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EDITORIAL

The Silent Warrior

Leonard J. Press, OD, FAAO, FOVDR
Editor-in-Chief

I wrote a blog recently[1] about a middle-aged man with a long-standing intermittent esotropia of the divergence insufficiency type who was decompensating both visually and psychologically, though it's not always easy to separate the two. Nor should we think that it would be, given that the eyes, brain, and body are intertwined. The patient gave the following description: "I feel like my whole body contracts when my eyes start to turn inward. It's like my body is holding the stress, but it's centered in my eyes. The process is so fatiguing to keep from seeing double, that I often look first and then process the information afterward." And while describing the contortion that he experiences from head to toe, the patient choreographed his feeling as illustrated (he gave permission to share this).

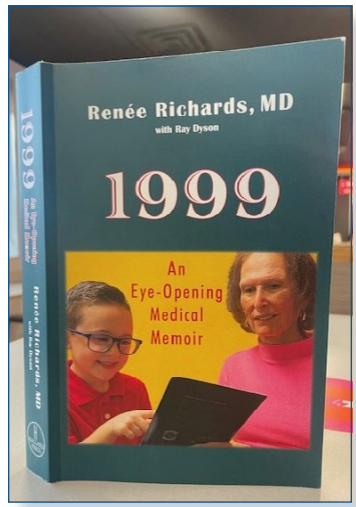
I prescribed a low amount (one diopter) of yoked prism bases down in addition to +0.50 sphere OU to be used in conjunction with his contact lenses that provided the patient with a great deal of calm. It was more than a placebo effect, as the patient has had an ample number of interventions in the past, ophthalmic and otherwise, that provided little if any relief. Plus I had done an A/B single subject crossover design by interposing bases down versus bases up prism chairside, and the patient could easily discriminate a difference in sensation.



The patient called and asked if I could increase the amount of prism. It's a natural tendency for patients (and some doctors) to think that if a little bit of something is good, a greater amount will be better. Rather than an arbitrary increase, I asked the patient to return so that we could probe what higher amounts would do. I used the same A/B crossover design except this time it involved magnitude rather than direction. The patient preferred 2.0^ to 1.0^, and then 3.0^ to 2.0^. Each time I interposed the prism, the patient would close his eyes, take a deep breath, exhale, and then open his eyes.

I noticed something on his second visit that I had overlooked on the first. The patient had a subtle "V" pattern intermittent esotropia, with slightly higher eso on downgaze as compared to upgaze. So I re-wrote the Rx as 3^ yoked bases down, and the patient will follow-up with a colleague nearby who has the perfect holistic approach to optometric vision therapy needed in this case. At the end of our visit the patient remarked: "It's such a pleasure to explain how I feel to you. You're the first doctor who took the time to really listen, and who speaks my language. You're a silent warrior, doc."

The silent warrior theme brought to mind a book authored by Dr. Renée Richards, published in 2021. It is titled 1999: An Eye-Opening Medical Memoir, and that it is[2] For those who don't recall or are not aware, Dr. Richards specialized in pediatric ophthalmology and strabismus as Dr. Richard Raskind, prior to undergoing a gender-transforming operation in the mid-1970s. An accomplished tennis player, she took five years off to pursue her dream and returned to the practice of ophthalmology in Manhattan in 1982.



Dr. Richards notes that colleagues considered her to have a boutique practice, and she recognized that as a solo practitioner she was a dinosaur. When asked in 1999 much longer she intended to practice (she was 65 years old then) she replied: "As long as I can still contribute and as long as I enjoy it, I'll keep going."

No apologies are made, in her book, about strabismus surgery being as much an art as a science. She writes: "That's where the skill and the judgment of doing eye muscle surgery come in. The numbers are not etched in stone for how much you move the muscle for how many degrees of turn of the eye you've measured. Many things come into account: whether it's a large myopic eye or a very small child's eye; whether there has been previous surgery; what the condition of the muscle is when we expose it and look at it — many, many possibilities for variation in the result from the predicted amount of surgery."

The year that Dr. Richards returned to Manhattan Eye and Ear Hospital (1982) was the year that I came to Manhattan to serve as Chief of the Vision Therapy Service at SUNY Optometry. Our in-house strabismus consultant was Dr. Norman Medow, a close colleague of Dr. Richards, who collaborated with my optometric colleague Dr. Jeffrey Cooper on a landmark paper about intermittent exotropia.[3] Dr. Medow and Dr. Cooper were silent warriors in their own right.

What is lacking in Dr. Richards' revealing medical diary for the year 1999 is any mention of collaboration with optometrists. While there are a number of cases she shares that needed prism to neutralize diplopia, there is scant mention of the role of orthoptists who at one time rendered therapeutic services within ophthalmologic practices. They too, Dr. Richards notes, have become dinosaurs with regard to their original function, with most transitioning to medical assisting in a technician role.

Part of the problem in underutilization of optometric therapeutic services (OVT) by fellow professionals stems from the misconception that viable OVT is limited to orthoptics. If that is the case, then there would be no need to refer a patient to an optometrist. The ophthalmologist/orthoptist collaboration suffices. That misconception persists to this day, as evidenced by a recent article by Huhtanen et al.[4] To improve the utilization of a pediatric ophthalmologist's time, four groups were identified by the Ophthalmology Department at Royal Children's Hospital (RCH) in Melbourne, Australia who could be well managed in Primary Care Optometry. The authors state that patients with amblyopia and strabismus were not included, "due to being in a separate, similar collaborative care program between RCH and the Discipline of Orthoptics at La Trobe University."

As a COVD working group thirty years ago, Jim Bosse, Dave Cook, Lou Hoffman and I co-authored an article on how ophthalmologists and optometrists should have the same synergistic relationship when it comes to managing strabismus as exists for cataract co-management.[5] The model of cataract co-management has continued to evolve in the ensuing years since that article was published. Dr. Arthur Benjamin, a California cataract surgeon, summarizes that synergy beautifully on his website when he writes: "Co-management in eye care represents a seamless, patient-focused approach that leverages the specialized skills of both ophthalmologists and optometrists. It's a partnership that ensures patients receive high-quality, comprehensive care."[6] He adds that the symbiosis combines the strengths of both professions, and that he upholds that relationship with pride and commitment. In that regard, Dr. Benjamin is a silent warrior.

Which brings me to the OVDRA online CE session on September 29 given by the optometrist Samantha Del Campo and the strabismus surgeon Thomas Lenart regarding Collaborative Management of Strabismus.

The original silent warrior in the relationship with Dr. Lenart was our colleague, Dr. Nancy Torgerson. They lectured about the synergy in their collaboration at the COVD meeting in 2014 in San Diego, and at the SECO meeting in Atlanta the same year. Dr. Curt Baxstrom, a fellow Washington state colleague of Dr. Torgerson, has also collaborated closely with Dr. Lenart. It is wonderful that working models of the symbiotic relationship exist, but they are too few and far between. Although the symbiosis between our two professions is now commonplace regarding cataract care, it remains scant in regard to optometric vision therapy.

To remedy that, Dr. Del Campo and Dr. Lenart co-authored a chapter last year on their symbiosis in the only periodical that represents a joint venture

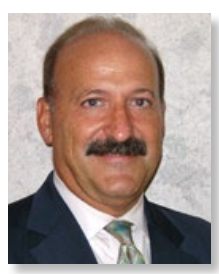


between Optometry and Ophthalmology.[7] That is an important step in the right direction of leveraging the specialized skills of both ophthalmologists and optometrists, as Dr. Benjamin phrased it. Its clear when it comes to vision development and rehabilitation that OVDRA optometrists and therapists have a skill set not possessed by most ophthalmologists and orthoptists.

At some point, hopefully in our lifetime, presentations such as the joint CE on Collaborative Management of Strabismus will be welcomed in primary care optometric settings and in a variety of medical forums including pediatrics, pediatric ophthalmology, and rehabilitation medicine. While being a silent warrior is a badge of honor in some respects, it is one that we would rather not have to wear.

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PERSPECTIVE

The Spatial Transformation Properties of Prism and Their Clinical Applications

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ABSTRACT

Prisms have traditionally been prescribed to compensate for ocular misalignment by shifting the apparent location of retinal images. This paper examines evidence that prism effects extend beyond chief-ray deviation and include spatial transformations arising from asymmetric angular deviation and nonlinear magnification. These optical properties can produce changes in perceived slant, curvature, spatial rotation, size, and distance.

The paper reconciles longstanding disagreements regarding prism-induced compression and expansion of space by distinguishing two-dimensional optical interpretations from three-dimensional perceptual frameworks. It further reviews how these spatial transformations may influence posture, movement, and visual behavior, with applications in vision therapy, neurorehabilitation, traumatic brain injury, autism, proprioceptive dysfunction, and learning-related visual disorders.

A broader understanding of prism optics and perception may allow clinicians to prescribe prisms more effectively, using the smallest amount necessary to achieve compensatory or therapeutic goals while optimizing visual behavior, orientation, posture, and sensorimotor organization.

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INTRODUCTION

What do prisms do? This is a question that has intrigued optometrists for many years. Traditionally, prisms have been and still are used to compensate for ocular misalignment. However, beginning in the 1960's, optometrists such as Dr. Bruce Wolff were incorporating them into their vision therapy programs to influence visual behavior.[1] More recently their application has been extended to the TBI population for rehabilitative purposes.[2]

During this discussion we will be addressing the aberrations created by the spatial properties of prism. When these are troublesome, patients are told to ignore them and eventually they will appear to go away. We will also touch upon the perceived compression or expansion of space which can potentially be beneficial. Seemingly there are paradoxes surrounding these observations. Specifically, how can there be compression or expansion in the same section of the prism, i.e. compression and expansion in the base or compression and expansion in the apex. The discussion will include knowing about and then using these aberrative properties as additional tools to consider when prescribing prism to treat perceptual and behavioral dysfunctions, and to affect a desired behavioral response in our patients.

Regarding the clinical application of prism specifically to observe behavioral changes, Dr. Robert Kraskin famously stated, "It's not what a lens does to a person, but what a person does with the lens".[3] This invites some specific questions. What

are the spatial properties of prism that affords a behavioral change in a person? Is changing the spatial location the only property that may affect a change in behavior? What role do aberrations of prisms and spatial transformations play in "what a person does with the lens", or prism in this case?

These questions have been a topic of discussion for many years and have sparked many lively debates, most notably between Dr. Paul Harris and Dr. John Streff with regard to the concept of expansion and compression of space. Dr. Harris wrote that when he looked through a prism, he observed that there was an expansion of space in the apex and compression of space in the base.[4] Dr. Streff wrote that he observed that there was a compression of space in the apex and expansion of space in the base.[5] These are very interesting concepts considering that the basic understanding of a prism is that it just shifts or transfers an image from one point in space to another creating an ocular rotation to adjust to that new position.

In this article we offer additional thoughts regarding the clinical benefits that these spatial transformations, as noted by Drs. Harris and Streff, create through the aberrations of the prism. A deeper understanding of these spatial transformation concepts and perceptual possibilities aids in the prediction of behavior when applying a prism. Utilizing this information becomes instrumental in designing or developing a more specific protocol when prescribing prism to affect the direction or development of a desired behavior pattern.

Summarizing to this point, the most commonly understood property of prism, as it applies to ophthalmic use, is to refract and redirect light with the amount of refraction value referred to as the total deviation or minimal angle. But there are questions associated with this that we will explore, such as: How does this simple property of redirecting light create the behavior changes observed in a person? How does the prism create the illusion of expansion and compression of space? How does the prism create the illusion of spatial distance changes?

THE FUNDAMENTAL PROPERTIES OF PRISM

Prism can be conceived as a refracting medium with surfaces that are inclined at a specific angle relative to each other. The primary spatial transformation property of prism is refraction or chief ray shifting.[6] In a prism, the chief ray is always refracted towards the base with the image appearing to shift in the direction of the apex. The ray incident to the front surface of the prism is refracted towards the base of the prism and the exiting ray is refracted towards the base of the prism. The total angle of deviation is defined when the chief ray passes through the prism where the angle of incidence is equal to the angle of emergence.

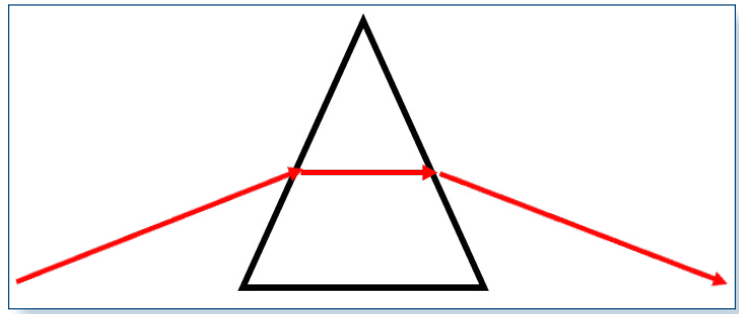


Figure 1. Minimal Ray Light Path

The total deviation is also referred to as the minimal angle or the minimal ray. When discussing prism, why does the conversation generally surround the minimal ray? Because the minimal ray light path is the most common property of prism as it applies to ophthalmic use (see Figure 1). The minimal or chief ray is determined when the path of the light ray is symmetrical, such that the angle of incidence to the (front surface) is identical to the angle of refraction (back surface) and parallel to the prism base as it traverses the prism. The optical strength or dioptric value of the prism is referenced by the total deviation or dioptric value. A one diopter value is defined as a refracting medium that produces an apparent displacement of 1 cm of the chief ray when measured at a distance of 1 meter.

The total deviation or minimal ray light path only applies to a prism placed in front of the eye when the face of the prism, the surface closest to

the object, is perpendicular to the direction of the incident light ray path or when the posterior surface of the prism is perpendicular to the eye (person). Other orientations produce a variable dioptric shift of the image depending on the angle of the prism in front of the eye and the resultant prismatic value is always more. While this may not be a major issue with smaller prism values, it does become a major issue with larger amounts of prism. This is also a point of consideration when attempting to measure an angle of ocular deviation and the imprecision of the resulting dioptric measurement which depends on how the prism is positioned in front of the patient.

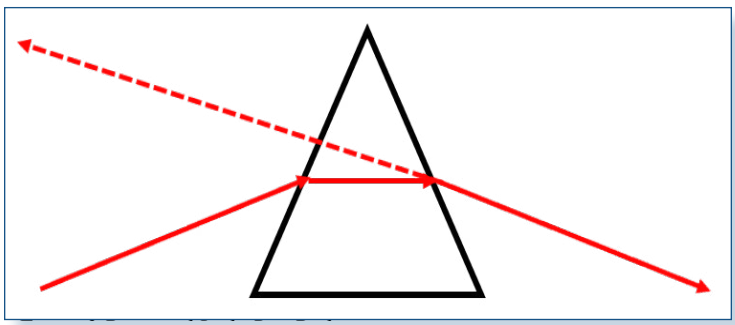


Figure 2. Projected Light Ray Path

An observer looking at an object through the prism will project the object to be displaced towards the apex of the prism (see Figure 2). The light path through the prism is refracted in the direction of the base, and the location of the object is perceived as shifted or translated in the direction of the apex.

We were taught how to calculate the amount of deviation. As noted earlier, one diopter of prism shifts the light path one centimeter in space for every meter of distance traveled through the air, which is convenient to know for taking tests and passing boards.

The conventional application of prism is therefore to shift the apparent location of images to match of the alignment of the two eyes so that they intersect in a manner that reduces the neuromuscular effort to maintain clear, single, and simultaneous binocular vision (CSSBV). However, as reviewed by Ogle,[7] there are other notable properties of prism that result in distortions related to nonuniform magnification. The distortion is characterized by: (1) an increasing angular magnification of the image toward the apex in the base-apex meridian, (2) a curvature of the images of lines perpendicular to the base-apex meridian, and (3) an increased slanting of the images of lines parallel to the base-apex line. Let's take a closer look.

Asymmetric Angular Deviation and Asymmetric Angular Magnification

In addition to what we have considered thus far, the rays of light approach the prism surface at different angles and are incident on different parts of the prism surface.[8] These rays of light have a different angle of incidence on the front surface. The variable thickness of the prism subtly affects the speed of transmission through the lens material. The longer the path through the medium of the lens the more it is slowed. As a result, the emerging rays of light all have different exiting angles.

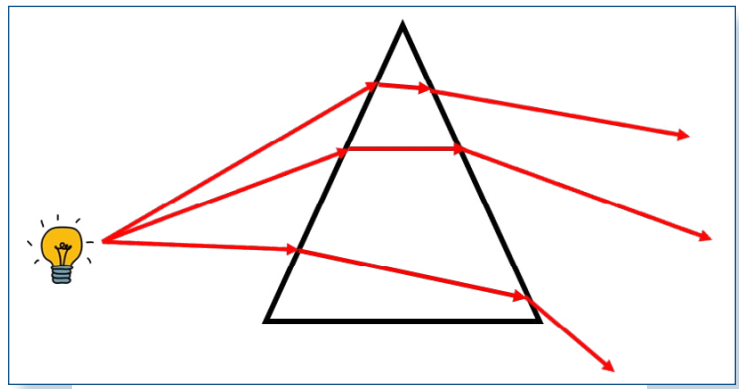


Figure 3. Asymmetric Angular Deviation

As can be seen in Figure 3, light emitted from the object point strikes the prism at different angles and the resultant deviation is different depending on the where the rays of light strike the prism. The deviated rays of light have greater deviation toward the base and less deviation toward the apex. This is referred to as asymmetric angular deviation.

Figure 4 shows that as the object is viewed through the prism, the projected angles of deviation of the image would have the opposite perception of the deviated side. In other words, the deviation would be perceived as a larger or expanded image

toward the apex and a smaller or contracted image toward the base.

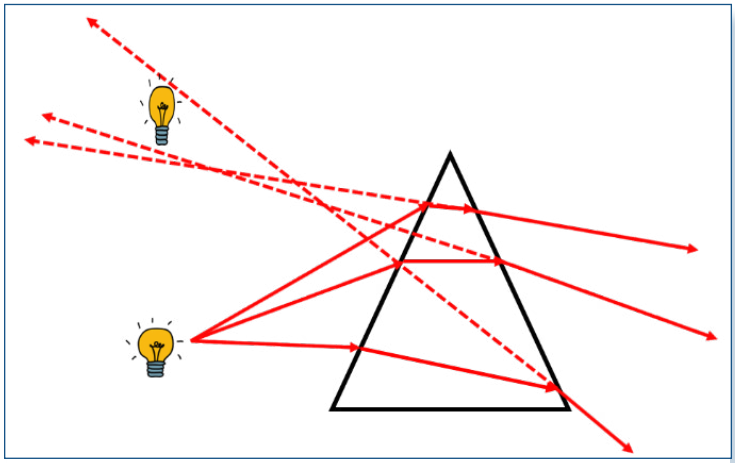


Figure 4. Asymmetric Angular Magnification

In the examples in the following discussion, we will primarily reference the spatial changes noted through a horizontal base left prism. Understanding the spatial changes with this prism, spatial observations can then be extrapolated to prism in any direction and applied to any prismatic diopter power, keeping in mind that the conscious awareness of the spatial change is generally more noticed with prism values greater than 5 diopters.

Compression and Expansion

Applying the principles of asymmetric angular magnification, there will be the perception of a compression of the square wave towards the base and expansion of the square wave towards the apex. This is demonstrated by looking through a base left prism at a square wave grating. When viewing the square wave grating in Figure 5a through the prism, look through any point on the base apex line, and without moving your eyes from that fixation point, compare the base side to the apex side of your fixation point. The square wave grating should appear as in Figure 5b, with perceived compression of space in the region of the base, and expansion of space in the region of the apex.

Slant

Along the line perpendicular to the base-apex line there is a simultaneous non-linear shrinkage (compression) of the object image toward the apex and increased size (expansion) towards the base. Projected images appear to change in height where the height is taller towards the base side of the center reference point and shorter towards the apex, as noted in Figure 5b. The behavioral observation of this size change would be that the floor or ceiling appears to tilt or slant. Since down space is easier to observe, the subjective observation would be uphill for a base down prism or downhill for a base up prism or a slant up to the right or left depending on the base direction of the prism. The slant is always upwards towards the apex of the prism, as in Figure 5b.

