges were within normal limits, but vergence facility was reduced for both base-in and base-out. No suppression was reported. Visual fields (HS-Octopus 600) and pupil responses were normal. The King-Devick test score corresponded to an 11-year-old level, and the BIVSS score was 62. During the VOMS, symptoms increased with pursuits, saccades, vestibulo-ocular reflex testing, and visual motion. The Test of Visual Perceptual Skills (TVPS) was partially administered, with low scores in visual discrimination (age equivalent: 9), visual memory (5 years 10 months), and spatial relations (6 years). Anterior and posterior segment findings were unremarkable.

Based on the low amplitude of accommodation and increased MEM lag of accommodation,

Table 3. Summary of Visual Evaluation Findings Before and After Vision Therapy (Case 2)

	Pre-treatment findings		Post-treatment findings	
	Right eye	Left eye	Right eye	Left eye
First visit since the last concussion	5 years (2017, 2018)			
Previous treatment	Glasses, PT, medical treatment			
Uncorrected VA LogMAR	0.00-	0.10+	0.00	0.00
Habitual prescription (D	-0.75 -0.50x015	-0.75 -0.50x180	plano -0.25x010	plano -0.50x025
Cycloplegic refraction (D)	+0.25 -0.25x010	+0.50 -0.75x180		
Corrected VA	0.00	0.00	0.00	0.00
New Rx for distance (D)	plano -0.25x010	plano -0.50x025		
New Rx for near (D)	+0.75 -0.25x010	+0.75 -0.50x025	+0.75 -0.25x010	+0.75 -0.50x025
CT (distance, $^{\Delta}$)	ortho		ortho	
CT (near, $^{\Delta}$)	2 esophoria		ortho	
Associated phoria (distance, $^{\Delta}$)	No prism			
Associated phoria (near, $^{\Delta}$)	No prism			
AC/A	4/1			
NPC (cm)	TTN		TTN	
AOA (D)	6	4	10	10
BAF (cycles/minute)	Failed +, took 10 sec to clear minus		9 cpm	
MAF (cycles/minute)	Failed +	11	12	14
MEM (D)	+1.00	+0.75	+0.75	+0.25
NRA (D)	+1.50		+2.25	
PRA (D)	-1.25		-2.25	
Vergences (distance, $^{\Delta}$)	BI: 4/8/4	BO: x/25/12	BI: x/8/6	BO: x/25/20
Vergences (near, $^{\Delta}$)	BI: 6/16/12	BO: x/30/25	BI: 6/14/12	BO: x/30/25
VF (cycles/minute)	6 (slow with base in and base out)		14	
KD (yr equiv)	11		13	
BIVSS	62		28	
VOMS increase in symptoms	Saccades, pursuits, VOR, & visual motion		Very light increase in dizziness during visual motion	
Saccades (Maples)	Head movement: 3 Ability: 4 Accuracy: 2		Head movement: 5 Ability: 5 Accuracy: 5	
Pursuits (Maples)	Head movement: 3 Ability: 4 Accuracy: 3		Head movement: 5 Ability: 5 Accuracy: 5	
OVT sessions	12 recommended		12 sessions completed	

accommodative insufficiency was diagnosed.[14] Based on her BAF and MAF difficulty and poor NRA, the patient was diagnosed with accommodative excess.[14] These discrepancies are further addressed in the discussion. The patient failed with plus and minus lenses on BAF, and both NRA and positive relative accommodation (PRA) results were abnormal. Based on the low near vergence facility and unstable distance phoria, the patient was diagnosed with fusional vergence dysfunction.[13] Because of reading complaints, poor King Devick results, poor pursuits results, and increased symptoms during saccades and pursuits in the VOMS test, oculomotor dysfunction was diagnosed.[14] Based on her complaints of headaches during the VOMS test, the patient was diagnosed with vestibulo-ocular dysfunction.[4] Based on the low TVPS score, she was diagnosed with poor visual perception.[13]

The patient participated in twelve sessions of optometric vision therapy, which included accommodative, vergence, oculomotor, vestibulo-ocular reflex, and visual perceptual therapy. This patient was very symptomatic and used many medications for additional diagnoses. Despite her progress, she started to report improvement in her symptoms only after the 8th session.

The patient reported feeling "less clumsy" with her distance prescription but discontinued its use following completion of vision therapy. She continued using reading glasses and distance prescription sunglasses, which she found beneficial. Reading performance improved, enabling her to engage with previously challenging texts. Improvements included an increase in amplitude of accommodation (from 6 to 10 D in the right eye and 4 to 10 D in the left eye, respectively), binocular and monocular accommodative facility (9 cpm and 11-12 cpm, respectively), NRA (increased by 0.75 D), PRA (increased by 1.00 D), vergence facility (from 6 cpm to 14 cpm), and King Devick (from normative age of 11 to 13 years). Symptom severity decreased, as reflected by the improved BIVSS scores (28, 55%), and visual processing performance increased on TVPS subtests: visual discrimination (age equivalent >18 years), visual memory (>15 years), and spatial relations (>16 years). A very small increase in dizziness was observed during vestibulo-ocular reflex testing.

Case Report 3

A 34-year-old female sustained football-related concussions in 2017 and 2022, both involving head trauma from collisions with other players. Although she remained conscious, the second concussion caused disorientation and removal from play. Assuming symptoms would resolve as with the first injury, she did not seek immediate medical attention. Persistent symptoms led to a physiotherapy referral in August 2022. Ongoing complaints included reading difficulty, ocular misalignment, fatigue, emotional dysregulation, head pressure, motion sensitivity, headaches, and photophobia, prompting referral for a binocular vision evaluation.

The patient was evaluated in January 2023. Table 4 summarizes the findings. Refraction revealed emmetropia with -0.08 D OD; 0.00^{+} OS LogMAR visual acuity. Cycloplegic refraction measured +0.50 D binocularly. Near point of convergence break was 5 cm, with fusion recovery at 7 cm. Cover testing showed orthophoria at distance and near. On associated phoria testing, the patient reported constant target movement. She had difficulty clearing +1.00 D in the monocular accommodative facility test and failed both plus and minus lenses binocularly. NRA and PRA were -1.50 D and +1.75 D, respectively. The King-Devick score was equivalent to a 10-year-old, and the BIVSS score was 61. During VOMS, symptoms of headache and dizziness increased during horizontal/vertical VOR and visual motion tasks. Horizontal saccades involved undershoots, and pursuits were jerky.

Spectacles were prescribed with +0.25 D OD and +0.50 D OS, as the patient reported immediate relief and visual comfort. An FL-41 migraine filter was incorporated into the distance prescription following successful in-clinic testing, initiated at the patient's request after her own online research. Internal and external ocular surface examinations were unremarkable.

A diagnosis of accommodative excess was made based on reduced binocular and monocular accommodative facility with plus lenses.[14] Findings of low vergence reserves, reduced fusional vergence facility, poor binocular accommodative facility with both plus and minus lenses, decreased NRA and PRA, and unstable fixation during the associated phoria test supported a diagnosis of fusional vergence dysfunction.[14] Increased symptoms during VOMS indicated vestibular-ocular dysfunction.[14] Additionally, her impaired performance on oculomotor assessments was consistent with oculomotor dysfunction.[14] OVT was recommended to alleviate accommodative excess, enhance vergence function, improve oculomotor function, and reduce symptoms.

Table 4. Summary of Visual Evaluation Findings Before and After Vision Therapy (Case 3)

	Pre-treatment findings		Post treatment findings	
	Right eye	Left eye	Right eye	Left eye
First visit since the last concussion	1 year (2017, 2022)			
Previous treatment	PT			
Uncorrected VA LogMAR	-0.08	0.00+	-0.08	-0.08
Habitual prescription (D)	----	----	+0.25	+0.50
Cycloplegic refraction (D)	+0.50	+0.50		
Corrected VA	-0.08	0.00+	-0.08	-0.08-
New Rx for distance (D)	+0.25	+0.50		
CT (distance, Δ)	ortho		ortho	
CT (near, Δ)	ortho		ortho	
Associated phoria (distance, Δ)	No prism			
Associated phoria (near, Δ)	No prism			
AC/A	4/1			
NPC (cm)	5/7		TTN	
AOA (D)	5	5	8	7
BAF (cycles/minute)	2 (difficulty with + and - lenses)			
MAF (cycles/minute)	4 (difficulty with +)	4 (difficulty with +)	10	10
MEM (D)	+1.00	+0.75	+0.75	+0.25
NRA (D)	+1.50		+2.25	
PRA (D)	-1.25		-2.25	
Vergences (distance, Δ)	BI: x/6/4	BO: x/16/10	BI: x/8/6	BO: x/25/20
Vergences (near, Δ)	BI: 4/6/4	BO: x/16/14	BI: 4/8/6	BO: 12/35/30
VF (cycles/minute)	6 (slow with base in and base out)		13	
KD (years of age)	10		13	
BIVSS	61		27	
VOMS increase in symptoms	VOR & visual motion		No increase in symptoms reported	
Saccades (Maples)	Head movement: 3 Ability: 4 Accuracy: 2		Head movement: 5 Ability: 5 Accuracy: 5	
Pursuits (Maples)	Head movement: 2 Ability: 4 Accuracy: 2		Head movement: 5 Ability: 5 Accuracy: 5	
OVT sessions	12 recommended		VT 11 sessions completed	

Following 11 therapy sessions, the patient demonstrated improvement in accommodative facility (from 4 cpm to 10 cpm monocularly; from 2 cpm to 8 cpm binocularly), NRA and PRA (increased by 0.50 D), some improvement of convergence ranges at distance and near (by 9Δ and 19Δ, respectively), and vergence facility (from 6 to 13 cpm). She reported faster refocusing at varying distances, reduced headache frequency, and a marked improvement in subjective symptoms (BIVSS score decreased to 27, 56% improvement). Although she resumed biking and running, she had not yet returned to playing football.

Optometric Vision Therapy Protocol

This optometric vision therapy protocol was applied to all three patients, with individual adaptations as described above. All patients were treated by optometric vision therapy protocols based on Scheiman and Wick[14] and Scheiman and Rouse,[15] consisting of 12 in-office sessions (40 minutes each) and 30 minutes of daily home exercises.

Accommodative therapy began with lens sorting, with an end point in the range of +5.00 D to -8.00 D in jumps of 0.50 D. These were followed by loose lens rock, where lens flippers gradually increased until the patient could use +2.50/-6.00 flipper monocularly. If the patient was between 30-35 years old, our end point was a +/-2.00 D flipper. The subsequent procedure was Hart chart, and the final stage was binocular accommodative rock with polaroid (bar reader and polaroid glasses), where the end point was the ability to use a flipper of +/-2.50 D at 20 cycles per minute. For patients between 30-35 years of age, the end point was a +/-2.00 D flipper.

Vergence therapy included Brock string to develop an awareness of eye position in convergence and divergence and vectograms and tranaglyphs to normalize positive and negative fusional vergence (PFV and NFV), used until the patient reached 30Δ base out and 12Δ base in. The aperture rule was used until card number 12 for convergence and 7 for divergence were reached.

Oculomotor therapy included Hart chart saccades, wall saccades, computer HTS program, Groffman tracings, letter tracing, multiple Brock strings, Marsden ball, Multi Matrix, Spot-It games, saccadic workbooks (Bernell), pegboard rotator, and MacDonald peripheral cards.

Visual perception therapy included parquetry blocks, geoboard, memory workbooks, visual discrimination workbooks, and a tachistoscope,

combined based on the patients' abilities to increase visual perceptual skills.

Oculo-vestibular therapy included work on the vestibular-ocular reflex on activation and inhibition, Marsden ball, and the walking rail.

Home exercises included loose lens rock, binocular accommodative rock, Brock string, Multi Matrix, wall saccades, Marsden ball, Spot-It game, saccadic workbooks, MacDonald peripheral card, tachistoscope, geoboard, memory workbooks, visual discrimination workbooks, eccentric circles, and barrel and lifesaver cards, with combinations of loose lenses at more advanced levels.

Discussion

Although visual symptoms are very common in post-concussion patients, visual examinations by eye care professionals do not typically involve the assessment of accommodative, binocular, oculomotor, and oculo-vestibular systems.[16] In all three of these cases, patients were previously seen by an eye care practitioner, who examined ocular health and refraction but not binocularity. The patients were subsequently referred for a binocular vision evaluation by a physiotherapist or cognitive therapist due to ongoing visual complaints persisting even months after the concussion. Additionally, all cases improved in both objective clinical measures as well as symptoms after a course of vision therapy, highlighting the importance of evaluating and treating binocular vision dysfunctions in addition to ocular health and refraction, especially in patients who have a history of concussion. These comprehensive evaluations should occur soon after injury to provide a better prognosis and improved quality of life.[17]

These positive outcomes collectively demonstrated the effectiveness of OVT in addressing the individuals' visual challenges. Among all the therapies available today, OVT is still not one of the commonly used, although vision plays a significant role in the patient's symptoms and quality of life after a concussion. Further, active treatments are more effective than rest-based approaches.[18]

The observed improvements in the cases described demonstrate the efficacy of OVT in mitigating post-concussion visual dysfunction. Evidence suggests that active interventions, including vision therapy, are more effective than passive, rest-based approaches.[18] Despite this, OVT appears underutilized.[19]

The present findings demonstrating the effectiveness of OVT after sport-related concussion align with those of Morton,[20] who reported a similar case of a 20-year-old athlete requiring 34 OVT sessions beginning eight days post-injury. In contrast, the three cases described here required only 11–12 sessions, possibly due to later initiation of therapy and the absence of CI, which was present alongside accommodative spasm in Morton's case. Earlier intervention in these cases may have mitigated long-term visual symptoms. Future studies should compare outcomes between patients treated soon after concussion and those treated at a later stage to assess the effect of early detection and intervention on visual dysfunction.

Patient 2 presented the greatest treatment challenge, likely due to the combined effects of 18 concurrent medications prescribed for coexisting Ehlers-Danlos syndrome, attention deficit/hyperactivity disorder, and postural tachycardia syndrome. The patient denied history of these diagnoses prior to her concussions. Thus, it is unclear whether these comorbidities represent distinct primary disorders or are manifestations secondary to the brain injury. Such inconsistencies may reflect a broader phenomenon in post-concussion populations, where diagnostic frameworks established in non-injured cohorts may not fully apply.[21]

Similarly, Patient 2 presented with low accommodative amplitude and high lag, consistent with accommodative insufficiency, yet demonstrated the ability to clear minus lenses rapidly while failing with plus lenses, suggestive of accommodative excess. These conflicting findings may reflect medication side effects; several of her prescribed drugs (e.g., bisoprolol, Elvanse, pregabalin, sertraline, prednisone, Xeomin) are known to affect visual function. Alternatively, such inconsistencies may reflect a broader phenomenon in post-concussion populations, where standard diagnostic criteria—derived from non-concussed cohorts—may not apply. Wiecek et al.[22] similarly reported conflicting accommodative findings in a retrospective study of 116 post-concussion patients, noting that 50% failed plus lens facility despite 54% having reduced accommodative amplitude. Together, these findings suggest the need to reconsider diagnostic frameworks for binocular and accommodative dysfunctions in concussed individuals.

An additional observation concerns increased myopic prescriptions in this population, often driven by patient complaints of reduced distance clarity. While patients may prefer stronger minus lenses

due to perceived contrast improvement,[23] over-minus correction can exacerbate symptoms and lead to headaches by overloading the visual system. A recommended approach is cycloplegic refraction, followed by post-cycloplegic refraction after four days, with prescriptions aimed at visual comfort rather than maximal acuity.

Conclusions

These cases underscore the critical role of optometrists in post-concussion care. Comprehensive assessment of visual function and interdisciplinary collaboration among optometrists, ophthalmologists, neurologists, physiotherapists, and psychologists are essential for effective management. The findings demonstrate that vision therapy can be beneficial even when initiated long after a sport-related concussion.

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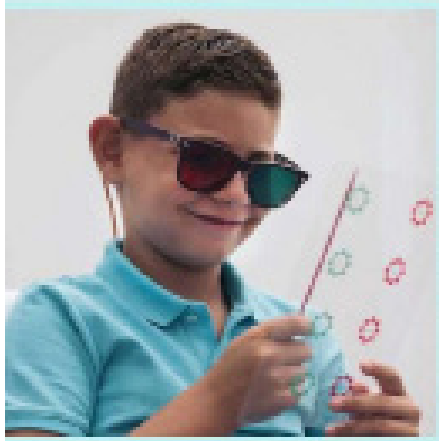

Centering
Antigravity
Identification
Speech-Auditory
Vision

Randy Schulman, MS, OD, FCOVD, FCSO

Article • Investigating the Factors that Hinder School-Based Vision Screening Programs at Egor Local Government Area, Benin City, Nigeria

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Okechukwu Clinton Ifeanyi, OD • University of Benin • Edo, Nigeria

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Doctor of Optometry, University of Benin.

Work emphasis, school-based vision screening, rural eye care, preventable blindness, health education, and community eye health programs.

Supported school- and community-based health interventions across Edo, Lagos, and Delta States, with experience in vision screening, outreach coordination, stakeholder engagement, and health communication.

ABSTRACT

Background: Many children worldwide experience vision impairment, much of which is preventable with early intervention. School-based vision screening programs are vital for early detection and improved academic performance. However, in countries like Nigeria, these programs often face challenges, such as poor awareness, limited resources, and low stakeholder involvement. This study aimed to evaluate the prevalence, barriers, and benefits of school-based vision screening programs in Egor LGA, focusing on identifying actionable strategies for improvement.

Methods: A 14-question survey was conducted among 291 respondents, including students, teachers, administrators, and parents. The questionnaire assessed the frequency of vision screenings, barriers to implementation, perceived benefits, and recommendations for enhancing program participation.

Results: Most respondents (79.7%) reported that no vision screenings had been conducted in their schools over the past year. The frequency varied among the schools conducting screenings, with only 1.7% performing screenings more than twice annually. Key barriers included a lack of awareness (33.7%), a lack of resources (23.7%), and a perceived lack of need (19.9%). Despite these challenges, respondents highlighted significant benefits, such as early detection of vision problems (49.5%) and improved academic performance (22.0%). Recommended strategies for improvement included increasing awareness campaigns (35.1%) and providing additional resources (25.1%).

Conclusion: The findings reveal critical gaps in the prevalence and consistency of school-based vision screening programs. Addressing barriers through targeted awareness, resource allocation, and stakeholder engagement can significantly enhance their implementation, leading to improved vision health and student educational outcomes.

Keywords: school-based programmemes, students, vision-screening

Introduction

Vision is a crucial sense for learning and development, particularly for children who depend on their eyesight to acquire knowledge and skills. According to the World Health Organization (WHO), approximately 19 million children worldwide are visually impaired, with 12 million experiencing conditions that could be prevented or treated.[1] Unaddressed vision problems can adversely affect academic performance, social interactions, and overall well-being. Early detection and treatment of vision issues lead to better educational outcomes and improved quality of life for students.[2,3]

One strategy to address this problem is school-based vision screening (SBVS), a process designed to identify children with potential vision problems and refer them to appropriate eye care services.[4] SBVS is widely recognized as an effective, cost-efficient, and feasible intervention, especially in low- and middle-income countries, where access to eye care is often limited.[5] Schools provide an ideal setting for vision screening due to their accessibility and ability to reach large populations of children who may not otherwise receive regular eye examinations.[4,6]

Globally, several countries have implemented successful vision screening models, offering valuable lessons for program design and scalability. For instance, the Vision for Baltimore program in the United States provides free eye exams and eyeglasses to disadvantaged children, significantly improving academic performance and stakeholder engagement.[7] Similarly, India's School Eye Health Program demonstrated the cost-effectiveness of training teachers to conduct initial screenings, reducing the burden on healthcare professionals.[8,9] In Africa, the Peek Vision Program in Kenya and Botswana leverages mobile technology to conduct screenings via smartphone-based applications, improving referral rates and access to specialist care.[5] Meanwhile, large-scale budget impact analyses in Cambodia and Ghana indicate that scaling up SBVS is financially viable when integrated into existing school health programs.[6]

Despite these successes, barriers to implementation persist. Socioeconomic limitations, lack of trained personnel, parental resistance, and misconceptions about spectacle use frequently hinder program effectiveness.[5,10,11] Addressing these challenges through policy interventions, awareness campaigns, and sustainable funding mechanisms is critical for maximizing the impact of school vision programs.[5,11]

In Nigeria, SBVS is a component of the National School Health Policy and Program, which aims to promote the health and well-being of school-age children.[9] However, program coverage and quality vary across states and local government areas (LGAs). Egor LGA, located in Nigeria's South-South geopolitical zone, is one such area where these disparities are evident. With a population of approximately 339,899, including a significant number of school-age children, the region comprises diverse ethnic, cultural, and religious communities that can influence health-seeking behaviors and attitudes toward vision care. Vision problems among Nigerian children include refractive errors such as myopia, hyperopia, and astigmatism, as well as strabismus, amblyopia, cataracts, glaucoma, corneal diseases, and vitamin A deficiency.[1] According to WHO, at least 2.2 billion people worldwide suffer from vision impairment, with regional and national variations in prevalence due to differences in eye care accessibility, environmental factors, and socioeconomic conditions.[1] In Nigeria, uncorrected refractive errors, congenital ocular defects, corneal scarring, and cerebral visual impairment are leading causes of moderate-to-severe vision impairment and blindness.[1]

Despite the recognized benefits of SBVS programs, various challenges impede their effective implementation in Egor LGA. These include limited financial resources, lack of trained personnel, poor referral systems, low parental engagement, and cultural beliefs about spectacle use.[5,10,11] Additionally, logistical challenges such as inadequate screening equipment, lack of school infrastructure, and weak follow-up systems further hinder success.[5,11] One notable concern is the low parental involvement in SBVS programs. Studies indicate that parental resistance to vision screening interventions, coupled with low awareness of eye health issues, significantly affects program acceptance and follow-up compliance.[5,10,11] These programs are usually conducted by trained personnel, such as school nurses, teachers, or volunteers, using various methods and instruments to assess visual acuity, refractive errors, ocular alignment, and other ocular health indicators.[12] The objectives of SBVS programs include detecting and preventing amblyopia, reducing the prevalence and impact of uncorrected refractive errors, identifying and referring children with other eye diseases or conditions, and promoting eye health awareness and education among children, parents, teachers, and communities.[8]

SBVS programs offer several benefits for children's eye health and well-being. Schools provide access to many children who may not have regular access to eye care services due to geographic, economic, or cultural barriers. These programs also use simple, low-cost, and portable tools that yield accurate results, reducing the workload for eye care professionals by filtering out children who do not need further evaluation.[13] Furthermore, they improve the detection and treatment of vision problems that affect children's learning, development, and quality of life, preventing complications associated with untreated eye conditions.[14]

Children with untreated vision problems often struggle to engage with educational materials, which adversely affects their academic performance. Research has shown a correlation between poor performance on standardized assessments and visual impairment. For example, research found that among third-grade students with normal visual acuity (VA), 89.5% had satisfactory academic performance, whereas only 75% of those with impaired VA did ($p = 0.015$).[15] This suggests a significant association

between low VA and poor academic performance, indicating that visual impairments can negatively impact students' learning and academic outcomes, including information processing and reading comprehension.

The effectiveness of SBVS programs is influenced by multiple factors. Socioeconomic barriers, such as limited financial resources, lack of health insurance, and transportation challenges, often prevent families from accessing follow-up care. Cultural beliefs and misconceptions about refractive errors and vision correction can also reduce acceptance and compliance with these programs. Additionally, lack of awareness among parents and teachers about the importance of vision screening can result in missed opportunities for early detection and treatment. Logistical challenges, including shortages of trained personnel, inadequate resources, and a lack of infrastructure, further hinder the implementation of these programs. Difficulties in follow-up, such as the cost of treatment, parental awareness, and school policies, also affect program outcomes.[16]

Materials and Methods

A convenience sampling technique was used, selecting participants who were readily available and who consented. This study was conducted at five schools: Benin Technical College (BTC), Calvary Crown Academy (CCA), Gabus International College (GIC), Russell Group of Schools, and GSTC, all located in Egor LGA, Benin City, Edo State. These individuals were selected based on their eligibility for the study.

A survey questionnaire was used to collect data from administrators, teachers, students, and parents. Quantitative data were collected through a survey questionnaire administered to a sample of school teachers, students, and parents in Egor LGA, Edo State. The survey questionnaire was designed to collect information on current practices and policies for SBVS programs, as well as the factors that hinder their implementation. The survey also gathered data on these stakeholders' perceptions and experiences with SBVS programs, as well as their suggestions for improving the programs to better address the vision needs of school-aged children.

Ethical Considerations

Before this study, informed consent was sought, and ethical approval was obtained from the University, where the research team is affiliated with the REC approval number: EC/UBEN/LSC.OPT/23/78. All procedures performed in this study were conducted in accordance with the Tenets of the Declaration of Helsinki for the protection of human subjects.

Data Analysis

The data obtained from this study were analyzed using the Statistical Package for Social Sciences (SPSS) version 22.0. Descriptive statistics, frequency tables, and percentages were used in the data analysis and interpretation.

Results

The study surveyed 291 participants, predominantly students (80.1%), teachers (14.8%), administrators (3.4%), and parents (1.7%). Respondents were drawn from six schools in Egor LGA, with Benin Technical College contributing the largest share (57.7%), followed by Calvary Crown Academy (15.5%), GSTC (14.4%), and Gabus International College (10.3%), with minimal representation from Russell Group of Schools (1.0% each). The distribution of respondents from these schools provides a comprehensive view of the status of vision screening programs in the region.

Regarding the status of SBVS programs, only 59 respondents (20.3%) reported that their schools conducted such vision screenings in the past year. In contrast, a significant majority (79.7%) indicated that no vision screenings occurred. The frequency varied across schools that conducted screenings: 3.4% of respondents reported that vision screenings were held once a year, 4.1% twice a year, and 1.7% more than twice a year. Notably, 11.0% of respondents were unsure of the frequency of these vision screenings.

Respondents provided several explanations for the absence of vision screening programs. Specifically, 33.7% attributed the lack of screenings to insufficient awareness, 23.7% to inadequate resources, and 19.9% to the perception that there was no need for vision screening. A smaller proportion of respondents reported combined reasons: 3.1% cited all three factors, 2.1% indicated a lack of resources and awareness, and 0.3% noted a combination of a lack of understanding and a perceived lack of need. Additionally, 17.2% of respondents had no opinion on the matter.

Regarding attitudes toward these programs, 55.3% of respondents expressed an optimistic view, 15.5% were neutral, 4.1% were negative, and 25.1% remained uncertain. The study also assessed knowledge of SBVS, revealing that only

39.2% of respondents had previously heard of such programs, while 60.8% were unaware of them. Among those familiar with the programs, awareness was primarily gained through other sources (25.1%), followed by teachers (10.0%), students (9.3%), school administration (5.2%), and parents (4.5%). When evaluating the perceived importance and benefits of vision screening programs, most respondents (238 out of 291) believed that such programs are essential for students. This belief is supported by the potential benefits, with 49.5% of respondents identifying early detection of vision problems as the key advantage, 22.0% noting improved academic performance, and 14.4% mentioning an enhanced quality of life. A further 10.3% indicated that all listed benefits applied, while only minimal percentages chose various combinations of benefits or reported no benefit. These promising findings underscore the potential positive impact of SBVS programs.

Finally, the study examined the challenges of implementing vision screening programs. The most commonly reported challenge was a lack of resources, cited by 41.9% of respondents, followed by a shortage of trained personnel (28.9%). Additionally, 11.0% cited insufficient parental support, and 3.1% cited cultural barriers. These challenges are real and significant, and the study aims to address them. Regarding strategies to improve participation, 35.1% of respondents recommended increasing awareness through targeted campaigns, while 25.1% emphasized the need for additional resources. Other suggestions included parental involvement and innovative screening tools, each noted by 11.0% of respondents. These findings highlight the multifaceted challenges and potential strategies for enhancing the delivery of SBVS programs in Egor LGA.

Discussion

Table 1 indicates that 79.7% of respondents reported no vision screening programs in the past year, while only 20.3% reported screenings. Among those, the frequency was low: only 1.7% conducted screenings more than twice annually. These findings reveal a significant gap in the implementation of the SBVS program in Egor LGA. This gap is wider than studies reported, which found that over 60% of basic schools in a Ghanaian municipality had some form of eye health intervention in place.[16] Similarly, a study in Armenia found higher screening coverage due to a standardized national framework.[10] The stark contrast suggests that Egor LGA lags in policy enforcement and operational readiness for SBVS.

Table 1. Frequency of Vision Screening Programmes

Response	Frequency	Percent
Once a year	10	3.4
Twice a year	12	4.1
More than twice a year	5	1.7
Don't know	32	11.0
N/A	232	79.7

Table 2. Reasons for Absence of Vision Screening

Response	Frequency	Percent
Lack of resources	69	23.7
Lack of awareness	98	33.7
No need for vision screening	58	19.9
A combination of the three above reasons	9	3.1
Lack of resources and awareness	6	2.1
Lack of awareness and no need for vision screening	1	0.3
No opinion	50	17.2

Table 3. Benefits of Vision Screening Programmes

Response	Frequency	Percent
Early detection of vision problems	144	49.5
Improved academic performance	64	22.0
Improved quality of life	42	14.4
All options	30	10.3
Options 2 & 3	1	0.3
Options 1 & 2	6	2.1
Options 1 & 3	2	0.7
No benefit	2	0.7

As shown in Table 2, the primary barriers cited for the absence of SBVS were lack of awareness, insufficient resources, and a perceived lack of need. These findings align with the rapid review study, which identified awareness, resource limitations, and stakeholder engagement as consistent global challenges in school-based eye care delivery.[12] Likewise, a qualitative study in Ontario found that awareness among educators and parents significantly affected program implementation and follow-up care.[13] Our results reinforce that awareness campaigns and educational outreach are critical starting points for intervention.

Table 3 outlines the perceived benefits of SBVS. The most frequently cited were early detection of vision problems, improved academic performance, and enhanced quality of life. These findings are supported by multiple studies. For example, Toledo

Table 4. Challenges to Vision Screening Programmes

Response	Frequency	Percent
Lack of resources	122	41.9
Lack of trained personnel	84	28.9
Lack of parental support	32	11.0
Cultural barriers	9	3.1
All options	9	3.1
Options 1 & 2	14	4.8
Options 1 & 3	3	1.0
Options 1 & 4	1	0.3
Option 1, 3, & 4	1	0.3
Option 2, 3, 4	1	0.3
Option 1, 2, & 3	10	3.4
Option 3 & 4	1	0.3
Others (Lack of awareness)	1	0.3
No benefit	3	1.0

Table 5. Suggestions to Improve Participation

Response	Frequency	Percent
Increase awareness	102	35.1
Provide more resources	73	25.1
Involve parents	32	11.0
Use innovative screening tools	32	11.0
All options	20	6.9
Options 1 & 2	5	1.7
Options 1 & 3	3	1.0
Options 1 & 4	5	1.7
Option 1, 3, & 4	3	1.0
Option 2, 3, 4	3	1.0
Option 1, 2, & 3	11	3.7
Option 3 & 4	1	0.3
Options 2 & 3	2	0.7

et al. found a strong association between corrected visual acuity and academic success among school-aged children, where students with normal vision achieved significantly higher academic scores.[17] The Vision for Baltimore initiative also linked vision correction through SBVS to improved classroom engagement and school attendance.[7] Our study validates these claims and underlines the potential of SBVS to improve not only health but also education outcomes.

A small proportion of respondents expressed skepticism or were unaware of these benefits. This suggests the presence of attitudinal barriers and underlines the importance of targeted stakeholder education. These attitudes could be influenced by cultural norms or misinformation, consistent with findings from a study that noted resistance to spectacle use among schoolchildren in parts of India due to social stigma and misconceptions.[8]

Table 4 identifies the most pressing challenges during SBVS implementation: insufficient resources and insufficient trained personnel. These findings mirror a study on mobile vision screening in Botswana, which emphasized the importance of equipping schools with both physical tools and adequately trained staff to deliver consistent and reliable screenings.[5] Similarly, a study on the cost and budget impact analysis emphasized that sustainable funding and workforce development are essential for scalability in low-resource settings.[6]

Parental support was another challenge. The frequency is notably lower than anticipated, yet crucial, as parental involvement significantly affects program uptake and compliance with referrals. A study identified a lack of parental follow-through as a major bottleneck in Canada's school-wide screening programs.[15] Our findings align with this concern and suggest the need for structured communication between schools and parents, possibly through community health workers or parent-teacher associations.

As seen in Table 5, recommended strategies for improving SBVS included increasing awareness, providing more resources, and engaging parents. These proposals reflect global best practices, such as those implemented in Cambodia and Ghana, where it was demonstrated that combining financial investments with education and parental engagement significantly improved SBVS participation.[6] Similarly, a study concluded in their systematic review that integrating SBVS into broader school health policies and using cost-effective tools enhances both reach and impact.[18]

In summary, this study's findings highlight systemic gaps in SBVS delivery in Egor LGA and offer a path forward through increased awareness, resource mobilization, and stakeholder education. When viewed alongside global data, our study adds valuable regional insight to the growing evidence base on the challenges and opportunities in school vision screening implementation.

Study Limitation

One limitation of this study is the low representation of parents among survey respondents, which limits insights into parental attitudes and barriers to SBVS. Possible reasons for this low participation include lack of awareness, socioeconomic constraints, cultural beliefs about

spectacle use, and limited communication between schools and parents regarding vision care. Since parental involvement is crucial for successful screening programs, future efforts should focus on raising awareness, improving school-parent communication, and addressing cultural misconceptions to enhance participation and referral compliance.

Conclusion

This study identified various challenges affecting the effectiveness of the SBVS in Egor Local Government Area, Benin City, Nigeria, mirroring similar issues found in other contexts. It highlights critical gaps in the provision of vision screening programs, including low prevalence, resource constraints, and lack of awareness. At the same time, it emphasizes the significant potential benefits of such programs and provides actionable recommendations to address the barriers identified. The findings emphasize the importance of targeted strategies to increase awareness, allocate resources effectively, and engage key stakeholders in ensuring that vision screening becomes an integral part of school health initiatives.

To address these challenges, recommendations include allocating adequate government resources to fund equipment, trained professionals, and materials; implementing staff training programs; fostering parental engagement; and establishing continuous monitoring and evaluation to improve the program's impact and sustainability in enhancing students' eye health outcomes.

Ethics approval: Ethical approval for this study was obtained from the Research and Ethics Committee of the Department of Optometry, University of Benin, with the REC approval number EC/UBEN/LSC. OPT/23/78. All procedures performed in this study were conducted in accordance with the Tenets of the Declaration of Helsinki for human subjects.

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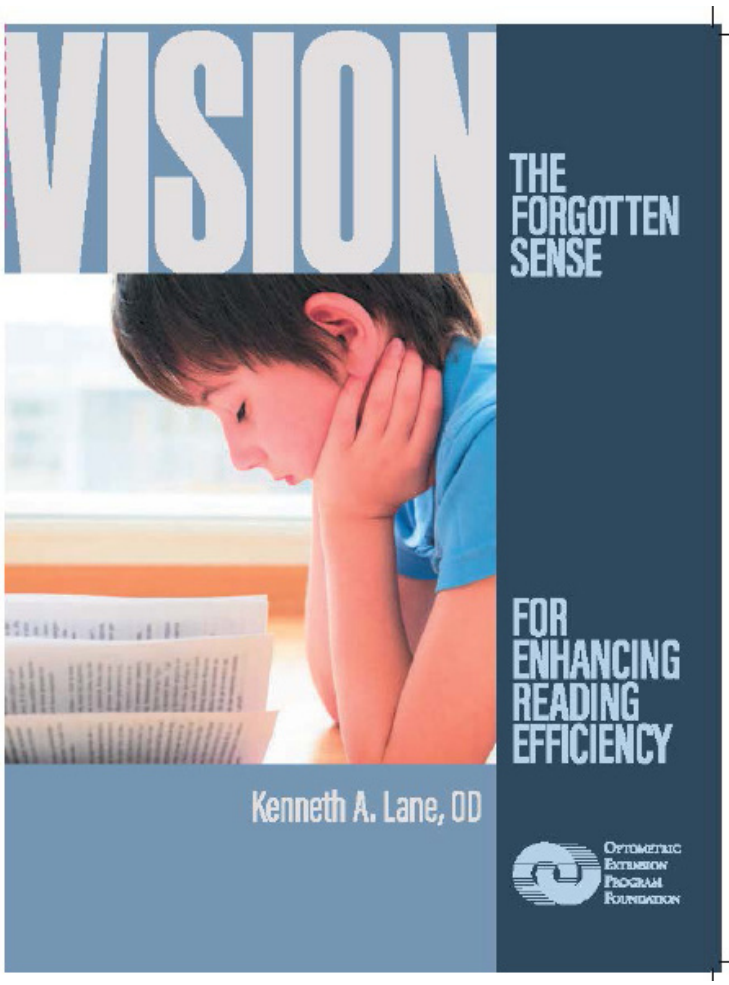

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Article • Effect of Optometric Multisensory Training (OMST) on a Patient with Post-Concussive Visual Snow Syndrome

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ABSTRACT

Background: Optometric Multisensory Training (OMST) is a unique method involving the simultaneous presentation of sensory stimulations in a safe and controlled clinical situation. This includes syntonic optometric phototherapy, vestibular (motion) stimulation, and auditory stimulation. The purpose of this study is to evaluate the effectiveness of OMST in improving visual and post-concussive symptoms in a patient diagnosed with post-concussive visual snow syndrome (VSS) following mild traumatic brain injury (mTBI).

Case Report: A 57-year-old female with a history of mTBI presented with persistent post-concussion syndrome (PCS), including VSS, oculomotor dysfunction, vestibular disturbances, cognitive deficits, and migraines. The patient underwent a 30-day OMST program consisting of in-office multisensory therapy combined with at-home syntonic phototherapy. Objective eye movement metrics and subjective symptom reports were assessed pre- and post-intervention.

Results: Using the RightEye standardized testing protocol, significant improvements were observed in eye movement performance, with overall scores improving from an average of 24 pre-treatment to 61 post-treatment. Using the Brain Injury Vision Symptom Survey (BIVSS), the patient reported an 80% reduction in visual snow symptoms and a 60% reduction in overall PCS symptoms, along with improvements in sleep quality, vestibular function, and cognitive processing.

Conclusion: OMST appears to be a promising neuro-optometric rehabilitation approach for patients with post-concussive VSS, demonstrating both objective and subjective clinical improvements.

Keywords: neuro-optometric rehabilitation, optometric multisensory training, post-concussion syndrome, syntonic phototherapy, visual snow syndrome

Introduction

Visual snow syndrome (VSS) is a neurological condition characterized by the persistent perception of a dynamic, pixelated visual disturbance across the entire visual field. It is often associated with post-concussion syndrome (PCS) and can significantly impair quality of life.

Patients frequently report additional symptoms, such as photophobia, palinopsia, headaches, and cognitive fatigue.[1]

Mild traumatic brain injury (mTBI) is a common precipitating factor for PCS and can lead to a wide range of visual and sensory dysfunctions, including oculomotor deficits, vestibular dysfunction, and impaired visual processing.[2,3] Traditional in-office vision therapy for PCS often falls short in addressing the complex multisensory integration deficits observed in these patients.

Optometric multisensory training (OMST) represents an emerging neuro-rehabilitation approach designed to address these deficits through simultaneous stimulation of visual, vestibular, auditory, and somatosensory systems.[4,5] This therapy incorporates syntonic phototherapy, using specific wavelengths of colored light alongside coordinated sensory inputs in a controlled clinical environment.[6,7]

This case report highlights the effectiveness of OMST in improving both objective visual function

and subjective symptomatology in a patient with post-concussive VSS.

Case Report

Patient History

HB is a 57-year-old African American female with a history of mild traumatic brain injury sustained at age 52 after being struck by an object in a school hallway, where she worked as a counselor. Following the injury, she developed persistent PCS. The patient's quality of life was significantly compromised by a complex constellation of neurological, visual, and systemic conditions. Persistent symptoms of visual snow created a continuous visual disturbance, interfering with reading, screen use, and overall visual comfort, while chronic migraines further limited daily functioning through pain, light sensitivity, and fatigue. Vestibular and oculomotor dysfunction contributed to imbalance, dizziness, and difficulty with eye movements, making tasks such as walking in busy environments or sustaining visual attention particularly challenging. These physical symptoms were compounded by cognitive deficits, including reduced concentration and processing speed, which impaired academic, occupational, and daily activities. Additionally, the presence of depression and anxiety exacerbated her functional limitations and emotional well-being, while obstructive sleep apnea led to poor sleep quality and daytime fatigue, further diminishing resilience and recovery. Hearing impairment added another layer of sensory difficulty, complicating communication and social interaction. Together, these overlapping conditions created a substantial and persistent burden, significantly reducing her independence, productivity, and overall quality of life.

The patient's post-therapy goals were centered on restoring functional independence and improving day-to-day performance across visual and cognitive domains. A primary objective was to reduce the intensity and intrusiveness of visual snow symptoms, allowing for greater visual clarity and comfort in both static and dynamic environments. She also aimed to improve reading stamina and comprehension, with the expectation of being able to sustain prolonged near work without fatigue, loss of place, or decreased understanding. This was critical for academic, professional, and personal tasks. In addition, enhancing cognitive processing speed and memory was a key goal, as she sought to think more clearly, to retain information more effectively, and to engage in complex tasks with greater efficiency. Collectively, these goals reflected a desire not only for symptom reduction, but also for meaningful improvements in functional capacity, productivity, and overall quality of life.

Table 1. Selected Pre-Treatment Exam Data (6/2/25)

Refraction	OD: 0.00-1.50x085 20/20 OS: -0.25-0.75x080 20/20
RightEye	Pursuits: 26 Saccades: 8 Fixation: 29 Overall Average: 24 (Figure 1)
Near Convergence	OD, OS unable to perform

Clinical Findings

Table 1 shows the exam data from the initial visit on 6/2/2025. The anterior and posterior segments were healthy and clear of disease processes.

Pre-treatment objective assessments revealed significant oculomotor dysfunction. These findings were consistent with the impaired visual tracking and fixation instability commonly observed in post-concussive patients.

Treatment Protocol

The patient participated in a structured 30-day therapy program known as optometric multisensory training (OMST), an integrative approach designed

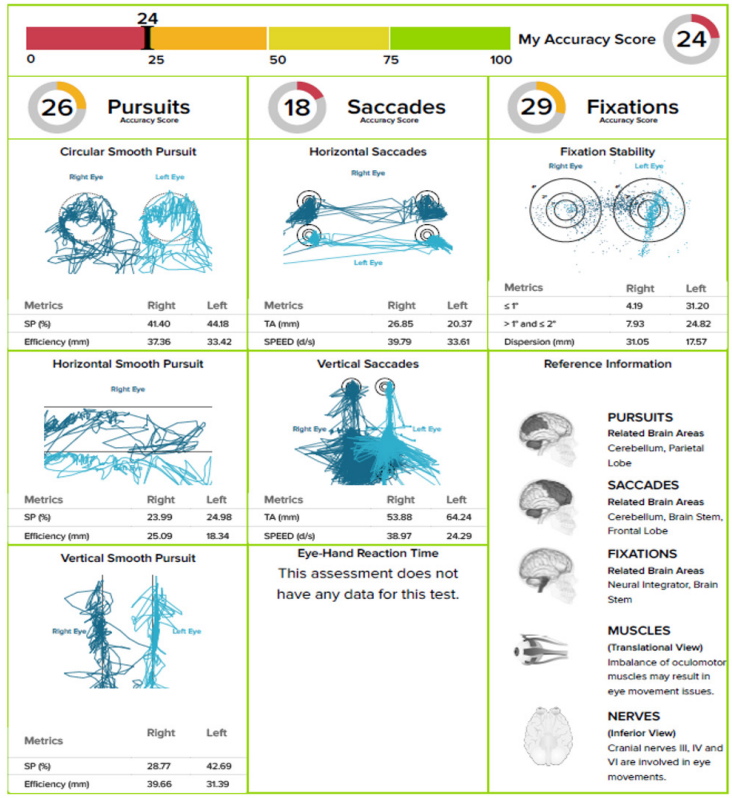

Figure 1. RightEye pre-treatment

Figure 2. Optometric multisensory table

to improve how the brain processes and coordinates information from multiple sensory systems. For the first 12 days, she completed daily one-hour, in-clinic sessions that combined visual, vestibular (balance), auditory, and somatosensory inputs in a carefully sequenced manner to promote neuroplasticity, the brain's ability to reorganize and to form new connections (Figure 2).[8,9] A central component of the program was syntonic optometric phototherapy, which uses specific wavelengths of colored light to influence visual processing and autonomic balance.[10] The therapy followed a progression of light frequencies, beginning with magenta and advancing through red, ruby-red, blue, green, yellow, and violet based on the premise that different wavelengths can modulate retinal pathways and downstream neural activity.

In addition to visual stimulation, vestibular input was incorporated through controlled body movements, alternating between side-to-side and head-to-toe directions, with daily changes in orientation. This type of stimulation is known to influence spatial orientation, postural control, and visual-vestibular integration, which are often disrupted in patients with neurological conditions.[11] Auditory stimulation was also included, with music delivered asymmetrically, 100% volume to the right ear and 50% to the left, to encourage right ear dominance and preferential activation of the left hemisphere, which is typically associated with language processing.[12] This binaural music was presented asymmetrically through headphones to encourage right-ear dominance and to stimulate left-hemispheric language and auditory processing centers. If the master volume was set at level 20, the right ear received the full intensity (volume level 20), while the left ear simultaneously received 50% reduced intensity (volume level 10). The auditory input remained comfortably soft and nonintrusive, comparable to low-level background or "elevator" music, allowing the patient easily to maintain normal conversational interaction with the clinician throughout therapy despite wearing headphones. These inputs were combined with somatosensory integration activities, engaging the tactile and proprioceptive systems to reinforce multisensory coordination further and to improve overall neural efficiency.[13]

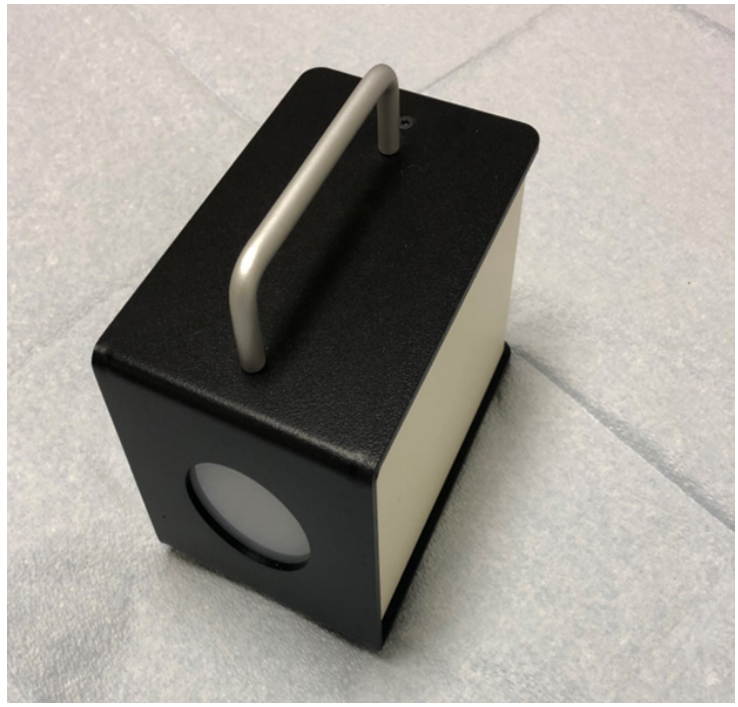

Figure 3. Syntonic phototherapy home unit

Outside of the clinic, the patient continued therapy at home using a lightbox for syntonic phototherapy (Figure 3), completing two 20-minute sessions over the weekend followed by 18 additional days of daily magenta light exposure. This home-based

component provided consistent reinforcement of the in-clinic interventions and supported cumulative neural adaptation through repetition. Importantly, no other therapeutic interventions were introduced during this period, allowing the observed outcomes to be more directly attributed to OMST. Overall, this comprehensive, multisensory approach is grounded in established principles of neuroplasticity and sensory integration, aiming to restore more efficient processing across visual, vestibular, auditory, and cognitive systems.[14]

Objective Outcomes

Following treatment, the patient demonstrated profound and clinically meaningful improvements across both objective measures and subjective experiences, reflecting a substantial enhancement in overall quality of life (Table 2). Post-treatment eye movement testing revealed greater than a 150% improvement in oculomotor performance, indicating significantly improved accuracy, speed, and coordination of saccades, pursuits, and fixation stability. These changes suggest more efficient integration of visual pathways and improved communication between the cortical and subcortical systems responsible for eye movement control. Functionally, this level of improvement reduces the effort required for visual tasks, allowing the patient to engage in daily activities with less strain and greater visual comfort.

These objective findings were strongly supported by the patient's subjective reports, which highlighted the real-world impact of the intervention. The patient noted an approximately 80% reduction in visual snow symptoms, representing a dramatic decrease in the constant visual disturbance that previously interfered with clarity, comfort, and environmental awareness. This reduction alone contributed significantly to decreased visual fatigue and improved tolerance for both near and distance tasks. In addition, there was a 60% reduction in overall post-concussive symptoms, indicating broad improvement in neurological function and a meaningful reduction in the symptom burden that often affects physical, cognitive, and emotional well-being.

Importantly, the patient also reported improved sleep quality and duration, a critical factor in neurological recovery and overall health. Better sleep likely supported enhanced daytime functioning, reduced fatigue, and improved emotional regulation. Vestibular symptoms, including dizziness and motion sensitivity, were also reduced, allowing the patient to move more comfortably within her environment and to tolerate activities such as walking, driving, or navigating visually complex spaces with greater confidence and stability.

Table 2. Post-treatment Exam Data (12/12/25)

Refraction	OD: 0.00-1.50x085 20/20 OS: -0.25-0.75x085 20/20
RightEye	Pursuits: 58 Saccades: 48 Fixation: 78 Overall Average: 61 (Figure 4)
Near Convergence	OD: x/25/20 OS: x/18/14

Cognitive improvements were another key outcome, with the patient reporting enhanced processing speed and mental clarity. This suggests improved efficiency in higher-level neural processing, likely supported by better sensory integration and reduced neurological interference. These gains translated into functional benefits, including mild but meaningful improvements in reading comprehension and stamina. The patient was able to sustain attention for longer periods and process written information with less effort, indicating early but important progress in academic and occupational readiness.