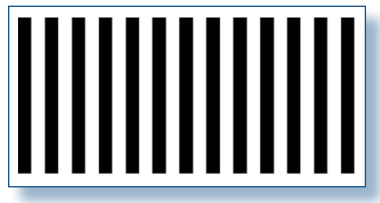


Figure 5a. Square Wave Grating

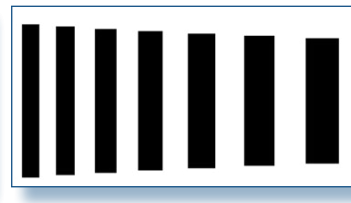


Figure 5b. Square Wave Grating viewd with Base Left Prism

Curvature

Another property of prism is curvature.[9] Along the line perpendicular to the base-apex line or parallel to the base, there is the perception of curvature. This is because light entering the prism from above or below has a different angle of incidence than light entering from straight ahead, such that different regions of a vertical line will be displaced differentially. The curvature also increases towards the apex of the prism as seen in Figure 6.

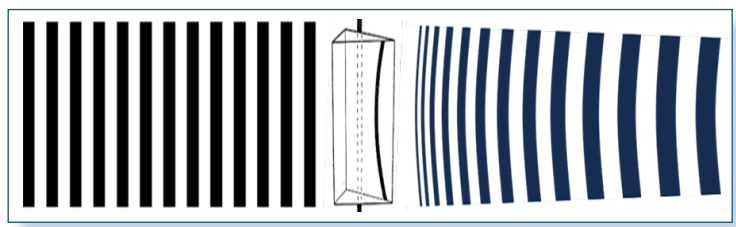


Figure 6. Shows the non-linear angular magnification along the line perpendicular to the base-apex line showing the gradual expansion of space from the base to the apex; the gradual increase in curvature from the base to the apex; the downward tilt towards the apex of the horizontal above horizontal center and the upward tilt towards the apex; and the perceived expansion of the spatial height in the base with compression of spatial height in the apex.

Thickness of Object Space

Up to this point we have described the non-linear relationships in the frontal plane, side-to-side and up-down. But how does a prism affect the space along a line perpendicular to a person, often referred to as the z-axis? If we could step out and look down on the object space, while someone is using a prism, what might we observe? Keeping with the illustration of using a base left prism and considering the spatial transformation properties noted above, and using the dashed lines representing the asymmetric angular magnification and the spatial perception previously illustrated, there would be a widening or expansion of space in the base and a narrowing or compression of space in the apex, as diagrammed in Figure 7. Doesn't this look like a prism?

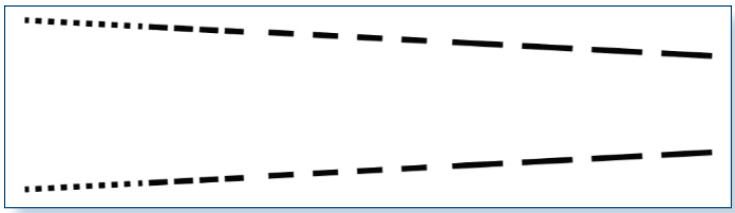


Figure 7. Expansion and Compression of Space

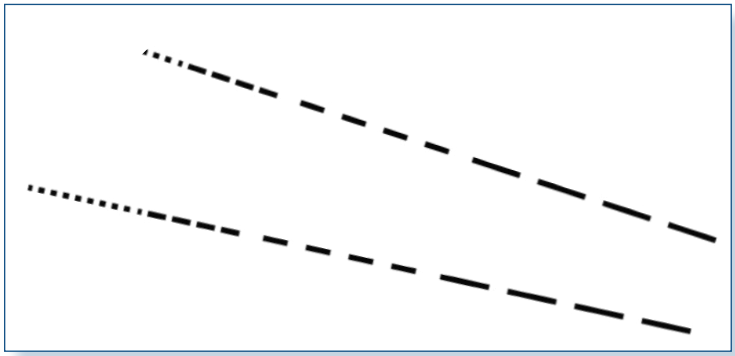


Figure 8. Spatial Rotation of Space

Rotational Relationship

Another spatial relationship associated with prism is the transformational spatial rotation of space. Objects do not just move side-to-side or up-to-down or appear closer or farther, rather there is a spatial rotation of the object space.

With a base left prism and looking at the object space from above, there is a rotation of space, as depicted in Figure 8.

After Effect

When wearing prism lenses for a period of time, adaptation occurs to these spatial transformations. While the observer might initially make errors in judgement based on their habitual frame of reference, perception ultimately shifts or recalibrates. Upon removal of the prism, the opposite perception occurs for a period of time until their baseline perception is restored.

Dioptric Values

Another property of prism is their interaction with dioptric power. Streff observed that students with myopia tended to rotate their heads so that they were looking through the apex of the prism for maximum clarity. Conversely those with hyperopia generally rotated their heads to look through the base of the prism to obtain maximum clarity. He addressed the optics of plano prisms through curved surface of +6.00 diopters front surface curvature, pointing out that there were dioptric changes that occur from the base to the apex. He found that in a 10-diopter prism there was an effective minus power through the apex and effective plus power

Table 1: Dioptric values of a 10-diopter prism

Angle of View for 10 Diopter Prism	Angle of View	Power at Axis 180	Power at Axis 90	Amount of Cylinder	Dioptrics
	40	+1.20	+0.54	+0.66	+1.20 -0.66 x 180
Towards the Base	30	+0.86	+0.41	+0.45	+0.86 -0.45 x 180
	20	+0.53	+0.26	+0.27	+0.53 -0.27 x 180
	10	+0.21	+0.10	+0.10	+0.21 -0.10 x 180
	0	-0.13	-0.06	-0.08	-0.06 -0.08 x 090
Towards the Apex	10	-0.52	-0.23	-0.29	-0.23 -0.29 x 090
	20	-0.98	-0.40	-0.58	-0.40 -0.58 x 090
	30	-1.52	-0.59	-0.93	-0.50 -0.93 x 090
	40	-2.13	-0.77	-1.36	-0.77 -1.36 x090

through the base.[10] (See Table 1) NOTE: for any prism value different than 10 prism diopters, these effects are still in play and are linearly proportional to the amount of prism used.

Streff's conclusion, in part, is that patients with myopia tended to prefer looking toward the apex due it affording them a view of the world through a lens with the spherical equivalent of -1.50. Looking through the apex gives the appearance of space moving closer to the individual. Patients with hyperopia look through the base because of the induced plus power that results from the oblique incidence of the rays hitting the lens. At the extreme, this is about +0.90 diopters.

BEHAVIORAL RESPONSE TO PRISMS

One can conceive of expected or canonic responses to prism on a monocular or yoked basis involving typical spatial shifts. This spatial transformation typically results in a shift in the center of gravity, with accompanying postural changes and pelvic rotation to the left or to the right with horizontal prism and postural shifts and pelvic rotation upward or downward with vertical prism, as noted in Table 2.

Table 2: Postural responses to the application of Prism. Adapted from Table 7.1, Canonic Responses to Prisms. Harris PA. The Use of Lenses to Improve Quality of Life After Brain Injury. In: P Suter and L. Harvey, eds. Vision Rehabilitation: Multidisciplinary Care of the Patient Following Brain Injury. Boca Raton: CRC Press, 2011:235.

	Eye Movement	Pelvis Shift
Base Down	upward	with center of gravity shift or postural change upward and pelvic rotation downward.
Base Up	downward	with center of gravity shift or postural change downward and pelvic rotation upward.
Base Right	leftward	with center of gravity shift or postural change and pelvic rotation to the right
Base Left	rightward	with center of gravity shift or postural change and pelvic rotation to the left

The Paradox in Compression vs. Expansion of Space

In the context of the optical, spatial, and behavioral properties of prism reviewed above, let's return to the apparent discrepancy between Dr. Paul Harris and Dr. John Streff regarding the compression and expansion of space.[11] As noted earlier, Dr. Harris wrote that there was expansion of space in the apex and compression of space in the base. Dr. Streff wrote that there was expansion of space in the base and compression of space in the apex. How can we reconcile such diametrically opposed views? What many have not understood is that Harris was working at the time primarily with flat prisms and all of Streff's work was done with curved prisms. It turns out that the induced plus and minus power, which is associated with curved prisms, is a major factor in how Streff described prismatic effects, which were absent in those prisms Harris worked with. Once understood, the differences melted away and both agreed on the primary spatial effects of prisms.

The resolution to this confusion lies in understanding the relationship between the two dimensional (2-D) spatial relationship of the physical optical properties of prism and a three dimensional 3-D perceptual interpretation spatial framework. In a conventional 2-D linear framework, prisms follow the physical properties referenced earlier. There is a shift of the image toward the apex and expansion or compression space differentially from the base to the apex in horizontal, vertical and z-axis space from the person outward. In a 3-D spatial relationship, the entire visual field undergoes spatial transformation across all directions and dimensions. This volume of 3-D space is represented in object and personal space by the frontal (vertical) plane, transverse (horizontal) plane and the sagittal (depth) plane with the corresponding x, y and z-axes. As the rays of light approach the prism, as illustrated in Figure 3, they encounter the prism surface varying angles and emerge at correspondingly different angles. This spatial transformation influences both the physical spatial property, as demonstrated in Figures 7 and 8, and the perceived spatial property, as illustrated in Figure 10. With prism, the spatial transformation property is the same in terms of quality but differs in terms of amount of deviation.

We'll delve more into that shortly, but before doing so a comment on terminology is in order. For orientation purposes, we will refer to the terms

of centrifugal and centripetal to describe perceived movements toward or away from the individual. The word centrifugal describes a force or an actual or perceived movement away from a center or axis plane, in optometry typically used as away from the self. Centripetal on the other hand describes a force or an actual or perceived movement toward a center or axis plane, in optometry typically used as toward the self. As an example, when one shifts from a distant part of the string to a place closer to them on the Brock String, and the eyes rotate to converge, there is a centripetal movement toward the individual. This is referenced as objects appearing to move inward or closer and matches what occurs through the apex of a base out prism. Conversely, centrifugal movement is referenced as objects appearing to move outward or further from the individual. The angular rotation of the eyes reflects a divergence movement and matches what occurs through a base in prism. In essence this type of distortion through the apex or base is an illusory translocation of space.

With regard to 3-D space, relational cues of relative size also come into play. The individual perceives that the expanded parts appear closer to us which would be a centripetal perception. The compressed parts appear further away and are referred to as a centrifugal perception.

The 3-D Spatial Relationship

Referencing a 3-D framework, Streff noted that there is the illusion of the compression of space toward the apex and expansion of space towards the base. His observation was related to the appearance of a spatial rotation. He reported that parts of an object (a wall for example in the frontal plane) appeared to look closer to him through the apex and further away in the base when looking through a large value base down prism. This illusion of the spatial rotation is a concept that involves the utilization of the perceptual cues of relative size and size constancy.

In a 3-D framework, cues of relative size result in the interpretation of the enlarged parts appearing closer and the compressed parts appearing further away. In essence, along with the displacement of the image, this type of distortion through the apex or base creates an illusory rotational translocation of space.

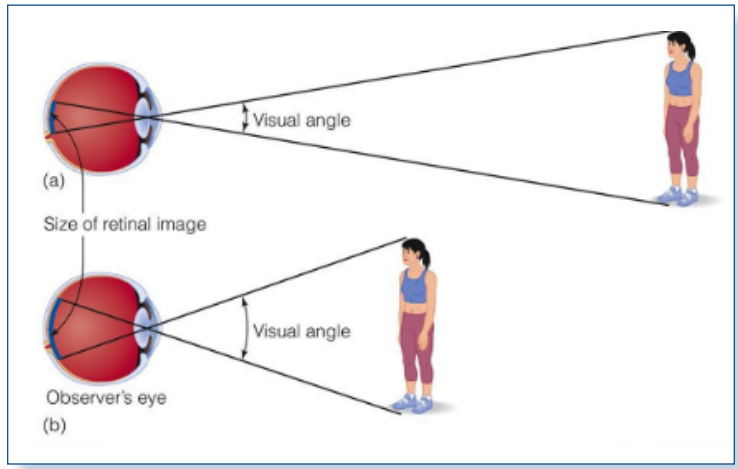
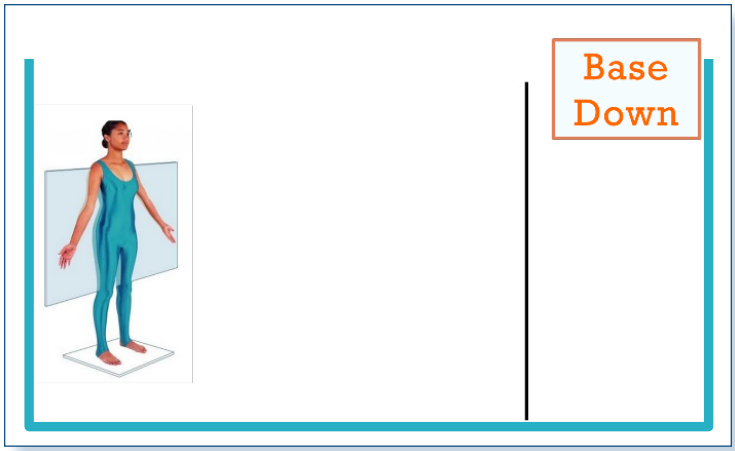


Figure 9. Relationship between visual angle and apparent distance.

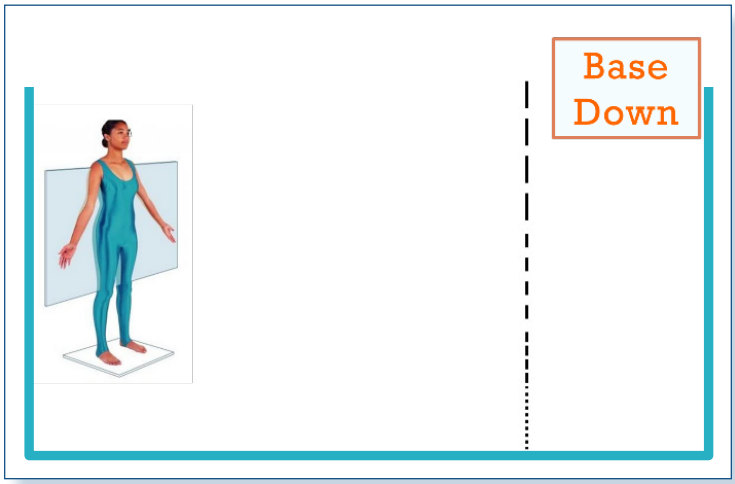
Relative size and size constancy dictates that when viewing an object that appears smaller in size, we perceive that object as farther away from us. When that same object appears larger, we perceive it as being closer to us, not larger. (See Figures 10 a and b).

When looking through the prism at the same object there are parts of that object that appear smaller and others that appear larger. Hence the illusion of compression or expansion of space along the base-apex line in a 3-D framework. Why is this? Recall our previous discussion about the non-linear magnification properties of the prism. In reference to where you are looking through the prism, the base side compresses space and the apex side expands space. Due to relative and size constancy perception, the parts of the object that look larger appear to be closer and the parts that look smaller are perceived to be further away. When looking at objects in the frontal plane you will note that there is a rotational tilt. With a base left prism, the left side of the object will look further away and the right side of the object will look closer to the person. With a base down prism, the lower area of regard will look further away (see Figure 1).

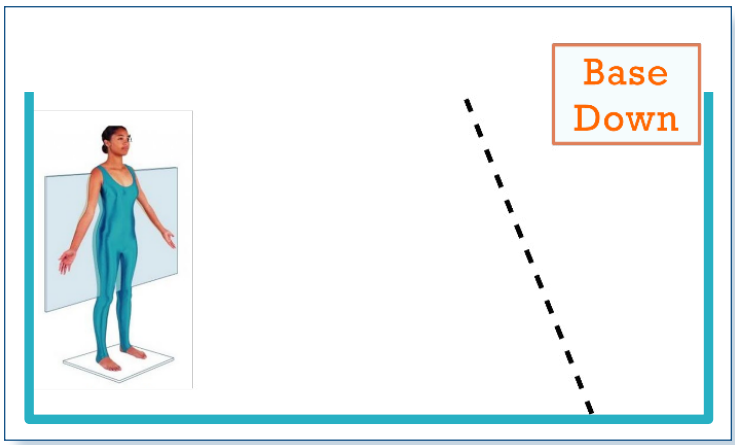
Streff wasn't the only one to write about this 3-D concept. Rock[12] in his book, The Nature of Perceptual Adaptation and Templeton and Howard[13] in their book, *Human Spatial Orientation*



In 10a, a person stands in front of a wall, represented by the black line, wearing a base down yoked prism.



In 10b, the wall, represented by the dotted line, is the non-linear spatial transformation created by the prism.



In 10c, the tilt of the wall is represented by the tilted dotted line and due to size constancy and relative size perception the wall appears uniform again.

Figure 10. The illusory effect of base down prism on perceived distance in the frontal plane.

also reference the illusion of a tilt in the frontal plane where the projected image appeared to be closer through the apex and further away through the base. The change in perceived space through the prism therefore depends on whether a linear or spatial framework is adopted, as depicted in Table 3.

Table 3: Illusory effects o prism.

Prism	Linear (2-D)	Spatial (3-D)
Apex	Wider (expansion)	Closer (centripetal)
Center	Neutral	Neutral
Base	Narrow (constriction)	Farther (centrifugal)

CONSIDERATIONS IN PRESCRIBING PRISMS

The standard conventional, or classical approach to prescribing prism for compensatory purposes is oriented toward neutralizing diplopia, or to satisfy comfort and performance criteria based on measurements of vergence dysfunction. The behavioral, developmental, and neuro-rehabilitative approach to prescribing prism for therapeutic purposes relies more heavily on the clinical observation of improvements in behavior and performance as prism is applied, bearing in mind the spatial transformation properties and postural behaviors noted above. It is noteworthy that the spatial transformation effect will most likely be perceived by the patient in the higher amounts of prism and generally used in active therapy. Although therapeutic prisms incorporated into the patient's prescription in lower amounts generally have less perceivable spatial transformation effects, they can still exert significant influence as will be discussed below.

Compensatory vs. Therapeutic Applications

Dr. Mel Kaplan[14] wrote that the two principal effects of prism, chief ray shifting used for compensatory purposes, and spatial transformation properties used for therapeutic purposes, cannot be separated from one another. They co-occur, and in some cases are interdependent. He noted that Skeffington, Getman, Apell, Streff and others reported that small lens powers may help some

patients make more profound changes at times than larger lens powers. This is because large powers tend to be more dissociative, with two prism diopters in any direction appearing to be the limit before some spatial distortion is present. Kaplan emphasized the application of yoked prisms, with bases in the same direction, and developed a specific protocol involving motor activities to gauge and refine the patient's response to prism.

As Harris notes, "The chief ray shifting effect of prisms cannot be separated from the spatial transformation of prisms. In many instances, the benefits of the prescribed prisms may result more from the spatial transformation than from the image shift."[2]

From a spatial transformation standpoint larger amounts might be used in vision therapy to induce positive disruptive changes in orientation and mobility whereas smaller amounts might be prescribed to influence proprioceptive orientation to provide a positive improvement in visual behavior for an improved relationship between where am I (Antigravity) and where is it (Centering). Some prism amounts and directions afford the patient an improvement in their performance where others show no change or a decrement in performance.

The decision to utilize prism therefore depends on their purpose. Prism may be purposefully disruptive at the outset, guiding the patient to re-organize visually. Therapeutic prism typically modifies the patient's behavior and performance and is designed to help the patient develop a different output with the anticipation that the prism may not be needed in the future. Therapeutic prism is often used in conjunction with an office-based vision therapy program.