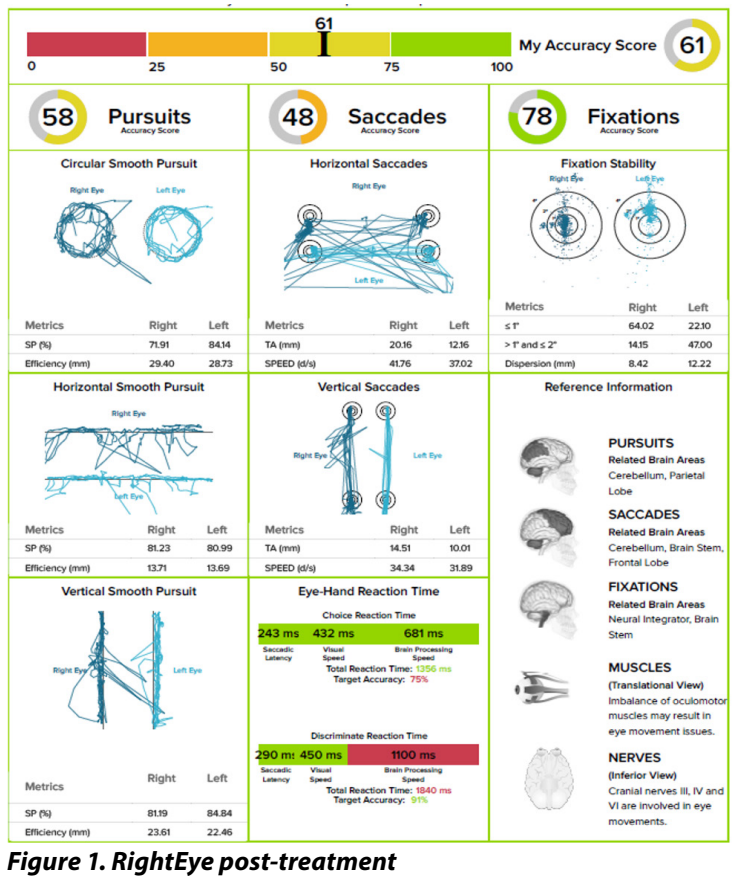

Figure 1. RightEye post-treatment

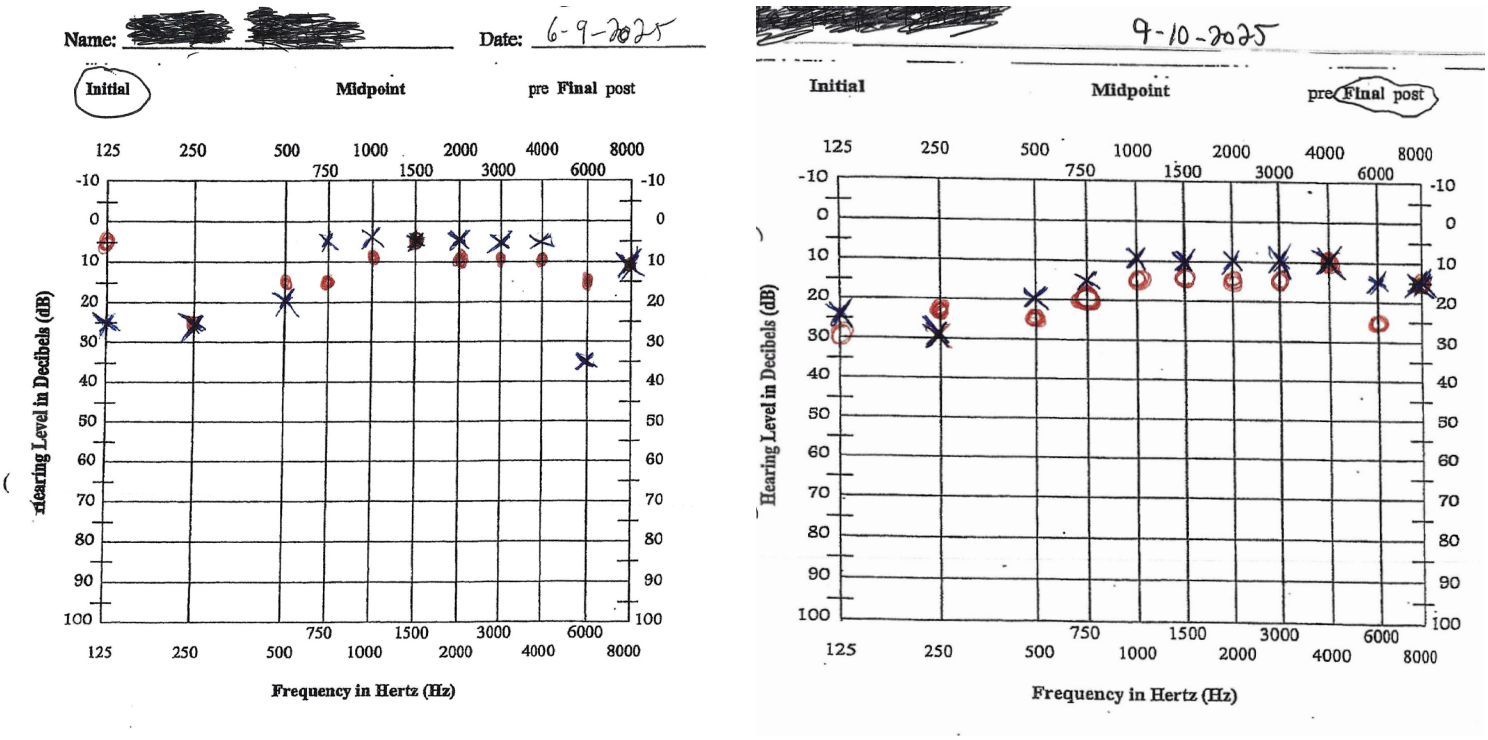

Figure 5. Listening profile pre- and post-treatment. Red dot=right eye, X= left eye

Overall, the close correlation between objective clinical improvements and subjective symptom reduction underscores the effectiveness of the treatment approach. The patient's gains extend beyond isolated clinical metrics, reflecting a holistic improvement in daily functioning. Reduced visual and vestibular symptoms, enhanced cognitive performance, better sleep, and improved tolerance for reading and visual tasks collectively contribute to a significantly improved quality of life. The patient is now better equipped to participate in work, academic, and social activities with increased independence, confidence, and comfort, representing a meaningful and impactful recovery trajectory.

Comparison of the pre- and post-treatment listening profiles demonstrated improved auditory threshold organization, increased interaural symmetry, and reduction of high-frequency irregularities following OMST intervention (Figure 5). These changes may reflect adaptive neuroplastic reorganization within multisensory cortical and subcortical networks. Given the integrated nature of auditory, vestibular, and visual processing systems, the observed improvements support the hypothesis that OMST facilitates enhanced sensory integration and neural efficiency in patients with post-concussive VSS.

Discussion

This case demonstrates the potential effectiveness of OMST as a comprehensive neuro-optometric rehabilitation approach for patients presenting with post-concussive visual symptoms, including VSS. The significant improvements observed in eye movement performance suggest enhanced integration and coordination of visual pathways. Given that oculomotor dysfunction is a well-established contributor to symptoms such as reading difficulty, reduced visual endurance, and visual fatigue,[15] the gains in saccadic accuracy, fixation stability, and tracking ability likely played a central role in the patient's functional improvements.

These findings are consistent with a growing body of case reports and clinical observations supporting OMST as an effective intervention for patients with concussion, traumatic brain injury, and developmental visual disorders.[16]

OMST's distinguishing feature is its multisensory design, which simultaneously engages visual, vestibular, auditory, and somatosensory systems. This integrated approach may be critical to its success, as it promotes neuroplasticity and facilitates more efficient neural processing. The observed outcomes align with previously published work suggesting that bottom-up sensory integration strategies can accelerate recovery in post-concussion patients by strengthening foundational sensory processing before advancing to higher-level visual tasks.[17]

The incorporation of syntonic phototherapy, a core component of OMST, may further enhance treatment outcomes by modulating autonomic balance and improving visual processing efficiency.[18]

In this case, syntonic intervention may have contributed to improved visual comfort and reduced sensory overload, thereby allowing the patient to better tolerate and engage in multisensory training tasks.

Additionally, the inclusion of at-home lightbox therapy provided important continuity of care and likely reinforced treatment effects through consistent repetition and ongoing neural stimulation.[19] The home lightbox was specifically designed to deliver magenta light only, aligning with the initial phase of the syntonic phototherapy sequence used during in-office treatment. The patient was given the lightbox and was instructed to use the device twice daily for 20 minutes per session over 18 consecutive days, creating a structured and intensive home-based intervention. This frequency and consistency of exposure supported sustained neuromodulation of the visual and autonomic systems, helping to stabilize and to extend the therapeutic gains achieved during clinic sessions. By maintaining regular stimulation outside of the clinical environment, this home-based component enhanced neuroplastic adaptation, reduced regression between visits, and contributed to the overall durability of treatment outcomes.[20] Such an approach underscores the value of integrating targeted, protocol-driven home therapy to complement in-office care and promotes longer-term functional improvement.

When considered together, OMST and syntonic phototherapy appear to offer a complementary therapeutic framework. OMST actively trains sensorimotor integration through dynamic, task-oriented activities, while syntonics may optimize the underlying physiological state of the visual system by regulating autonomic tone and enhancing baseline processing efficiency. Clinically, this combination may allow for more rapid progression through therapy and improved patient tolerance, particularly in individuals with heightened sensory sensitivity following neurological insult.

Importantly, the absence of other concurrent therapies in this case strengthens the likelihood that the observed improvements were attributable to OMST and its associated components. Both objective findings and subjective symptom reduction were substantial, supporting a meaningful clinical response rather than a coincidental or placebo effect.

While this report represents a single case, the magnitude of improvement, particularly in oculomotor performance and functional visual outcomes, supports further investigation into OMST as a viable treatment modality for patients with VSS and PCS. Future research should focus on larger cohort studies, standardized outcome measures, and controlled trials to define treatment protocols better, to isolate contributing components such as syntonic phototherapy, and to validate the reproducibility of these results.

Conclusion

OMST demonstrated significant clinical benefits in a patient with post-concussive visual snow syndrome, with meaningful improvements observed in both objective oculomotor performance and subjective symptom reduction. Enhanced saccadic accuracy, fixation stability, and pursuit tracking were strongly associated with reductions in visual disturbance, perceptual noise, and overall post-concussive symptom burden. As eye movement efficiency improved, the patient experienced greater visual clarity, improved tolerance for sustained visual tasks, and enhanced daily functioning, emphasizing the important relationship between oculomotor control and functional recovery following mTBI.

The findings also highlight the value of OMST's multisensory rehabilitation approach, which integrates visual, vestibular, auditory, and somatosensory stimulation to promote neuroplasticity and more efficient neural processing. Improvements in vestibular symptoms, cognitive function, sleep quality, and overall quality of life suggest that OMST may exert broader regulatory effects on central nervous system integration and autonomic balance. As a structured, yet adaptable, non-invasive neuro-optometric intervention, OMST may represent a promising treatment option for patients with persistent post-concussive symptoms who have not fully responded to traditional therapies.

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Article • Digital Therapies for Amblyopia

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ABSTRACT

Amblyopia is a developmental visual disorder with consequences beyond reduced visual acuity, affecting stereoacuity, contrast sensitivity, oculomotor function, and eye-hand coordination. While patching remains the longstanding standard of care, adherence challenges and its inherently monocular approach have driven the development of a new generation of digital and perceptual learning-based therapies. This article reviews five clinically relevant technologies—Luminopia, CureSight, AmblyoPlay, Bynocs, and RevitalVision—examining their mechanisms of action, treatment protocols, and supporting clinical evidence. Collectively, these platforms represent a meaningful expansion of the amblyopia treatment toolbox, offering patients and clinicians options that are engaging, home-based, and in many cases, capable of addressing the binocular and cortical deficits that patching alone does not target. Selecting the right tool requires an understanding of each platform's unique delivery, its evidence base, and the patient's individual visual profile.

Keywords: amblyopia, dichoptic therapy, perceptual learning, digital therapeutics

Introduction

Amblyopia is a developmental visual disorder resulting from atypical binocular experience in early childhood, leading to abnormal visual cortex development and vision impairment. Recovery relies on neuroplasticity in the visual cortex.[1] While reduced visual acuity is the hallmark most clinicians recognize, amblyopia's impact extends far beyond the size of a letter on the eye chart. Affected individuals demonstrate deficits in contrast sensitivity, stereoacuity, spatial localization, spatial resolution, oculomotor function (including pursuits, saccades, and fixation stability), accommodation, vergence, suppression patterns, eye-hand coordination, motion perception, and temporal processing.[2,3]

For decades, the primary evidence-based treatment has been patching. The landmark PEDIG Amblyopia Treatment Studies, published over twenty years ago in 2003, established the foundational dosing parameters still used today: ATS 2A demonstrated that 6 hours per day of patching was equally effective as full-time patching for severe amblyopia, and ATS 2B showed that 2 hours per day was equally effective as 6 hours for mild to moderate amblyopia. These findings were groundbreaking at the time and remain clinically relevant, but they also define the lower bound of what we ask of our patients: a minimum of 2 hours per day with a patch over a child's eye. Anyone who has treated amblyopia with patching knows the main obstacle isn't the prescription, it is compliance. Adherence to patching has been reported to be between 40 and 60% by week 6 and may fall to approximately 30% by week 12.[4] A treatment that children resist wearing, that carries social stigma, and that doesn't quite address the binocular nature of the condition leaves room for improvement.

The good news is that the gap is being filled. A new generation of digital and perceptual learning-based therapies is reshaping how we approach amblyopia treatment. In this article, I will review five clinically relevant emerging technologies—Luminopia, CureSight, AmblyoPlay (EyeHero), Bynocs, and RevitalVision—examining the science behind each, the evidence supporting their use, and what they mean for the future of amblyopia care.

Luminopia

Luminopia is a prescription digital therapeutic for amblyopia, the first FDA-cleared device of its kind. It received FDA clearance in 2021, with a commercial launch in 2023, and is indicated for children ages 4–12 with amblyopia secondary to anisometropia or mild strabismus (under 5 prism diopters).

The core mechanism is dichoptic therapy: presenting different images to the right and left eye simultaneously to reduce the brain's suppression of the amblyopic eye and to encourage both eyes to work together. What makes Luminopia unique in its delivery is that, rather than games or abstract exercises, children watch their own selected TV shows and movies through a virtual reality headset. The software modifies streaming content in real time, applying a global contrast reduction to the fellow eye so that the brain must rely on the amblyopic eye's input to perceive the full image. The result is a treatment that feels less like therapy and more like screen time.

The standard prescribed dose is one hour per day, six days per week. Parents have access to a monitoring portal that lets them track their child's daily usage and select content from an approved library, providing families with both oversight and a degree of personalization.

Xiao et al.[5] conducted the pivotal Phase 3 randomized controlled trial, enrolling 105 children ages 4–7 with unilateral amblyopia from anisometropia, strabismus, or both, with amblyopic eye visual acuity between 20/40 and 20/200. Children were randomized 1:1 to Luminopia plus glasses versus glasses alone for 12 weeks. The Luminopia group improved by 1.8 lines in visual acuity compared to 0.8 lines in the glasses-only group, a statistically significant difference of 1.0 line. Notably, 62% of Luminopia-treated children achieved two or more lines of improvement, compared to 33% in the control group (glasses only), and median adherence reached 88%. These results were compelling enough that the trial was stopped early for success and formed the basis for FDA clearance.

CureSight

CureSight is a prescription, FDA-cleared, home-based dichoptic treatment for amblyopia that combines real-time eye tracking technology, red-blue anaglyph glasses, and streamed video content. It is indicated for children ages 4 to under 9 with anisometropic, small-angle strabismic (up to 5 prism diopters) or mixed-mechanism amblyopia, with amblyopic eye visual acuity between 20/32 and 20/100.[6,7]

As with Luminopia, children watch their own selected TV shows and movies, but the technology behind CureSight is distinctly different. Rather than applying a global contrast reduction to the fellow eye, CureSight uses a built-in eye tracker on an 11.6-inch computer monitor to continuously track each eye's gaze in real time. Using this gaze data, the software applies a targeted, dynamic blur specifically around the fovea of the stronger fellow eye—only where that eye is actually looking, moment by moment—while leaving the amblyopic eye's view completely sharp and unaffected. The blur diameter and intensity are automatically calibrated to each child's measured visual acuity: the worse the amblyopia, the larger and stronger the blur applied to the fellow eye. The prescribed dose is 90 minutes per day, five days per week for 16 weeks, totaling 120 hours of treatment.

The pivotal evidence comes from a prospective, multicenter, randomized, evaluator-masked, controlled noninferiority trial across six sites in Israel that led directly to FDA clearance.[8] Children (n=103) were randomized 1:1 to CureSight versus standard patching for 16 weeks. CureSight achieved a mean VA improvement of 2.8 lines, compared with 2.3 lines in the patching group, with 79% of CureSight-treated children achieving 2 or more lines of improvement, versus 61% in the patching group. CureSight also demonstrated significant improvement in stereoacuity and binocular VA, an important distinction, as Luminopia's pivotal RCT did not show a significant stereoacuity benefit. It is worth noting, however, that the patching group in this study showed an essentially identical stereoacuity improvement of 0.40 log arcseconds. Adherence was 91% for CureSight versus 83% for patching.

Wygnanski-Jaffe et al.[8] followed the original RCT patients for one year after treatment stopped, with no additional amblyopia treatment permitted. VA gains were fully maintained at 12 weeks post-treatment with no regression. At one year, children lost less than one line from their end-of-treatment peak but retained approximately two full lines of improvement compared to baseline, a residual gain that remained statistically significant ($p<0.0001$). Stereoacuity and binocular VA gains were similarly maintained, and amblyopia recurrence occurred in only 5 of 27 patients at the one-year mark.

AmblyoPlay (Eyehero)

AmblyoPlay (Eyehero) is a gamified, app-based dichoptic vision therapy platform, developed by Smart Optometry in Slovenia and designed for home use in children with amblyopia. It is downloaded as an app and used on a tablet or computer screen. Children wear red-green anaglyph glasses to separate the images presented to each eye. The amblyopic eye receives high-contrast stimuli, while the fellow eye receives attenuated input through the red filter, and interactive games are played during therapy. AmblyoPlay is commercially available but is not FDA-cleared.

The platform operates on dichoptic principles, presenting complementary stimuli to each eye under conditions that require binocular cooperation to reduce interocular suppression and to promote functional binocularity. A proprietary adaptive algorithm continuously adjusts game content and difficulty in real time based on the child's accuracy, reaction time, and consistency. Importantly, AmblyoPlay is designed to target not only visual acuity but a broader range of visual functions, including oculomotor control, selective attention, and hand-eye coordination. This reflects the understanding that amblyopia is a multisystem neurodevelopmental disorder. The prescribed dose is 30 minutes per day, five days per week, for six months.

The clinical evidence comes from a prospective non-randomized controlled study that had 29 children ages 7–13 with anisometropic amblyopia were enrolled: 14 in the treatment group and 15 age- and sex-matched typically developing controls who received no intervention.[3] What distinguished this study from most amblyopia trials was its multimodal assessment approach. They measured not only visual acuity and stereopsis but also oculomotor function via videonystagmography, motor proficiency via the BOT-2, and postural stability via the Sensory Organization Test.

Visual acuity in the amblyopic eye improved progressively across all four time points, with a total gain of approximately 1.5 lines at six months. Stereoacuity improved from a median of 300 arcseconds at baseline to 45 arcseconds at six months. That is a three-octave gain in binocular depth perception. Oculomotor improvements were equally compelling: saccadic latency decreased from 235 ms to 213 ms, smooth pursuit gain improved across all tested frequencies, and optokinetic gain improved from 0.65 to 0.73 in the amblyopic eye. Perhaps most strikingly, BOT-2 upper extremity coordination scores improved by nearly 40%, from 22.55 to 31.00 ($p<0.001$), and balance scores improved significantly as well. Controls showed no significant changes in any measured domain.

Bynocs

Bynocs is a cloud-based dichoptic vision therapy platform designed to treat amblyopia through gamified, computer-based training. It runs in a standard internet browser on any screen larger than 11 inches and requires only an internet connection and a pair of red-blue anaglyph glasses. Bynocs has been studied in children as young as 4 years old and in adults up to 30 years old, across multiple amblyopia types, including anisometropic, isometropic bilateral, strabismic, and deprivational amblyopia.

The platform uses red-blue anaglyph glasses to separate the images presented to each eye. What distinguishes Bynocs is its figure-ground stimulus design: the key game elements requiring active attention are visible only to the amblyopic eye, while the fellow eye sees the broader game environment. Color contrast between the two eyes is automatically adjusted so that the weaker eye perceives higher-contrast stimuli, and the stimuli presented to the amblyopic eye undergo continuous dynamic adjustments in size, spatial frequency, temporal frequency, and target speed as the patient improves. A notable feature is built-in remote supervision. Patients are monitored by a Bynocs optometrist via video call, with visual performance and compliance assessed after every ten sessions. The prescribed dose is 30 minutes per day, five days per week for six weeks.

There was a retrospective study from Mumbai analyzing data from 161 children ages 4–13, including both anisometropic (127 children) and isometropic bilateral amblyopia (34 children), a population typically excluded from other dichoptic studies.[6] With no control group, BCVA in the amblyopic eye improved by a mean of 0.39 logMAR, approximately four lines. The percentage of patients without measurable stereopsis dropped from 65.8% to 11.8%, and the percentage without flat fusion on Worth 4-dot fell from 61.5% to 6.3%. One limitation worth noting is that stereopsis was measured using the "Bynocs Randot Test," the platform's built-in tool, which raises a potential conflict of interest when assessing the platform's outcomes.

A study published in the Journal of Pediatric Ophthalmology and Strabismus by Ganesh,[7] conducted at Aravind Eye Hospital in Coimbatore, evaluated 59 patients ages 5–30 across a broader range of amblyopia types. Of 74 eyes treated, 82.4% showed improved visual acuity, and 98.6% improved in at least one measured parameter. Stereopsis was assessed using independent validated tests, the TNO test for near and polarized random dot stereograms for distance, and the results were compelling: patients without measurable near stereopsis dropped from 47.5% to 11.9%, and 48% achieved normal near stereoacuity of 60 arcseconds or better. Among the 36 patients with residual amblyopia after failed patching, Bynocs produced a two-line improvement in VA (0.40 to 0.20 logMAR), compared to only a half-line improvement that those same patients had achieved with patching. Improvement was statistically significant both in patients ten years and under and in those older than ten, with no significant difference between age groups (p=0.123), directly challenging the assumption that amblyopia treatment is ineffective beyond the critical period. Durability data at a mean of 5.5 months post-treatment showed that most patients maintained their gains, with regression occurring in only 4 eyes (12.1%), primarily in strabismic cases.

RevitalVision

RevitalVision is an FDA-cleared perceptual learning software program developed by Talshir Medical Technologies in Israel, indicated for adults with amblyopia. It is software-only. No special glasses, headset, or hardware beyond a standard computer monitor is required. Beyond amblyopia, it has been reported to improve vision in several other conditions, including presbyopia, low myopia, post-cataract surgery, and post-LASIK surgery.[9]

Unlike the dichoptic platforms described above, RevitalVision is a monocular perceptual learning program that works by directly retraining the visual cortex.[10] In amblyopia, abnormal development of the primary visual cortex leads to disrupted lateral interactions among orientation-selective neurons, characterized by reduced facilitation and an abnormally extended range of inhibition.[10] RevitalVision addresses this by training the visual cortex using Gabor patches, which are gray-level sinusoidal gratings mathematically designed to match the receptive field structure of neurons in the primary visual cortex, eliciting a unique neural response in the exact cells that are dysfunctional in amblyopia.

During each session, the patient sits 1.5 meters from their computer screen in a dark room wearing their optical correction, with the fellow eye blurred using a semi-transparent cover. They are shown two brief sequential displays and must identify which display contains a target Gabor patch flanked by collinear high-contrast flankers, in a two-alternative forced-choice contrast-detection task. This configuration forces the brain to engage its lateral cortical interactions to detect the target. The program progressively increases the challenge across sessions in terms of contrast, spatial frequency, and orientation, systematically reducing pathological lateral inhibition and restoring facilitatory neural connections. The prescribed protocol is 30-minute sessions three to four times per week, for a total of 40 to 60 sessions over approximately four months.

The foundational evidence comes from a prospective, randomized, masked, placebo-controlled trial.[10] Seventy-seven amblyopic patients ages 9–55 were enrolled: 63 in the treatment group and 14 in a placebo control group that practiced a superficially similar task without the active flanker stimulation. There were 16 normal-vision controls. Both anisometropic and strabismic amblyopia were included, with strabismic subjects limited to less than 8 prism diopters of deviation. The treatment group achieved a 78% average improvement in visual acuity, approximately 2.5 lines, after a mean of 45 sessions, while the control group showed no improvement whatsoever. Of treated patients, 68% achieved two or more ETDRS lines of improvement, and 83% of patients who started with visual acuity worse than 20/40 ended treatment with visual acuity better than 20/40. Contrast sensitivity improved by a factor of 2 to 4 times across all spatial frequencies. Critically, improvement was not significantly dependent on age. Adults responded as well as older children, and gains were fully retained at 12-month follow-up, with additional improvement at higher spatial frequencies.

Discussion

The amblyopia treatment landscape has expanded well beyond the patch. The five technologies reviewed here—Luminopia, CureSight, AmblyoPlay, Bynocs, and RevitalVision—each bring a distinct mechanism, evidence base, and patient experience to the table, and none is universally superior to the

others. Choosing the right tool requires knowing your patient's exam findings, interest, and needs.

Delivery format matters. A child who is motivated by gaming may thrive with AmblyoPlay or Bynocs, while one who simply wants to watch their favorite show may be better suited for Luminopia or CureSight. A patient whose amblyopia persists into adulthood may gravitate toward RevitalVision's cortical retraining approach. Whether treatment is delivered through a virtual reality headset or in free space with anaglyph glasses may also influence your patient's binocularity, comfort, acceptance, and ultimately adherence. Your choice depends on your knowledge of each tool, its limitations, and, above all, your patient's individual needs—because the right tool for one patient may not be the right one for the next.

The clinical profile of each patient should also guide selection. A child struggling specifically with stereoacuity may benefit from a treatment with demonstrated outcomes in stereopsis. One with documented oculomotor deficits or poor hand-eye coordination may be best served by a platform that targets those functions directly. Understanding the age ranges studied in each clinical trial helps us match the evidence to the patient sitting in our chair.

Conclusion

As clinicians, our role is no longer simply to prescribe a patch and monitor acuity. We are now equipped to be thoughtful about which technology best addresses each patient's specific visual profile, preferences, and goals. The more fluent we become in these tools, including their mechanisms, their evidence, and their limitations, the better positioned we are to individualize care. Amblyopia treatment has entered a new era, and our patients are better off for it.

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Article • Clinical Guide to Einsteinian Low Vision Rehabilitation: Part 2 of 4

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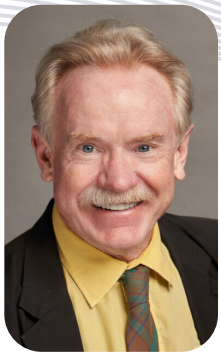

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The Longest Journey Begins with the First Step

"The noblest distinction of man is his passion for knowledge."
–Will Durant

"Professor McIver, we have a question for you," said the spokesperson for the small cadre of low vision students at the office door. "May we come in?"

"Certainly." Replied Professor McIver. "What is on your mind?"

"Well," began the first student hesitatingly, "we have a conundrum. It seems to us that everything we have been taught so far regarding visual acuity (VA) is that 20/20 is perfect vision, and that 20/20 should be our goal for each patient, but that seems impossible for patients with low vision, and on top of that, we don't even know where 20/20 came from."

"Well, first, 20/20 is not nor ever has been 'perfect vision,' and it is not even vision, it is sight. There is a difference between the two, with sight being the ability to distinguish angular subtense and vision being the brain's cognitive gestalt. Definitions aside for now, however, I think the best way for you to gain a good understanding of '20/20' is to dive deep into 'The Source'. There is a very interesting story of how we came to 20/20 and what it really means. Let's go meet Robert Hooke from history."[7]

"Come in," Robert Hooke said to the group of applicants. "As you should know from the notice I posted, I am looking for an assistant with good sight to help me with my studies of the star systems." It was the late 17th century, and the nascent concept of experimental scientific method was rising. The Royal Society of London was home to a number of great scientists, including Robert Boyle and Isaac Newton in addition to Robert Hooke.

It was clear and dark that night, unusual for London, as Robert Hooke took his apprentice applicants out to view the night sky. Taking them aside, one by one, he directed their attention to Ursa Major (Big Dipper) and asked them to count the constituent stars.

"I am sure there are seven," many of the hopefuls said one after another, only to hear, "Thank you, but you are not correct. You may go," Robert Hooke disappointedly said, again and again.

"I count eight," one candidate finally announced, and he was hired on the spot.

At last!" Hooke exclaimed, "I have found an assistant with good sight! I can always count on Alcor and Mizar to be arbiters of good sight, as the stars are static and will always be available to me in the future when I need new apprentices."

"What are Alcor and Mizar?" the student spokesperson chimed in, bringing the group back to the present.

"Why, those are what look like a binary star in the handle of the Big Dipper," Professor McIver said, stepping away from The Source. "You see, the angular separation, combined with the brightness differential between Alcor and Mizar, is 1' of arc, or the minimum angle of resolution of a 20/20 letter. Just imagine for a moment how odd it is that our definition of good visual acuity goes back to an employment test from almost four centuries ago," Professor McIver told his study group. "20/20 is nothing more than the essential angular subtense between the stars Alcor and Mizar in the Big Dipper used by Robert Hooke to qualify an apprentice for employment. But it marked an important point of departure by defining what good sight is and establishing a standard that could be replicated anywhere in the Northern Hemisphere."

"I don't believe I have ever heard much of Robert Hooke," another student exclaimed, "even though I was a science major as an undergraduate student."

"That is not surprising," Professor McIver explained, "as Robert Hooke had a significant falling out with Isaac Newton, accusing Newton of plagiarizing his work. As you might imagine, that did not sit well with the powerful Newton, who, as head of the Royal Society, it has been suggested, canceled Hooke even to the point of having his portrait 'disappeared'. Whether that story is true or just apocryphal, we now know that

Robert Hooke's scientific work with astronomy and microscopy was groundbreaking, and we owe a lot to him, including our modern-day definition of good visual acuity. Remember, however, 20/20 is arbitrary and the result of an old employment test; it is not an absolute standard of 'good sight,' so do not, as good doctors with psychometric integrity, use it as a cutoff for refraction, an ideal goal acuity, or best-possible vision."

Noticing

"You can observe a lot just by watching."
–Yogi Berra

"Have you ever noticed," Professor McIver began to the class, "that so much of what is done in the health care professions is that which was done in the past and handed down from generation to generation of practitioners without question? Don't get me wrong, there is a lot of good, evidence-based practice being done, but so much of our daily care has not changed significantly in decades, as there is just not good evidence for everything we do."

"Why are you telling us this?" Hugh from the back asked. "If it's been done for decades, shouldn't it be just fine to continue?"

"There it is!" Professor McIver exclaimed. "While it might be "fine", we need to keep our eyes open for changes in our process of care if we are to consistently improve. There is a word for continuous improvement in Japanese: it is 'kaizen.' It is a mindset, a philosophy, and a methodology of making ongoing step-by-step progress and requires an open mind to keep watching for opportunities. Most steps of improvement are small and incremental, but sometimes those steps of improvement are large and might even be big enough to provide a chance for a punctuation of the equilibrium. For instance, back in the 1970s, a revolution in measuring visual acuity was largely sidelined by the inertia of years and years of recording acuity in the established, classic manner. Even though the old way no longer met the high standards of psychometric integrity, it had always been considered 'fine' and so endured (Bailey I, Lovie-Kitchin J, 2013). That should no longer be considered satisfactory for modern optometrists who adhere to psychometric integrity."

"Listen, why don't we open up 'The Source' and see where the revolution came from and how we can still access its power in caring for our partially sighted patients?"

"That sounds intriguing," Hugh said, speaking for many in the class, "but until it can be demonstrated as superior, I still think 'fine' is 'fine.'"

"We'll see if you still think so when I am done. So, here we go," Professor McIver replied. "Math was advancing in the late 16th century when John Napier discovered logarithms and used them as an aid in calculating the large numbers needed for astronomy. By the early 17th century, he invented the calculating tool known as Napier's Bones as a forerunner to the slide rule. The slide rule was invented by William Oughtred[8] and eventually allowed three decimals of accuracy once Isaac Newton, Robert Hooke's nemesis, added a slide a few decades later. The slide rule made modern industrial society possible and was de rigueur for all professions until the early 1970s, when affordable electronic calculators came on the scene as inexpensive and widespread for performing complex calculations. 'So what', you think, right?" Professor McIver asked rhetorically.

"Exactly," Hugh sighed, waiting for the end of the session. "It's been a nice history lesson, but what does it have to do with patient care?"

"Glad you asked," Professor McIver continued, "as it has everything to do with rational decision-making in the clinic. Recall that, as you have been taught so far, we have vision charts and refraction techniques which were developed for fully sighted patients. The large, irregular-letter-size jumps on the chart and the 0.25 D lens steps were fortuitously workable for them in refracting the fully sighted. When we begin dealing with partially sighted patients, however, those systems break down quickly, causing frustration for both patients and doctors, and is one of the main reasons, in my opinion, that many practitioners shun low vision care."

"So, let's continue to explore how math can be our friend in low vision rehabilitation. In the 19th century, Hans Weber observed that human sensory ability to detect change was proportional to the original stimulus. The original stimulus for measuring sight is the 1' of arc that Robert Hooke posited as good vision. Proportional change can be thought of as changes in letter size as well as dioptric power. Do you see the logarithmic function? Logarithms allow math to be simplified by making proportional change into additive change. Now, since it is easier for human brains to add and subtract rather than to multiply and divide, we can glimpse an avenue to lever the principles of 'good sight' that Robert Hooke defined with the rational chart letter sizes and power progression that Weber discovered. It has since been

determined that the proportional change for humans to detect a change—a just noticeable difference, or JND, if you will—is when the stimulus changes by 0.1 log from a baseline. If we take Robert Hooke's 1′ of arc subtense as standard good sight, we can quickly build a visual acuity chart, a rational vision chart, with steps between letter sizes of 0.1 log steps. Bailey and Lovie did exactly that in the late 1970s with the Bailey-Lovie chart, the first rationally designed vision chart that incorporated human abilities as the guiding principle. The ETDRS chart then slightly modified the Bailey-Lovie chart and became the standard way to measure visual acuity ever since. In actuality, when we think deeper about how these charts were developed with logMAR principles, we find that they are calculating devices like a slide rule! Slide rules were the calculating devices that made possible the industrial revolution. They ruled the calculating world until the 1970s, when electronic devices took over. Therefore, using the slide rule properties of the logMAR, we can make accurate predictions of power, working distance, and telescopic power with nothing but a logMAR chart. But I am getting ahead of the progression of knowledge here; that is a lesson for another time. For now, it must be recognized that old charts and paradigms for measuring visual acuity only worked satisfactorily for fully sighted patients due to fortuitous circumstances, not scientific rigor. In order to properly take care of all of our patients, we need to break out of the old confines and embrace the power of logMAR thinking."

Hugh was quiet a moment, as was the whole class; then he said, "I for one cannot wait to see how to apply this, even though it seems a bit esoteric at this point."

"That is 'fine' for now," Professor McIver smiled. "The following lessons will explain more and introduce techniques, processes, and methods to apply logMAR thinking. Class dismissed."

Rationalizing Near Visual Acuity

"It ain't what you know that gets you into trouble, it's what you know for sure that just ain't so."

–Mark Twain

"I am glad we got this little study group together," Professor McIver began. "You fourth-year students have had a decent amount of clinical experience by now and no doubt believe that you have a good grasp of visual acuity, what it means, and how to use it in patient exams. Distance VA (dVA) is pretty well standardized as being a Snellen fraction with numerator of object distance over a denominator of object size, always making sure we keep our units the same: i.e., metric vs. imperial. We commonly take that to be when tested at a distance of 20 feet, the patient can resolve a 20-foot letter: 20/20. Simple enough, and we all get that. We have already seen what that means in the most precise and psychometrically sound manner in an earlier lesson when we use non-standard test distances. As a related aside, it should be mentioned that "count fingers" is an abomination to psychometric integrity and should never be seen on a record as a form of visual acuity. Count fingers is an anachronism to be left for people who don't know what visual acuity means. You see, there are drastically different sizes of fingers, from 120 foot letter to 200 foot letter, as many different color fingers as there are people, and different contrasting backgrounds such as dark scrubs or a white clinic coat. If you can't get an acuity on the ETDRS chart at 2 meters, just grab a Feinbloom distance chart, and move closer to the patient. The Feinbloom chart goes all the way up to a 700 foot letter with high contrast and recognition numerals, so it is acceptable for very poor acuities. There is also the Berkeley Rudimentary Vision Chart for very reduced acuities. So, if I ever find you using "count fingers", I will require you to turn in your psychometric integrity membership card. As you might surmise by now, "count fingers" is a trigger for your Professor McIver, so avoid using it at all costs. That being said, today, I want this group to focus on near VA (nVA) and apply the principles of dVA to nVA.

You have each been working with near visual acuity for a while, and I want to hear your take on the various nomenclatures. We know there are four separate nomenclature terminologies in general use for recording near visual acuity, but we are interested in knowing what their differences are, their advantages and disadvantages, and which makes the most sense to use. Those four systems are:

1. Reduced Snellen (RS)
2. Point or 'n' units
3. Jaeger, aka J-System
4. M-Units

"While you may have had classes, labs, or clinics together, I want to make sure you know each other. By way of introduction, we have today in our group Rochelle, Ahn, Jesse Johnson (J.J.), and Logan," Professor McIver said. "Let's begin with reduced Snellen."

"Oh," exclaimed Rochelle, "let me take that. I find reduced Snellen so much easier than the other systems because it closely resembles distance Snellen, and I can know exactly what it means. The comfort level is great for me, and people outside our field can quickly

determine what our report letters mean. Besides, a lot of electronic health records contain drop-downs in the nVA fields for reduced Snellen. RS just makes so much sense."

"I hear you saying, Rochelle," Professor McIver began, "that the convenience and emotional comfort of using RS makes it the best choice for you. I get that, and many practitioners prefer it too for just those reasons. Also, people outside optometry, such as nurses, teachers, OTs, and primary care physicians, have a warm feeling for thinking they know what RS means. But what actually does RS mean? It only has psychometric validity if the chart is held at the prescribed distance, but in standardized recording of RS, there is no place to note the working distance. This is a trap into which many people fall: not ensuring the working distance is the one specified on the near VA chart. Sadly, I see this happening more often than not. Let me give you an example: I observed a post-op tech who was assigned to give a one-day cataract patient her acuity test with a near chart. The tech removed the surgical pressure patch, handed the patient the near VA chart, telling her to hold the chart where she saw it best, and read the lowest line she could. Well, the patient proceeded to hold the chart at about 20 cm and read off the 20/25 line. The tech then recorded 20/25 on the patient's chart. It was at this point that I interrupted and told the tech that was not the correct acuity to record, and she rebutted with that was how the surgeon taught her. Clearly, neither understood nVA. I sensed a teachable moment and asked the tech to read the top of the chart where it was written, 'To be used at 16 inches or 40 cm.' The ensuing corrected working distance yielded a nVA of 20/50 that made much more sense given the state of dry maculopathy the patient had in that eye."

"Oh, I see what you are saying," Rochelle said thoughtfully, "Unless I record the working distance with RS, no one could know the actual nVA. I see now that it is a potentially misleading nomenclature if we are using RS without recording working distance to take a baseline acuity or follow up on disease progression."

"You got it!" exclaimed Professor McIver. "RS is only valid if it is recorded with the actual working distance. If it is not recorded that way, the value of the RS should be discounted. There is no way of knowing the true VA unless we know actual working distance and actual size of the optotype. So, to summarize, RS is a good-news, bad-news situation, where the good news is that we have a high comfort level using it, but the bad news is that RS is a trap waiting to happen if we do not record working distance with it."

"Now, on to the next nVA system of nomenclature, the point system, also known as 'n'. Ahn, would you take the advocate role, please?" asked Professor McIver.

"Certainly, and thanks for assigning me this one," Ahn commenced. "The point system originated centuries ago, when physical type sets on printing presses were used. That means it has a long history of being used in and by printing presses, books, librarians, and publishers. A single point is 1/72", so it is an actual size, not like RS, where we do not know the actual size or working distance. Interestingly, in word processors, the font size is roughly equivalent to 'n', so in communications with teachers of the visually impaired or librarians, it would not be far off to equate font and point size."

"Very good, Ahn," Professor McIver interjected. "It is important to have an actual size when dealing with true VA, but that is only half the equation. What about the actual working distance, such as when someone records nVA as 10 point?"

"Not a problem; we just say, for example: 10 pt at 40 cm," Ahn proudly proclaimed. Now we have an actual working distance and an actual size. That makes the 'n' system better than RS, right?"

"Indeed, it does, Ahn," Professor McIver said, "but how does one record that to make comparison with dVA easy and quick, especially when you mix metric and imperial systems?"

"Oh," Ahn pondered. "I see what you mean. Creating a Snellen fraction would be awkward at best."

"Not to worry," Professor McIver assured him. "At least with an actual working distance and actual an size of the letter, we can eventually extrapolate a psychometrically valid nVA. For now, know that any 'n' measurement must also be recorded with the working distance.

"On to the J-system," Professor McIver said. "Jesse Johnson, I hear you are an advocate of using the J-system for nVA. Would you please explain to the group why you find it so compelling?"

"I got this," Jesse eagerly exclaimed, "but just call me J.J., please. "Jaeger has been used all over the world since the mid-19th century. Developed in 1867, originally a set of paragraphs of variable size. Nowadays, it is usually seen on a near-point card with single letters. All the surgeons I know use it, and electronic health records frequently have it in drop-down menus. That gives J-system the advantages of widespread use over many years."

"Nicely stated, J.J." Professor McIver complimented. "It is indeed a commonly used nVA system that has been around a very long time. Before I begin my

critique of the Jaeger system, do any of your classmates wish to chime in?"

"Yes, I would." Ahn interjected. "Much like the point system, just because a test has been around a long time does not mean it is valid or reliable."

Rochelle then jumped in with, "And I do not see any working distance recorded when any J number is recorded, like J1 or J2, and so on."

"Excellent, both of you!" Professor McIver asserted. "In addition, J size is not standardized between chart manufacturers, and there is no accounting for letter readability or stroke height commonality for letters that go above or below the line of text, so even if one records the working distance of a J nVA, one is unable to record a Snellen fraction with an actual letter size because the size is not known. Therefore, the J-system is useless for someone interested in test task equivalence or psychometric integrity."

"But why do so many people use the J-system in practice then?" J.J. asked, exasperated.

"For that answer," Professor McIver replied, "one has to look at psychosocial reasons, not psychometric ones. Think back to when we discussed 'The Source'. Recall the levels of confidence we have in each stage of evidence and the weight we assign to them. Also, recognize that the amount of material students and interns/residents must absorb inhibits them from drilling down to the foundation of each bit of new knowledge. Professors with stellar credentials and great respect for their work hold forth on the subjects of their expertise, and interns/residents memorize what they are told. Some of those interns/residents then go on to distinguished careers of their own and teach their students what they learned from their mentors, and so on, from generation to generation."

"But that is not the scientific method," Rochelle interjected, stating the thoughts of each of the students in the group. "It is more like quackery, methodology where one does not test the claims of the proponent."[9]

"Absolutely," Professor McIver agreed. "Quackery is a methodology that places the burden of proof on the skeptic, not the proponent, which is the exact opposite of the scientific method. You see its use much too often in crazy theories on the internet, which subsume the naive with what sounds like science. But, in fairness to the busy intern or resident in health care, there is not always enough time in the moment to fully test all the pronouncements of the sages. Still, we need to take the time to think critically and test the common canons we use on a regular basis. Regarding the three ways we have looked at so far for measuring and recording nVA, specifically reduced Snellen, the point system, and Jaeger, we find that they are all incomplete and potentially misleading. Clearly, they do not meet the standards that our psychometric integrity demands, nor do they allow us confidence in test task equivalence."

"Logan, may I ask you to tell us about the fourth system of measuring and recording nVA with its advantages and disadvantages?" Professor McIver said, turning to the fourth student in the group.

"Of course," Logan replied. "I must say that I love the M-Unit system. It uses letters with an actual size. By definition, a 1 M letter subtends 5 minutes of arc at 1 meter. Since it is metric, we can set up a rational Snellen fraction with the test distance in meters over the M letter size. This allows us to compare nVA with dVA to see if the results make sense. Also, the letters have been chosen for equivalent readability, further enhancing our level of confidence in the results."

"That sounds like the M-Unit system is a slam-dunk for becoming the standard for measuring and recording nVA," Professor McIver stated, "but are there any drawbacks to the M-Unit system?"

"None that I can see," Logan smugly replied. "It is clearly the best psychometric system for measuring and recording nVA."

"Hold on," J.J. cautioned after a thoughtful pause. "I learned today that psychometric advantage is not always what drives people, even the smartest ones. Psychosocial forces are powerful determinants of behavior and can at times override the scientific factors. While now I see that in a logical world, the standard for nVA would clearly be the M-Unit system, it is not used commonly in practice. Contemplating why it is not the worldwide standard leads me to believe that the inertia and habit of the psychosocial domain when using the other three nVA systems inhibits the widespread use of the most rational and soundest system in psychometric terms, M-Units."

"Well thought out and well stated, J.J.," Professor McIver congratulated. "Therefore, in order to break fully from what we have established are the inadequate and misleading nVA systems of reduced Snellen, the point system, and Jaeger, going forward, we will henceforth always record nVA in a Snellen fraction, with the numerator being the actual test distance in meters and the denominator as the actual size of the smallest letter read in M-Units. Further along, we will learn how to use logMAR thinking to convert imperial measurements to metric and create Snellen fractions for reduced Snellen and point measurements, as long as they are recorded with test distance."

Down for the Count

"The problem is never how to get new, innovative thoughts into your mind, but how to get the old ones out."

–Dee Hock

"Okay, Hugh," Professor McIver began the session, "having established that visual acuity exams were arbitrarily developed based on an employment test for apprentice astronomers hundreds of years ago, are you ready to delve into logMAR thinking and expand your practice horizons?"

"Actually, Professor McIver, I would like to hand this off to my classmate with the long name; we just call him Einstein as he seems to get things earlier than most of us. He is the one in our class who actually believes that this 'logMAR' thinking paradigm of yours might be promising."

Turning to the student nicknamed Einstein, Professor McIver quipped, "I am pleasantly surprised to hear that. Almost everyone who first hears of logMAR thinking initially has their eyes glaze over in flashbacks to high school math class, where logarithms initially confounded them before they slowly backed away to hide. Do please tell me why you think it has potential."

"Sure, it's pretty simple really: I just hate memorizing formulas and heard that this logMAR thinking is just about counting steps without having to memorize much. That's about it. I like things simple."

"You know, I don't like memorizing formulae either," Professor McIver said, smiling, "and the original Einstein is purported to have said not to memorize anything you could look up, and that is exactly what logMAR thinking is about: minimal memorizing and maximum impact. In fact, that is how the title of this little book came about! Low vision success with minimal memorizing; it's so Einsteinian.

"Now, if you can remember just a few simple principles and can count to ten, you too can be as successful with patients who have partial vision as you are with fully sighted patients. Let's start counting."

"Reviewing 'The Source' quickly," Professor McIver eagerly said, "we already know good sight has been arbitrarily though not unreasonably described as the ability to just recognize a letter that subtends a minimum angle of resolution of 1' arc. Also, Weber showed us that human sensory ability to detect change is a log progression of 0.1 log steps. Putting those two principles together is what Ian Bailey and Jan Lovie-Kitchin did to create the first rational visual acuity chart, the Bailey-Lovie Chart. Let's build one here to lock in those principles, and once we have that, we can proceed to unlock the power of logMAR thinking. I need another student to assist me. You, what is your name?"

"Well sir, my name is Edward Neville, but people just call me E. N."

"Makes sense," Professor McIver acknowledged. "If you are 'down for the count', we are going to begin at the beginning to build our logMAR chart; it really is a calculator just like slide rules of old in that one can do more complex math by just counting steps. Recalling that our starting point of good sight will be an MAR of 1' arc, and its attendant VA of 20/20, what is the log of the MAR?"

"Easy start," exclaimed E. N., "High school math says that the log of 1 would be zero!"

"Always good to start easy," Professor McIver smiled. "Now, knowing what Weber discovered, what would be the next reasonable size letter as a JND larger than 20/20's MAR of 1' arc?"

"Weber's Law says that a 0.1 log increase in size would yield a JND, and that would mean that starting from 20/20's logMAR of 0.0, we would add 0.1, which then equals 0.1 total. If we take the anti-log of 0.1, we get an MAR of ~1.25, and that yields a VA of 20/25," E. N. said confidently. That fits perfectly with our vision charts for fully sighted patients, so no big deal, right?"

"Perhaps," countered Professor McIver, "it's no big deal…yet. Let's keep building our logMAR chart/calculator. What is the next JND step, E. N.?"

"Let's do a bunch at once," E. N. suggested. "We would climb the logMAR scale 0.1 steps at a time, so: 0.2, 0.3, 0.4, 0.5, 0.6, 0.7, 0.8, 0.9, and 1.0. Taking their anti-logs gives us their MAR: 1.6, 2.0, 2.5, 3.2, 4.0, 5.0, 6.3, 8.0, and 10.

We can translate the MAR into visual acuity by putting '20' in the numerator and multiplying the MAR by 20, then inserting it into the denominator, like so: 20/32, 20/40, 20/50, 20/63, 20/80, 20/100, 20/125, 20/160 and 20/200."

"My name is Phoebe, and oh, my gosh!" she exclaimed. "I thought this logMAR thinking system was supposed to make low vision easier, and all I see is a lot of crisscrossing numbers that make my head spin."

"Hang on, Phoebe," Professor McIver interjected, "we just have to get past this hump to show why logMAR is so powerful, then we can begin using the paradigm to make clinical measures easier. Let's see if we can make a vertical table out of our numbers above to indicate how the logMAR chart/calculator can be used."

"I see it easier now," Phoebe began, "but it still just looks like a bunch of numbers on a regular acuity chart."

"They are!" Professor McIver said excitedly. "But because they are derived from Weber's Law, they have

LogMAR	MAR	VA
0.0	1.0	20/20
0.1	1.25	20/25
0.2	1.6	20/32
0.3	2.0	20/40
0.4	2.5	20/50
0.5	3.2	20/63
0.6	4.0	20/80
0.7	5.0	20/100
0.8	6.3	20/125
0.9	8.0	20/160
1.0	10.0	20/200
1.1	12.5	20/250
1.2	16.0	20/320
1.3	20.0	20/400

tremendous power if we just notice a few things and memorize a few constants. Then we can introduce predictive analysis and make clinical work easier. Remember that every single line or step is a just-noticeable difference, or JND, so every line is a level of significance different than the one above or below. That extends not just to visual acuity, but to dioptric power and telescopic power as well. A JND is a JND is a JND."

"Wait," E. N. said slowly, "I think I see a pattern…it seems that in the VA column, every three steps down, the denominator doubles. Oh, and same thing in the MAR column! But the numbers aren't exact sometimes. The 32 doubles to 63 and the 63 to 125."

"Yes, E. N., correct on both counts. The first is one of the few things I want you to memorize," Professor McIver commented. "Let's visualize that in the columns of our chart, starting with 20/20 and dropping down three lines, or JND steps. You can see that we land on 20/40. Three more lines/steps down, and we are at 20/80, then 20/160. I want you to memorize that: on a logMAR chart, every three steps either doubles or halves the value of the denominator, depending on the direction you go. The same pattern exists in the MAR column. Here it is highlighted to make it more visible. Regarding your second observation, E.N., for those cases that are not exact, it is because of rounding extra and unneeded digits to make the concept easier to use in clinic without affecting significance of the JND."

"You may recall me saying something about being able to count to ten." Professor McIver continued. "Well, you might also notice on the chart that not only does every three steps double or halve, but every ten

LogMAR	MAR	VA
0	1	20/20
0.1	**1.25**	20/25
0.2	1.6	20/32
0.3	2.0	20/40
0.4	**2.5**	20/50
0.5	3.2	20/63
0.6	4.0	20/80
0.7	**5.0**	20/100
0.8	6.3	20/125
0.9	8.0	20/160
1.0	**10.0**	20/200
1.1	12.5	20/250
1.2	16.0	20/320
1.3	20.0	20/400

steps move the decimal one place. This means that if you ever have to use the logMAR scale and it goes off the available numbers on the chart, you can just move the decimal to continue. We will work on that more later, but for now, memorize that ten steps move the decimal place."

"I see what you are saying now, but what good is it in practice?" Phoebe asked.