Particularly as related to yoked prism, an overarching goal is to develop a more flexible relationship between the visual streams mediating "where am I" and "where is it" through neuro-muscular re-education. In his article on a rationale for the use of prisms in the vision therapy room, Dr. Robert Fox[15] focuses on the use of prisms in the range of 5 to 15 prism diopters and writes: "Prism is a powerful tool. The various optical distortions it provides allow for neural changes that affect the patient's sensory-motor and vestibular-anti-gravity systems." The basis for the induced distortions to which he refers are the spatial transformations noted above. In addition to making spatial adjustments for image shift, the person must allow for the illusion of tilts and slants of the visual world as well as size changes afforded by the prism. In this case, prisms alter the perceived reality to the point that the habitual visually directed neuromotor organization is no longer valid. The individual then interacts with the world to organize new spatial localization percepts using proprioceptive and kinesthetic feedback. Together with visual processing, ongoing activity to develop a more efficient and flexible relationship between the "where and I" and "where is it" systems is developed and monitored.

REPRESENTATIVE CLINICAL APPLICATIONS

A variety of tests and protocols have been proposed for determining the direction and amount of prism for therapeutic purposes, particularly as it pertains to yoked prism. Our intent is to provide some representative approaches, rather than to undertake an exhaustive literature review.

Egocentric Localization

Ciuffreda and Ludlam[16] address the spatial effects of yoked prism in terms of egocentric localization, citing the work of Padula as related to visual midline shift. In a variety of contexts, they have pointed out that visual midline shift is a misnomer because it is not a shift per se (e.g. lateral with respect to the body midline), but rather a polar-based, rotational deviation from the normal, midline-based reference point, thus reflecting one's three-dimensional, egocentrically-based, directional map of visual space.

Ciuffreda and Ludlam also address work done in conjunction with Suchoff using prism probes while the patient navigates the center of a long hallway. The application to hemianopia, with or without inattention, is beyond the scope of this paper. Their methodology highlights an important principle with regard to yoked prism in neuro-rehabilitation. The key point is that we as optometrists primarily are

concerned with how people move through space and watching their response to sets of prisms with them moving is necessary to seeing how the person can use them. The maximum amount of yoked prism they use is up to 10^ at distance and 15^ at near, but the typical values prescribed are in the 2^ to 6^ range. After a no-prism trial in navigating the hallway, the doctor or therapist observes the effect of yoked prism in various directions and amounts on improvement in ambulation and visual scanning and elicits subjective responses from the patient as well. They note that the minimum amount of prism that accomplishes this goal should be used, as larger amounts may cause spatial distortion due to the nonuniform magnification properties of prisms and/or substantial sensorimotor adaptation problems.

Visual Midline Shift

A midline shift is observed when the patient leans (tilts) to one side or the other or forwards or backwards, as originally introduced by Padula in the framework of the Visual Midline Shift Syndrome (VMSS).[17] It is thought that the VMSS is due to body control on the side of the neurological insult. Through the application prism, the individual may be observed to have better posture, have a more efficient gait and demonstrate more confidence with movement and motion. If this is truly a muscle weakness, how does the application of yoked prism assist the patient to walk in a more upright posture? As an alternative, VMSS may reflect alteration in visual perception which is different than muscle weakness. Applying the spatial transformation properties of prism noted above, a prism can be designed to alter the spatial perception to allow the person to see a more upright world and the person can then alter their posture to be more upright and erect.

Proprioceptive Dysfunction

Da Silva has streamlined a protocol for what is termed Postural Dysfunction Syndrome (PDS).[18] Formerly known as Proprioception Dysfunction Syndrome, this is an approach to prism prescribing which we might conceive of as a spatial vector at oblique axes, The amount of prism prescribed tends to be in the 1 to 4 prism diopter range with 2 to 3 prism diopters as the most frequently prescribed amount. The protocol involves observations of posture and movement ranges of the head, arms and feet in addition to visual observations of vergence and spot or sector scotomas. The prism orientations are typically in the oblique direction of 125 degrees in the right and 55 degrees in the left. In addition to the proprioception qualities of the prism values in his protocol there is also the consideration of the spatial transformation properties of prism that may be participating in the patient's overall behavior performance to the prism.

Postural Shifts

Yoked prisms are sometimes prescribed for specific populations to address particular visual characteristics.[19] Kaplan popularized this approach for children on the autistic spectrum. As reviewed by Dr. Stacey Coulter and her colleagues, yoked prism lenses are an optometric intervention that aims to improve visual spatial organization and visual integration abilities.[20] Consistent with our overview of the spatial transformations involved, they note that yoked prism lenses impact vision several ways. Generally larger amounts of prism values are considered for treatment in the autism population. They shift the field of vision, encourage a gaze shift toward the prism apex, and create a non-uniform change in magnification across the visual field, all of which alters their peripheral awareness. In addition, the spatial transformation properties create a visual perception that is vastly different than their habitual view of the world so now they have to "take inventory" and look around and begin to pay attention to their visual world.

With regard to children on the autistic spectrum who exhibit toe walking, in which their center of gravity is shifted forward, the theory is that yoked prism bases down will result in a postural shift upward thereby re-positioning the center of gravity. One might conceive of this as anterior visual midline shift. Additionally, there is the consideration that other spatial transformation

properties may play a role in shifting the center of gravity. With base down prism, the floor, being on the transverse plane, will now appear to slant upward and frontal plane space will have the illusion that it is up space is tilted towards them so the patient makes a postural counterbalance to right himself to having a world with parallel walls and floors. There is a school of thought that posits the inverse, namely that yoked prism bases up will result in transfer weight bearing from the toes to heels resulting in normalized heel-to-toe walking. This is because the initial shift in center of gravity will result in a postural shift forward that is destabilizing, and the child counteracts this by adopting a postural shift upward. With base up prism, the visual perceptual awareness skew in that the floor appears to tilt downwards and the frontal plane up space is perceived to tilt away from them. This creates an initial shift in center of gravity that results in a postural shift forward that is destabilizing, and the child counteracts this by adopting a postural shift upward.

The different schools of thought in using yoked prism as disruptive or stress-inducing, versus counter-stress, and their effects on perceived space and postural adjustment, were reviewed by Press.[21]

Visual-Auditory Localization

The Z-Bell test is a probe of visual-auditory localization developed by Dr. Deborah Zelinsky.[22] The patient is seated in various positions and asked to close their eyes. With eyes closed they are instructed to reach out and touch the bell after hearing the sound using different hands. The bell is placed in a variety of different positions relative to the person. Then lenses or prisms are applied and the patient is retested. The accuracy of spatial localization helps to determine the prism dioptric value and base direction as well as a lens value that synchronizes and balances visual-auditory spatial localization. This is reportedly effective with a variety of patients including those who have experienced stroke or traumatic brain injury (TBI). The mechanism through which prism changes localization has been posited to be that the low-level light impacting the non-image forming signals to the brainstem for controlling such processes as posture and spatial awareness.

Vision and Learning

In a set of articles with emphasis on vision and learning, Dr. Leonard Press proposed a protocol for the prescribing of small amounts of prism in the vertical and horizontal directions.[21] His approach utilizes a combination of associated and dissociated phoria measurements comparing distance to near findings in free space. Press noted that children who have vision-based reading difficulties often have a compression of visual space resulting in reduced vertical vergence ranges in both directions. This may be indicative of a crowding phenomenon, and when symmetrical can be an indication for the prescribing of small amounts of yoked prism. When combined with prism in the horizontal direction as indicated by testing, prism may result in a neuro-therapeutic effect based on stabilization as well as recalibration of visual space. Please see Appendix A for ancillary concepts that supplement these clinical applications.

CONCLUSION

We have discussed the spatial transformation properties of prism in the context of three principal factors. One is the deviation of light as a dioptric function of the angle relative to the eyes. The second is asymmetric angular deviation that results from differences in incident angles of reflected light relative to ray point origins. The third factor is asymmetric or nonlinear magnification. These properties result in the perception of compression or expansion of visual space along the base-apex line that vary depending on whether one adopts a 2-D or 3-D framework.

Beyond traditional considerations of prism that facilitates fusion and shifting of the visual field, the behavioral and performance changes that occur with spatial transformation properties of prism can be related in part to changes in angular rotation of the eye, adaptive shifts in the head and neck region, or postural changes translated through pelvic rotation. (See Table 3). This can occur with

prism in any orientation, but is most dramatic with yoked prism, particularly in the vertical direction.

Yoked base down prisms theoretically elevates visual gaze and stimulate an anterior rotation of the pelvis to create an extension and a more open body posture. In addition, there is the illusion of the expansion of space in lower space and compression of space in upper space. Yoked base up prisms theoretically depresses visual gaze, stimulate a posterior rotation of the pelvis to create a flexion in body posture to stimulate a more centered or grounded body posture and compress perceptual space.

We have reviewed how clinicians harness the various properties of prism in a variety of contexts, amounts, and directions based principally on their perceived alteration or transformation of visual space. The applications to asthenopia, diplopia, and visual field changes based on the chief ray light path, which is the conventional shifting of the image in space, have been amply covered elsewhere.[31,32]

NOTE: This article is based in part on a presentation given by Dr. Dean at the 50th Annual Meeting of COVD titled *Spatial Transformation Properties of Prism*. Available as General Session #8 at **https://tinyurl.com/3wvvspk2**

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Dr. Dean is a member of the Optometric Vision Development and Rehabilitation Association (OVDRA), in 2010 he completed the fellowship process. Members of OVDRA are optometrists who specialize in working with children and adults experiencing difficulties in the areas of binocular vision, focusing, eye teaming, strabismus and amblyopia and visual processing skill.

Dr. Dean coordinates with parents and educators in writing IEP programs and helping to write accommodations and 504 modifications. He also coordinates sensory integration programs with Occupational Therapy, Physical Therapy and Behavior Medical Specialties.

Dr. Dean is a member of the Neuro-Optometric Rehabilitative Association (NORA). Neuro-Optometric Rehabilitation is an individualize treatment regimen for patients with visual deficits as a direct result of physical disabilities, traumatic brain injury and other neurological insults. He provides treatment for people with visual field loss, double vision, visual balance disorders, visual midline shift syndrome, post trauma vision syndrome, light sensitivity and reading disorders.

Dr. Dean is also a member of the American Optometric Association (AOA); South Carolina Optometric Physicians Association (SCOPA); and the Optometric Extension Program Foundation (OEPF).

Spatial Transformation Effects

The spatial transformation effect depends on: base curve; distance from the eye; prismatic power; thickness of the prism and the orientation of the prism before the eye.

The World is a Prism, if You Had a Prism

The visual world can be conceptualized as behaving like a triangular prism because when viewed through a triangular prism, the entire visual space undergoes a systematic spatial transformation that correspond to the geometry of the prism itself. In a 2-D space, the reduction in object-space dimensions from the prism base toward the apex produces the perception of tilted spatial planes. Specifically, the floor appears to tilt upward towards the apex and the ceiling appears to tilt downward towards the apex, creating a visual configuration resembling the triangular geometry of a prism.

In 3-D space, the transformation extends across all spatial dimensions, with the perceived visual field becoming expanded and wider toward the prism base and compressed and narrower toward the apex. This altered spatial organization mirrors the three-dimensional structure of a triangular prism. When the frontal plane undergoes apparent tilt, the resulting spatial orientation reflects the angular characteristics of the prism geometry. Therefore, the shape of the prism itself provides a visual model for understanding how a prism transforms perceived space.

The Magnitude of Prism

A survey of COVD members on the prescribing of vertical yoked prism for their clinical population showed that among 123 respondents, 9.3% prescribed less than one prism diopter, 33.6% prescribed one prism diopter, and 41.1% prescribed two prism diopters.[22] This reinforces the concept that when the decision is made to prescribe prism, the least amount should be used that provided the maximum compensatory or therapeutic effect. It is not unusual to measure a large value only to have a small prism value allow the patient to have better performance.

Orientation and Mobility

It is common in low vision rehabilitation to test and train orientation and mobility, particularly when a patient is acclimating to optical aids. A widespread application of this is "the hallway test" in which the patient is asked to walk a corridor with and without yoked prisms in place to assess its effect of visually guided movement. It can be helpful to video-document these trials for demonstration purposes, and Merrill Bowan, OD has a YouTube channel dedicated to such demonstrations which includes the hallway test as well also other applications of microprism, generally defined as prism in the region of 1 to 2 prism diopters.[25]

Undesirable Prismatic Effects

It is well known that progressive addition lenses (PALs) induce varying degrees of peripheral spatial distortion, as well as variable gain in the vestibulo-ocular reflex (VOR) contributing to adaptive difficulties for some patients.[26] Lesser known is the fact that many ophthalmic laboratories incorporate yoked prism down as a lens thinning procedure to improve the aesthetic appearance and reduce the weight of the lens.[27] The amount of yoked prism is approximately two-thirds of the add power. That means the induced yoked prism base down effect on PALs will range from one-half to two prism diopters. It could well be that the adaptive difficulty encountered by some patients with PALs is related to the spatial transformation properties induced by yoked prism incorporated into lenses. Parenthetically a toric contact lens typically incorporates base down prism to minimize lens rotation, inducing anywhere from 0.52^ to 1.15^ through the optical center according to one study.[28]

Guided Illusory Effects

Given Prentice's Rule, any spectacle lens has an induced prismatic effect when looking away from the optical center. Analogously, in light of our earlier discussion about transformations of space, it should be clear that all prisms have induced spatial effects that simulate the size properties of dioptric lenses when looking away from the center of the prism (see Table 4). The clinician can harness this when the effect of a plus lens is desired, but the patient doesn't exhibit plus lens acceptance due to distance blur factors, or AC/A variables.

Typically, when prescribing a plus lens, the desired result is to create a centrifugal spatial shift where the virtual image is further away. To simulate that effect, the clinician can prescribe prism that results in expansion of space in the base. Petrosyan noted that yoked base down prism shares some spatial characteristics with plus lenses in that it expands visual space volume.[29] Although he didn't address the spatial characteristics of prism, Peckham identified one hundred years ago that base-in prism had a salient effect on increasing the patient's plus lens acceptance.[30] However, due to the differential effects of looking through the apex versus the prism, as well as the differential behavioral responses to prisms, clinical probes are necessary prior to prescribing, as the inverse of what is expected sometimes occurs.

PERSPECTIVE

Head Tilt Secondary to Vertical Heterophoria: An Overlooked Cause of Neck and Back Pain in Ocular-Cervical Postural Syndrome (OCPS)

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ABSTRACT

A Binocular vision disorder that is frequently untested and therefore undetected may contribute to significant morbidity within the general population. While patients with Ocular-Cervical Postural Syndrome (OCPS) may or may not have visual symptoms, they often seek care from diverse medical specialists for neck and back disorders.

A vertical heterophoria accompanied by a compensatory head tilt adopted to maintain binocular alignment results in OCPS. Individuals with this condition commonly report chronic neck and upper back pain and/or reduced cervical mobility. The underlying etiology of these symptoms is often undiagnosed, misdiagnosed, attributed to stress or considered a normal baseline variant.

Optometrists are uniquely positioned to identify this condition through evaluation of vertical heterophoria and to provide treatment through compensatory prism correction, vision therapy, and visual hygiene interventions.

Interdisciplinary collaboration is encouraged. Medical professionals encountering patients with unexplained cervical discomfort or persistent head tilt should consider referral to optometrists specializing in binocular vision disorders for further evaluation.

A hypothesis is also presented suggesting that the prevalence of vertical heterophoria may be increasing, potentially associated with prolonged digital device use and forward head posture, commonly referred to as "Tech Neck."

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INTRODUCTION

Vertical heterophoria, often overlooked in general eye care, may contribute to neck and upper back morbidity in both visually symptomatic and asymptomatic individuals.[1] The term *individuals* is emphasized rather than *patients* because many affected people may not present for eye care. The term Ocular-Cervical Postural Syndrome(OCPS) is proposed to categorize this syndrome.

Many practitioners of Binocular Vision Dysfunction (BVD) limit its clinical consequences to asthenopia, visual processing interference, avoidance of reading and/or pseudomyopia.

However, a review of the anatomy and physiology of the visual, vestibular, neurologic, and cervical systems suggests that these systems are highly interconnected and function in a symbiotic relationship.[2]

It is well documented that the visual system maintains complex interrelationships with other bodily systems.[3] Many ocular structures share embryonic origins with other organs and connective tissues, and therefore may be subject to similar

pathologic processes. Eye care practitioners routinely observe and manage the significant effects of systemic diseases, such as diabetes and thyroid eye disease, on the eyes, adnexia, and binocular system.

The significant interrelationship between the eyes, brain, vestibular system, balance, and posture has contributed to the continued evolution and expansion of the subspecialty of neuro-optometry.

While these systemic influences on ocular health are well recognized and routinely addressed in clinical practice, the potential impact of musculoskeletal dysfunction on binocular alignment — and conversely, the influence of binocular dysfunction on musculoskeletal health — remains largely underrecognized.

A review of the peer-reviewed literature reveals limited discussion of the potential effects of vertical heterophoria on the musculoskeletal system in *visually asymptomatic individuals*. If such relationships exist, eye care practitioners may be able to expand their role in interdisciplinary patient care by identifying and treating a visually induced contributor to musculoskeletal morbidity.

Therefore, an important question remains: *what is the relationship between relatively benign, often overlooked vertical heterophoria-with its often associated compensatory head tilt-and disorders of the neck and upper back?*

The primary purpose of this paper is to inform eyecare practitioners about the importance of addressing patients with vertical heterophoria, whether or not they present with visual symptoms. A secondary purpose is to raise awareness among other medical specialties that the visual system may contribute to chronic neck pain and upper back morbidity.

Vertical Heterophoria

Vertical heterophoria is a subtype of binocular vision disorder resulting from a vertical misalignment of the eyes. The incidence of vertical heterophoria has been estimated to range from 7% to 52%.[4] While this range appears excessive to many experienced clinicians, it nevertheless suggests that vertical heterophoria is not a rare finding.

Within the specialty of optometric management of head trauma, the incidence of vertical heterophoria is undoubtedly higher, secondary to damage to the Trochlear nerve.[5]

Vertical heterophoria may result from several etiologies, including orbital asymmetry, cranial nerve damage, tendon sheath disorders, neurologic disease, systemic conditions, vestibular disorders, and a newly theorized origin commonly referred to as "tech neck".

It is well documented that vertical heterophoria can cause a variety of symptoms, including headaches, diplopia, dizziness, anxiety, and other psychological manifestations.[6] Correction through the use of compensatory prism can ameliorate these symptoms.[6]

Head Tilt

Head tilt, often called torticollis, is an abnormal posture where the head leans towards one shoulder.

Musculoskeletal conditions such as Congenital Muscular Torticollis, Cervical Dystonia (Spasmodic Torticollis), arthritis, herniated discs as well as central neurological disorders could be a cause.

Ocular torticollis is a compensatory head posture tilt, turn or lift adopted to fix vision issues. It is most commonly associated with vertical heterophoria, strabismus, Fourth Nerve Palsy, partial Third Nerve palsy, orbital asymmetries, and Infantile Nystagmus syndrome.

Vestibular disorders such as Benign Paroxysmal Positional Vertigo and Meniere's Disease may produce head tilt due to disturbances in spatial orientation. Loss of hearing in one ear can cause a person to tilt their head to the better hearing ear.

Neck Pain

Neck pain is a major global cause of disability. According to Physiopedia, a widely used physiotherapy reference resource, neck pain ranks fourth among causes of disability worldwide. The incidence of neck pain is reported to range between 10%-20%.[7] Prevalence is higher in women than men, higher in high-income countries, higher in urban areas and peaks around 45 years of age.[7]

It is likely that most individuals have experienced neck pain at some point in their lives. The incidence of neck pain is likely increasing due to modern societal demands. Humans are upright animals, and the visual system most likely evolved primarily for horizontal distance viewing, such as that required for hunting and navigation.

Accordingly, the neck experiences the least musculoskeletal stress when the head is maintained in a neutral, forward-facing position. A review of the anatomy and physiology of the neck and spine in the following discussion clearly supports this premise.

As survival is paramount in all species the practicality for directional mobility is built into the cervical anatomy and physiology.

However, modern lifestyles increasingly require individuals to spend prolonged periods of time looking downward at desks, computers, and digital devices. According to TechNewsWorld, an e-commerce technology news outlet, the average daily screen time for individuals in the United States is approximately 6 hours and 40 minutes.[8]

Therefore, it is not surprising that the excessive mechanical stress on the musculoskeletal system from an abnormal head posture may create cervical problems.

The Connection

It may be injudicious to assume that the visual system, and therefore binocular function, operate independently of the musculo-skeletal and postural systems.

Behavioral optometrists have long proposed connections between the visual system and the rest of the body. In 1963, A.M. Skeffington introduced his well known "four circle" model, which conceptualizes the visual system as interconnected with balance(anti-gravity), movement and speech.[9]

Behavioral optometrists have noted that foot direction (inward or outward pointing) may sometimes mirror ocular alignment patterns, such as under- or over-convergence. There are studies that support these clinical observations.[10] Likewise, some reports have suggested relationships between posture and refractive error.[11]

A brief review of the neck anatomy and physiology may help illustrate the complex relationship between visual input, head posture, and musculoskeletal alignment. The purpose of this discussion is not to fully integrate cervical biomechanics into optometric care, but rather to highlight the interconnected nature of these systems and their potential influence on each other.

The cervical spine consists of the first seven vertebrae (C1-C7). It provides structural support for the head, protects the spinal cord, and allows for a wide range of head movements necessary for visual orientation in space. The first cervical vertebrae (C1), known as the atlas, articulates with the base of the skull and supports much of the weight of the human head, which averages 10-13 pounds. The second vertebrae (C2), known as the axis, allows (C1) to pivot, facilitating rotational movements of the head that are essential for coordinating gaze shifts and maintaining visual fixation during head movement.

The cervical vertebrae are connected by facet joints that allow forward, backward, and rotational movements of the neck and head. Surrounding the spine is a complex network of muscles, nerves, tendons, and ligaments, while shock-absorbing intervertebral discs lie between adjacent vertebrae. Major muscles associated with the cervical spine include the sternocleidomastoid, trapezius, levator scapulae, erector spinae, deep cervical flexors and the suboccipital muscle group.

These muscles contain an exceptionally high density of proprioceptive receptors, including muscle spindles and Golgi tendon organs, embedded within the muscle tissue. Sensory input from these receptors contributes to vestibular reflex pathways that are essential for detecting head position relative to the trunk, coordinating eye movements, stabilizing visual input, and maintaining balance.[12]

Together, the cervical vertebrae, facet joints, proprioceptive receptors, and associated musculature contribute to the maintenance of optimal posture and head position. Postural control is further regulated through the integration of sensory information from multiple systems, including visual, vestibular, and

somatosensory inputs. Among these, visual input plays a critical role in postural stabilization by providing spatial orientation cues that guide head positioning and facilitate balance during both static and dynamic activities.[13]

The spinal column is not perfectly straight. The upper spine is curved inward toward the nose while the lower spine has the reverse curve. This normal cervical curvature is called cervical lordosis. This lordosis serves to maintain structural stability, shock absorption, and efficient support of the head while allowing normal mobility. Proper alignment of the head over the cervical spine allows the visual axis to remain relatively stable during fixation and binocular tasks. When the normal cervical curvature of the spine is lost the weight of the head is no longer properly balanced.[14]

It makes sense that if the head has an abnormal shifting the weight is unequally distributed. Head tilting drastically increases gravitational forces on the neck, turning a manageable 11 pound head into a 27-60 pound weight at various angles.[15] Additionally, the activity of the vestibulo-ocular reflexes are altered which can lead to vertigo, dizziness and balance issues.[16]

This causes strain and potential anatomical changes in cervical and thoracic muscles, spinal discs, ligaments and joints leading to pain, stiffness, headaches and potential nerve damage as the body struggles to support the extra load.[17]

Electromyography (EMG) studies of the splenius and upper trapezius muscles have demonstrated altered muscle activity in individuals with forward head tilt. These individuals reported chronic neck pain compared to a control group.[18] Such findings suggest that abnormal head posture stresses the cervical muscles.

Tech Neck

The relevance of discussing tech neck in this context is to illustrate a parallel syndrome in which a visually driven abnormal head posture results in secondary musculoskeletal consequences. A further hypothesis is that these may not represent parallel systems at all, but rather components of a highly interconnected system.

"Tech Neck" is a visually driven syndrome that has been more widely described in other medical specialties. The term was first coined in 2008 by Dr. Dean Fishman, a chiropractor in Florida. According to the University of Nebraska Medical Center it is classified as an overuse syndrome.[19]

Tech neck refers to chronic neck pain resulting from prolonged downward head posture during the use of mobile phones, tablets and other electronic devices. This persistent strain on the bones, nerves and muscles can result in muscle stiffness, joint inflammation, pinched nerves and potentially even herniated discs.[20]

The degree of forward head posture associated with tech neck can be quantified by measuring the angle of the tragus (the cartilaginous prominence anterior to the ear canal) and the C7 vertebra using a protractor like device and/or postural imagining techniques. An angle measuring less than 50 degrees was considered an abnormal forward head tilt.[21]

Mechanical stress in the cervical spine can induce poor posture and abnormal body alignment. A variety of anatomical and physiologic adaptations have been observed in association with prolonged neck flexion, including changes in the length and strength of cervical musculature and weakening of the thoracic wall and middle back muscles.[22] Many chiropractors add impaired balance, altered respiration, digestive disorders and anxiety disorders as further consequences.

The more one researches tech neck, the more one encounters research from numerous other professions and its effects on other bodily functions.

Case Studies

It is well recognized that patients with a vertical ocular misalignment may experience diplopia unless compensatory mechanisms are employed. The first line of compensation is most likely the use of vertical fusional vergence through increased extra ocular muscle activity. This strain on the vergence system can lead to symptoms commonly associated with Binocular Vision Dysfunction (BVD).

Another compensatory mechanism to avoid both diplopia and reduce asthenopia is a down-

ward head tilt, usually to the side with the hyper deviation. This posture is frequently observed clinically and should be considered a potential red flag for vertical heterophoria. Such a strategy appears logical, as most striated muscles fatigue and diplopia isn't well tolerated.

As previously discussed a persistent head tilt could disrupt the intrinsic balance of the muscles, joints, tendons, ligaments in the cervical region and the vestibulo-ocular reflexes.

While many symptoms may arise from this persistent head tilt[23] neck and upper back morbidity is discussed in these case studies.

The two cases discussed in this paper were selected because they were visually asymptomatic except for refractive error. Both were seen in a private practice setting for routine refraction and ocular health evaluation. Presumably, these patients did not mention cervical symptoms during the case history because they were unaware of any potential connection between their visual system and neuromuscular neck status.

Case One

A 23 year-old white female presented to a private practice for a routine eye examination. Her ocular history was remarkable for low myopia, and she denied any visual asthenopia.

Upon entering the examination room and sitting in the chair, an orbital asymmetry was noted. Her right eye appeared to be positioned a few millimeters higher than her left. The patient was completely unaware of this asymmetry and was also unaware that she intermittently tilted her head downward to the right.

Her corrected visual acuity at both distance and near was 20/20 in each eye with a refractive correction of -0.75 DS in the right eye and -0.50 DS in the left eye. Ocular health findings were unremarkable, and her general health was reported to be good.

At the conclusion of the examination, the patient was asked whether she experienced any neck or back discomfort. She responded enthusiastically, asking, "How did you know?" It was explained that binocular vertical asymmetry can often lead to a compensatory head tilt, which can contribute to chronic neck strain.

She reported a longstanding history of chronic neck and upper back discomfort. To manage these symptoms, she receives weekly chiropractic adjustments and massage therapy. While these treatments temporarily reduce her symptoms, they typically recur several days later. She rated her neck discomfort as approximately 3.5 on the 10 point subjective pain scale.

The option of incorporating a small amount of vertical prism into her spectacle correction was discussed as a potential method to reduce the compensatory head tilt and possibly alleviate her neck discomfort. She agreed to proceed with a prism correction. In this clinician's practice, the need and amount for prism correction in a non-stabismic patient is typically guided by fixation disparity testing.

Based on these findings, a prescription incorporating 2 prism diopters of base-down prism in the right eye was trial framed. The patient reported that her visual acuity appeared sharper and that both her eyes and neck felt more relaxed.

Her final prescription included her myopic correction with 2 diopter base-down prism in the right eye. She was asked to return in 3 months for follow-up.

Although she scheduled the follow-up appointment, she did not return. She was later contacted by telephone and expressed appreciation for the prism correction in her glasses. She reported that her neck and back pain had decreased to approximately "1" on the subjective pain scale and that she was visiting both the chiropractor and massage therapist less frequently.

Case Two

A 34 year-old Asian male in his fourth year of medical school presented to a private practice for a routine eye examination. His ocular and systemic health histories were unremarkable.

No obvious orbital asymmetry or history of head trauma was reported, and initially there was no apparent head tilt. Best corrected visual acuity was 20/20 in each eye at both distance and near

with a refractive correction of -2.75-0.50x20 in the right eye and -3.00-0.25x75 in the left eye.

Initial cover testing revealed no obvious vertical heterophoria, and the examination proceeded uneventfully. At the completion of monocular refraction, both phoropter occluders were opened and the patient was asked about the clarity of his binocular vision. He responded that the vision was very clear but concurrently asked whether he should be seeing two vertically separated Snellen lines.

Surprised by this comment, a repeat cover test was performed at distance while the patient maintained a straight head posture. This revealed a definitive 4 prism diopter right hyperphoria. As these findings were discussed, the patient began tilting his head downward to the right.

He was unaware of this head posture, however, his wife later confirmed that he frequently demonstrated this head tilt.

The patient was then asked whether he experienced neck or upper back discomfort.

Similar to the first patient, he appeared surprised and asked, "How did you know?" He rated his neck discomfort as approximately 4 on the 10 point pain scale and attributed it to the prolonged studying required during his medical school studies.

The findings of vertical heterophoria, compensatory extra-ocular-muscle effort, and resulting head tilt were explained. A trial of 2 prism diopter base-down prism over his habitual right eye correction was demonstrated. He reported an immediate subjective improvement in both clarity and comfort.

A recommendation was made to incorporate a total of 2 prism diopters of vertical prism split between the two eyes in his spectacle prescription as a possible method of reducing his neck discomfort. It was emphasized that no guarantees could be made regarding improvement in neck pain, as such discomfort can have many etiologies.

He agreed enthusiastically, stating, "If I can reduce this neck discomfort and tightness , I am willing to try."

Two weeks later, he returned to pick up the glasses and requested to speak with the author. He reported that his vision was clear and that his eyes felt more comfortable. Five weeks later, he responded to a text message reporting that his subjective neck pain had decreased to approximately "2". He also expressed that he believed continued use of the prismatic glasses might lead to further improvement over time.

Tech Neck and Vertical Heterophoria

Based on clinical experience there appears to be an increase in the incidence of vertical heterophoria unrelated to cranial nerve disease or anatomical asymmetry.

This raises the question of whether there may be a correlation between the relatively new "Tech Neck" syndrome and a possible increase in vertical heterophoria.

Important mechanisms linking head posture and the musculoskeletal system are the vestibulo-ocular reflex (VOR), the Cervico-Ocular Reflex (COR) and the vestibulo-spinal reflex (VSR).

The VOR is an involuntary reflex that stabilizes vision by moving the eyes in the opposite direction of head movements.[24]

The COR uses proprioceptive signals from the neck joints and neck muscles to stabilize gaze by coordinating eye movements in response to neck stretches and neck twists.[24]

The VSR is a critical involuntary mechanism that maintains balance, posture and stability by inducing muscle contractions in the limbs and trunk in response to head movements.[25]

These reflexes work in synergy to keep you stable and oriented when moving.

The condition benign paroxysmal positional vertigo (BPPV) illustrates the clinical implications of these connections.

Within the semicircular canals, calcium carbonate particles (otoconia) normally move freely within the endolymphatic fluid and stimulate hair-like cilia, transmitting information about head position and balance to the brain. In BPPV, these particles become trapped and cannot move freely to compensate for head movement. When the patient suddenly sits up, tilts the head back, or turns to the side, abnormal signals are transmitted to the brain.

As a result, normal vestibulo-ocular coordination may be disrupted, producing symptoms such as vertigo, dizziness, lightheadedness, nausea, blurred vision and nystagmus. This condition typically resolves within several days, and canalith repositioning procedures are often curative.[26]

A hypothesis is therefore proposed that the sustained maladaptive head posture associated with tech neck may affect the vestibular system, which plays an important role in positional stabilization and ocular alignment. Disruption of this system could potentially contribute to the development of vertical heterophoria in patients without cranial nerve pathology or anatomical asymmetry. Thus, a possible physiologic mechanism for the apparent increase in vertical heterophoria may exist.

Alternative physiologic mechanisms are proposed by Ross Hauser, et al.[27] In this paper they propose that multiple ocular pathologies can be induced by forward head position (Tech Neck) common in today's societal demands. They propose that cervical structural dysfunctions induced by extended forward head position causes carotid sheath compression. This compression can lead to impaired venolymphatic drainage of the eye and brain by compressing the internal jugular veins. This can also cause ocular dysautonomia from a disruption in the parasympathetic/sympathetic system balance. Irregular blood flow and or drainage may impair the cranial nerves and or the extra-ocular muscle function which may impair binocular alignment.[27]

There are optometric case reports describing the development of a significant and symptomatic vertical heterophoria following trauma to the cervical area in several patients. The optometrist hypothesized a mechanism where the cervical damage caused the vertical heterophoria. The reporting optometrist subsequently began co-managing certain patients with a local chiropractor.[28]

If this hypothesis proves correct, an adjunctive treatment approach for some cases of vertical heterophoria may involve repositioning digital devices to promote a more natural gaze posture. Such ergonomic repositioning is commonly recommended by specialists in the management of tech neck syndrome.

Optometry is a Human Service

Optometry is not just a science; it is a human service. While we specialize in eye health and the visual system, we can aid the population in many other ways.

Optometrists can recommend supplements that support both visual function and overall systemic health. We can screen for conditions such as high blood pressure and counsel patients with diabetes on the importance of healthy eating and increased physical activity. In visual rehabilitation, we often recommend consultations for children with attention disorders, and we frequently work with school systems to assist with appropriate child placement, special needs support, and learning aids.

In addition, optometrists may aid patients experiencing neck or upper back pain by testing for vertical heterophoria and /or being observant for consistent head tilts. Besides assigning a possible cause and or symbiotic relationship between the vertical heterophoria and neck/back problems we can ameliorate these conditions by simple prismatic correction.

Raising awareness by interdisciplinary education can further aid both our profession and sufferers of unexplained cervical morbidity.

If the theorized origin in some cases is 'tech neck," we can recommend ergonomic modifications, such as raising digital devices or lowering chair height to improve posture and reduce strain.

Visually it is well established that constant close work can lead to accommodative-binocular dysfunctions as well as dry eye disease. Systemically, prolonged sedentary near work does not support a healthy cardiovascular system.[29] It may also contribute to cervical discomfort, decreased cervical mobility, and potentially irreversible musculoskeletal dysfunction.

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CASE REPORT

Vision Therapy and Vision Rehabilitation in a Child with ABCA4 Retinal Dystrophy

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ABSTRACT

Background

Diagnosis of gradual onset visual impairment can be complicated, with some testing results inconclusive. Additionally, patients with visual impairment can have concurrent binocular vision and accommodative problems that need attention. We report successful application of vision therapy in a young patient to reduce asthenopia when reading. When visual acuity became more reduced, macular pathology was diagnosed and vision rehabilitation was provided.

Case Report

An 18-year-old patient was followed for the last 10 years. During that time, she was successfully treated with vision therapy to alleviate blurred near vision and horizontal diplopia, which improved reading performance. Later, she required vision rehabilitation as her vision declined due to macular changes. Finally, the diagnosis of ABCA4, likely cone-rod dystrophy was made. Counseling the child on the visual diagnosis was managed uniquely due to significant concurrent life events that challenged routine patient education.

Conclusions

Young patients with visual impairment also need to be assessed for binocular vision and accommodative disorders. Vision therapy and rehabilitation can be provided to these patients for best outcomes and enhanced quality of life. Diagnosis and delivering the serious news of vision loss can be complicated in pediatric populations. Families will take the lead in how and when the news is delivered to the child.

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Keywords: convergence insufficiency; inherited retinal disease; vision rehabilitation; vision therapy

INTRODUCTION

Blurred vision, eyestrain, diplopia, and difficulty reading can present as symptoms of both accommodative and binocular vision disorders. Vision therapy is used to successfully treat convergence insufficiency and accommodative disorders.[1,2] Macular pathology can cause similar visual symptoms and should be ruled out before commencing vision therapy. It is important to realize that macular pathology and binocular/accommodative disorders occasionally occur simultaneously.[3] Vision therapy has been shown to be successful in patients with visual impairment.[4] We present a child who successfully underwent vision therapy for convergence and accommodative disorders. However, her intermittently blurred vision worsened

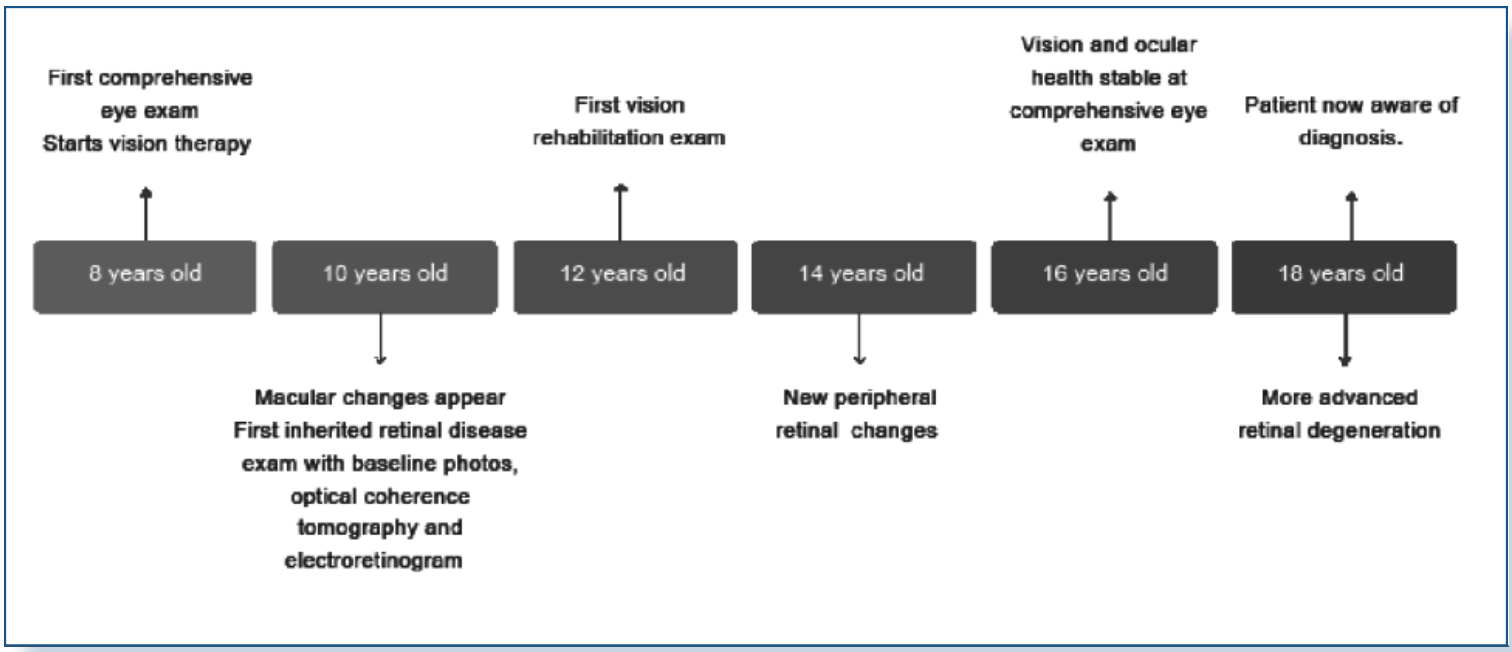


Figure 1. Timeline of patient care.

and only later was she found to have an inherited retinal disease. Delivering serious news in the pediatric population is complicated and difficult. In this case, for several years after the patient's retinal diagnosis, her legal guardian declined to inform her of the diagnosis to allow her to deal with other major psychological trauma in her life. No identifiable health information was included in this case report.