"Excellent question, and an even better segue, Phoebe," Professor McIver began. "We know that the discovery of logarithms and the subsequent development of the slide rule led to the ability to do complex calculations easily, and an explosion in the

LogMAR	MAR	VA
0.0	1.0	20/20
0.1	1.25	20/25
0.2	1.6	20/32
0.3	2.0	20/40
0.4	2.5	20/50
0.5	3.2	20/63
0.6	4.0	20/80
0.7	5.0	20/100
0.8	6.3	20/125
0.9	8.0	20/160
1.0	10.0	20/200
1.1	12.5	20/250
1.2	16.0	20/320
1.3	20.0	20/400

sciences took place, from architecture to navigation to physics, chemistry, construction, and myriad other disciplines. A slide rule even went to the moon on the Apollo missions in the 1970s. Our logMAR charts are just like slide rules in that they allow quick and accurate calculations: in this case, knowing what a patient will see based on a single good measurement of visual acuity. I call it predictive analysis, and it is what gives logMAR thinking its clinical power."

"Sounds intriguing," E. N. began hesitatingly, "but if it is so powerful and has been around since the 1970s, why isn't it the standard of low vision care?"

Pausing a bit before answering, Professor McIver said, "I too have wondered that, E.N., and it wasn't until I applied the social thinking of punctuated equilibrium that an answer began coalescing in my mind. I think that logMAR processing was just too disruptive in that it required a substantial front loading to change paradigms before benefits would be visible, and people already in the field were satisfied with their own models while teaching them to their students. That is not to say that their ways were wrong or bad, but logMAR thinking allows more degrees of freedom, fewer assumptions, and makes formula-free practice possible. Whatever the reason for its non-dominant position today, I want to present to you predictive analysis and let you be the judge of its utility for when you are in practice. Let's begin."

"There are only four parts to predictive analysis:

1. Measure and record VA with actual letter size and actual test distance, using consistent units.
2. Set a VA goal for improvement to meet the patient's needs.
3. Count the steps on the logMAR chart between measured and goal.
4. Implement with working distance and dioptric power for near or telescope power for distance."

"Let's look at the parts one at a time in sequence," Professor McIver said. "First is measure and record VA. Sounds easy, right? But we have seen how inappropriate and sloppy measurements, and old but commonly used systems, can lead to psychometrically invalid results. In order to maximize the power of logMAR thinking properly, it is best to use M-Units for letter size and meters for test distance: actual letter size and actual test distance. Once we have a psychometrically valid visual acuity, we ask ourselves whether that is good enough for the patient's needs. If the answer is no, then we need to set a goal for the letter size that will meet the patient's needs. For our purposes here, we will be dealing with near point reading distances, though soon enough, we will see how the same principles can be applied to distance with telescopes. Always keep in mind that we are looking at JND steps on the logMAR charts."

"How about an example, Professor McIver?" Remi asked from the front of the room. "This sounds fascinating, but I don't see how to apply it."

"Great prompt, Remi," Professor McIver said. "Everyone get out their logMAR near point card and follow along. Ready? Okay, for part one of predictive analysis, let's say a patient wants to read regular size printed material, which we have already established as 1 M, but can only read 1.6 M size letter at 40 cm with their current +2.50 D add. We record the acuity as 0.4 m/1.6 M.

Now, part two: we ask with our inner voice, 'Is that good enough?' Since the patient has reduced visual acuity, the answer usually is of course not. For this example, let's set our goal at 1.0 M print size since that is what the patient expressed as their reading need.

Part three says that we count the JND steps between measured nVA and goal nVA, which in this case is two steps: 1.6 M the measured—>1.0 M the goal. Now comes the fun part of predictive analysis. Since we need two JNDs of improvement to get from the measured VA to the goal VA, we need two JNDs

closer working distance and two JNDs of higher dioptric power."

"What do you mean about JNDs of working distance and dioptric power?" E. N. asked.

Professor McIver pointed to the logMAR chart/calculator and said, "Every step between lines is a JND, and it doesn't matter if the JND is an acuity, a working distance, or a dioptric power; a JND is a JND is a JND. When we want to improve acuity to a desired goal from the measured baseline, we have to reduce working distance and increase dioptric power by the same number of JNDs as is the number of steps from measured to goal. In our example above, we know the patient is reading at 40 cm. By finding 40 on the chart, we see two steps smaller for a closer working distance is 25 cm. So, we must move the chart to 25 cm for the patient to see it. However, since most of our low vision patients are presbyopic, we also need to adjust the add power upwards. The dioptric equivalent of the original working distance is 2.50 D. Making that higher by the two JND steps necessary takes us to 4.00 D. If you then put in a 4.00 D add and hold the chart at 25 cm, all other things being equal, the patient will be able to read 1.0 M. No calculations, no formula, no guessing; just using the slide rule-like power of the logMAR chart's progression unleashes the power of predictive analysis."

"That is so unexpectedly easy," Remi stated. "All we have to do is measure properly, set a goal, count the steps between measured and goal, then implement based on JND. Are you saying that now I can just use predictive analysis for my low vision patients and not be so nervous?"

"Yes, Remi, it is easy, but most people I have encountered over the years try to make it more difficult or think it is some kind of 'voodoo'," Professor McIver said. "Just a few simple steps takes you from current VA to goal-meeting without fuss. The key is to do the parts in order, and since there are only four parts, it is simple to remember."

"What about distance vision?" E. N. interjected. "Many patients with partial vision want to see better for travel, television, or other distance tasks like driving. How do we help them when distance is, by definition, zero diopters?"

"Great question, E. N." Professor McIver said, turning toward him. "The same principles apply, except instead of diopters, we use telescopic power or times (x). Follow me on this: when we have fully corrected a patient to their best distance visual acuity, what is the telescopic power in play? Anyone? Trick question, actually, as it involves the multiplicative identity of anything times one is itself. Therefore, our fully corrected patient in our exam chair is using their natural telescope power of 'one'. We still take best visual acuity with actual letter size and actual test distance, part one of predictive analysis, and if it is not good enough, we set a goal, which is part two, before counting the steps between our measurement acuity and goal acuity, that being our part three. Then, for part four, we implement it, but instead of starting at the working distance or diopters, we start at 1.0, which is the patient's incoming telescopic power, and count up the chart. Here is an example: a patient sees 20/100 but wants to see 20/40, the common visual acuity needed for obtaining a driver's license. From the logMAR chart/calculator, we see that it takes four JND steps to get from 20/100 to 20/40. Now, go to 1.0 on your logMAR chart/calculator and count up four steps: 1.0—>1.25—>1.6—>2.0—>2.5, and there it is, we need a 2.5 x telescope to get our example patient to see their goal. Again, no formulae, no calculations, no guessing and no voodoo. By the way, the most common error here is to mistake telescopic power of x for dioptric power or 'D'; don't fall into that trap."

"My goodness, Professor McIver, that is a lot to take in," Hugh slowly said. "No wonder it hasn't caught on."

"I get your point, Hugh," Professor McIver said understandingly. "But I believe that trying to memorize reams of formulae that have many assumptions built in is really the harder path. Once you recognize the power and simplicity of logMAR thinking and predictive analysis, and how they free you from formulae, calculating, and guessing, I think you will wonder how you practiced low vision without it. All you have to do is practice with predictive analysis and become familiar with it in order to appreciate its utility and power."

"That's it for now."

Another Added 'Bennettefit'

"Science advances by the relentless examination of the commonplace; that some of its greatest discoveries have been made through fascination with what other men have regarded as not worthy of note."
–Jules Henry

"Well, if it isn't Logan Mars," Professor McIver said, looking up from his desk during office hours. "Please come in. Is something on your mind?"

"Good afternoon, Professor McIver," Logan began. "I am intrigued by the applications of logMAR thinking and predictive analysis and see how they can rationalize my approach to low vision exams, but

it seems there must be more to it. I mean, I see how to use the logMAR chart/calculator to quickly get the appropriate add to meet patient's reading goals and also to use it for choosing telescope power for distance, but not everyone uses M-Units or metric distances. What happens then?"

"Very good observation, Logan. I have planned that for today's lecture and workshop. Let's get to the lecture hall and address your questions with the whole class," Professor McIver said. "Walk with me."

"Logan brought a good question to me during office hours," Professor McIver said, opening class time. "He is interested in going beyond the basics of logMAR thinking and predictive analysis, especially regarding non-metric measurements such as reduced Snellen acuity measurements or the 'n' (point) system. Let's jump in. First, there are another few things for you to memorize."

"Wait, Professor McIver, you said we didn't have to memorize stuff with LogMAR thinking," protested Karen. "Now you say we need to memorize more. That sounds like a bait and switch."

"Karen, what I said was you don't have to memorize as much," countered Professor McIver. "Besides, it is on the logMAR near chart/calculator if you forget something, so memorizing just helps you along but is not essential. Let's look at what I mean. Everyone, pull out your handy near logMAR chart and note what it says at the top: 16 inches or 40 cm. Now notice that those are numbers on the chart that are four steps apart. Now you can easily transpose inches to cm as they are a constant ratio like the logMAR chart/calculator! Since the number of cm is always larger than the number of inches for the same distance, you always know which way to go on the logMAR chart. For instance, 10 inches is 25 cm. Therefore, if someone tells you they measured a near acuity at 10 inches, you know the numerator of the reduced Snellen fraction is 0.25 m."

"That is useful, alright," Karen conceded, "but that only gets us the numerator. What about the denominator in cases where the reported near visual acuity is in reduced Snellen or point?"

"Let's solve for that now!" Professor McIver exclaimed, happy for the lead-in to the next part. "look at your near point logMAR chart and find the 1.0 M size row. Got it? Ok, now look across the row, and you should see 20/50. If someone reports a reduced Snellen acuity of 20/50, you know the letter size was 1.0 M; that is your denominator. Now, hopefully, they also record a working distance. If that is 40 cm, then your actual psychometrically accurate reduced Snellen fraction would be: 0.4 m/1.0 M. If, however, the working distance is reported as 10 inches, you can quickly count up four steps to 25 cm and record the VA as: 0.25 m/1.0 M."

"Now, the point system uses an actual size (1 pt = 1/72 inch), and you have to memorize this: 8 pt = 1.0 M, but once you have that, you can see they are only separated by a single step with a decimal place moved. To transpose from point to M-Units, you just go one step up the chart and move the decimal to the left. Also, you recall from above that since 1.0 M = 20/50 (on a standard logMAR near chart), then 8 pt = 1.0 M = 20/50; another of the few things useful to memorize.

"So, if a near acuity is reported as being 20/100 at 10 inches, in the past you might have been flummoxed as to what the near visual acuity actually is, but now, using those minimal memorized factors and the power of the logMAR chart/calculator, you can determine the actual working distance as 25 cm and the actual size as 2.0 M, giving you a psychometrically valid reduced Snellen fraction of 0.25 m/2.0 M."

"Is that all you can do with logMAR thinking and predictive analysis?" came the smart aleck anonymous voice from the back of the classroom.

"While sarcasm has not incorrectly been described as an example of intelligence poorly used," Professor McIver retorted, "there is another useful bit of logMAR magic you might find charming. In my travels, it has come in quite handy. Specifically, one can translate miles to kilometers and back, as they are separated by only two steps on the logMAR chart/calculator; i.e., 100 km = 63 miles or 40 kilometers = 25 miles. This is especially good to know when one has a speed limit to mind in a foreign land."

"Is that it?" came the plaintive voice again. "Is that all that you have for us?"

"As a matter of fact, no, there is another added logMAR 'Bennettefit' that is something you might find much more useful in clinical practice than just miles to kilometers and back," Professor McIver again countered. "Reciprocals of diopters and meters are built into our logMAR chart/calculator. Once again, take another look at your near point logMAR chart/calculator and find the 1.0 M. Got it? Okay, now go one step up to find 1.25 and one step down from 1.0 to find 0.8; those are reciprocals. 8.00 D focuses at 12.5 cm, and 1.25 D focuses at 80 cm. Now, return to 1.0 and count three steps up from there to find 2.0, then three steps down from 1.0 to see 0.5: 5.00 D focuses at 20 cm and 2.00 D focuses at 50 cm. Quickly you can see that reciprocals are symmetric about 1.0! No more

"That's enough added 'Bennettefits' for today," Professor McIver said, glancing at the back of the room for any more questions on the power of logMAR thinking or the logMAR chart/calculator. "Okay, if there's nothing else, dismissed."

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Article • The Extensive Relationship Between Eyes and Mind: Understanding the Psychology Behind Your Vision

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ABSTRACT

The perception of sight, commonly regarded as a direct and objective interpretation of reality, is, in fact, a deeply subjective process actively modulated by an individual's psychological state. This article explores the burgeoning field of psychosomatic ophthalmology, which investigates the profound impact of human psychology on visual health and perception. It synthesizes evidence from ophthalmology, neuroscience, and psychology to argue for a holistic, mind-body approach to eye care.

The article details the neurobiological pathways through which psychological distress, particularly chronic stress and anxiety, adversely affects ocular health. It elaborates on the role of the hypothalamic-pituitary-adrenal (HPA) axis and the sustained release of cortisol, which can impair blood flow, increase inflammation, and disrupt the blood-retinal barrier. These mechanisms are discussed as contributing factors in the progression of sight-threatening diseases such as glaucoma, age-related macular degeneration (AMD), diabetic retinopathy, and dry eye disease.

Furthermore, the review examines how transient emotional states and cognitive processes like attention actively shape visual perception. Evidence is presented demonstrating that mood can alter the perceived saturation of color, while attentional biases, influenced by fears and desires, can change how we interpret visual scenes and even perceive physical space. The influence of stable personality traits, such as neuroticism and optimism, on perceptual biases and long-term visual health outcomes is also considered.

Keywords: brain**,** eye psychology, perception, visual psychology

Introduction

The act of seeing, for most of us, feels effortless and immediate. We open our eyes, and the world simply appears, a faithful, high-definition stream of reality delivered directly to our consciousness. But this perception of an unvarnished truth is a masterfully constructed illusion. A growing and compelling body of scientific evidence reveals that our vision is not a passive reception of light, but an active, dynamic process deeply intertwined with our psychological landscape. The intricate workings of our minds—our stress levels, emotional states, attentional focus, and even our core personality traits—profoundly shape, tint, and, at times, fundamentally transform what we see. This emerging understanding, situated at the crossroads of both optometry and ophthalmology and its inter-disciplinary relations to neuroscience and the subject of psychology itself, is ushering in a new era of "psychosomatic ophthalmology," demanding a more holistic and integrated approach to preserving the precious gift of sight.[1]

The term psychosomatic ophthalmology was first mentioned in the literature in 1947 by Harrington.[2] Interestingly, there are approximately 20 articles published on this topic up to 2012, and from 2012 to 2025, approximately 325 articles were found on online databases like Pubmed and the Cohrane Library.

The Stressed-Out Synapse: Neurobiological Pathways from Mind to Eye

The connection between psychological distress and vision is not merely circumstantial; it is engraved into our very neurobiology. When we experience chronic stress or anxiety, our body's ancient

survival mechanisms are thrown into overdrive. The hypothalamic-pituitary-adrenal (HPA) axis, our central stress response system, floods the body with glucocorticoids, most notably cortisol.[3] While essential for short-term survival, sustained high levels of cortisol can be profoundly damaging to the delicate and highly vascularized structures of the eye. This stress-induced hormonal cascade can impair blood flow to the retina and optic nerve, increase inflammation, and disrupt the function of the blood-retinal barrier, a critical structure that protects the eye's sensitive neural tissue.[1]

This process is a key suspect in the progression of several sight-threatening conditions. In glaucoma, a disease characterized by damage to the optic nerve, elevated cortisol has been shown to correlate with increased intraocular pressure, a primary risk factor for the disease.[4] The autonomic nervous system, perpetually in a "fight-or-flight" state under chronic stress, further constricts blood vessels, potentially starving the optic nerve of vital oxygen and nutrients and accelerating nerve fiber degeneration.[5]

Stress impacts more than glaucoma.[6] Research links stress and depression to increased risk of advancing from dry to wet age-related macular degeneration, which involves new, abnormal blood vessel growth.[7] Stress-driven inflammation is likely key in this shift. In diabetic retinopathy, psychological distress can worsen the disease by harming glycemic control and causing inflammation that damages retinal vessels.[8] Even dry eye discomfort rises with stress and anxiety, likely through changes in tears and surface inflammation.

Many studies have also suggested potential correlations between psychological stress,[9] psychoneuroimmunology,[10] and female hormones,[11] all of which might directly or indirectly affect vision and visual function.

The Colors of Emotion and the Focus of Attention

Beyond the progression of disease, our psychological state can alter the very fabric of our moment-to-moment visual experience. Our emotions, for instance, act as a filter, subtly tinting our perception of the world. Pioneering studies have demonstrated that sadness can genuinely dull our perception of color, making the world appear more gray and less vibrant. This phenomenon, which has been linked to alterations in the brain's processing of chromatic information along the blue-yellow axis, suggests that our emotional state can directly modulate the neural signals that constitute our experience of color.[8] Conversely, a positive mood can enhance the saturation of colors, making them appear richer and more vivid, a perceptual echo of our inner joy.[12]

This subjective experience is powerfully shaped by the cognitive process of attention. Our visual system is constantly inundated with an overwhelming amount of information, far more than we can consciously process. Attention acts as a crucial gatekeeper, a spotlight that selects and prioritizes information deemed relevant by our brain.[12] This selection is not always a conscious choice; it is heavily influenced by our goals, fears, and desires. An individual with arachnophobia, for example, will have an attentional system primed to detect the faintest hint of a spider's leg in a cluttered room, often spotting it before anyone else.[13]

This "motivated perception" extends beyond our fears. In a fascinating study, researchers found that desired objects, such as a bottle of water to a thirsty person, are perceived as being physically closer than less-desired objects, a phenomenon dubbed "wishful seeing."[14] Our minds, it seems, bend the rules of visual space to bring our goals within perceived reach. This highlights a critical truth: we do not see the world as it is, but as it is useful for us to see it. This has profound implications for everything from eyewitness testimony, where emotional trauma can drastically alter the memory of what was seen, to the everyday interpretation of ambiguous social cues.

The Anxious Eye and the Optimist's Gaze

Our deep-seated personality traits also cast a long shadow over our visual world. Individuals high in neuroticism, a trait characterized by a tendency toward anxiety, worry, and negative emotions, are more likely to interpret neutral or ambiguous visual stimuli in a threatening way.[15] Their perceptual systems are, in a sense, biased toward detecting danger, a trait that may be evolutionarily advantageous but can contribute to a state of chronic hypervigilance and stress.

In contrast, optimism appears to confer a degree of perceptual resilience. Optimists are more likely to direct their attention away from negative or threatening images and toward positive ones. This attentional bias may not only contribute to their emotional well-being but could also have long-term benefits for physical health, potentially by mitigating the physiological cascade of the stress response.[16]

While more research is needed, it is plausible that an optimistic disposition could be a protective factor against the psychosomatic components of eye diseases.

Healing the Mind to Heal the Eye: A New Therapeutic Frontier

The profound connection between mind and vision opens up a new and vital therapeutic frontier. If psychological distress can cause and exacerbate eye disease, then psychological interventions should be a core component of comprehensive eye care. This moves beyond simply acknowledging the emotional burden of vision loss and toward actively using psychological tools to protect and improve visual health.

Mindfulness-based stress reduction (MBSR), a structured program that teaches patients to cultivate present-moment awareness, has shown remarkable promise. In a randomized controlled trial involving patients with glaucoma, an eight-week MBSR program resulted in significant reductions in intraocular pressure and lower levels of stress biomarkers, such as cortisol and pro-inflammatory cytokines, compared to a control group.[17] These findings provide powerful evidence that training the mind can induce measurable physiological changes in the eye.

Cognitive behavioral therapy (CBT) is another powerful tool. CBT helps individuals identify and reframe the negative thought patterns and behaviors that contribute to stress and anxiety. For a patient struggling with the diagnosis of a progressive eye disease, CBT can help them manage their fears, improve coping strategies, and reduce the very stress that might be accelerating their condition.[18] Biofeedback techniques, which allow patients to gain conscious control over physiological processes like muscle tension and heart rate, can also be employed to help patients manage the physical manifestations of anxiety that impact vision.[5]

The future of both optometry and ophthalmology lies in embracing this integrated mind-body model. It calls for greater collaboration between eye care professionals and mental health specialists, mainly with psychologists and therapists. It means that screening for depression and anxiety in all optometry and ophthalmology clinics should become as routine as measuring intraocular pressure. It suggests that prescribing stress-management techniques alongside eye drops is not alternative medicine, but evidence-based, personalized care, and can lead to very satisfied patients.[19]

The journey of a photon from an object to the back of our retina is only the first step in the complex miracle of sight. The final image we perceive is a collaborative masterpiece, co-authored by our eyes and our minds. By understanding and respecting the profound influence of our psychology—from the neurochemical storms of stress to the subtle biases of our personality—we can move beyond a purely mechanistic view of vision. We can begin treating the whole person, fostering not only healthier eyes but also a calmer, more resilient mind capable of seeing the world in its fullest, most vibrant light. There is a strong need for an interdisciplinary approach to the treatment of these patients, and eye care professionals should play a leading role.

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Article • A Comparative Cross-sectional Study on Amblyopia and its Relationship with Retinal Nerve Fiber Layer Thickness

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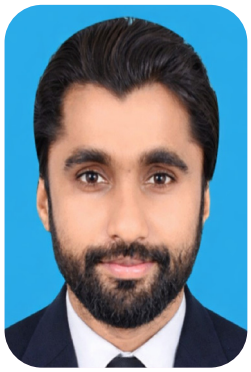

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ABSTRACT

Background: Amblyopia affects approximately 1.75% of the world's population. Atrophic alterations in the lateral geniculate nucleus have been observed in amblyopic eyes, leading to the hypothesis that retinal nerve fiber layer (RNFL) thickening is caused by a limitation of normal postnatal ganglionic cell reduction. This study was conducted to determine and compare the thickness of the RNFL in all types of amblyopic and non-amblyopic eyes.

Methods: A comparative cross-sectional study was conducted at Al-Shifa Trust Eye Hospital Rawalpindi over six months. Data were collected from 70 patients (140 eyes) using a clinical structured questionnaire. RNFL thickness was measured using optical coherence tomography. All amblyopic patients were included in this study. A one-way ANOVA and paired sample t-test were applied to compare the RNFL thickness between amblyopic and non-amblyopic eyes.

Results: The mean age of the patients was 10.20±2.811, ranging from 5 to 16 years. The mean thickness of RNFL globally in strabismic amblyopia was 108.1±14.1, whereas in anisometropic and bilateral refractive amblyopia, the RNFL thickness was 105.74±11.042 and 107.84±12.329, respectively. There was no statistical mean difference between thicknesses of strabismus, anisometropic, and bilateral amblyopia. There was no statistically significant difference found between the thicknesses among all quadrants of anisometropic amblyopia.

Conclusion: There was no significant difference in RNFL thickness between different types of amblyopia. Similarly, there was no significant difference in the thickness of RNFL in amblyopic and non-amblyopic eyes.

Keywords: amblyopia, optical coherence tomography, retinal nerve fiber thickness

Introduction

An estimated 285 million people around the world are visually impaired; nineteen million are children below the age of 14 years. Refractive errors, which are the most common cause of vision impairment in children, affect about 43% of this population.[1] In 2020, 16 million in Pakistan suffered distance vision loss, while over 10 million suffered from near-sightedness.[2] The prevalence of amblyopia is 1% to 5% globally, while Asians have a 3% prevalence.[3]

Refractive errors and amblyopia are the most common reasons for visual loss in children.[4] Amblyopia, which comes from the Greek phrase "dullness of vision," is the leading cause of vision loss in infants.[5] Amblyopia is a neurodevelopmental and neurophysiological ocular condition that causes poor vision but has no associated pathology. It can be treated most effectively if started at an early stage.[6]

However, recent research indicates that older patients may benefit from treatment since the visual system's plasticity persists into adulthood. Additionally, children and adults with amblyopia may benefit from treatment that focuses on the suppressive interactions within the visual cortex to enhance both binocular and monocular visual performance.[3]

Amblyopia is defined by diminished visual acuity in one or both eyes as a result of visual deprivation or aberrant interaction during vision development.[7] There are three main types: strabismic, anisometropic, and deprivation.[8,9] Atrophic alterations in the lateral geniculate nucleus have been observed in amblyopic eyes, leading to the hypothesis that retinal nerve fiber layer (RNFL) thickening is caused by a limitation of normal postnatal ganglionic cell reduction.[10] Between the inner limiting membrane and the retinal ganglion cell layer, the RNFL is densely packed. It is made up of ganglion cell axons, astrocytes, retinal arteries, and Müller cell processes, constituting the inner limiting membrane, which covers the nerve fiber layer's surface.[11] Between roughly 18 and 30 weeks of gestation, the total population of cells in the ganglion cell layer in humans reaches its peak (2.2-2.5 million cells). The cell number rapidly decreases to 1.5 to 1.7 million cells. In the adult human optic nerve, there are 1.1 million axons.[12] The RNFL thickness continues to diminish with advancing age.

The assessment of the RNFL is of high significance for the identification of glaucoma, optic nerve abnormalities, and other diseases. Optical coherence tomography (OCT) can be used to assess RNFL.[13] If amblyopia affects the process of postnatal reduction of ganglionic cells, RNFL thickness may be affected. Abnormal RNFL could lead to other diseases, such as glaucoma, optic neuropathies, and optic neuritis. Therefore, the purpose of this study was to find an association between amblyopia and RNFL thickness, as there are conflicting results in previous studies, and no such study has been conducted in Pakistan. This study will help us to determine the relationship between RNFL and amblyopia.

Methods

This study was conducted at the Orthoptics Department of Al-Shifa Trust Eye Hospital, Rawalpindi, a tertiary eye care facility. This comparative cross-sectional study included patients of both genders, ages 5 to 16 years, with amblyopia. This study included strabismic and refractive (anisometropic, isometropic, and meridional) amblyopia, as well as unilateral and bilateral amblyopia. Unilateral amblyopia was characterized as a ≥two-line difference in best VA, when <20/30 in the poorer seeing eye and with presence of past or current strabismus and/or anisometropia. Bilateral amblyopia was characterized as best-corrected visual acuity (BCVA) in the two eyes <20/40 within the sight of factors that aggravated the amblyopia.[14] All procedures and methods of data collection were conducted following the Declaration of Helsinki.