CASE PRESENTATION

Figure 1 provides an overview of this case report. An 8-year-old female presented to the Pediatrics/Binocular Vision Center of the Illinois Eye Institute complaining of reduced reading performance. She became sleepy when reading and occasionally experienced horizontal diplopia and blurred vision during near work. The child's medical history was unremarkable. There was a family history of macular degeneration causing vision loss in her maternal grandfather and maternal aunt before age 40. Her mother was seriously ill, contributing to a stressful home environment. Entering Snellen visual acuity with habitual spectacles for mild astigmatism was 20/40 right eye, 20/50 left eye at distance and 20/20 each eye at near. Pupils, extraocular muscles, and confrontation fields were normal. Cover test through habitual correction revealed orthophoria at far and 14 Δ intermittent alternating exotropia at near with low frequency and good control. Stereopsis was 20 sec. arc lateral disparity and 250 sec. arc global. Dry refraction yielded visual acuity of 20/25 in each eye at distance through +0.25 -1.00 x 180. Cycloplegic refraction with 0.5% cyclopentolate, 0.5% tropicamide and 2.5% phenylephrine spray revealed +1.25 -1.00 x 180 bilaterally. Intraocular pressures, anterior segment, and dilated fundus examination were unremarkable, with healthy optic nerves, cup/disc ratios of 0.3 and clear maculae. The dry refraction findings were prescribed for full-time wear; the patient was to return for 1 month follow-up.

The patient returned 4 months later, wearing her new spectacles. Aided acuities were 20/40+ at distance and 20/30 at near. Through these spectacles, cover test showed 2 Δ exophoria at far and 25 Δ intermittent alternating exotropia at near. Near point of convergence was 7 centimeters with alternating suppression and no recovery, using an accommodative target, and 7/10 centimeters (break/recovery) using a penlight with red lens. Worth 4-dot showed fusion at 40 centimeters and alternating suppression at greater distances (hence, central suppression based on smaller angular subtense of the target). Prism bar vergences at near measured x/10/4 for divergence and x/10/6

for convergence. Minus lens accommodative amplitudes were 4.00 right eye and 4.50 left eye. Accommodative facility binocularly using +/-2.00 D lens flippers showed suppression through plus (unfortunately accommodative facility was not tested monocularly). The Developmental Eye Movement test yielded normal results for the patient's age. Reduction in corrected distance acuity was attributed to over-use of accommodation to control the exotropia; thus, refraction was not repeated.

VISION THERAPY

The patient was diagnosed with intermittent alternating exotropia at near, of the convergence insufficiency type. This diagnosis was based on the small exophoria at distance and the large intermittent exotropia at near with poor convergence along with symptoms of intermittent diplopia, blur, and sleepiness with close work. (Unfortunately, a Convergence insufficiency Symptom Survey (CISS) was not administered.) She also was diagnosed with accommodative insufficiency, based on low accommodative amplitudes. Fluctuations in best-corrected acuity were attributed at that time to the accommodative disorder. Vision therapy was recommended for these conditions. Full time use of glasses was recommended.

The vision therapy program utilized weekly in-office vision therapy supplemented with daily home therapy. For the first 3 months of vision therapy, the patient performed monocular accommodative activities intended to improve amplitude and flexibility (plus lens push-away, near/far rock, minus lens rock, plus/minus lens flippers). She experienced intermittent difficulty clearing plus lenses and seeing distance targets clearly. She also worked on gross convergence skills using binocular push-up activities with suppression monitoring, such as beads and string. Similar activities were prescribed for home. Participation in weekly office visits was excellent. Compliance with home therapy was moderate (home activities were completed 4-5 times per week); there was a great deal of stress at home due to the mother's illness. The patient's uncle arranged for professional tutoring in reading and math, which improved the child's school performance.

Vision therapy continued for 6 additional months, during which time the patient made slow progress despite moderately good compliance. In addition to accommodative activities, she worked on convergence and divergence using activities such as Vectograms, Aperture Rule, and free-space fusion cards (Bernell Corp., Mishawaka, IN). The patient's home situation became more stressful, as her mother passed away, and the patient was subjected to a custody battle between family members. Nevertheless, the patient reported that her initial diplopia and sleepiness had decreased. She continued to experience fluctuating distance vision with acuities ranging from 20/25- to 20/60 through habitual spectacles on different visits. Cover test showed orthophoria at far and 16Δ exophoria at near. Near divergence range was x/16/14; convergence range was 18/30/25. Pull-away accommodative amplitudes were 12.5 diopters in the right eye and 14 in the left. The vision therapy program ended due to the disrupted home situation and the patient's psychological distress. The patient received grief counseling.

The patient returned for comprehensive eye examination 3 months later, with corrected acuity of 20/50 in each eye at distance and 20/40 at near. Refraction provided no improvement. Cover test showed 2 Δ exophoria at far and 10^{Δ} exophoria at near. Near point of convergence was 5/7 centimeters, using an accommodative target. Because of the inconsistencies in visual acuity testing, the patient underwent another thorough ocular health evaluation, including dilated fundus examination. Ocular health findings, remained completely normal. Psychogenic vision loss was suspected, based on tubular results on tangent screen visual fields. Follow-up in 2 months was recommended with electrodiagnostic testing to be considered.

At a follow-up two months later (age 10) best corrected distance acuities were 20/40 right, 20/70 left. Near point of convergence was to nose; near divergence range was x/16/14, convergence was x/25/16. Push-up accommodative amplitudes

measured 8 diopters in each eye. For the first time, the fundus examination revealed mild macular retinal pigmented epithelium atrophy and flecks in the posterior pole bilaterally. Optical coherence tomography demonstrated retinal pigmented epithelium disruption in both maculae. The patient was referred for an inherited retinal disease examination and vision rehabilitation. Due to the psychological trauma the patient was experiencing from her family situation, her uncle (now her legal guardian) did not want her to be told of her retinal diagnosis or prognosis. We believed the macular disease had been developing for some time but had not previously been evident on fundus examinations.

INHERITED RETINAL DISEASE EXAMINATION

The inherited retinal disease examination at the Illinois Eye Institute included vision loss history, pedigree analysis, dilated fundus examination, electroretinogram, and review of recent optical coherence tomography. History indicated reduced visual acuity, but the patient denied photophobia, nyctalopia, scotoma, or color vision problems. Pedigree analysis showed likely autosomal recessive juvenile macular degeneration, with both maternal aunt and maternal grandfather losing central vision before age 40. Dilated examination revealed atrophic macular changes with drusenoid-like changes throughout the posterior pole; the peripheral retina was unaffected. The electroretinogram was not well tolerated but did show abnormal scotopic and photopic responses. Optical coherence tomography review confirmed retinal epithelium disruption and thinning of the macular region.

Following the inherited retinal disease examination, there was a gap in care for 1.5 years. During that time, five phone conversations were documented regarding the diagnosis and options. Subsequently, the patient returned for comprehensive eye examination. At that visit, her best corrected visual acuity was 20/70 in each eye, and the family consented to having a Vision Rehabilitation consultation.

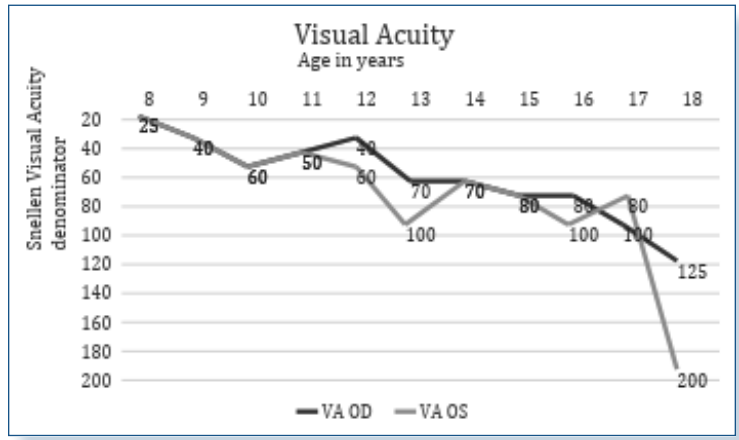


Figure 2. Best corrected distance visual acuity showing the gradual decline as patient ages.

VISION REHABILITATION

The patient was seen five times in the Vision Rehabilitation clinic over the course of the next 6.5 years. Figure 2 shows the gradual decline in her visual acuity over the course of her care. Ancillary testing was difficult to obtain due to the legal guardian's hesitation. The patient continued to engage in psychological counseling at school and family counseling outside school. The primary concern was blurry distance vision. She reported no fatigue with near work. It is possible that her previous work in vision therapy had helped with reading ability. Her grades were above average at a gifted school with no accommodations. She was best corrected to 20/70 in each eye at distance and .4/2M at near. Her habitual near working distance was recorded as 20 centimeters; she pulled material closer to take advantage of relative distance magnification. The predictions for devices were 3.5x distance magnification and +10 D equivalent power at near. Trials of optical and electronic magnification found a preference for a 3.25x25 handheld telescope (20/25 vision), 2.1x mounted telescopes, and +4 D reading glasses with base in prism (.25/.8M). Although the patient did not report near fatigue at the initial Vision Rehabilitation examination, it is common for children with visual impairment to have reduced accommodation and to pull reading material closer to gain a larger retinal image size.[5,6] These two factors can lead to trouble with reading. The

+4 reading glasses were accepted by the patient and put in place as her visual acuity was expected to get worse and near fatigue to increase. These devices were dispensed, and a school report was completed recommending an Individual Education Plan with a Teacher of the Visually Impaired, front row seating, testing accommodations, Orientation and Mobility consultation, and assistive technology.

At the next visit 1.5 years later, the patient's main concern was continued blur at distance with new concerns of fatigue after 20 minutes of near work, but no diplopia. She denied any problems with glare or mobility. School performance was stable. She reported having the Individual Education Plan with the previous recommendations in place. Refraction was stable and distance acuities were now 20/80 in each eye with .2/1M at near. Her magnification devices were still appropriate. Bar magnifiers with an equivalent power of +6 diopters were added to help her read printed material. Accommodation and confrontation visual fields were normal. Dilated fundus examination showed continued atrophic degeneration of macular area and new findings of diffuse bilateral peripheral temporal retinal pigment clumping and dropout. Optical coherence tomography results were like previous, showing expanding atrophic outer retina. The legal guardian deferred additional testing.

The patient returned 13 months later with continued concern of blur. She denied nyctalopia. She reported that her devices were helpful and she benefited from front row seating and testing accommodations. Best corrected vision was 20/80 in each eye at far and .2/1.6M at near. She was now an 8th grader and doing well in school. Refraction was stable, accommodation normal and confrontation visual fields full. Cover test showed orthophoria at far and 20Δ constant alternating exotropia at near. As her visual acuity reduced with increasing macular changes, her fatigue with reading at near was increasing. She continued to maintain a 20-25 centimeter near working distance and use

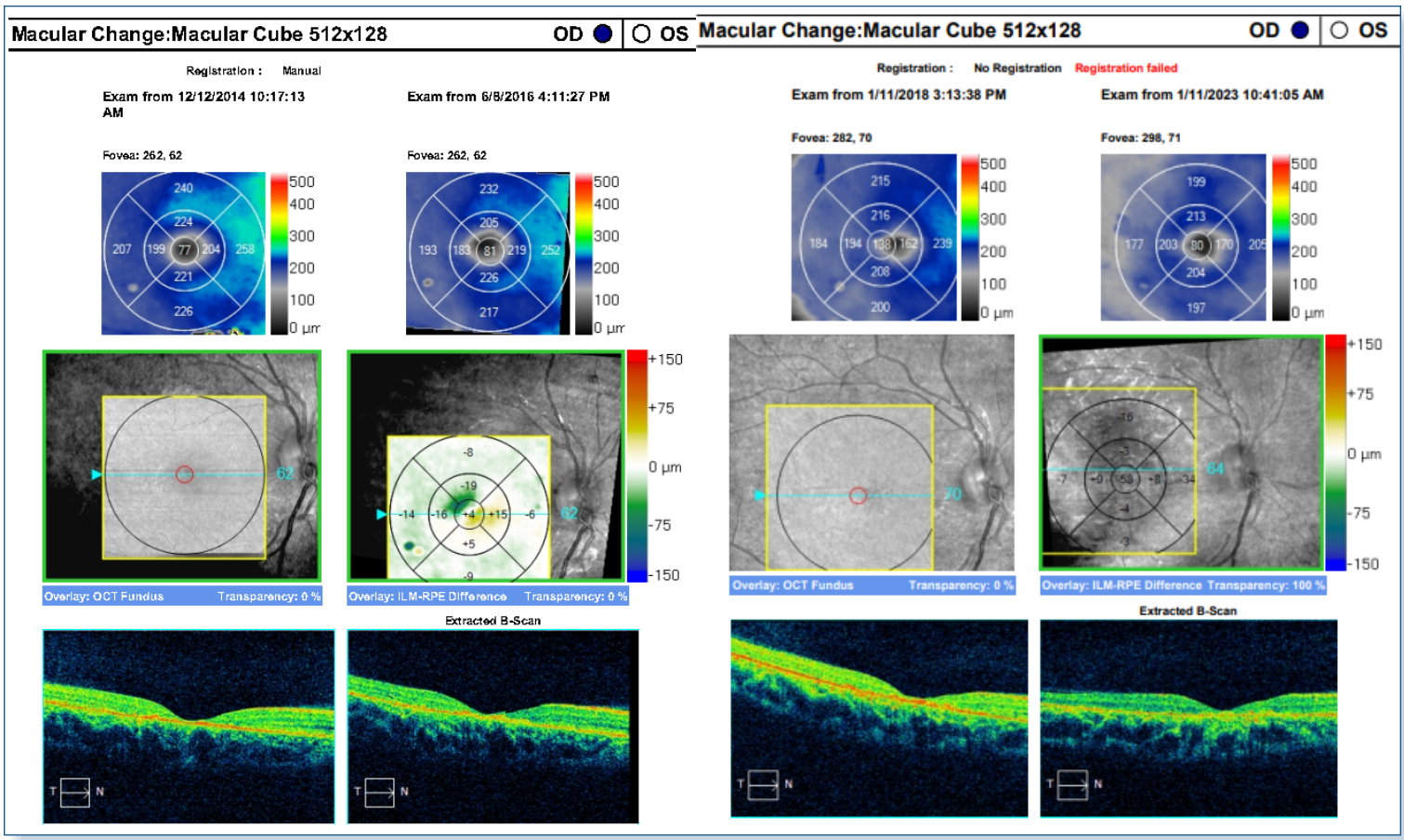


Figure 3. Serial images of the ganglion cell analysis of the macular cube from 2014 to 2023 of the right eye. Significant thinning was evident in 2014 at 77 microns and was stable to 80 in 2023. Foveal pit anatomy shows atrophy. Eccentric viewing by patient may have caused slightly eccentric mapping of the fovea.

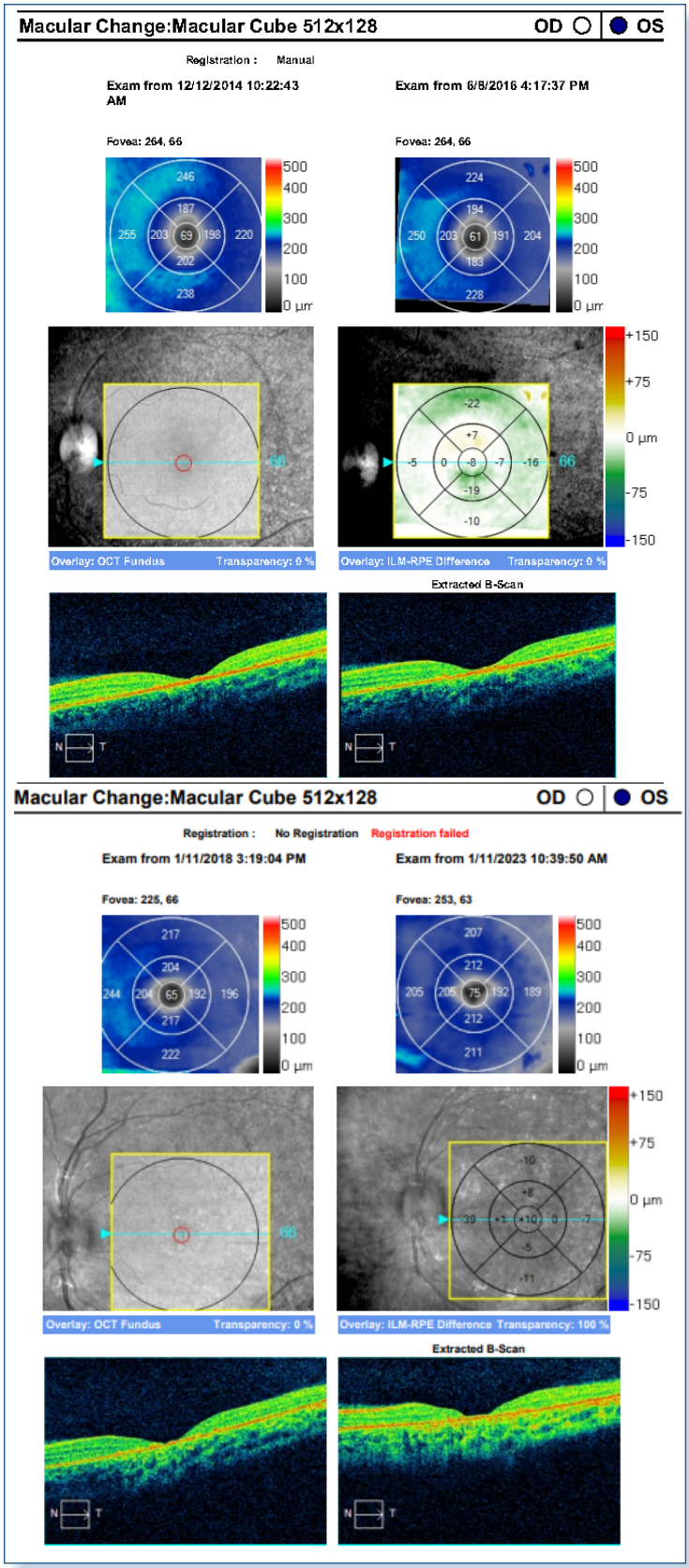


Figure 4. Serial images of the ganglion cell analysis of the macular cube from 2014 to 2023 of the left eye. Significant thinning was evident in 2014 at 69 microns and was stable to 75 in 2023. Foveal pit anatomy shows atrophy. Eccentric viewing by patient may have caused slightly eccentric mapping of the fovea.

the +4 reading glasses. To help compensate, print size was enlarged on her screens (phone/tablet/computer) to enable her to read. She also started to prefer white print on a black background for visual reading. Additionally, likely the poor acuity and close working distance required when using high plus reading glasses had led to a return of this large exo posture. Fortunately, she did not experience diplopia due to suppression. Dilated fundus examination revealed consistent results with both central and peripheral degenerative changes. Recommendations included: prescription glasses, updated school report for services, continued use of devices; additional resources were discussed including technology and audio learning. The legal guardian deferred additional ocular health testing.

The most recent visit occurred at age 18. The patient's main concern was whether she could obtain a driver's license. She was a high school senior, class president and in the top 5% of her class. She was active in many extracurricular activities including dance and theater. She still used her optical magnification devices occasionally but relied more on assistive technology consisting of enlargement and contrast enhancement while using computers and her smartphone. The family was now openly discussing the diagnosis of an inherited retinal disease and was open to genetic testing.