Sample size

The sample size of this study was calculated by using the WHO calculator at the confidence interval of 95%. The power of the test was 90%, and the population standard deviation was 16.2. The test value of the population mean was 107.2, and the anticipated population mean of the pilot study was 113.8. The sample size was 64. Seventy patients were included for this study.

Sample selection criteria

All types of amblyopia and both genders were included. Patients with any ocular pathology, such as glaucoma, optic nerve disease, cataract, or macular disease, as well as those not willing to participate, were excluded from the study.

Ethical consideration

Approval was given by the Ethical Review Committee at Al-Shifa Trust Eye Hospital (ERC-71/AST-21) and by the head of the department of the hospital before data collection. Moreover, verbal informed consent was also taken from every individual before they participated in this study. Face and content validity was checked by circulating it to experts in the field. Confidentiality of the patient's data was maintained, and ethical values of research were properly considered and followed at every step of the study.

Data collection procedure

Data collection for the study was done at the Paediatric and Diagnostic Department of Al-Shifa Trust Eye Hospital, Rawalpindi. By using the universal non-probability sampling technique, data was collected from the patients for six months, from October 2021 to March 2022. This was done to ensure the inclusion of all patients fulfilling the study criteria during the study duration, as it would allow a more accurate analysis of the variables. A clinical Performa was used,

which was based on the previous studies and clinical findings. The first part of the Performa consisted of socio-demographic characteristics; the second part contained ocular parameters such as visual acuity and objective refraction. The last part consisted of RNFL thickness, including nasal, temporal, supra-nasal, supra-temporal, intranasal, and infra-temporal, which was measured by OCT.

The responses recorded in the Performa were recorded and analyzed using Statistical Package for social sciences (SPSS) version 21. The descriptive analysis was done on the categorical and continuous variables. Percentages and frequencies were reported for categorical variables, and mean and standard deviation were recorded for continuous variables. As the data were normally distributed, a parametric approach was used during inferential analysis. In inferential analysis, all statistical tests used a confidence interval of 95%, and a p-value of <0.05 was considered significant. Therefore, one-way ANOVA and paired sample t-test were used to compare the mean difference of RNFL thickness in different types of amblyopia and to compare the RNFL thickness of amblyopic and non-amblyopic eyes.

Results

A total of 140 eyes of 70 patients were included in the study. The mean age of participants included in the study was 10.20 (+/-2.811), ranging from 5 to 16 years. Both genders were included in the study, out of which males outnumbered females. Out of the total, 37 patients were from urban areas. Treatment included: 34 (48.6%) were using only glasses, while 36 (51.4%) were using both glasses and patching therapy (Table 1).

Table 1. Socio-Demographic Characteristics

Socio-Demographic Characteristics	Frequency	Percentage (%)
Gender		
Male	40	57.1
Female	30	42.9
Area of residence		
Urban	37	52.9
Rural	33	47.1
Other activities		
Outdoor activities	44	62.9
Indoor activities	26	37.9
Refractive error		
Myopia	14	20.0
Hyperopia	18	25.7
Astigmatism	38	54.3
Treatment		
Glasses only	34	48.6
Glasses with patching	36	51.4

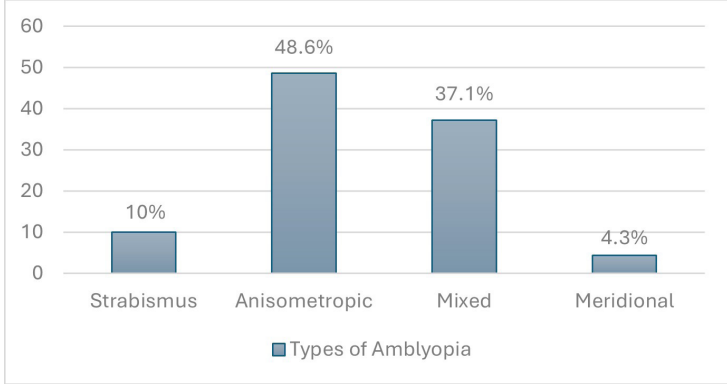

Figure 1. Types of amblyopia

Table 2. Ocular Parameters

Variables	N	Mean	Standard Deviation
BCVA in Amblyopic Eyes	95	0.81	0.570
BCVA in Non-Amblyopic Eyes	45	0.12	0.145

A majority of the patients (54; 77.1%) presented with a family history of amblyopia (Figure 1). The mean and standard deviation of best-corrected visual acuity were 0.81±0.570 logMAR in the amblyopic eyes, whereas the best-corrected visual acuity in the non-amblyopic eyes was 0.12±0.145 logMAR (Table 2). The most common presenting complaint was blurred vision (74.3%), as shown in Figure 2.

The one-way ANOVA was used to compare the mean difference in RNFL thickness among different types of amblyopia. There was no statistically significant difference between the various types of amblyopia (Table 3). The paired sample t-test was used to compare the RNFL thickness of the amblyopic and non-amblyopic eyes. Although the amblyopic RNFL was thicker than that of non-amblyopic eyes, no statistically significant difference was found (Table 4).

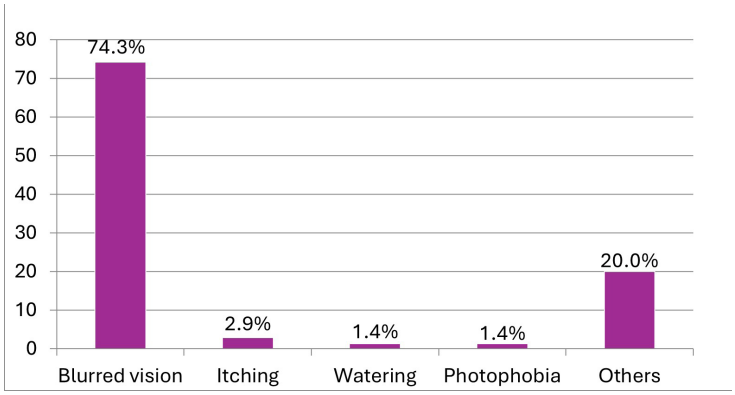

Figure 2. Chief complaints of amblyopic patients

Table 3. RNFL Thickness in Different Types of Amblyopia

RNFL Thickness	Strabismic Amblyopia		Anisometropic Amblyopia		Bilateral Amblyopia		Meridional Amblyopia		F	P-value
	Mean	SD	Mean	SD	Mean	SD	Mean	SD		
Nasal Quadrant	80.82	12.17	85.82	20.77	94.65	24.46	81.20	20.42	1.792	0.15
Temporal Quadrant	69.88	9.84	75.67	14.53	68.09	13.61	75.20	9.83	2.266	0.86
Supra-Temporal Quadrant	132.63	20.56	135.38	16.47	133.84	21.84	133.60	11.78	0.071	0.97
Supra-Nasal Quadrant	142.38	25.66	121.05	30.23	122.23	27.53	125.80	23.67	1.313	0.27
Infero-Nasal Quadrant	150.13	27.28	126.18	28.52	136.37	25.44	148.00	50.79	2.343	0.078
Infero-Temporal Quadrant	139.75	23.87	143.92	17.16	145.07	26.05	140.60	10.04	0.173	0.915
Globally	108.13	14.11	105.74	11.04	107.84	12.32	105.80	12.65	0.251	0.860

Table 4. RNFL Thickness Between Amblyopic and Non-Amblyopic Eyes

RNFL Thickness	Amblyopic Eyes		Non-amblyopic Eyes		T	P-value
	Mean	SD	Mean	SD		
Nasal Quadrant	86.89	18.70	82.80	17.46	-1.275	0.209
Temporal Quadrant	75.87	14.17	74.27	10.54	-0.949	0.348
Supra-Temporal Quadrant	140.89	19.21	138.81	18.73	-0.791	0.433
Supra-Nasal Quadrant	132.57	27.07	129.47	27.85	-0.970	0.337
Infero-Nasal Quadrant	139.13	32.17	136.87	32.27	-0.750	0.457
Infero-Temporal Quadrant	147.18	22.15	146.11	20.74	-0.337	0.737
Globally	105.22	23.64	108.07	10.11	-0.877	0.385

The one-way ANOVA was used to compare the mean difference in RNFL thickness among different types of refractive errors. There was no statistically significant difference between various types of refractive error in all quadrants except supra-nasal. RNFL thickness for the supra-nasal quadrant in hyperopes was thicker amongst the refractive errors given (Table 5). The one-way ANOVA was also used to compare the mean difference in RNFL thickness among amblyopic patients with and without using patching therapy. No statistically significant difference was found in all quadrants, as shown in Table 6.

Discussion

The main objective of this study was to determine the RNFL thickness and compare it between different types of amblyopia and fellow non-amblyopic eyes. In the present study, the mean RNFL thickness was not statistically significantly different between amblyopic and non-amblyopic eyes, which is in accordance with the study conducted by Kee et al.,[15] in which the thicknesses of the superior, inferior, nasal, and temporal quadrants of the retinal nerve fiber layer between normal children and children with amblyopia were also not statistically significant. In a study by Yen et al.,[16] the difference in RNFL thickness between strabismic amblyopic eyes and fellow eyes did not reach statistical significance, so it was concluded that amblyopia is not linked with RNFL thickness.

In thirty individuals with aniso-hypermetropic amblyopia, Yoon et al.[17] used the OCT to evaluate both peripapillary and macular RNFL thickness. They found that anisometropic amblyopic eyes have thicker peripapillary RNFL than normal eyes. Because of the tiny size of the eye, those with high hypermetropia may have thicker RNFL, and the apparently increased thickness of RNFL may be attributable to this structural alteration in the eye rather than the effect of amblyopia.

Table 5. RNFL Thickness in Different Types of Amblyopia

RNFL Thickness	Myopic Amblyopia		Hypermetropic Amblyopia		Astigmatic Amblyopia		F	P-value
	Mean	SD	Mean	SD	Mean	SD		
Nasal Quadrant	91.07	30.78	94.11	22.202	89.08	18.720	0.305	0.738
Temporal Quadrant	72.67	11.98	70.39	18.449	69.32	13.973	0.274	0.761
Supra-Temporal Quadrant	140.60	23.874	132.89	18.739	130.47	15.305	1.662	0.197
Supra-Nasal Quadrant	103.93	22.864	134.50	29.159	122.03	25.251	5.756	**0.005**
Infero-Nasal Quadrant	127.80	26.138	137.17	26.320	136.74	30.674	0.591	0.556
Infero-Temporal Quadrant	146.80	13.241	138.72	22.533	143.18	25.628	0.524	0.595
Globally	106.07	12.157	108.17	14.738	103.13	18.425	0.613	0.545

Table 6. RNFL Thickness Between Amblyopic and Non-Amblyopic Eyes

RNFL Thickness	Glasses without patching		Glasses with patching		T	P-value
	Mean	SD	Mean	SD		
Nasal Quadrant	90.66	23.551	90.89	21.423	0.002	0.966
Temporal Quadrant	73.09	15.779	67.58	13.244	2.539	0.116
Supra-Temporal Quadrant	136.11	19.165	130.42	17.451	1.717	0.194
Supra-Nasal Quadrant	124.60	24.997	118.22	29.798	0.952	0.333
Infero-Nasal Quadrant	132.60	28.907	137.25	28.454	0.467	0.497
Infero-Temporal Quadrant	140.40	18.820	145.17	25.905	0.783	0.379
Globally	106.91	10.430	103.19	20.499	0.921	0.341

Another study conducted by Chen et al.[18] used Spectral Domain (SD)-OCT to compare the thickness of the macula and RNFL in children with anisometropic amblyopia. They discovered that the average thickness of the outer macular ring and RNFL was substantially thicker in anisometropic amblyopic eyes than in normal eyes. Furthermore, the macular thickness of both treated amblyopia and untreated amblyopia were the same. They observed that macular and RNFL thicknesses appeared to be more strongly linked with axial length and refraction variations than with amblyopic development.

Atsushi Miki and Motohiro Shirakashi[10] investigated 26 individuals with persistent unilateral amblyopia and 25 patients with treated unilateral amblyopia. The average RNFL thickness for the consistently amblyopic eyes was 105.5±14.0 µm. This value did not differ substantially from that of the normal eyes (105.2±13.0 µm) or the treated amblyopic eyes (107.1±11.7 µm). Hence, this study also proved that RNFL thickness is not associated with amblyopia.

Walker and Rubab[12] studied thirty patients older than 18 years. They did not find a significant difference in RNFL thickness between amblyopic and fellow eyes in adults. The average RNFL thickness was 90.6±9.6 µm in amblyopic eyes and 90.1±12.1 µm in fellow eyes. Another study was conducted to find choroidal and RNFL thickness in adults with anisometropic amblyopia by Aylin et al.[19] No significant difference in peripapillary RNFL thickness was found between amblyopic and fellow or control eyes. The results of the present study indicate that RNFL thickness does not differ between amblyopic and fellow eyes across all types of amblyopia.

Another study was conducted on 150 participants with myopia, hyperopia, or emmetropia using the OCT. The participants were 15-30 years old, were free from ocular disease, and had not undergone any surgery. The ANOVA revealed that the difference

between the myopes vs. hyperopes and emmetropes was statistically significant ($p<0.05$) as compared to hyperopes vs. emmetropes ($p=0.152$). The mean values of RNFL thickness were thinner for myopes vs. hyperopes ($p<0.05$) in all quadrants. The mean value of RNFL thickness was greater in emmetropes than hyperopes in both the temporal and superior quadrants.[20] The findings of the present study indicate that RNFL thickness for the supra-nasal quadrant in hyperopic amblyopia was thicker amongst the refractive errors. There was no statistically significant difference between various types of refractive error in other quadrants.

Conclusion

This study offers a significant and surprising insight into the relationship between RNFL thickness and amblyopia. Our results contradict the widely held belief that identifiable structural alterations in the RNFL are a consequence of amblyopia, a disorder generally linked to aberrant visual development. The RNFL thickness of amblyopic eyes was found to be similar to that of their fellow (non-affected) eyes in all retinal quadrants. This held true regardless of the type of amblyopia. These findings directly contradict the conventional belief that amblyopia leads to thinning of the RNFL, which has been thought to reflect underlying retinal or optic nerve pathology. Our study suggests that the structural integrity of the RNFL may remain unaffected in amblyopia, even in the presence of significant visual deficits. This calls for assessing the existing theories of the pathophysiology of amblyopia, especially with regard to its assumed impact on retinal structure.

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REGIONAL CONFERENCES

Tri-State Vision Conference 2026
November 15 @ 9:00 am - 3:00 pm
Topic: Vision & Learning
Speakers: Drs. Jennifer Simonson, Barry Tannen, & Jessica Zwilling

Michigan Vision Therapy Study Group 2027
January 22 - 23, 2027

Southwestern Congress of Optometry 2027
January 23 - 24, 2027

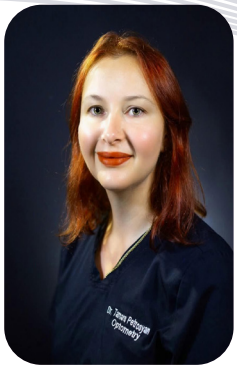
The Studt Practicum 2027
February 21, 2027 @ 8:00 am - 2:00 pm

Great Lakes Congress 2027
March 7 - 8, 2027

International Congress of Behavioral Optometry 2028
March 1 - 4, 2028

Student Corner • An Approach to Understanding Amblyopia Etiology and Diagnosis

Tamara Petrosyan, OD • SUNY College of Optometry • New York, New York



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Introduction

Amblyopia, derived from the Greek words "amblys" (blunt or dull) and "ops" (eye), literally translates to "blunt eye." This ancient term captures the essence of a condition in which vision is significantly reduced despite the absence of any obvious structural or pathological abnormality in the eye itself. Hippocrates, around 450 B.C.E., provided one of the earliest recorded descriptions in his aphorisms: "When the doctor sees nothing and the patient sees very little, the diagnosis is amblyopia." This observation remains remarkably relevant today, highlighting the functional nature of the disorder—a diagnosis of exclusion where the clinician observes no overt ocular anomaly, yet best-corrected visual acuity (BCVA) is impaired, typically defined as less than 20/20 (or a ≥2-line interocular difference) in one or both eyes.

Clinically, amblyopia is classified into functional (neurodevelopmental, reversible with appropriate intervention) and organic (resulting from identifiable structural or pathological damage to the visual pathway). Functional amblyopia arises from abnormal visual experience during early development, most commonly due to strabismus, anisometropia, high isoametropia, or form deprivation. Organic amblyopia stems from conditions such as optic neuropathy, retinal dystrophies, or neoplasms that mimic the presentation but require entirely different management. Distinguishing between these is a cornerstone of accurate diagnosis.

Amblyopia ranks among the most prevalent and consequential neurodevelopmental visual disorders in clinical practice. It affects 2–5% of the global population and, according to the Pediatric Eye Disease Investigator Group (PEDIG), is the leading cause of unilateral vision loss in adults aged 20–70+, surpassing trauma, infections, and other ocular diseases combined. In children under 20 years of age, amblyopia accounts for more vision loss than all other pediatric ocular diseases and physical trauma combined. In the United States, the Multi-Ethnic Pediatric Eye Disease Study (MEPEDS) and the Vision in Preschoolers (VIP) study report a prevalence of approximately 3–4% among preschool-aged children, with roughly 500,000 Americans living with visual impairment attributable to amblyopia.

The consequences extend far beyond reduced visual acuity in the affected eye. Individuals with amblyopia typically rely on a single "good" eye, placing them at a >25% lifetime risk of vision impairment in the better-seeing eye from unrelated causes such as age-related macular degeneration, glaucoma, retinal detachment, or accidental trauma. Dependence on a single functioning eye dramatically increases vulnerability to bilateral functional blindness. Additional lifelong consequences include increased risk of accidents, falls, and off-axis injuries due to impaired depth perception, contrast sensitivity, and peripheral awareness; educational and occupational limitations stemming from persistent deficits in reading speed, visual attention, fine motor coordination, and perceptual skills; and psychosocial effects, including social stigma, reduced self-esteem, and restricted participation in activities requiring excellent binocular vision.

Amblyopia imposes a disproportionate public health burden. Research demonstrates that it generates two to three times more disability-adjusted life years (DALYs) than congenital cataracts or childhood glaucoma. Validated patient-reported outcome measures highlight the breadth of impact: the PedEyeQ (developed by PEDIG) shows that children with residual amblyopia score significantly worse than peers in functional vision, social interactions, eye-related bother, and emotional frustration domains; the ATS QOL and NEI-VFQ-25 reveal that adults experience ongoing difficulties with daily visual tasks, reduced confidence, and lower overall quality of life.

Socioeconomic disparities show that children from low-income families and underserved communities experience higher prevalence, later diagnosis, denser amblyopia, and poorer long-term outcomes. Delayed intervention permits potentially irreversible cortical changes, perpetuating cycles of visual impairment and diminished opportunity.

Amblyopia affects the entire family unit. Children may face teasing, lowered self-esteem, social withdrawal, or activity limitations. Academic underperformance stemming from visual-perceptual deficits can lead to frustration, anxiety, or behavioral concerns. Parents frequently experience elevated stress, guilt, and worry regarding their child's vision and treatment adherence. Quality-of-life studies consistently show lower scores in emotional, social, and family functioning domains among parents of children with amblyopia. These psychosocial burdens often contribute to poor compliance with traditional therapies, underscoring the value of family-centered and binocular approaches that minimize disruption to daily life.

For nearly 300 years, amblyopia management centered on monocular punitive approaches—occlusion (patching) or pharmacological penalization of the fellow eye. Landmark research by Birch (2013) and others reframed amblyopia as a fundamentally binocular neurodevelopmental disorder driven by abnormal interocular suppression and disrupted binocular integration rather than simple monocular deprivation. This shift, supported by neurophysiological evidence from Hubel and Wiesel's Nobel Prize-winning work on critical periods and ocular dominance columns, emphasizes cortical plasticity, fellow-eye deficits, and global visual processing abnormalities. Contemporary approaches increasingly prioritize binocular therapies that restore balanced input between the eyes.

Neurodevelopment and Pathophysiology of Amblyopia

Amblyopia is a neurodevelopmental disorder of the visual cortex and higher visual pathways resulting from abnormal binocular visual experience during early life. It is not a disease of the eye itself, but the brain's adaptive—and ultimately maladaptive—response to discordant, blurred, or absent input from one or both eyes. To understand how the different types of functional amblyopia develop, we must first review the normal visual pathway and the critical role of experience-dependent plasticity.

After a visual stimulus strikes the retina, both image-forming and non-image-forming information is transmitted by retinal ganglion cells through the optic nerve, optic chiasm, and optic tract to the lateral geniculate nucleus (LGN) of the thalamus. The majority of fibers then project from the LGN to the primary visual cortex (V1) in the occipital lobe.

Within V1, neurons are organized into ocular dominance columns—alternating strips of cortex that respond preferentially to input from the right eye, the left eye, or both eyes equally. These columns compare the visual input between the eyes and also integrate binocular input. At birth, the foveal cones are immature and continue developing over the first 24 months, with further maturation extending into childhood. The formation and refinement of ocular dominance columns therefore depend on both nature (molecular guidance cues from retinal ganglion cells) and nurture (visual clarity and experience). Synaptic connections are strengthened by correlated activity between the two eyes and weakened by uncorrelated activity. This experience-dependent plasticity is most pronounced during a critical period (approximately birth to 8 years in humans), after which the system becomes less modifiable.

The presence of an amblyogenic risk factor does not guarantee that amblyopia will develop. The condition is more likely and more severe when multiple risk factors coexist; however, some children with significant anisometropia, high isoametropia, or even constant unilateral strabismus never develop amblyopia, possibly due to protective factors such as emmetropization/spontaneous refractive improvement, intermittent rather than constant deviation, stronger baseline binocular function, or individual variations in cortical plasticity and resilience.

Functional vs. Organic Amblyopia

Functional amblyopia is a purely neurodevelopmental condition caused by abnormal visual experience during the critical or sensitive period, without any structural or pathological damage to the eye or visual pathway. It is reversible to varying degrees with appropriate intervention because the deficit is cortical rather than ocular. Best-corrected visual acuity is reduced, yet the eye and visual pathway appear structurally normal on examination.

Organic amblyopia results from identifiable structural or pathological abnormalities (e.g., retinal dystrophy, optic neuropathy, tumor, toxicity, nutritional deficiency, etc., causing permanent

damage). It is not reversible by visual experience alone and requires treatment of the underlying disease. The key clinical differentiators are the presence of visible or detectable pathology on dilated fundus exam, OCT of the optic nerve and macula, ERG, VEP, and/or systemic workup, including bloodwork and neuroimaging; severity that does not match the amblyogenic factor; and lack of expected response to optical correction or binocular therapy. Any atypical, bilateral, progressive, or non-responsive case should prompt immediate investigation for organic causes.

Critical, Sensitive, Susceptible, and Residual Plasticity Periods

Period	*Age Range*	*Characteristics & Clinical Implications*
Critical Period	Birth – ~6 months	Binocular circuits are rapidly forming. Highest plasticity and vulnerability; even brief deprivation may cause changes, while prolonged deprivation can cause profound, sometimes irreversible cortical reorganization, where gains with treatment might be made, but some life-long residual amblyopia might remain post-treatment.
Sensitive Period	~6 months – 8 years	Primary window for most clinical amblyopia; excellent recovery potential with timely intervention.
Susceptible Period	~8 – 10 years	Declining plasticity; amblyopia can still develop or worsen, but response to treatment is slower and less complete.
Residual Plasticity Period	Adolescence ---> Adulthood	If amblyopia has not already formed, it will not develop; meaningful rehabilitation gains remain possible with intensive, targeted binocular approaches.

Pathophysiology of Functional Amblyopia by Type

Although the final common pathway is cortical suppression and imbalanced binocular input, each functional amblyogenic factor produces characteristic patterns of disruption.

Amblyogenic Refractive Errors	*Isometropia*	*Anisometropia*
Myopia	> 8.00 D	> 3.00 D
Hypermetropia	> 5.00 D	> 1.00 D
Astigmatism	> 2.50 D	> 1.50 D

Isometropic Amblyopia (Bilateral Refractive)

During the critical and sensitive periods of visual development, the visual cortex depends on sharp, high-contrast, patterned input from both eyes to properly refine ocular dominance columns, sharpen receptive fields, and establish normal binocular connections. When both eyes are equally blurred, there is no competitive advantage between them, but the cortex is deprived of the clear, detailed visual signals it needs for normal maturation. As a result, the normal experience-dependent synaptic pruning and strengthening processes are disrupted bilaterally. Ocular dominance columns remain poorly refined, receptive fields stay enlarged, and higher-order visual processing areas (V2, V3, and beyond) never receive the high-fidelity input required for proper spatial tuning and contrast sensitivity. Even after full optical correction is provided later, the cortical architecture has already developed abnormally, leading to bilateral reduced best-corrected visual acuity, increased crowding, and other perceptual deficits.

Anisometropic Amblyopia (Unilateral Refractive)

Uncorrected refractive error in one eye produces a chronically blurred or size-mismatched (aniseikonic) image on that retina. The visual system actively inhibits or suppresses the blurrier input to avoid confusion. This leads to shrinkage of ocular dominance columns serving the affected eye and expansion of those serving the fellow eye. The result is a progressive, unilateral loss of visual acuity and cortical spatial changes that favor the clearer eye.

Strabismic Amblyopia

Constant unilateral misalignment means each fovea receives a different image. The strabismus must be constant; if intermittent, then the brain will, at some point in space/time receive clear and fused images from both eyes, and amblyopia will not develop. If it is present the majority of the time, binocular visual input remains chronically asymmetric. The strabismus must be unilateral; if alternating fixation, each eye individually will at some space/time send clear foveal images to the brain, so while binocularity and stereopsis might be affected, monocular amblyopia will not develop. In constant unilateral strabismus, even if the deviating eye is optically capable of 20/20 vision, the brain applies strong active suppression of the foveal input to avoid the constant diplopia caused by misalignment, leading to cortical reorganization, shrinkage of ocular dominance columns for that eye, and functional amblyopia despite the eye's normal retinal and refractive potential. This often leads to deeper binocular dysfunction than purely refractive cases.