Refraction was stable and best corrected vision at distance was reduced to 20/125 right and 20/200 left with .23/2M at near. She had significant loss of log contrast sensitivity to 0.64 right, 0.52 left and 0.57 both eyes. Confrontation visual fields remained full. Dilated fundus examination revealed consistent results. Optical coherence tomography, seen in Figures 3 and 4, found thinning of the ganglion cell layer and macular atrophy consistent with ABCA4.[7] Fundus photos, seen in Figure 5, showed diffuse macular atrophy, mildly attenuated vessels and granular hypopigmentation to mid-periphery and temporal pigment clumping. All these findings are consistent with severe ABCA4.[8,9] A buccal genetic sample was collected. Recommendations included: prescription glasses, updated school report, continued use of devices, discussion of accommodations and services at

the university level. There was a discussion about driving and not meeting the state requirements. Alternative transportation services were discussed. The plan was to follow up in the clinic after the genetic results were received.

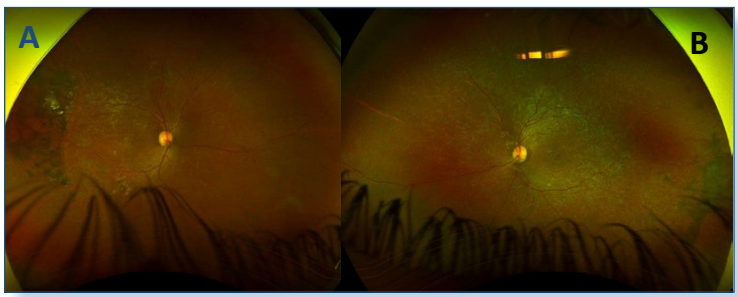


Figure 5. Fundus photograph of (A) right and (B) left eyes at age 18 showing mildly attenuated vessels, macular atrophy, widespread granular hypopigmentation of the retinal pigment epithelium and pigment clumping temporally.

Genetic testing was done using the My Retina Tracker® program through the Foundation Fighting Blindness, revealing that the patient is heterozygous for ABCA4 c.4954T>G, p.(Tyr1625Asp). Reviewing this genetic variant in NIH National Library of Medicine, National Center for Biotechnology Information, ClinVar, it is classified to be pathogenic.[10] In review of all clinical testing, this case appears to be ABCA4 retinal dystrophy leaning towards a cone-rod dystrophy. The patient was contacted multiple times for follow-up but has not yet returned to the clinic.

DISCUSSION

Convergence insufficiency is a common binocular vision disorder in which the patient shows an exodeviation greater at near with inadequate convergence ability for near work. Common symptoms include blurry vision, diplopia, difficulty reading, and eye strain. All these symptoms were evident with our patient. This condition responds well to vision therapy.[11,12] In this case, vision therapy did help but ultimately could not address the fluctuating visual acuity. It was considered during the vision therapy program that her fluctuating near vision might be attributed to an accommodative disorder, with overaccommodation potentially occurring secondary to the large near exodeviation and her efforts to achieve fusion.[13] Nonorganic vision loss from psychosocial stress can also cause vision loss in children.[14,15] This was considered as our patient had the significant psychological trauma of the illness and death of her mother. Treatment for nonorganic vision loss includes mental health counseling, which the patient was already actively receiving. Although ocular disease had also been suspected due to fluctuating visual acuity, there was no funduscopic evidence for many months. In retrospect, OCT could have been performed sooner, but at the time of diagnosis, OCT was not commonly used for children due to lack of access and lack of normative data.

When managing suspected vision loss, it is critical to collect data to confirm the diagnosis. Clinical data may be ambiguous, or in this case, difficult to obtain. In our case there were two barriers: the legal guardian's reluctance to allow additional clinical testing and the nature of the child's condition changing over time, making it difficult to confirm or verify the diagnosis. Since there was no ocular health emergency, the family's decision was respected. We carefully followed the child, supported the family, and encouraged ongoing mental health therapy.

When inherited retinal disease is suspected, clinical data collected should include family pedigree, history with emphasis on pattern of visual loss, visual acuity, contrast sensitivity, color vision, visual field including far peripheral with kinetic perimetry and central with microperimetry, optical coherence tomography, fundus photos, electrophysiology, genetic testing and counseling. In this case, testing supported a diagnosis of inherited retinal degeneration likely involving both cones and rods. The genetic testing panel initially reported a variant of uncertain significance, but upon further investigation, the variant was documented to be pathogenic. More thorough genetic testing, beyond the free testing available, may provide more information. Other testing was deferred by the legal guardian. Ideally, repeated electrophysiology and visual field testing would be helpful for clinical diagnosis.

Once a diagnosis has been determined it is important to keep apprised of emerging treatments

and clinical trials in which a patient may opt to participate if they are eligible. For ABCA4 retinopathies, there are multiple treatments being studied including gene therapy and visual cycle vitamin A modulation. Both treatments are currently in clinical trials.[16]

One study, "A Phase 2 Multicenter, Double-Masked, Randomized, Placebo-Controlled Study to Investigate the Long Term Safety, Tolerability, Pharmacokinetics and Effects of ALK-001 on the Progression of Stargardt Disease" is referred to as the TEASE study. It is evaluating modifying the vitamin A cycle to prevent retinal degeneration. The investigational drug ALK-001, Gildeuretinol Acetate, is a deuterated vitamin A derivative and is being administered orally for 24 months versus a placebo at eight study locations.[17] Another study also investigating the vitamin A cycle is "Phase 3, Multicenter, Randomized, Double-Masked, Placebo-Controlled Study to Evaluate the Safety and Efficacy of Tinlarebant in the Treatment of Stargardt Disease in Adolescent Subjects" and is referred to as the DRAGON study. This study is investigating the oral drug tinlarebant versus a placebo for 24 months to slow retinal lesion growth. The DRAGON study has sites worldwide.[18]

A study investigating gene therapy is entitled "ACDN-01-001: Open-Label, Single Ascending Dose Study to Evaluate the Safety, Tolerability, and Preliminary Efficacy of Subretinal ACDN-01 in Participants With ABCA4-related Retinopathy." This study is referred to as STELLAR and is currently in recruitment at 10 locations with a study duration of 5 years. It seeks to evaluate the use of ACDN-01, an investigational RNA exon editor gene therapy, to restore full length ABCA4 mRNA in patients with ABCA4-related retinopathies.[19]

Low vision rehabilitation has been shown to maximize visual function and improve quality of life in the pediatric population.[20,21,22] During our patient's visits in the low vision rehabilitation clinic, magnification, and assistive technology were provided. Additionally, she was connected to her school's vision services and taught the importance of self-advocacy.

The legal guardian requested that the diagnosis of vision loss not be discussed with the patient. During the visits, no retinal diagnosis was discussed; communication included simply that her eyes needed a little more help including magnification and technology. The child never questioned what was done in the examinations or any of the recommendations. Sometime between the final two visits, the patient had been told about her diagnosis and by age 18 she appeared well-adapted and was thriving. Her guardian confirmed that she was doing well at school, socially, and at home.

A clinician relies on their background, experiences, and training to develop patient communication skills. Importantly, clinicians need to be aware of protocols that have been developed to deliver serious news. Guidelines from these protocols can be tailored to each situation. This will lead to productive interactions and increased ability of the patient to understand their diagnosis and participate in their ongoing care. It is also important to remember that families will reference their own culture, beliefs, and values when considering additional testing and telling their children about the diagnosis. When involving a child, first deliver the serious news to the parent/guardian, who should decide how to deliver the news to the child.

Several well-known communication protocols for delivering serious news have been developed, including SPIKES[23], ABCDE[24] and BREAKS.[25] Common themes amongst these protocols include creating a private, quiet space and allowing enough time for the conversation in person. Explore what the family understands and provide clear information without medical jargon so it can be digested. Provide as much information as the family wants, without overwhelming them. Acknowledge and validate the family's emotional responses with empathy. Provide resources and next steps to the family, a roadmap of where they are going. How the provider delivers the serious news of vision loss can have a lasting impact on how the patient accepts their visual impairment.[26]

CONCLUSION

This case illustrates the importance of treating the visual problems that are diagnosed and being aware of changes in vision that uncover additional problems requiring management. This case shows that binocular vision and accommodative problems can coexist and be successfully managed in patients that also have visual impairment. The case also highlights the challenges of delivering serious news in the pediatric population, while being sensitive to a family's desire to control the information, helping the pediatric patient to understand and accept the information when they are ready. Our patient received continued mental health counseling and vision rehabilitation and currently is a thriving university student.

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CASE REPORT

Impact of Refractive Correction on Ocular Alignment in Accommodative Esotropia

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ABSTRACT

Background

Accommodative esotropia is a type of esotropia in which the eye turns inward either constantly or intermittently, that is commonly seen in children. It is usually associated with uncorrected hypermetropia and an increased AC/A ratio. In this condition, the patient applies excessive accommodative effort to achieve clear vision, and this increased accommodation is accompanied by accommodative convergence, leading to inward eye deviation. The underlying cause is the abnormal relationship between accommodation and convergence initiated by excessive focusing effort due to uncorrected refractive error.

Case Report

A mother presented her five-year-old daughter who had been experiencing inward deviation of both eyes for six months. This deviation was especially apparent when performing near visual tasks. Clinical assessment revealed significant uncorrected hypermetropia. Accommodative esotropia resulted from the excessive accommodating effort generated by the uncorrected hypermetropia, which also increased accommodative convergence. Following a comprehensive ocular examination that included cycloplegic refraction, full hypermetropic spectacle correction was advised. At the follow-up, the patient's ocular alignment had improved and they had good compliance with their glasses. In the absence of glasses, the esodeviation continued, indicating how accommodation contributed to the deviation.

Conclusion

This case indicates that accurate cycloplegic refractive correction plays a critical role in the effective management of accommodative esotropia. Early detection of the condition and timely initiation of optical treatment, with spectacles, can help restore proper ocular alignment and support the normal development of binocular vision in children.

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Keywords: accommodative esotropia; binocular vision; cycloplegic refraction; esodeviation; hypermetropia; pediatric strabismus

INTRODUCTION

Accommodative esotropia is one of the most common forms of convergent strabismus encountered during early childhood and is characterized by inward deviation of one or both eyes associated with accommodative effort.[1,2,3,4] The condition is commonly associated with uncorrected hypermetropia, in which excessive accommodation required to maintain clear

retinal image formation induces increased convergence due to the normal neurological relationship between accommodation and convergence mechanisms.[2,5,6] The accommodative convergence to accommodation (AC/A) ratio plays a significant role in the pathogenesis and clinical presentation of accommodative esotropia.[5,7]

The condition commonly develops between 2 and 6 years of age, although the onset may vary depending on the magnitude of hypermetropia and the visual demands of the child.[8,9] Clinically, the deviation is usually more noticeable during near fixation tasks such as reading or prolonged close work and may occasionally be present during distance fixation.[8-10] Parents commonly observe intermittent or constant inward turning of the eyes, which may gradually increase over time if left untreated.[8,10] Accommodative esotropia is broadly classified into fully accommodative esotropia, partially accommodative esotropia, and non-accommodative esotropia.[4,9,10] Understanding the underlying accommodative mechanism is essential for appropriate diagnosis and management.

Uncorrected hypermetropia is the main cause behind fully accommodative esotropia. In this case, excessive accommodative effort results in increased accommodative convergence which leads to ocular misalignment. In these cases, complete correction of the esodeviation occurs after proper hyperopic spectacle correction. Patients commonly present with inward deviation of the eyes particularly during near tasks, which often leads to asthenopia, headache, and difficulty in reading. Because of sensory adaptation mechanisms such as suppression, diplopia is uncommon in children.[5,11] Hypermetropia, normal extraocular muscle motility, and, in some cases, amblyopia have been reported in accommodative esotropia as key clinical findings.[8,11]

The diagnosis of accommodative esotropia is primarily established through comprehensive ophthalmic evaluation, including visual acuity assessment, cycloplegic refraction, ocular motility examination, and cover test evaluation.[3,12] The determination of the nature of the ocular deviation can be done through refractive correction which is essential for confirming the accommodative component of the esotropia.[3] Management mainly involves prescription of full cycloplegic hypermetropic correction to reduce accommodative demand and thereby decrease the associated convergence response.[5,13] In patients with a high AC/A ratio, bifocal lenses may be considered to control residual near deviation.[9] Timely management and early diagnosis are essential to prevent amblyopia and to promote proper binocular visual development and stable ocular alignment.[11,13]

CASE REPORT

A 5-year-old female child presented with her mother, with chief complaint of inward deviation of both eyes, more noticeable during near visual activities, for the past 6 months. There was no history of ocular trauma or previous ophthalmic consultation. There was no history of developmental milestone abnormalities. According to the parent, the deviation became more evident during reading and close fixation tasks.

On visual assessment at the initial visit, unaided distance visual acuity was recorded as 20/200 in the right eye and 20/200 in the left eye, while near visual acuity was N6 at 40 cm in both eyes. Dry/manifest retinoscopy revealed +5.00 DS hypermetropia in both eyes, with distance visual acuity improving to 20/100 OD and 20/100 OS. Atropine cycloplegic refraction revealed +7.00 DS in both eyes. With full +7.00 DS correction, visual acuity improved further to 20/80 OD and 20/80 OS. During the initial assessment, the esodeviation reduced with full hypermetropic correction; however, complete resolution of the deviation was confirmed after a one-week trial of full-time spectacle wear. Full cycloplegic correction was therefore prescribed. Along with full correction, Hart chart exercises were advised twice daily for 2 months as supportive therapy to improve accommodative flexibility and visual performance.[5,12,13]

Extraocular motility assessment performed at the initial evaluation revealed full, free, and painless ocular movements in all directions of gaze. Hirschberg corneal reflex testing suggested

esodeviation, which was subsequently quantified using prism bar cover testing. Orthoptic assessment using the cover-uncover test revealed a small manifest esodeviation, predominantly involving the right eye (see Figure 1). Such moderate angle esodeviation is frequently reported in accommodative esotropia associated with high hypermetropia.[5,8] Prism bar cover testing revealed approximately 30 prism diopters of esodeviation. Important sensory tests such as stereopsis and the Worth four-dot test (WFDT) were attempted, but the child was unable to respond reliably.



Figure 1. Eso-deviation of right eye on First visit

Anterior segment examination by using slit lamp biomicroscopy showed normal eyelids and adnexal structures within the normal limit. The conjunctiva was clear with no evidence of congestion or discharge. The cornea was transparent in both eyes, with no signs of ulceration or opacity. The anterior chamber was of normal depth and quiet. The pupillary reactions of both eyes are regular and reactive. The crystalline lens was transparent bilaterally. Posterior segment evaluation using the ophthalmoscope revealed healthy optic discs with distinct margins, and the cup-to-disc ratio in both eyes was 0.4. Retinal vasculature, macula, and peripheral retina appeared within normal limits in both eyes.

Based on the clinical findings, we diagnosed a case of fully accommodative esotropia with bilateral high hypermetropia. This was confirmed through complete resolution of the esodeviation after a one-week trial of full-time spectacle wear with full cycloplegic correction of +7.00 DS OU. The patient was advised to continue full-time spectacle wear. The importance of compliance was explained to the parent to ensure proper visual development and maintenance of ocular alignment.[5,14,15]

At the 2-month follow-up visit, the patient's mother reported good compliance with regular spectacle wear. At follow-up visit, best-corrected distance visual acuity was 20/30 OD and 20/30 OS, and near visual acuity was N5 at 40 cm in both eyes. At the 2-month follow-up, orthophoria was observed with spectacle correction at both distance and near, while examination without glasses showed approximately 20 prism diopters of esotropia at both distance and near. Extraocular movements remained full, free, and painless in all directions of gaze. Cycloplegic refraction was repeated, and the result was the same +7.00 DS as the initial refraction. We recommended wearing the same prescription glasses and returning for a follow-up after 2 months. At that time, the patient's ocular findings were stable and showed no discernible alterations in alignment (Figure 2). Written informed consent was obtained from the patient's parent for documentation of clinical details and photographs.

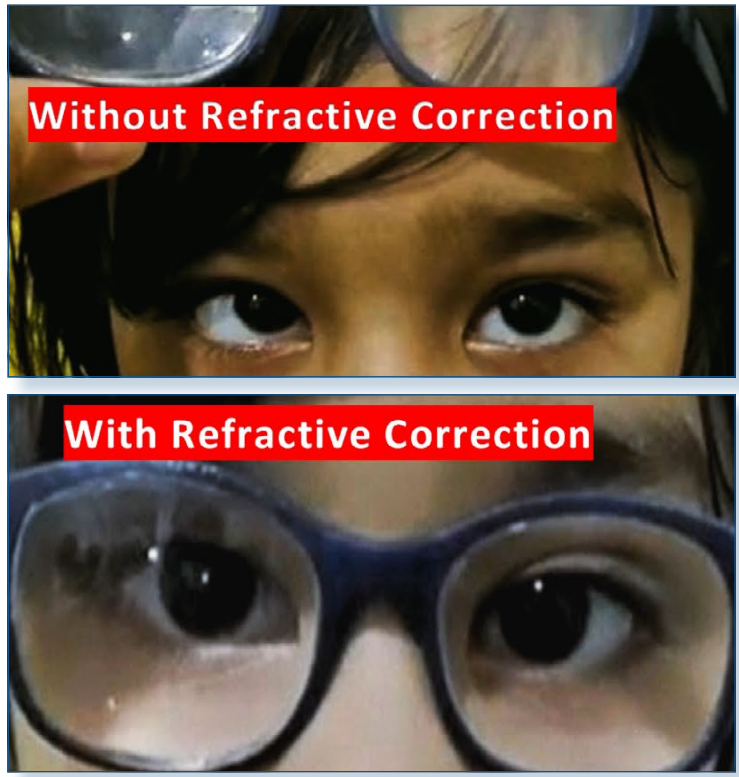


Figure 2. Demonstrations of reduced esodeviation with refractive correction.

DISCUSSION

This case highlights the importance of comprehensive refractive assessment in children presenting

with esotropia and high hypermetropia. Although dry/manifest retinoscopy revealed +5.00 DS OU, atropine cycloplegic refraction demonstrated a higher latent hypermetropic component of +7.00 DS OU, indicating that accommodation may have masked the full refractive error during the initial assessment. Therefore, cycloplegic refraction helped determine the maximum hypermetropic correction required for optical management. With full spectacle correction, the esodeviation improved to orthophoria, supporting the diagnosis of refractive accommodative esotropia. Early detection and timely initiation of appropriate optical correction can assist in restoring ocular alignment and may support binocular visual development in children.[1,5,7] Under normal physiological conditions, accommodation required for near fixation is accompanied by a proportional amount of convergence through the accommodative convergence mechanism. However, in children with significant hypermetropia, excessive accommodation is required even for clear distance vision, leading to increased accommodative convergence and consequent inward deviation of the eyes.[1,5,7]

The patient demonstrated orthophoria with spectacle correction while esotropia persisted in the absence of glasses, confirming the diagnosis of fully accommodative esotropia. Similar findings have been reported in several clinical studies evaluating the role of optical correction in accommodative esotropia management.[5,13,16]

Accommodative esotropia commonly presents between 2 and 6 years of age and is often initially noticed by parents as intermittent or constant inward deviation of one or both eyes.[8,9,10,17] The deviation is usually more pronounced during near fixation tasks such as reading or focusing on small objects. Extraocular movements are generally full and unrestricted, with no associated neurological or paralytic pathology.[9,10] Based on the clinical response to refractive correction, accommodative esotropia is broadly classified into fully accommodative, partially accommodative, and non-accommodative types.[4,9,10]

Uncorrected hypermetropia is the main cause of fully accommodative esotropia, a kind of esodeviation in which an aberrant relationship between accommodation and convergence causes the eyes to deviate inward. Usually, full hypermetropic spectacle correction fully corrects this abnormality. Patients with this condition usually become orthophoric while wearing glasses and exhibit normal ocular alignment.[2,5] In contrast, another type partially accommodative esotropia shows only partial improvement even after appropriate spectacle correction. The deviation is not fully resolved, indicating the involvement of additional non-accommodative factors.[4,10,17] Therefore, unlike fully accommodative esotropia, this condition often requires a different management approach, such as orthoptic therapy or surgical intervention.[10,17,18]

The present case demonstrates several classical features of accommodative esotropia. The patient, a 5-year-old child, falls within the commonly reported age group for onset of the condition. The history of inward deviation becoming more noticeable during near activities strongly supports the role of excessive accommodative demand in the development of the esodeviation. Additionally, cycloplegic refraction revealed high bilateral hypermetropia (+7.00 DS), which is a major contributing factor in the pathogenesis of accommodative esotropia.[8,10,17]

Another important clinical feature in this case was the presence of full, smooth, and painless extraocular movements in all directions of gaze. This observation effectively ruled out paralytic or restrictive causes of strabismus and indicated that the deviation was concomitant in nature. Furthermore, the anterior and posterior segment evaluations were found to be within normal limits, suggesting that the esotropia was purely refractive and functional, with no underlying structural abnormality of the eye.[9,10] Birch et al. reported that prompt optical correction can significantly improve binocular sensory outcomes and stereopsis development in children with accommodative esotropia.[11]

The detection of high bilateral hypermetropia during cycloplegic refraction was an important clinical finding in this case.[7,16,18] This patient's

assessed refractive error was noticeably high, which increased the case's clinical significance.

Although accommodative esotropia is usually associated with a moderate degree of hypermetropia, the extremely high hypermetropic correction in this case requires significant accommodating effort for clear vision.[16,18] This leads to a significant increase in accommodative convergence, which increases the likelihood of a more pronounced inward deviation of the eyes, especially if therapy is postponed or neglected.

The significance of doing accurate and comprehensive cycloplegic refraction in paediatric patients is highlighted by this example. In addition to preventing under-correction, accurate refractive error estimate guarantees the proper prescription of glasses.[3,12,15] Maintaining appropriate ocular alignment and successfully managing accommodative esotropia depend heavily on prompt and accurate optical correction.

An additional significant aspect of this case was the clear and complete response to optical correction. The patient demonstrated orthophoria with full hypermetropic correction, while the esodeviation persisted in the absence of spectacles.[5,14] This finding confirmed the diagnosis of fully accommodative esotropia. Good compliance with spectacle wear, as reported by the parent, also contributed substantially to the favourable treatment outcome.[5,13,14,16] In many pediatric cases, poor compliance with spectacles may compromise alignment and binocular visual development. The recommended Hart chart exercise was used as supportive therapy to improve accommodative flexibility and visual performance along with refractive correction (Figure 3).[5,12,13]

The deviation measured approximately 30 prism diopters, indicating a moderate angle of esotropia, which is commonly observed in accommodative cases but remains clinically significant enough to warrant early intervention.[8,11,19] Delay in diagnosis and management may predispose the child to amblyopia, suppression, and loss of binocular vision.[8,11,19] In the present case, early diagnosis and prompt prescription of full cycloplegic correction resulted in stable ocular alignment and prevention

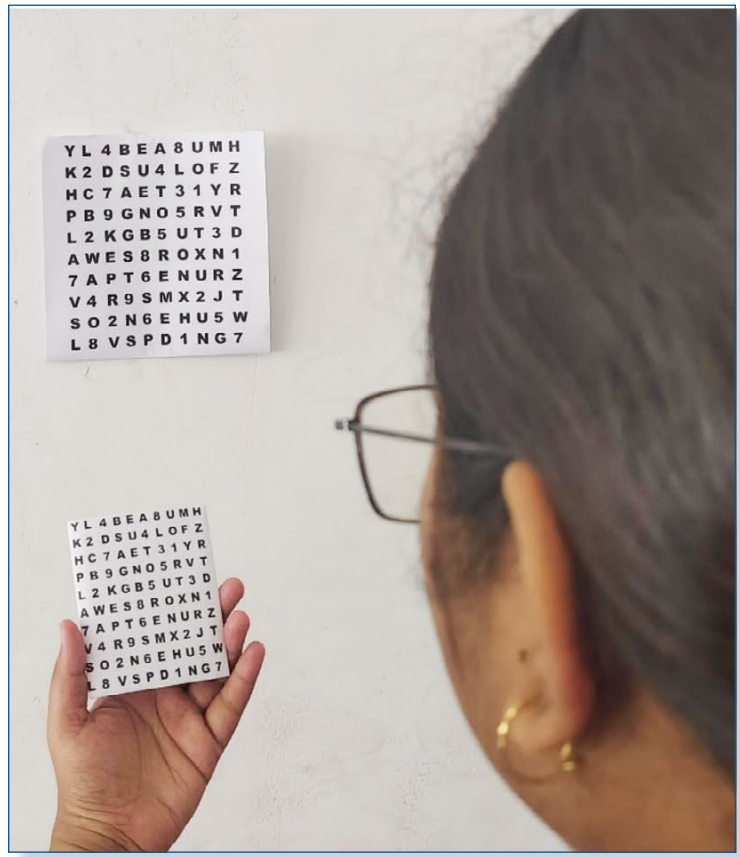


Figure 3. Hart Chart Exercise

of potential sensory complications. Comparison of findings before and after refractive correction is demonstrated by Table 1.

The present case also emphasizes the importance of pediatric binocular vision assessment and vision therapy support in improving visual function.[20,21] Recent studies by Chakraborty et al. demonstrated the effectiveness of active vision therapy and binocular rehabilitation in pediatric visual disorders, particularly amblyopia and binocular vision dysfunction.[20,21] Additionally, consistent spectacle wear was associated with stable ocular alignment during followup. Longterm compliance with prescribed hyperopic correction has been identified as one of the most important prognostic factors in maintaining ocular alignment in accommodative esotropia.[5,13,16]

As seen in comparing Figures 1 and 2, it is common for the esotropic angle without glasses to increase relative to the angle prior to wearing glasses. This most plausible explanation with high bilateral hyperopia is that prior to spectacle wear, the child does not accommodate fully to attain

Table 1: Comparison of Findings Before and After Refractive Correction

Parameter	Initial Visit / Pre-Correction	After Full spectacle Correction / Follow-up
Unaided Distance VA	20/200 OU	20/200 OU
Best-Corrected Distance VA with +7.00 DS	20/80 OU	20/30 OU at 2-month follow-up
Near Vision	N6 at 40 cm OU	N5 at 40 cm OU
Cycloplegic Refraction	+7.00 DS OU	Stable at +7.00 DS OU
Ocular Alignment without Glasses	Approximately 30 PD esotropia	Approximately 20 PD esotropia at 2-month follow-up
Ocular Alignment with +7.00 DS Glasses	Esodeviation reduced initially; complete resolution confirmed after 1-week spectacle trial	Orthophoria at distance and near
Extraocular Movements	Full, free, and painless	Full, free, and painless
Final Diagnosis	Fully accommodative esotropia with bilateral high hypermetropia	Stable fully accommodative esotropia with good spectacle compliance

Abbreviations: OU – Both eyes; DS – Diopter sphere; PD – Prism diopters.

sharp visual acuity. That under-accommodation and de-focus may be purposeful in tolerating some level of blur in exchange for the avoidance of diplopia. Once the child has worn the spectacles that helps improve visual acuity, the accommodative reflex operates more fully when the glasses are removed and the AC/A response increases the esotropic angle. Parents should be advised to expect to see the child's eye turn increase without the glasses relative to the angle before receiving the Rx. They should be reassured that this is normal, and that what is more important is alignment and binocular vision with the glasses on.

This case is an example of accommodative esotropia. It highlights that proper refraction especially cycloplegic refraction is extremely important. Furthermore, appropriate management and consistent use of prescribed spectacles are essential. From this case, it can be understood that full hypermetropic correction is the most effective treatment for accommodative esotropia. Our patient in this case report exhibited residual mild bilateral refractive amblyopia after a two-month period of wearing her full cycloplegic prescription. Bilateral visual acuity tends to continuously improve over the course of the first year with full-time wear of the prescription in high bilateral hyperopia in young patients, as demonstrated by one of the PEDIG studies.[22] We added Hart Chart accommodative therapy, as in our clinical experience, this may accelerate the process.

CONCLUSION

This case demonstrates a typical presentation of fully accommodative esotropia in a 5-year-old girl with bilateral high hypermetropia. Comprehensive ocular examination, particularly cycloplegic refraction, helped identify the full hypermetropic correction required for optical management. With consistent full-time spectacle wear, the esodeviation resolved and orthophoria was maintained. This case supports the role of early diagnosis, accurate refractive correction, compliance with spectacle wear, and regular follow-up in the management of accommodative esotropia and prevention of binocular vision complications in children.

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CASE REPORT

The Use of Yoked Prism and Neuro-Optometric Rehabilitation Therapy to Treat Abnormal Egocentric Localization in a Patient with Traumatic Brain Injury

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ABSTRACT

Background

Abnormal egocentric localization is a common yet often underdiagnosed consequence of traumatic brain injury (TBI). Affected individuals may experience dizziness, vestibular deficits, spatial disorientation, eye strain, and headaches. Egocentric localization refers to a person's perceived vertical midline relative to their body position. When this internal sense of midline is shifted to one side, it can disrupt spatial orientation and postural control. Yoked prism lenses are used as an intervention to realign the perceived visual midline, thereby reducing disorientation and improving balance and navigation in daily environments. In many cases, additional treatment through neuro-optometric rehabilitation therapy (NORT) is recommended to improve visual-motor integration processing and spatial awareness leading to an enhancement of overall visual function.

Case Report

A 43-year-old woman sustained a TBI after an accident where a wooden plank struck her head. The patient presented with dizziness, photosensitivity, eyestrain, and difficulty navigating crowded environments. After 6 initial sessions of NORT, the patient did not experience any reduction of her symptoms. Upon further evaluation, she was found to have abnormal egocentric localization. The patient reported marked improvement in symptoms and functionality following the introduction of yoked prism.

Conclusion

Accurate detection and diagnosis of abnormal egocentric localization is essential when examining patients who have sustained a TBI. Delayed diagnosis can prolong symptoms of disorientation and dizziness and severely impact a patient's quality of life. Evaluation of egocentric localization should be considered as part of the guidelines for initial examination when working with this population.

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Keywords: egocentric localization; neuro-optometric rehabilitation; yoked prism;

BACKGROUND

Egocentric localization is described by Ciuffreda and Ludlam as a disruption of the egocenter, the internal reference point that defines an individual's perceived midline.[1] This can lead to significant functional sequelae, including spatial disorientation, imbalance, and difficulty navigating the environment. These disturbances are thought to arise from altered visual–vestibular integration and oculomotor dysfunction, which shift the patient's perceived spatial midline away from true body alignment.[1] In this case report, the patient's symptoms and findings demonstrate how abnormal egocentric localization

can contribute to postural instability, dizziness, difficulty reading, and discomfort following traumatic brain injury (TBI). Recognition of this deficit allowed for targeted intervention with yoked prism, which facilitated perceptual realignment and improved functional mobility and patient comfort.

This altered egocenter reflects a misalignment between the perceived and actual body midline, a mechanism that closely relates to but is distinct from visual midline shift syndrome (VMSS). Padula and Argyris characterized VMSS as a post-trauma visual disorder in which the visual midline itself is displaced, resulting in shifts in perceived spatial orientation and postural control. They characterize VMSS by hemiplegia, hemiparesis, flexion, and side neglect.[2] While VMSS primarily describes the perceptual shift in visual space, abnormal egocentric localization represents the underlying disruption in how the individual integrates visual, vestibular, and proprioceptive cues to establish themselves in space. Thus, abnormal egocentric localization can be understood as a critical component contributing to the development and persistence of VMSS.

Following TBI, patients often experience a broad range of visual and perceptual disturbances, many of which can be attributed to an underlying disruption in egocentric localization. These symptoms often coexist with oculomotor dysfunctions such as convergence or accommodative insufficiency, saccadic inaccuracy, and poor binocular coordination, which further destabilize spatial perception.[1] Clinically, individuals may describe feeling as though they are being pulled to one side, that objects appear tilted or shifted, that they don't feel grounded, or that their body alignment does not match the visual world around them. When unrecognized, abnormal egocentric localization can contribute to persistent imbalance, visual fatigue, and lack of confidence during ambulation. Patients may feel wary of daily activities that require accurate spatial awareness, such as walking or driving.[1] Recognition of this dysfunction is therefore essential, as targeted interventions such as yoked prism lenses and neuro-optometric rehabilitation therapy (NORT) can help restore perceptual alignment and improve overall spatial orientation.

The perception of "straight ahead" is a fundamental aspect of egocentric localization, and its neural basis has been closely linked to parietal and occipital lobe function. Ferber and Karnath have suggested that damage within these cortical regions can lead to significant distortions in perceived straight-ahead orientation.[3] Their findings support the view that accurate egocentric localization depends on the integration of visual and proprioceptive inputs mediated by these cortical areas. In the context of a TBI, disruption to these neural networks can produce similar perceptual shifts, manifesting clinically as abnormal egocentric localization or visual midline displacement.

A simple and accessible test of egocentric localization involves the examiner slowly moving an object, such as a pen or pencil held about 15 inches away, from the patient's periphery towards their midline. The patient should be instructed to verbalize when they believe the object is aligned with their perceived visual midline. It is important that the examiner is positioned off to the side of the patient so as not to interfere with the patient's midline perception. Testing is repeated several times from both horizontal and vertical directions to assess consistency and determine whether a reproducible shift is present. This test allows the examiner to obtain a gross understanding of the patient's subjective straight-ahead position. Standardized procedures for assessing egocentric localization remain limited, however more quantifiable methods have been described.[1] These include the Bernell Spatial Location Board – Height Adjustable Model and the Bernell Vision Disc, the latter of which was not intended for this measurement but can be adapted. Quantification of egocentric localization may also allow for more precise assessment of changes following yoked prism intervention.

Binocular vision dysfunctions, including accommodative, vergence, and oculomotor disorders, are extremely common in patients who have experienced a TBI, and a full work-up to assess their sensorimotor status is necessary in this population.[4] These underlying dysfunctions can compound and exacerbate the symptoms associated with abnormal egocentric localization. Once the patient's visual

skills have been fully evaluated and considered, yoked prism can serve as a treatment option for abnormal egocentric localization to shift the visual field towards the direction of the patient's perceived vertical or horizontal midline. For example, a patient with egocentric localization shifted to their left would benefit from base right prism. By shifting the patient's visual field into the position that corresponds to their perceived midline, the prism correction may help reorient their spatial awareness and improve their ability to navigate their surroundings.[5] In addition to treatment with yoked prism, neuro-optometric rehabilitation therapy may be utilized to improve sensorimotor function and further reduce symptoms when indicated.

CASE REPORT

A 43-year-old female presented after sustaining a TBI due to a building accident where a 2x4 wooden plank struck her head. The patient was seen in the Vision Rehabilitation Service at the University Eye Center. The patient reported that it was a closed head injury, and she did not experience any loss of consciousness. She was evaluated by her primary care physician following the event, who diagnosed her with a concussion. At her initial presentation, 6 months after the injury, the patient reported photosensitivity that was exacerbated by moving or swaying lights. The Post-Traumatic Visual Symptom Survey (PTVS) was administered at this visit to determine the patient's symptoms and the severity at which she was experiencing them. The patient reported a score of 3 (the highest possible score per category, indicating a nearly constant occurrence) for the following symptoms: dizziness, restricted field of vision, bumping into objects on one side more than the other, sensation of the floor or ceiling tilting, difficulty moving or turning eyes, pain with eye movement, poor posture, bothered by movement/crowded environments, photosensitivity, unable to sustain near work, headaches and loss of place when reading. Her total PTVS score was 87 of 120 points, indicating that she was highly symptomatic. She reported taking 60 mg of methylphenidate twice a day to help with fatigue secondary to her TBI. Her systemic health was otherwise unremarkable. She completed 12 sessions of physical therapy to address pain and stiffness in her back and began to see an acupuncturist weekly to manage her back pain.

The patient presented without spectacle correction and denied a history of spectacle wear. Upon evaluation, her cover test revealed a variable 2-4PD esophoria at near with a 2PD esophoria at distance. No tropia was present and the patient was not symptomatic for diplopia. The patient's accommodative near point of convergence was receded with a 5-inch break and 12-inch recovery. The non-accommodative near point of convergence was also receded with a 24-inch break and no recovery. The patient's fixation was unsteady OD and OS, and both pursuits and saccades were jerky with frequent fixation losses and excessive blinking, a sign of motor overflow. The patient manifested a low compound astigmatic refractive error in each eye, with a small near add based on her age. The final prescription was +1.00 -0.75 at axis 85 in the right eye, +0.50 -0.75 at axis 90 in the left eye, with a +0.75 add. The patient's distance base out vergence ranges showed a 25PD break and 12PD recovery, and her base in vergence ranges showed a 4PD break and 2PD recovery. Her near base out vergence ranges showed a 30PD break and 12PD recovery, and her base in vergence ranges showed a 10PD break and 6PD recovery. Phorias were measured with Modified Thorington and testing revealed a 5PD eso at distance, and 3PD eso at near with no measurable vertical deviation. The Developmental Eye Movement (DEM) Test was administered and the patient scored less than 1% on both the horizontal and ratio components using the highest norms available for a 13 year old. The patient scored in the 10%ile on the Visual Tracing Test (VTT) using the highest norms available for a 12 year old. Keystone Visual Skills and Van Orden Star testing showed unstable binocular findings and alternate suppression at both distance and near. Due to the severity of her symptoms of nausea and disequilibrium, visual-vestibular testing was not performed. The patient was diagnosed with basic esophoria, oculomotor dysfunction, and visual-vestibular dysfunction. New glasses for distance

and near were prescribed, and neuro-optometric rehabilitation therapy (NORT) was recommended.

In the early stages of NORT, techniques were utilized that stressed oculomotor and vergence skills to address the signs and symptoms of oculomotor dysfunction and basic esophoria. She was initially too symptomatic to incorporate therapies that addressed her visual motion sensitivity and visual-vestibular dysfunction. Oculomotor techniques included visual tracing, Michigan Tracking, and Hart Chart saccades. The patient also attempted monocular thumb pursuits and binocular thumb jump ductions while seated, but was unable to perform them due to the exacerbation of her symptoms of dizziness and nausea. Peripheral awareness techniques via the Macdonald chart also induced these symptoms. In the office, the patient attempted computerized tracking, however the brightness of the computer screen was bothersome and induced eye strain and fatigue.

After completing 6 sessions of NORT, the patient was not progressing as expected and was reporting almost constant symptoms of dizziness and disequilibrium. During the 7th session, the patient shared a recent incident where she was at a festival and felt her entire world shifting to one side. Her partner was with her at the time and told her she had been leaning over and walking towards her right. She felt very disoriented during this experience and left the festival; it took her several days to recover at home. This prompted further testing, which included egocentric localization testing. A slight shift to the right was noted. Yoked prism was trialed, and the patient reported relief with base left prism. Both 1PD and 2PD yoked base left prism was trialed, and the patient felt most comfortable with 1PD base left. The patient was observed to have an unsteady gait when walking down the hallway with her habitual glasses, where she was leaning slightly to the right. She lost her balance when trying to turn around and put her hands out to maintain balance while walking. She demonstrated a much steadier gait with 1PD base left yoked prism over her habitual glasses; she was not leaning to her right and was able to consistently walk in a straight line. She reported a subjective improvement in her balance, and felt she had an improved awareness of her surroundings while

Table 1: Clinical findings at initial evaluation and re-evaluation following incorporation of 1 PD base-left yoked prism. Normative values adapted from Morgan's expected findings as reported by Scheiman and Wick.[13] These values are provided for reference and may vary based on testing method and patient age.

	Habitual Glasses	Yoked Prism Glasses	Normative Data
Cover test near	2–4$^{\Delta}$ EP	2$^{\Delta}$ EP	Orthophoria to 6$^{\Delta}$ XP
Cover test distance	2$^{\Delta}$ EP	Orthophoria	Orthophoria to 2$^{\Delta}$ XP
Near point of convergence break/recovery	12 cm / 30 cm	8 cm / 13 cm	≤5 cm / ≤7 cm
Distance base out vergence blur/break/recovery ($^{\Delta}$)	X/25/12	X/25/18	9/19/10
Distance base in vergence blur/break/recovery	X/4/2	X/6/4	X/7/4
Near base out vergence blur/break/recovery	14/30/12	16/40/35	17/21/11
Near base in vergence blur/break/recovery	X/10/6	X/18/16	13/21/13
Developmental Eye Movement Test	<1%ile	81%ile	Age-adjusted norms
Visual Tracing Test	10%ile	47%ile	Age-adjusted norms
Post-Traumatic Visual Symptom Survey score	87	26	Lower scores indicate fewer symptoms
Keystone Visual Skills	Unstable alignment	Expected alignment	Expected alignment
Van Orden Star	Alternate suppression	Normal, no suppression	No suppression
Visual-vestibular testing	Unable to complete	Normal	–

wearing the trialed prism. Her distance and near glasses were updated incorporating 1PD base left yoked prism into the prescription.

Once the patient received her glasses, she reported being much more comfortable navigating her environment, and she was able to read for longer periods of time. She no longer felt disoriented when scrolling through her phone when wearing the yoked prism glasses. A re-evaluation was performed through her yoked prism prescription and the findings are compared to her initial evaluation findings in Table 1. The patient now showed a 2PD esophoria at near and was orthophoric at distance on cover test. Her accommodative near point of convergence improved to a 3-inch break with a 5-inch recovery. In the phoropter, the patient's distance base out vergence ranges improved to a 25PD break and 18PD recovery, and her base in vergence ranges showed a 6PD break and 4PD recovery. Her near vergence ranges also improved and showed a 40PD break and 35PD recovery for base out, and her base in ranges showed a 18PD break and 16PD recovery. The DEM testing improved from a ratio of less than 1% at her initial evaluation to an 81%ile. Her Visual Tracing Test performance improved from 10%ile to 47%ile. The PTVS was re-administered, and the patient's total score decreased from 87 to 26. The frequency of her symptoms was no longer higher than a score of 2 (frequent occurrence). The only symptoms that she graded as a 2 were photosensitivity and decreased attention span when reading. Keystone Visual Skills and Van Orden Star testing revealed stable, expected findings indicating alignment at distance and near. Visual-vestibular testing including dynamic visual acuity was normal, as well as the Cohen Test (a clinical assessment involving viewing a stationary flat fusion target while the patient performs horizontal head shakes), which demonstrated no suppression or nystagmus. Notably, the patient did not feel nauseous or disoriented during and after visual-vestibular testing.

At her following NORT appointments, she continued to report that the visual symptoms and fatigue that she presented with, as well as her constant eye strain, pressure, and headaches, were greatly reduced once adjusting to the glasses. She reported that she was now able to read a book at night for over 30 minutes without getting fatigued, which she was unable to do since prior to her injury. She also noted an improvement in her reading comprehension, and reported she was able to recall specific details about what she had read and now engage in thoughtful conversation with her partner. Her symptoms of dizziness and disorientation were completely resolved, and her energy levels felt dramatically increased. She expressed that she had more free time in the evenings, as prior to receiving her prism glasses she normally spent her evenings recovering from the day's activities.

The patient ultimately completed 40 sessions of NORT. At her six-month follow-up, she demonstrated sustained improvement in her symptoms. Her egocentric localization was re-assessed and remained centered with her habitual 1PD base left prism glasses. Following these outcomes, the patient completed her NORT program and transitioned to routine annual comprehensive evaluations.

DISCUSSION

Although treating the sensorimotor disorders associated with TBI is integral to the rehabilitation of these patients, it is important not to overlook associated visual-spatial disorders such as abnormal egocentric localization. Yoked prism can be an effective method of treatment to address and correct for anomalous egocentric localization.[1] A proposed mechanism is that yoked prism influences the magnocellular processing involved in proprioceptive, kinesthetic, and vestibular integration. One case study sought to investigate the neural mechanisms that take place in patients with VMSS, both with and without prism correction via the use of event-related potentials from noninvasive electrophysiological recordings. The examiners found that without prisms, delayed response latencies indicated that there was inefficient magnocellular system processing in somatosensory, language, and executive brain regions. With prisms, electrophysiologic findings suggested faster and

more efficient visual processing and activation patterns typically involved in normal vision.[6] These findings suggest that corrective prism may facilitate neural reorganization in VMSS patients that contributes to the reduction in symptoms of disorientation, disequilibrium, and dizziness as well as an improvement in cognitive processing.[5] Although yoked prism correction is an important intervention when treating TBI patients with VMSS and therefore abnormal egocentric localization, it is also important to utilize NORT to address any underlying sensorimotor symptoms.

In this case, the decision to trial and ultimately prescribe 1PD base left yoked prism was guided by the patient's egocentric localization testing and by observing the patient's ambulation with and without the yoked prism. Small prism values are typically prescribed for abnormal egocentric localization, as they provide a subtle, yet clinically meaningful, shift of the visual field toward the patient's perceived midline, while minimizing disruption to binocular alignment and enhancing spatial orientation and postural stability.[7] The patient was given adequate time to assess each trial and report which prism power felt most comfortable. Observations of gait and balance with and without prism confirmed that even this small amount produced a noticeable improvement in her ability to navigate her environment safely.

Of the improved clinical findings upon re-evaluation after the incorporation of yoked prism, the change in DEM Test performance was particularly notable. Although the DEM Test is traditionally considered as a measure of saccadic efficiency and visual-verbal automaticity, more recent evidence suggests that performance on the test reflects a broader combination of visual processing speed, fixation behavior, and higher-order visuocognitive functions.[8] Leigh and Zee also describe saccadic eye movements as being supported by distributed cortical and subcortical networks, including parietal and frontal regions involved in spatial attention and visual guidance, rather than being purely low-level motor events.[9] In this case, the patient's improvement on the DEM Test may be best understood as a consequence of improved spatial stability following alignment of the egocenter with yoked prism. This reduction in spatial disorientation may have enhanced the efficiency with which visual information was processed during testing, suggesting that correction of abnormal egocentric localization may have downstream effects on visual processing. These findings are also consistent with the patient's reported improvement in reading endurance with use of her yoked prism glasses.

Although this patient experienced a substantial reduction in symptoms and demonstrated functional improvement following the introduction of 1PD base left yoked prism, it is important to consider that these improvements cannot solely be attributed to prism correction. Prior to prism correction, the patient had already completed 6 sessions of NORT, targeting oculomotor and vergence skills, which may have contributed to progressive neuroadaptation and partial symptom reduction that became more evident over time. It is also possible that the initial NORT sessions facilitated the patient's ability to subsequently adapt to and benefit from yoked prism correction. This concept may be loosely compared to the "sandwich" approach in strabismus management, where vision therapy is utilized both before and after surgery to prepare the sensorimotor system for change and then reinforce binocular integration and stability afterward.[10] Similarly, the early sessions in this case may have helped prepare the patient for the introduction of yoked prism and contributed to the improvements she reported in balance, navigation, reading endurance, and dizziness. Because the prism was introduced while therapy was ongoing, it is difficult to determine the exact contribution of each intervention independently, and this proposed relationship between early therapy and enhanced adaptation to yoked prism in cases of egocentric localization remains speculative. Re-evaluating the patient after stabilization of symptoms without the yoked prism may have provided additional insight into the specific effect of prism correction on egocentric localization and functional performance.

Another consideration in this case is the long-term necessity of continued yoked prism wear following completion of NORT. At present, there

is no standardized protocol defining when prism correction can be safely reduced or discontinued in cases of abnormal egocentric localization. Although NORT aims to improve the underlying visual-spatial deficits commonly present in the TBI population, some patients may continue to benefit from, or function comfortably with, continued use of yoked prism even after completion of therapy. In this case, the patient remained comfortable and functionally stable with 1PD base left yoked prism at both completion of NORT and her 6-month follow-up. Whether gradual reduction or discontinuation of prism would be appropriate over time remains uncertain and may depend on the persistence of egocentric localization findings under both prism and non-prism conditions at future exams, as well as the patient's real-world functional demands.

Abnormal egocentric localization can be an overlooked condition in the TBI patient population. Outside of vision rehabilitation providers, many consider egocentric localization to be an unproven concept, and the use of yoked prism and neuro-optometric rehabilitation therapy as a form of treatment remains controversial.[11] To ensure appropriate care, it is essential to increase awareness and acceptance of this condition among primary care optometrists and ophthalmologists. Greater familiarity can help clinicians feel more confident in identifying suspected cases and making timely referrals when indicated. Fortunately, testing for this condition is accessible, non-invasive, and can easily be performed in a clinical setting.

Without intervention, abnormal egocentric localization can significantly impair a patient's ability to function safely and independently. Individuals often struggle with walking and navigating their environment, leading to an increased risk of falls and other accidents.[12] With appropriate treatment, patients can regain their sense of spatial orientation, which improves their ability to perform daily activities and reduces their risk of accidents and injuries. Beyond physical risks, abnormal egocentric localization can also have a severe impact on a patient's mental health. If left untreated, patients may develop anxiety, depression, or other psychological symptoms related to their distorted sense of spatial orientation.[2] Early detection and targeted intervention are therefore essential to minimizing both the physical and psychological complications associated with this condition.

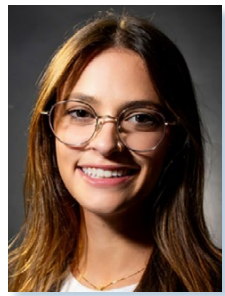
Conclusion:

Greater awareness and recognition of abnormal egocentric localization is essential to ensure that fewer patients go overlooked and untreated. Testing for egocentric localization should be incorporated into baseline evaluations for patients with a history of TBI. When left unrecognized, this condition disrupts daily activities such as walking, driving, and reading, and can increase the risk of falls and injury. Beyond the physical consequences, it can also contribute to significant psychological distress in the form of anxiety, frustration, and reduced confidence. As demonstrated in this case, improvements in visual performance measures such as reading endurance may reflect enhanced integration of visual-spatial information following realignment of the egocenter, with downstream effects on functional visual efficiency. With timely and appropriate intervention, including yoked prism and neuro-optometric rehabilitation therapy when indicated, patients with abnormal egocentric localization may regain balance and stability, reduce discomfort and anxiety, and experience meaningful improvements in overall visual function and quality of life.

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ORIGINAL STUDY

The Future of AI in Clinical Decision Support for the Neuro-Optometrist

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ABSTRACT

A retrospective pilot study was performed to look at the relationship between real life clinical neuro optometric vision therapy protocols performed by a group of licensed optometrists and treatment plans created by an artificial-intelligence (AI) model configured with neuro-optometric data, and related published text. The ultimate goal would be to create a widely used tool to help guide a rehabilitation team toward better treatment outcomes. It could help them decide if vision therapy is indicated for a patient and what types of therapy would be useful. We analyzed historical patient data (n= 8) to gather patient profiles, prescribed treatment plans, and patient outcomes. The patient profiles were input into the AI system, which generated personalized treatment protocols and predicted patients' outcomes. We compared the similarities between the clinician prescribed protocols and AI generated treatment plans. We also looked at how the AI suggested treatments and predicted outcomes compared to the actual patient outcomes. The AI model showed a strong agreement with the clinician strategies and, in some cases, identified additional treatments supported by peer-reviewed research. It also recommended collaborating with other professionals for the overall recovery of the patient. The findings indicate that AI-based neuro-optometric systems can mimic important aspects of expert clinical reasoning and may offer a scalable way to improve diagnosis, treatment, and patient access to neuro optometric vision therapy.

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INTRODUCTION

Oculomotor dysfunction and subsequent vision therapy techniques have had a strong role in injury recovery in a wide array of settings. Early findings of war time injury had a high incidence of coexisting visual dysfunction. Following the conflicts in Iraq and Afghanistan, studies of service members with mild traumatic brain injury (mTBI) demonstrated a high prevalence of visual dysfunction, with approximately 60-75% exhibiting oculomotor, accommodative, or binocular vision abnormalities.[1-3] Meanwhile, concussion management in sports medicine and neurology largely emphasized physical and cognitive rest until symptom resolution.[4]

Beginning in the early 2000's, renewed attention to visual dysfunction following traumatic brain injury, driven in part by military studies from the VA (Veterans Affairs) and DoD (Department of Defense), contributed to increased use of neuro-optometric rehabilitation. Contemporary programs incorporated computerized training systems, quantitative outcome measures, and multidisciplinary approaches that extended beyond traditional orthoptic exercises to include broader cognitive and rehabilitative interventions.[1,3,5] Some of these computerized programs include Vision Therapy System 4 (VTS 4), Sanet Visual Integrator (SVI) and

Neurotracker which provided much better metrics and data for more quantitative data points.

These newer programs feature a great many data and decision points that would be greatly assisted with the use of Large Language Models (LLM). The authors have explored this concept in "Using Large Language Models in Neuro-Optometry Rehabilitation Program."[6] The paper suggests the development of a "Rehabilitation Database". This database would be the exclusive source of relevant clinical information that the LLM would query to generate informed answers to rehabilitation clinicians.

Further work on a rehabilitation database led to the hypothesis of this pilot study. Namely, that AI models will be able to suggest appropriate treatment plans along with additional suggestions for complementary interdisciplinary recommendations for better recovery outcomes.

MATERIALS AND METHOD

The study design is a retrospective observational pilot study with de-identified records. The data input was de-identified patient records from the Edmonds Eye Associates Vision based Neuro Rehabilitation practice in Philadelphia, Pennsylvania.

Inclusion criteria included:

- Patients that were involved with some type of mTBI
- Patients that had enough sessions to interpret their outcome
- Patients that had a known outcome

Exclusion criteria included:

- Patients that had visual dysfunction related to anything other than mTBI, such as migraines suffers, or native convergence insufficiency
- Patients that were frequent no shows or refused clinical recommendations
- Patients that were lost to follow-up for any number of reasons

The Large Language Model (LLM) was a custom, proprietary version of Chat GPT-5 (Generative Pre-trained Transformer-5) (Open AI). The LLM was pointed to a private rehabilitation database of specific Neuro-Optometric content.[7-11] The LLM exclusively queries this database to answer clinical questions posed by the user. The information that was input into the LLM was a full printout of a patient's deidentified initial comprehensive eye examination and binocular vision evaluation. The only prompt given was simple: "Generated personalized treatment protocols and predicted patient outcomes". The cases were uploaded one after another and the model analyzed and provided output as seen in Addendum A.

The model did not have any access to outcomes, treatments, or progress notes, only the initial evaluation. The output was not refined for this pilot study, although the model is certainly capable of follow-up questions and additional information. There was no blinding of reviewers during this retroactive pilot study, but rather a comparison between the output and the actual patient outcomes.

Ethical Considerations

This pilot study involved a retrospective analysis of existing clinical records that were fully de-identified prior to analysis in accordance with the HIPAA Privacy Rule Safe Harbor method (45 CFR §164.514). No protected health information (PHI) was used, and no patient contact or intervention occurred. As such, this study did not constitute human subjects research under 45 CFR §46.102 and was exempt from Institutional Review Board (IRB) review under 45 CFR §46.104(d)(4).

Results:

Treatment Comparisons:

Review addendum A for full AI output

Case 1

The actual treatment course for this patient was a total of 11 sessions with a full recovery back to baseline visual comfort, and return to normal school activities documented. Initial sessions started with convergence, divergence, saccade, pursuit and accommodation therapies. They progressed to add in jump vergences and the other exercises'

difficulties were increased at each session. After 4 sessions Neurotracker was added and difficulty was increased session to session.

Overall, the actual therapy that was administered very closely follows what the AI model recommended. It recommended a three-phase approach estimating 16 weeks of therapy starting with stabilization and autonomic calming, followed by oculomotor and binocular integration, followed by functional load and school stimulation. The real-world therapy merged the phases together rather than separating them. The timing was very close to the prediction as well as the overall outcome.

Case 2

The actual therapy spanned 6 sessions. Initial session started with VTS hololens base in and base out, and basic saccade and pursuit training. Accommodation and jump vergences were quickly added and the speed and reaction times were quickly ramped up as they tolerated the sessions very well. Neurotracker was added and ramped up quickly as well. The quicker the patient was able to get through the exercises the more we added to keep them engaged and keep the progress momentum.

The AI model recommended the three-phase approach spanning 16-20 weeks with binocular calming and work integration. AI also suggested we add functional binocular stability including store aisle simulation tasks, visual motion desensitization. Although we did not add functional binocular stability this could have been beneficial for this patient. This was likely done on their own and they were able to return to full-time work. The predicted timeline was longer than needed in real-world recovery. The patient was able to return to their full baseline and did not require ongoing treatment.

Case 3

Actual therapy sessions totaling 5 sessions. The initial VTS hololens targets had to be non randot targets as they were unable to appreciate Random dot stereopsis (RDS) targets. They were only able to tolerate one round of base in and base out exercises. Saccade and pursuit activities were minimally tolerated. Session 2 was much better tolerated, and difficulty was increased gradually. Neurotracker was introduced after session 3.

The AI generated plan demonstrated its ability to conceptualize the overall therapy and break it up into phases of therapy. It recommended 12-16 weeks. Phases include visual efficiency stabilization; binocular and oculomotor endurance followed by functional integration and return to load. Recommendations included fluorescent light desensitization and extended reading simulations. This is typically something that would be recommended for the patient to do outside of therapy such as taking laps around the grocery store without actually shopping or working their way up from short passages in magazines and working toward longer books or increasing screen time. The expected outcomes were true to the real outcome.

Case 4

This patient had a total of 4 sessions before taking an extended break from therapy and then returned for 8 additional sessions. They were not discharged from therapy but ended up stopping attending sessions. This case was complicated by their need to attempt to return back to work or lose their job.

Their sessions started with free space activities to establish any type of binocularity and basic saccades and pursuits. When they returned after their extended break from therapy we started into VTS 4 hololens with maximum depth of field settings. This was tolerated but they were unable to keep consistent sessions to build any momentum. The patient ultimately returned to work but was unable to keep their position due to their ongoing symptoms.

The AI model predicted 20-24 weeks of therapy. It breaks down therapy into neurovisual stabilization and safety, then binocular re-establishment and oculomotor control, and finally function integration and mobility confidence. It made good recommendations for a slow and steady recovery as well as an accurate guarded prognosis.

Case 5

This case had a total of 9 sessions with a full recovery to baseline visual function. They were very eager to get better and push through their discomfort. We started with VTS 4, hololens, saccades and pursuits. We quickly escalated to adding jump vergences, central fixation, and balance board complexities during pursuit and saccades exercises as well as Neurotracker.

The AI model predicted 12-14 weeks of therapy. Phases included visual comfort and stability, then binocular and oculomotor endurance, followed by functional integration and driving confidence. It went with a more conservative approach whereas real world therapy was able to advance much more quickly.

Case 6

This case had a total of 9 sessions. Sessions began with VTS 4 and SVI with base in, base out, saccades and pursuit training. Difficulty was increased as tolerated by the patient. They graduated from therapy with ongoing headaches and some cognitive delay which they will be seeing cognitive therapy and their neurologist to further explore.

The AI model recommended 14-18 weeks of therapy. Its phases included headache reduction and binocular calming, then binocular stamina and oculomotor efficiency finishing with functional load and screen reintegration. The plan was similar to real world therapy.

Case 7

The total therapy sessions for this case were 6. Therapy started with RDS targets for base in and base out, and SVI for saccades and pursuits. We progressed through and added jump vergences, central fixation during saccades and pursuits, as well as use of a balance board through the exercises.

The final piece of the therapy was to update the glasses prescription.

The AI generated a well-constructed and appropriate management plan for this patient. It started with photophobia reduction and visual comfort followed by binocular and oculomotor endurance finishing with functional workplace integration. It recommended many additional tasks that could have been given for homework to reinforce the in-office therapy.

Case 8

This case had a total of 6 therapy sessions. Therapy started with VTS hololens with base in and base out activities as well as SVI for saccades and pursuits. Neurotracker was added toward the end of therapy to work on visual processing integration.

AI spaced out the phases much more than was actually performed in this case. It recommended 10-14 weeks of therapy. It recognized the surgical limitations and planned accordingly. Phases started with binocular awareness and stability, followed by binocular coordination and endurance finishing with functional binocular integration.

DISCUSSION

The AI model was able to largely replicate the treatment protocols of the experienced licensed optometrists. The recommendations based on the evaluation findings as well as the predicted timeline is what we find to be the standard of textbook cases of the diagnoses that were determined. The timelines in our