Form-Deprivation Amblyopia

Obstruction of the visual axis (e.g., large ptosis, corneal opacity, congenital cataract, dislocated

lens, vitreous hemorrhage) deprives the cortex of patterned input entirely. This causes the most severe and treatment-resistant cortical reorganization, often with nystagmus when bilateral or early-onset.

Neuroplasticity Across the Lifespan

Early intervention during the sensitive period maximizes recovery because the visual cortex is highly modifiable. However, neuroplasticity is not an all-or-nothing phenomenon. Even in adulthood, mechanisms such as synaptic strengthening, dendritic remodeling, and perceptual learning can still produce meaningful gains when stimulation is appropriately intensive and binocularly balanced. This residual plasticity explains documented improvements in motivated older patients and underpins current research into pharmacological agents (e.g., modulating GABA inhibition) and perceptual learning paradigms that aim to reopen or enhance plasticity windows.

Leading neuroscientists and vision scientists—notably Eileen Birch, Robert Hess, and Dennis Levi—have established that amblyopia is primarily a binocular problem. Abnormal interocular suppression and disrupted binocular integration, rather than simple monocular deprivation, drive the majority of sensory, motor, and perceptual deficits. This paradigm shift explains fellow-eye abnormalities, persistent deficits after monocular therapy, and the superiority of binocularly oriented approaches in both assessment and management. Understanding these mechanisms helps clinicians anticipate prognosis (deeper deprivation or earlier onset generally predicts more resistant amblyopia) and prioritize binocular metrics (stereopsis, suppression, polarized binocular acuity) during every evaluation.

Because amblyopia is largely asymptomatic in young children—who rarely complain of unilateral vision loss—systematic screening is essential. The InfantSEE program (American Optometric Association), which provides free comprehensive eye assessments for infants aged 6–12 months by volunteer optometrists, has significantly improved early detection of amblyogenic risk factors.

Clinical Presentation and Symptoms

Amblyopia is frequently described as the "silent thief" of vision in childhood. Many young patients remain asymptomatic or lack the language to describe their visual deficits, allowing the condition to progress undetected. Far beyond simple acuity reduction, amblyopia produces a broad spectrum of sensory, motor, perceptual, and functional impairments. A nuanced understanding of both classic and subtle presentations is essential for timely identification, particularly during routine examinations, where overt signs may be absent. Some of the below information builds on previous Student Corner publications, including an approach to understanding strabismus etiology and an approach to understanding strabismus diagnosis part 1 and part 2.

Reduced best-corrected visual acuity is the defining clinical feature, typically unilateral with a ≥2-line interocular difference, though it can occasionally be bilateral. Parents may observe squinting, eye closure, head tilting, or preferential use of one eye. However, children often adapt remarkably well and rarely voice complaints of blur.

Crowding/Contour Interaction: Contour interaction, also known as the crowding effect, refers to the well-documented phenomenon in which visual acuity for a target optotype (letter or symbol) is significantly reduced when it is surrounded by similar contours or neighboring optotypes compared with when the same target is presented in isolation. In individuals with normal vision, mild crowding occurs mainly in the peripheral visual field due to normal lateral inhibition and receptive field overlap in the visual cortex. In amblyopia, however, contour interaction is markedly exaggerated and extends into the central visual field of the affected eye. This occurs because abnormal visual experience during the critical or sensitive period leads to enlarged receptive fields, reduced cortical inhibition, and disrupted spatial summation in V1 and extrastriate areas.

The brain's imbalanced binocular input and weakened lateral interactions cause neighboring contours to interfere strongly with target recognition, making it difficult for the amblyopic visual system to isolate the target from its surroundings. As a result, patients perform substantially worse on crowded acuity charts than on isolated-letter tests—a finding that is one of the most consistent and diagnostically useful hallmarks of functional amblyopia. The severity of crowding also carries strong prognostic value: greater contour interaction typically indicates deeper cortical involvement and a more guarded response to treatment.

Contrast Sensitivity Loss: Contrast sensitivity is the ability of the visual system to detect subtle differences in luminance (shades of gray) across a range of spatial frequencies. In normal vision, it is mediated primarily by the parvocellular (P) pathway, which processes

high-spatial-frequency detail and fine edges. In amblyopia, contrast sensitivity is significantly and persistently reduced, particularly at higher spatial frequencies (finer details), even when visual acuity has been partially improved with optical correction or therapy. This stems from active cortical suppression of the amblyopic eye's input, leading to enlarged receptive fields, weakened lateral (inhibitory) circuits, and imbalanced drive in V1 and extrastriate areas. The parvocellular pathway is particularly impaired, while magnocellular deficits affect motion and low-contrast processing; these changes extend into higher-order visual areas, producing the hallmark features of crowding, spatial distortions, unsteady fixation, and reduced global form/motion integration. Importantly, the fellow ("good") eye is not entirely normal, showing subtle deficits in contrast sensitivity and binocular summation, underscoring that amblyopia is fundamentally a binocular cortical disorder rather than a purely monocular one.

Unsteady/Inaccurate Monocular Fixation: Unsteady and inaccurate monocular fixation in amblyopia—manifesting as fixational nystagmus, saccadic intrusions, or slow drifts when viewing exclusively with the amblyopic eye—is a direct consequence of cortical miswiring and weakened neural drive in the visual fixation control system. In normal vision, steady fixation is maintained by a finely tuned feedback loop involving precise signals from V1 ocular dominance columns to higher centers (frontal eye fields, superior colliculus, and brainstem fixation neurons). In amblyopia, chronic suppression of the weaker eye during the critical and sensitive periods leads to shrinkage of its ocular dominance columns in V1 and reduced cortical responsiveness. When the fellow eye is occluded, the brain must rely almost entirely on the degraded, suppressed input from the amblyopic eye. This produces unreliable, low-gain neural signals that are insufficient to hold the eyes steady.

As a result, the fixation system "hunts" for a stable position, leading to small, involuntary drifts, corrective microsaccades (saccadic intrusions), and sometimes a low-amplitude fixational nystagmus. These instabilities are far more pronounced when the amblyopic eye is tested alone because the stronger fellow eye normally provides compensatory binocular drive and stabilizes fixation through interocular summation. The problem is not mechanical (the muscles are normal); it is a central cortical issue stemming from imbalanced binocular input, enlarged receptive fields, and reduced inhibitory control in the visuomotor pathways. This explains why unsteady fixation is one of the most consistent associated deficits in amblyopia and why it often persists even after acuity has improved. It also contributes to poor tracking, reading difficulties, and visuomotor incoordination.

Poor Eye Tracking: Poor eye tracking in amblyopia is not caused by weakness of the extraocular muscles but by disrupted cortical control of the oculomotor system resulting from abnormal binocular visual experience during development. In normal vision, smooth pursuit and accurate saccades depend on high-fidelity visual feedback from V1 and higher cortical areas (especially the motion-sensitive area MT/V5 and parietal attention networks) that continuously update the brain about target position, velocity, and spatial location. This information is sent to the frontal eye fields, superior colliculus, and brainstem oculomotor nuclei to generate precise eye movements. In amblyopia, several interrelated cortical abnormalities break this feedback loop:

- **Unsteady fixation and degraded retinal image quality** create a constantly shifting, low-contrast image that provides unreliable velocity and position signals
- **Chronic suppression and imbalanced binocular input** reduce the strength and precision of input reaching MT/V5 and other motion-processing areas, resulting in jerky pursuits (the eyes fall behind and then make catch-up saccades)
- **Enlarged receptive fields, weakened lateral inhibition, and poor spatial localization in V1 and parietal cortex** delay saccadic latency; and inaccurate saccadic landing with frequent regressions impairs visual scanning and sustained reading, leading to reduced fluency and the classic complaint of skipping words or lines.

Even the fellow eye often shows subtle tracking deficits because amblyopia is a binocular cortical disorder—the entire visuomotor system has developed with abnormal interocular balance. These eye-movement abnormalities persist even after acuity improves because the underlying cortical reorganization in motion and attention networks is not fully reversed by optical correction or monocular therapy alone. Poor eye tracking is one of the most functionally disabling aspects of amblyopia and is a key reason why children may be misdiagnosed with attention or learning disorders. It is best assessed with tasks such as pursuits, saccades, and reading fluency

tests, and it improves most reliably with binocularly oriented vision therapy rather than monocular approaches.

Accommodative Inaccuracy: Accommodative inaccuracy in amblyopia is primarily a central (cortical) problem, not a peripheral issue with the ciliary muscle or lens. In normal vision, the brain uses clear, high-contrast retinal images and precise blur cues from both eyes to drive rapid and accurate accommodative responses via the near triad (accommodation, convergence, and miosis). In amblyopia, the amblyopic eye sends degraded, suppressed, or spatially distorted signals to the visual cortex. This results in unreliable blur detection and poor feedback to the midbrain and cortical centers that control accommodation. Unsteady fixation, increased fixational drift, and saccadic intrusions further degrade the quality of the retinal image, making it difficult for the brain to compute the exact amount of defocus.

Because amblyopia is fundamentally a binocular disorder, the normal synergistic linkage between accommodation and vergence is disrupted by interocular suppression and reduced binocular summation. The fellow eye often shows subtle accommodative facility or accuracy deficits due to abnormal cortical drive. Clinically, this manifests as inconsistent focusing at near, asthenopia, blur during sustained near tasks, reduced accommodative amplitude or facility on testing (MEM, NRA/PRA, flipper tests), and slower recovery from blur. These problems persist even after refractive correction because the root cause is cortical miswiring rather than optical blur itself.

Spatial Distortions and Perceptual Issues: Spatial distortions and perceptual issues in amblyopia—such as micropsia (objects appearing smaller), macropsia (objects appearing larger), perceived displacement of objects, and difficulty judging distances or aligning handwriting—are primarily caused by cortical miswiring in the visual processing hierarchy rather than by any problem with the eye itself. Chronic suppression of the amblyopic eye's input during the critical and sensitive periods leads to enlarged receptive fields and weakened lateral inhibition in V1 and extrastriate areas (V2, V3, and higher association cortices). This disrupts the brain's ability to accurately localize visual elements in space and to integrate local features into a coherent global percept. The visual system loses its normal precision in mapping retinal position to perceived location, resulting in spatial mislocalization and size misperception because the cortical representation of the visual field is distorted and less sharply tuned.

Higher-order deficits in the dorsal visual stream (parietal "where/how" pathway) further impair visuospatial processing, making it difficult to judge distances, maintain proper alignment (e.g., handwriting drifting off the line), or accurately reach for objects. Even the fellow eye often shows subtle deficits in spatial processing due to abnormal binocular integration. These perceptual distortions are not just subjective annoyances—they directly contribute to real-world difficulties such as poor reading fluency, clumsy eye-hand coordination, and academic challenges. They persist even after acuity has improved because the underlying cortical reorganization is not fully reversed by optical correction alone.

Visual Perceptual and Learning Difficulties: Visual perceptual and learning difficulties in amblyopia—such as skipping words or lines, reduced reading fluency, poor visual attention, figure-ground discrimination problems, and complaints that may masquerade as learning disabilities—stem directly from higher-order cortical dysfunction beyond the primary visual cortex (V1). Chronic suppression and abnormal binocular input lead to enlarged receptive fields, weakened lateral inhibition, and disrupted connectivity in extrastriate areas (V2, V3, V4) and higher association cortices. This impairs global form and motion integration, making it difficult for the brain to bind local letters into coherent words or lines of text. Figure-ground discrimination suffers because the visual system cannot efficiently segregate a target (a word or line) from surrounding clutter, leading to frequent skipping or losing place. Unsteady fixation, jerky pursuits, and increased saccadic latency further degrade smooth visual scanning, so the eyes cannot track text fluidly, resulting in reduced reading fluency and frequent regressions. Higher-order deficits in the dorsal visual stream also impair visual attention and visuospatial processing, causing the brain to struggle with sustained focus on sequential visual information and spatial organization (e.g., aligning handwriting or keeping place on a page).

These problems are not due to poor motivation or general cognitive issues—they are specific consequences of the same cortical reorganization that causes crowding and spatial distortions. Because the deficits are subtle and often occur in the presence of relatively preserved Snellen acuity, they

frequently go unrecognized as visual in origin and are mistakenly attributed to learning disabilities or attention disorders. Even the fellow eye may show subclinical impairments in attention and global processing, reinforcing that amblyopia is a binocular cortical disorder that affects the entire visual processing hierarchy. This is why many children with amblyopia are first identified in school settings for "reading problems" rather than in eye exams, and why addressing the underlying binocular and perceptual deficits is essential for true functional improvement.

Higher-Order Visual Processing Deficits: Higher-order visual processing deficits in amblyopia arise because the abnormal binocular experience during the critical and sensitive periods cascades beyond V1 into extrastriate areas (V2, V3, V4, MT/V5) and higher association cortices responsible for integrating, interpreting, and using visual information for perception and action. In normal development, balanced input from both eyes refines not only basic feature detection in V1 but also global form and motion integration, figure-ground segregation, object recognition, spatial attention, and visuomotor transformation in these higher areas. In amblyopia, the chronic suppression of the weaker eye's signals leads to imbalanced cortical drive, enlarged receptive fields, reduced lateral inhibition, and weakened feedforward and feedback connections between V1 and higher visual regions.

As a result, the amblyopic visual system struggles with global processing tasks such as perceiving coherent motion in noisy backgrounds, integrating local contours into whole objects (global form perception), segregating a target from a cluttered background, and maintaining efficient visual attention. These deficits manifest clinically as spatial distortions, difficulty judging complex scenes, impaired perceptual grouping, and problems with tasks that require rapid or accurate visual interpretation (e.g., reading fluency, face recognition in crowds, or navigating dynamic environments). The fellow eye often shows subtle higher-order impairments, reinforcing that amblyopia is a binocular cortical disorder that affects the entire visual processing hierarchy, not just local acuity in V1. This is why many patients continue to report perceptual and visuospatial difficulties long after Snellen acuity has improved.

Fine Motor and Coordination Problems: Amblyopia frequently leads to fine motor and coordination difficulties because it disrupts the precise visuomotor integration required for accurate eye-hand coordination, reaching, grasping, and visually guided movements. The core issue is not simply reduced acuity but a cascade of higher-order visual deficits that impair the brain's ability to use visual information effectively for motor planning and execution. Reduced stereopsis (depth perception) is a major contributor, as the amblyopic visual system struggles to judge distances and spatial relationships in three dimensions, making tasks such as threading a needle, catching a ball, or writing neatly more challenging. In addition, oculomotor dysfunction (unsteady fixation, jerky pursuits, increased saccadic latency, and fixational instability) prevents stable visual tracking of moving targets or precise localization of stationary ones during movement.

These problems are compounded by deficits in the dorsal visual stream, which handles global motion processing, visuospatial attention, and the transformation of visual information into motor commands. Higher cortical areas involved in perceptual grouping, figure-ground discrimination, and predictive eye-hand coordination receive unreliable or suppressed input, resulting in slower, less accurate, and more variable motor responses. The fellow eye often shows subclinical deficits in contrast sensitivity and motion processing, further degrading binocular visuomotor integration. Consequently, children and adults with amblyopia commonly exhibit clumsiness, slower fine-motor skills, increased fall risk, and difficulties with activities requiring precise visual guidance—deficits that persist even after acuity has improved and are directly linked to the underlying cortical reorganization rather than the eye itself.

Primitive Reflexes: Primitive reflexes are automatic, involuntary, unconscious movement patterns present in early infancy that serve as foundational building blocks for survival, early motor development, and wiring of the sensorimotor system (e.g., Moro reflex causing babies to startle and cry to loud noises, rooting reflex causing suckling motion when cheek/mouth is touched for feeding, and palmar grasp reflex causing baby to involuntarily curl their hand around an object placed in their palm). Under normal development, these reflexes are integrated and inhibited by higher cortical centers during the first year of life as voluntary motor control and postural stability emerge. In patients with functional binocular deficits and amblyopia, many developmental vision protocols screen for retention of these reflexes because early disruption of normal visual experience and sensorimotor integration can delay or prevent their maturation.

The Asymmetric Tonic Neck Reflex (ATNR) is the most commonly retained reflex in these patients. When the head turns to one side, the ATNR causes extension of the arm on that side and flexion of the opposite arm, physically linking head position with limb movement and eye gaze. Its persistence creates a motor roadblock that prevents the eyes from moving independently of the head and body, directly contributing to unsteady fixation, jerky pursuits, poor eye tracking, and impaired eye-hand coordination. Other frequently retained reflexes include the Symmetric Tonic Neck Reflex (STNR)—head flexion causes arm flexion and leg extension; head extension causes the opposite—which interferes with smooth transitions between flexion and extension and often causes poor posture and difficulty with quadruped positions; the Tonic Labyrinthine Reflex (TLR), triggered by head position relative to gravity and leading to poor balance and motion sensitivity; the Moro Reflex, linked to hypersensitivity and startle reactions; and the Spinal Galant Reflex, associated with poor posture, fidgeting, and interference with stable sitting or visual scanning during reading.

Temporal Mismatch and Asynchronization in Amblyopia: In amblyopia, visual information from the amblyopic eye reaches V1 with a measurable temporal delay relative to signals from the fellow eye. This interocular latency difference—often on the order of 5–20 msec or more—has been consistently demonstrated using visual evoked potentials (VEP), reverse-correlation psychophysics, and dichoptic temporal synchrony tasks. The delay arises from cortical suppression, reduced neural drive, enlarged receptive fields, and altered temporal tuning in the amblyopic pathway (particularly affecting the magnocellular system and higher-order processing). As a result, the temporal window of the amblyopic eye is typically flatter, broader, and delayed compared with the fellow eye. This temporal asynchronization creates profound difficulties with binocularity in the visual cortex.

Binocular neurons in V1 and extrastriate areas require precisely synchronized input from both eyes to achieve proper summation, fusion, and stereoscopic depth perception. When signals arrive out of phase, the cortex cannot effectively correlate and integrate the two inputs. The brain therefore defaults to suppression of the slower (amblyopic) eye's signals to avoid perceptual confusion or rivalry. This mechanism directly contributes to loss of stereopsis, reduced binocular summation, persistent interocular suppression, and impaired global motion/form integration. Even small timing mismatches disrupt the experience-dependent refinement of binocular circuits during the critical and sensitive periods, perpetuating the binocular dysfunction that defines modern understanding of amblyopia. Clinically, this explains why binocularly oriented therapies that compensate for or resynchronize interocular timing (e.g., via controlled temporal phase offsets) can improve fusion and stereopsis more effectively than purely monocular approaches.

Diagnosis: Criteria, Testing, Differential Diagnosis, and Masqueraders

According to the American Optometric Association and international consensus statements, amblyopia is diagnosed when all of the following criteria are met:

1. Reduced best-corrected visual acuity (usually <20/20 or a ≥2-line interocular difference) that cannot be fully explained by organic pathology.
2. Presence of a documented amblyogenic factor acting during the critical or sensitive period of visual development (typically before age 8).
3. Absence of structural or pathological abnormalities sufficient to account for the level of vision loss after comprehensive evaluation.

All 3 of the above criteria must be met for the diagnosis of functional amblyopia. The diagnosis is fundamentally one of inclusion (amblyogenic factor + compatible functional deficits) and exclusion (no organic cause).

Inclusion Criteria

- Confirmed amblyogenic factor (strabismic, refractive, or deprivational) with onset before age 8.
- Reduced BCVA that is consistent in severity and laterality with the amblyogenic factor.
- Evidence of neural adaptation, including crowding effect, suppression, eccentric fixation, or reduced stereopsis.
- Normal or near-normal ocular health on dilated examination and ancillary testing.

Exclusion Criteria

- Organic pathology that fully explains the vision loss.
- Severity mismatch (e.g., light perception or no light perception vision with only mild refractive error or intermittent strabismus).
- Adult-onset without prior history or progressive loss.

Severity	Visual Acuity Range	Common Amblyogenic Factors	Typical Prognostic Notes
Mild	20/30 – 20/40	Low-moderate anisometropia, intermittent strabismus	Excellent response potential
Moderate	20/50 – 20/100	Moderate-high anisometropia, constant strabismus	Good response with early intervention
Severe	Worse than 20/100	Dense form deprivation, high combined factors	Guarded; often requires intensive care

- Bilateral symmetric profound reduction without a clear bilateral deprivation history.

Mixed amblyopia (e.g., anisometropia combined with strabismus) is not uncommon and typically produces more severe deficits; the dominant factor should be clearly identified and documented. Severity must align with the amblyogenic factor for diagnostic confidence. Significant mismatch between observed severity and the amblyogenic factor is a critical red flag that demands investigation for masqueraders.

When to Suspect Amblyopia in Routine Exams: Red Flags by Age

- **Infants (0–12 months):** Asymmetric red reflexes (Bruckner test), poor visual attention, nystagmus, failure to fix and follow, or resistance to occlusion of one specific eye. Because subjective recognition-based optotypes are not yet viable, clinicians must rely on objective quantification to establish a baseline VA. Forced-Choice Preferential Looking (FCPL), such as Teller Acuity Cards or LEA paddles, leverages an infant's innate preference for patterned stimuli to estimate resolution acuity. Sweep Visual Evoked Potentials (Sweep VEP) can be utilized to record cortical responses to rapidly changing spatial frequencies, providing a fast and objective assessment of the visual system's functional integrity.
- **Toddlers (1–3 years):** Eye closure in bright light, preferential head turn or closure of one eye, frequent bumping into objects, or abnormal photoscreening results. Teller cards using FPL can be used to measure visual acuity.
- **Preschool (3–5 years):** Poor performance on LEA Symbols or HOTV (especially with crowding bars), reduced stereopsis, or parental concern regarding eye alignment or tracking.
- **School Age (6+ years):** Reading avoidance, headaches, declining academic performance, skipping words/lines, or unexplained unilateral acuity reduction despite normal ocular health.
- **Any Age:** Interocular acuity difference ≥2 lines, eccentric fixation on visuoscopy, suppression on Worth 4-dot or RDS, or acuity that fails to match the refractive error or ocular appearance.

Any child presenting with a strong amblyogenic risk factor warrants detailed evaluation even if initial acuity appears borderline. Testing results are not interpreted in isolation but synthesized to confirm or refute a functional diagnosis. Documenting these

Amblyopia Clinical Testing

Test / Procedure	Purpose	Equipment	Performance	Adaptations for Young / Uncooperative Patients	Key Considerations & Common Pitfalls	Interpretation & Clinical Implications
Case History & Patient-Reported Outcomes	Establish risk factors, timeline, functional impact, and psychosocial context	Intake form/ template, PedEyeQ, CISS, COVD QOL, ATS QOL / NEI-VFQ-25	-Open-ended: "What brings you in?" -Prenatal/perinatal: gestational age, birth weight, NICU, maternal smoking/substances -Developmental milestones (visual tracking, motor skills) -Family history of amblyopia/ strabismus/refractive error -Onset, constancy, laterality of eye turn or preference -Previous treatments & compliance -Symptom survey	Use parent-proxy heavily; use simple language and toys to engage child; break into short segments with rewards	Avoid leading questions; document compliance barriers; missing family history leads to underestimation of genetic risk	Early onset + constant unilateral deviation = higher risk of severe amblyopia; high PedEyeQ frustration scores predict poor compliance with penalization therapy (patching)

Visual Acuity Testing (with Crowding) and Contrast Sensitivity	Quantify deficit and assess contour interaction/ crowding effect	LEA Symbols, HOTV, Snellen/ ETDRS charts, crowding bars, translucent occluder/patch	-Explain as a game -Test binocularly first -Monocular at distance and near (use translucent occluder or fog with + if have eccentric fixation). -Full chart --> single line --> single letters with crowding bars -Record exactly: e.g., "20/40 WL with bars OD" -Compare monocular vs binocular -Monocular contrast sensitivity -Neutral density filter eval	Matching cards/ gestures for LEA/ HOTV; short segments with praise; use preferential looking if needed	Always test with crowding bars; never rely on single uncrowded letters; fatigue can falsely lower scores	≥2-line difference or marked crowding = amblyopia; worse crowded acuity = poorer prognosis; binocular summation = positive sign
Fixation Assessment: Visuoscopy	Detect & characterize eccentric fixation (present in ~80% strabismic cases)	Direct o-scope or visuoscope with bull's-eye target	-Dim lights; occlude non-tested eye; test better eye first -Show bull's-eye on your hand -Project bull's-eye onto fundus; ask patient to look at center dot -Observe 30 full seconds -Record: type (central/eccentric), direction, magnitude (degrees), stability, % foveation	Test very quickly; parental holding for stability	Test better eye first for comparison; poor cooperation can mimic unsteadiness	Central + high % foveation = good prognosis; unsteady peripheral EF = deep amblyopia; Expected VA ≈ 20/20 × (EF° + 1)
Refractive Assessment	Identify amblyogenic refractive error and establish baseline	Retinoscope, cycloplegic agents (1% cyclopentolate and/or tropicamide)	-Dry retinoscopy & subjective (if possible) -Instill cycloplegic (cyclo or tropicamide 2 drops, 5 min apart); Wait 30–40 min -Cycloplegic retinoscopy and refraction -Keratometry/topography for astigmatism/scissoring reflex -Consider aniseikonia testing	Get parental help with drops	Cycloplegia / Damp assessment is mandatory in amblyopia workup ; under-cycloplegia misses latent hyperopia	High anisometropia or isoametropia confirms amblyogenic factor; compare with BBP findings
Best Binocular Prescription (BBP)	Find lens powers that maximize binocular function (Sanet-Vergara)	Polarized acuity chart, trial frame, Worth 4-dot, stereopsis tests	-Place cycloplegic findings in trial frame -Use polarized chart binocularly -Reduce plus (esp. more hyperopic eye) in 0.25 D steps; stop at best binocular visual acuity + comfort -Confirm improvement with Worth 4-dot & stereopsis	Keep sessions short; use engaging targets; praise frequently	BBP often uses less plus than monocular maximum; do not over-plus	Improvement in binocular metrics even if one eye's monocular acuity drops slightly = successful BBP
Binocular & Sensorimotor Evaluation (**Please see previous student corner publications on strabismus)	Assess alignment, suppression, correspondence, stereopsis, motility, accommodation	Occluder, Worth 4-dot, red lens, stereopsis book, prism bar, flippers, MEM retinoscope	-Alignment: Hirschberg, Bruckner, cover test, 4^{Δ} base-out, modified Thorington -Suppression: Worth 4-dot (dist/near), Bagolini -Correspondence: red lens or Bagolini -Stereopsis/Vergence: NPC, phoria, ranges, facility, RDS, local stereo -Motility: 9 gazes for comitance -Accommodation: amplitude, facility, MEM, NRA/PRA	Use toys as fixation targets; short bursts; parent assistance	Perform binocular tests BEFORE prolonged occlusion; test both distance & near	Strong suppression + no stereopsis = poor binocular potential; no ARC + good stereo = favorable
Ocular Health & Ancillary Testing	Rule out masqueraders and organic causes	Slit lamp, dilated fundus, OCT, ERG/VEP, MRI	-Anterior segment; dilated fundus exam (mandatory) -Keratometry, OCT, OCT-A, fundus photos, FAF -Color vision and visual field -Electrophysiology (VEP/ERG) if atypical -MRI if optic nerve or intracranial signs	Use hand-held slit lamp, 20 D + indirect ophthalmoscope as needed; sedation if necessary for young children	Never skip ocular health with dilation in new/ unresponsive cases	Normal fundus does NOT rule out early dystrophy; bilateral/nystagmus/progressive = high masquerader risk

indicators at baseline enables realistic expectations and tailored counseling. Precise diagnosis of amblyopia rests on confirming an appropriate amblyogenic factor, compatible functional deficits, and exclusion of masqueraders. This structured approach ensures functional cases are identified promptly while organic conditions are not missed.

Visuoscopy

In amblyopia (particularly strabismic and mixed types), the amblyopic eye often develops eccentric fixation—the patient uses a non-foveal retinal point for fixation instead of the fovea when in a monocular state. Visuoscopy should be used to evaluate monocular fixation in suspected amblyopia, especially when strabismus is present. It is quick, inexpensive, and provides critical prognostic information that standard acuity testing cannot. Eccentric fixation directly correlates with poorer visual acuity and guarded prognosis. Visuoscopy lets you:

- Confirm whether fixation is central or eccentric
- Quantify direction, magnitude, stability, and percent foveation
- Predict expected best-corrected visual acuity
- Differentiate functional amblyopia from organic causes (organic cases rarely show eccentric fixation with normal fundus)

Procedure (takes ~1 minute per eye): Dim lights and shine bullseye on your hand, describe target and show patient where they need to look --> fully occlude affected eye --> project bull's eye target onto fundus of better eye using low illumination (just so you can see it in the eye but are not blinding the patient) to get a baseline for comparison and patient practice --> rotate dial to make the retina clear in your view, tell patient to look directly at the very center of the bull's-eye and observe for 30 seconds --> repeat on amblyopic eye.

While observing the foveal reflex, watch where it lands relative to the center of the bull's-eye and note any movement or instability. If fixation is unsteady on the amblyopic eye but steady on the fellow eye, this is a strong indicator of functional amblyopia rather than organic disease. Perform visuoscopy before prolonged occlusion to avoid inducing artifactual suppression. In young children, have the parent hold the child steady, praise frequently, have a toy or sticker ready as a treat after each test, and test for only 10-15 seconds. Record four key parameters for each eye:

- **Type of Fixation:**
  - **Central (foveal):** Foveal reflex is centered on the target --> best prognosis
  - **Eccentric:** Foveal reflex is off-center --> amblyopic eye is using a non-foveal point
- **Direction:** If the foveal reflex is displaced toward the patient's nose (nasal side of the target center), the patient is using a nasal retinal point to fixate --> nasal eccentric fixation. If the foveal reflex is displaced toward the patient's temple (temporal side of the target center), the patient is using a temporal retinal point to fixate --> temporal eccentric fixation. EF usually matches the strabismus: nasal EF with esotropia, temporal with exotropia. Can also be superior or inferior.
- **Magnitude** (in degrees from fovea): Each o-scope has a specially designed bull's-eye target with concentric rings that are calibrated in angular degrees from the center. This allows you to measure the exact eccentricity of fixation in real time on the fundus. Most standard visuoscopes have rings at 1°, 2°, 3°, and 5° from the center, but the manual should be checked for exact specifications.
  - Foveal off-center: ≤1°
  - Parafoveal: 1–3°
  - Paramacular: 3–5°
  - Peripheral: >5° (Most cases in amblyopia are <3°)
- **Stability & Percent Foveation:**
  - Steady vs. unsteady (drifts, saccadic intrusions)
  - % foveation = how often the fovea is used during the 30-second observation (e.g., 70% means the fovea is used 70% of the time)

Nasal EF direction is recorded as positive (+); temporal is noted as negative (-). The expected visual acuity can be calculated. The (+)/(-) are used for recording the direction only, not for the formula. Approximate BCVA ≈ **20/20 × (eccentric fixation in degrees + 1)**. Example: 3° temporal eccentric fixation --> expected VA ≈ 20/20 × (3 + 1) = 20/80.

If the patient has central fixation + high % foveation --> excellent prognosis. Unsteady peripheral eccentric fixation indicates a deeper amblyopia, more guarded prognosis, and likely a slower response to treatment.

In addition to direct visuoscopy, two entoptic phenomena—the Haidinger Brush and Maxwell's Spot—provide valuable, non-invasive confirmation of central versus eccentric fixation, particularly when patient cooperation with ophthalmoscopy is limited. Both phenomena are fovea-specific and provide an objective window into whether the patient is truly using the anatomical fovea for fixation. Macular disease can significantly interfere with both of these phenomena; if a patient cannot perceive the brush

or spot (or sees it distorted/absent) despite good instruction and proper technique, this is a strong red flag for organic macular disease rather than (or in addition to) eccentric fixation.

- **Haidinger Brush** is an entoptic phenomenon created by the radially arranged, dichroic (polarization-sensitive) macular pigment (xanthophylls) in the fovea. When the patient views a field of polarized blue light (through a rotating polarizer or specialized filter), the pigment differentially absorbs light depending on its polarization orientation, producing a visible propeller- or hourglass-shaped brush that appears to the patient to rotate with the polarizer. This brush is perceived only when the light stimulates the central fovea; therefore, with eccentric fixation, the brush appears displaced from the fixation target in the direction opposite the eccentric point.
- The **Maxwell's Spot** arises from the higher concentration of macular pigment (lutein and zeaxanthin) in the central fovea, which strongly absorbs short-wavelength (blue) light. When viewing a uniform blue field (e.g., through a blue filter or Maxwell's spot tester), the foveal region appears as a dark central spot or shadow due to this selective absorption. Because the spot marks the true anatomical fovea, any misalignment between the spot and the patient's reported fixation point directly confirms EF.

Neutral Density Filter Test

The neutral density (ND) filter test is a simple prognostic and diagnostic tool that helps confirm the functional (cortical) nature of amblyopia and differentiate it from organic causes of reduced vision. It exploits the fact that the amblyopic visual system is relatively resistant to overall luminance reduction because of cortical suppression and altered contrast processing. A ND filter decreases overall luminance without inducing a color change and produces a corresponding decrease in central visual acuity in healthy eyes. To perform the test, first measure BCVA of amblyopic eye monocularly with the optimal monocular refractive correction --> place a standardized ND filter over the corrected amblyopic eye (commonly 1.0 or 2.0 log units) --> remeasure acuity of amblyopic eye through filter.

In functional amblyopia, the amblyopic eye shows minimal or no drop in visual acuity (often <1 line), because the cortical suppression and altered contrast/luminance processing in V1 make the amblyopic system relatively insensitive to overall brightness reduction. In contrast, an organic lesion (e.g., optic neuropathy, macular dystrophy, or media opacity) typically causes a marked drop (2 or more lines) in acuity with the filter. This relative luminance sparing in the amblyopic eye strongly supports a functional diagnosis.

The ND filters can also be used to quantify the depth of suppression. The patient views the Worth 4-dot target through red-green filters to identify the current suppression pattern --> place ND filters of increasing log units over the fellow (non-amblyopic) eye to reduce its luminance until the interocular signal is balanced in the brain --> the specific density value at which the patient breaks suppression or achieves fusion—reporting all five dots if diplopic or four dots if fused—serves as the neutralization point, providing a precise metric for the strength of the cortical suppression.

Performing Sanet-Vergara-Press Best Binocular Prescription (BBP)

The Best Binocular Prescription (BBP) is a neurofunctional approach that prioritizes the lens powers that give the best overall binocular performance rather than the strongest monocular acuity in each eye. It is especially useful for hyperopic anisometropic amblyopia. The goal is to minimize binocular competition (suppression) and maximize binocular summation, fusion, and stereopsis to decrease the amount of cortical suppression (the cause of amblyopia and visual acuity loss) leading to improvement in visual acuity (the symptom of amblyopia).

Step 1: Obtain Accurate Baseline Refraction: Perform dry retinoscopy and subjective refraction (if possible) --> Perform full cycloplegic refraction (using cyclopentolate or 2 drops of tropicamide) --> Record the maximum plus accepted monocularly for each eye.

Step 2: Polarized Binocular Testing: Turn on the polarized acuity chart and use polarized glasses over the trial frame --> Place the cycloplegic (or dry) findings into the trial frame. Start with the full monocular prescription in both eyes. Instruct the patient to look at the letters with both eyes open and report when they are clearest and most comfortable --> Begin reducing the plus power (usually in the more hyperopic/amblyopic eye) in 0.25 D steps. After each adjustment, ask: Is the chart clearer, the same, or blurrier? Is it more comfortable? Continue reducing plus until you reach the point where binocular visual acuity is maximized and the patient reports best overall clarity/comfort. --> Fine-tune both eyes as needed (sometimes you

need to increase / decrease plus in the dominant eye as well).

Step 3: Confirm Binocular Balance and Improved Binocularity: Worth 4-dot test (distance and near)—look for improvement in suppression, optimally fusion with luster. Stereopsis Testing—aim for the Rx that gives you the best possible stereoacuity (goal is often 30–50 arc seconds or better). Cover test and associated phoria—check for improved alignment or reduced phoria.

Step 4: Finalize and Prescribe: The final BBP is the combination that gives the best binocular visual acuity, best fusion on Worth 4-dot, best stereopsis, and/or when the patient reports best overall comfort. Prescribe this prescription for full-time wear. Explain to the patient/parent: these glasses are balanced for both eyes working together, not just the strongest prescription for each eye separately.

Important Clinical Nuances & Tips:

- You will often reduce plus in the amblyopic (more hyperopic) eye and increase plus in the non-amblyopic eye to decrease the anisometropia in the prescription. This is counter-intuitive but central to the method.
- It is common for monocular acuity in the amblyopic eye to temporarily decrease slightly—this is acceptable if binocular function improves. The BBP is successful when the patient says that both eyes feel like they're working together better, even if one eye's monocular acuity is not at its theoretical maximum.

Category	Condition	Etiology / Mechanism	Key Clinical Features
Eyelid, Corneal, or Lenticular Anomalies	Congenital ptosis, corneal opacities/dystrophies, cataracts, lens dislocation (especially unilateral)	Structural obstruction or media haze that physically blocks or severely degrades patterned visual input to the retina during the critical/sensitive period, resulting in classic form-deprivation amblyopia	Media haze, leukocoria, asymmetric red reflex on Bruckner test; partial or unilateral cataracts, dense ptosis, or corneal scars; often presents with poor fixation and nystagmus if bilateral/early onset; dilated exam or slit-lamp reveals the obstructing lesion
Hereditary Retinal Disorders	Best's Disease	Autosomal dominant BEST1 gene mutation causing defective chloride channel in RPE, leading to lipofuscin accumulation and macular RPE dysfunction	Vitelliform "egg-yolk" macular lesion (early) that later scars or ruptures; abnormal EOG (reduced Arden ratio); gradual bilateral central vision decline starting in childhood/adolescence; normal or near-normal ERG initially
	Stargardt's Disease	Autosomal recessive ABCA4 mutation causing toxic lipofuscin (A2E) buildup in RPE, leading to photoreceptor degeneration	Flecked maculopathy with "beaten bronze" or "fish-tail" appearance at the posterior pole; central scotoma; progressive bilateral central vision loss in school-age children; "dark choroid" on fluorescein angiography; abnormal full-field ERG
	Cone-Rod Dystrophy	Genetic mutations (multiple genes) primarily affecting cone photoreceptors, with later rod involvement	Severe photophobia, nystagmus, color vision loss (especially red-green), and reduced visual acuity from early childhood; bull's-eye maculopathy on fundus exam; markedly abnormal photopic ERG with relatively preserved scotopic ERG early on
	X-Linked Juvenile Retinoschisis	RS1 gene mutation on X chromosome causing splitting of the inner retinal layers (schisis)	Spoke-wheel macular schisis on OCT; peripheral retinal schisis; reduced b-wave on ERG; primarily affects boys; decreased central vision and strabismus common; vitreous veils or hemorrhage possible
	Retinitis Pigmentosa	Mutations in >50 genes causing progressive photoreceptor (initially rods, later cones) degeneration and retinal pigment epithelial atrophy	Night blindness (nyctalopia) as earliest symptom; bone-spicule pigmentation, arteriolar attenuation, waxy pallor of optic disc; constricted visual fields; markedly abnormal ERG (reduced or extinguished)
	Achromatopsia / Cone Dysfunction Syndromes	Genetic mutations (e.g., CNGA3, CNGB3) causing complete or incomplete absence of functional cone photoreceptors	Severe photophobia, nystagmus, poor color vision (complete achromatopsia sees only shades of gray), and reduced acuity from birth/infancy; normal or near-normal fundus appearance initially; markedly abnormal photopic ERG with preserved scotopic ERG

Retinal Vascular Disease	Coats' Disease	Idiopathic retinal telangiectasia and capillary leakage with massive exudation (non-hereditary, sporadic)	Unilateral telangiectatic vessels with yellow-white lipid exudates in the peripheral retina; predominantly affects boys; often presents with strabismus, leukocoria, or xanthocoria; fluorescein angiography shows characteristic leakage; can mimic unilateral amblyopia
Optic Nerve Anomalies	Optic Atrophy	Genetic (dominant, recessive, or X-linked) or acquired damage to retinal ganglion cells and optic nerve axons	Pale optic discs (temporal or diffuse); nystagmus in recessive forms; vision loss often bilateral and symmetric; may have associated systemic neurologic findings
	Leber's Hereditary Optic Neuropathy	Mitochondrial DNA point mutations causing acute dysfunction of retinal ganglion cells	Sequential or simultaneous central/cecocentral scotomas; typically presents in young males (teens to 30s); disc hyperemia or pseudoedema acutely, followed by atrophy; maternal inheritance pattern
	Optic Nerve Hypoplasia	Incomplete intrauterine development of retinal ganglion cell axons (idiopathic or associated with septo-optic dysplasia, maternal diabetes, alcohol/drug exposure)	Small or pale optic disc with "double-ring" sign; vision loss often out of proportion to amblyogenic factor; may be unilateral or bilateral; endocrine dysfunction common (growth hormone deficiency, hypothyroidism); neuroimaging (MRI) and endocrine evaluation recommended
	Morning Glory Disc Anomaly	Congenital excavated optic disc malformation with central glial tuft and peripapillary retinal detachment risk	Enlarged, funnel-shaped excavated disc with central glial tissue and radial retinal folds; high risk of serous retinal detachment or choroidal neovascularization; usually unilateral; vision often severely reduced
Neoplasms of the Visual Pathway	Optic pathway glioma or craniopharyngioma	Benign or malignant tumors compressing or infiltrating the optic nerve, chiasm, or tracts	Slowly progressive vision loss (often asymmetric); disc pallor or swelling; endocrine abnormalities (craniopharyngioma); bitemporal field defects; MRI with contrast is essential for diagnosis
Cerebral / Cortical Visual Impairment	Cerebral Visual Impairment (CVI)	Damage to visual cortex, optic radiations, or higher visual association areas from perinatal brain injury (hypoxic-ischemic encephalopathy, periventricular leukomalacia, prematurity, hemorrhage, infection, or seizures)	Often bilateral or asymmetric; poor visual attention, dorsal-stream dysfunction (visuospatial/motion deficits), better vision in familiar environments; history of neurological insult or MRI abnormalities; normal ocular structures; vision loss disproportionate to any amblyogenic factor; frequently abnormal VEP
Other Masqueraders	Persistent Fetal Vasculature (PFV)	Failure of the fetal hyaloid vascular system to regress during embryonic development	Remnants of hyaloid artery/vitreous vessels causing media opacity, cataract, or retinal traction; typically unilateral; microphthalmia or elongated ciliary processes common; leukocoria or strabismus at presentation

- In deep anisometropia (e.g., plano/+8.00), you may need several follow-up visits to titrate the BBP gradually.
- Re-evaluate the BBP every 4–6 weeks initially, as it can change as binocular function improves.
- Contact lenses or Shaw lens can sometimes give an even better binocular response than spectacles (less aniseikonia and prism imbalance).

Masqueraders of Amblyopia: Any condition that reduces vision without obvious external signs can mimic amblyopia. Dilated fundus examination is mandatory in every case; atypical features should trigger prompt escalation.

Although a significant relative afferent pupillary defect (RAPD) typically signals organic pathology, research indicates that a dense functional amblyopia (20/400 or worse) can occasionally produce a very subtle RAPD. This phenomenon is a frequent point of confusion for clinicians attempting to differentiate functional neurodevelopmental deficits from structural organic loss.

In the majority of amblyopia cases, an APD does not occur, because the condition is fundamentally a neurodevelopmental disorder of the visual cortex rather than a disease of the eye or afferent visual pathway traveling from the eye to the brain. Because the retina and optic nerve remain structurally normal, the subcortical pupillary light reflex—which branches off the visual pathway before reaching V1—is typically unaffected. In exceptionally severe, dense cases, a slight APD may manifest due to a significant reduction

in neural drive or secondary retrograde changes in the visual pathway, though any marked defect should always be treated as a red flag requiring investigation for an organic cause.

While amblyopia does not produce classic visual field defects or restrictions, a syntonics or functional visual field might show deficits when both eyes are open. The development of this functional constriction is driven by the brain's attempt to manage discordant binocular input. Under monocular testing conditions, the lack of competition from the fellow eye allows the cortex to process the signals from the amblyopic eye, often revealing a full functional visual field. However, once the fellow eye is uncovered, its superior input triggers a competitive imbalance, and the visual cortex actively inhibits or suppresses the signal from the amblyopic eye that shuts down its central processing.

The "amblyopic pit" is a specialized clinical concept describing a functional, processing, and attentional, rather than structural, deficit or scotoma in the visual field. The foveal regions of the primary visual cortex (V1) are highly specialized for binocular integration and fine spatial detail. The pit typically forms here because the neurons responsible for central vision are the most vulnerable to the effects of abnormal interocular timing and suppression. The central suppression leads to enlarged receptive fields and weakened lateral inhibition, which the patient experiences as spatial mislocalization or "crowding". During binocular reading, the functional pit can cause words or lines to disappear or become jumbled, leading to frequent regressions and reduced reading fluency

Clinical Pearls and Decision-Making

Mastering amblyopia diagnosis requires more than collecting discrete test results. It demands the skillful synthesis of history, risk factors, refractive data, sensory findings, binocular status, and ocular health into a unified clinical picture. If any element is discordant, pause and investigate further rather than forcing a functional label. A confident diagnosis of functional amblyopia emerges when the following elements align:

- A clear amblyogenic factor with appropriate timing (before age 8)
- Reduced BCVA (with crowding) proportional to the factor's severity and duration
- Evidence of neural adaptation (crowding effect, suppression, eccentric fixation, or reduced stereopsis)
- Normal or near-normal ocular health on dilated examination and ancillary testing
- Positive or expected response to initial Best Binocular Prescription (BBP) optimization.

If all elements align (appropriate amblyogenic factor + compatible findings + normal health) --> functional amblyopia. Specify type and severity. If there is inconsistency present (severity mismatch, atypical progression, abnormal fundus, or lack of expected BBP response) --> suspect masquerader and escalate to electrophysiology or imaging.

Common Pitfalls and How to Avoid Them

- **Over-Reliance on Monocular Visual Acuity Alone:** Basing the diagnosis solely on uncrowded single-letter acuity. Always include crowding bars and polarized binocular comparisons. Binocular summation often reveals more than monocular data.
- **Missing Subtle Eccentric Fixation:** Assuming central fixation based on casual observation. Perform visuoscopy in every unilateral acuity reduction or strabismus case. Even small eccentricities (<3°) significantly affect prognosis.
- **Inadequate Masquerader Rule-Out:** Accepting a "normal" fundus without dilation or ancillary testing. Dilate routinely. Maintain a low threshold for ERG/VEP/MRI in bilateral, progressive, or atypical cases. Consider especially if no meaningful response after 3–4 months of appropriate optical, monocular and/or binocular therapy and rehabilitation.
- **Ignoring Psychosocial Context:** Focusing only on clinical metrics while overlooking family burden or compliance barriers. Integrate PedEyeQ or ATS QOL early to guide realistic, family-centered recommendations.
- **Age-Based Therapeutic Nihilism:** Dismissing potential improvement in older children or adults. Always assess residual binocular potential regardless of age.

The highest success rates occur in collaborative environments. A second opinion or co-management strengthens diagnostic confidence and builds patient/family trust. Reassess key binocular metrics at every visit—improvement in these often precedes monocular acuity gains and serves as an early indicator of successful intervention. By systematically integrating findings, avoiding common pitfalls, and applying a binocular-first mindset, clinicians can achieve precise diagnoses that translate directly into better patient outcomes.

Amblyopia stands as a quintessential model of experience-dependent neuroplasticity in the human visual system. It is a condition in which timely, mechanism-based identification and diagnosis can profoundly alter a patient's lifelong visual, perceptual, educational, and psychosocial trajectory. The modern paradigm shift from purely monocular punitive approaches to binocular thinking represents one of the most important conceptual advances in the field. By recognizing amblyopia as a disorder of interocular imbalance and suppression rather than simple monocular deprivation, clinicians are encouraged to restore balanced binocular input from the very first encounter. This perspective improves functional outcomes, reduces treatment burden on children and families, and aligns care more closely with the underlying neurophysiology. The goal is to transform amblyopia from a leading cause of preventable unilateral vision loss into a success story of early detection, precise diagnosis, and optimized visual development for every patient we serve.

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Petrosyan TS. An approach to understanding amblyopia etiology and diagnosis. Optom Vis Perf 2026;14(2):181-96.

Course Announcement

Pediatric Eye Care and Vision Therapy: From Theory to Practice with Tamara Petrosyan, OD & Marc B. Taub, OD, MS, EdD

A comprehensive exploration and overview of everything you were taught…but forgot!

Learn at your own pace and in the comfort of your own home.

This self-directed course is a broad overview of 12 different topics in the world of pediatrics and binocular vision in 14 hours of lecture. It starts with topics related to vision development and prescribing, makes its way through myopia control and strabismus, and finishes with visual information processing and acquired brain injury. Each lecture is accompanied by a resource section that includes many articles on the lecture topic. The course is meant to introduce or reintroduce you to ideas that you may never have learned, or perhaps you learned and forgot long ago.

eBooks from OEPF

Applied Concepts in Vision Therapy 2.0
Leonard Press, OD
Marc B. Taub, OD, MS, EdD
Pamela H. Schnell, OD

Vision The Forgotten Sense
Kenneth Lane, OD

Neuro-Visual Processing Rehabilitation: An Interdisciplinary Approach
William Padula, OD
Raquel Munitz
W. Michael Magrun

The Essential Playbook How to Maximize Outcomes in Optometric Vision Therapy
Nancy Torgerson, OD & Kristi Jensen, OD

The Power of Lenses – Vol 1
Marc B. Taub, OD, MS, EdD &
Pamela H. Schnell, OD

A Snapshot of Upcoming Events

Aging Eyes, Aging Vision: What Changes And What Can Be Done- Dr. Cathy Stern
July 8, 2026; 8:00 - 10:00 pm EDT

OEPF VT Morning Tea, Free Meeting, Moderated By Tom Headline, COEP-T
July 18, 2026; 12:00 - 2:00 pm EDT

Speciality Sports Vision
August 16, 2026--4 Hours
Speaker: JJ Lant, OD

Treatment Through Touch: Ocular Therapeutic Manipulation - Dr. Leonid Skorin, Jr.
August 19, 2026; 8:00 - 10:00 pm EDT

Visualization
August 22-23, 2026
Speaker: John Abbondanza, OD

Seeing Beyond the Surface: Ocular and Systemic Insights into Hereditary Connective Tissue Disorders - Dr. Vishwa Jain
September 9, 2026; 8:00 - 10:00 pm EDT

The Art & Science of Optometric Care – A Behavioral Perspective
Sept 18 - 19, 2026
Sept 28 -30, 2026
Speaker: JJ Lant, OD

Sports Vision
October 3 and 4, 2026
Speaker: JJ Lant, OD

6h Insights: Cerebellum And Introduction To Vestibular Rehabilitation-Dr. Cathy Stern
October 5, 2026; 8:00 - 10:00 pm EDT

VT/Visual Dysfunctions - (VT-1)
Part 1: October 17 and 18, 2026
Part 2: October 24 and 25, 2026
Speaker: Virginia Donati, OD

Speciality Sports Vision
November 1, 2026--4 Hours
Speaker: JJ Lant, OD

VT/Learning Related Visual Problems – (VT-2)
Nov 7 - 8, 2026
Nov 12 - 14, 2026
Speaker: Virginia Donati, OD

ABI/TBI with John Abbondanza and Marc Taub
November 15 and 22, 2026
11:00am - 7:00 pm EDT

VT/Strabismus and Amblyopia – (VT-3)
December 3 - 6; 2026
Speaker: John Abbondanza, OD

OEPF Monthly Study Group: Chat VT
July 21, 2026; 8:00 pm EDT
August 18, 2026; 8:00 pm EDT
September 20, 2026; 8:00 pm EDT
October 20, 2026; 8:00 pm EDT
November 17, 2026; 8:00 pm EDT
December 16, 2026; 8:00 pm EDT

For a complete list of events, please visit the OEP website at: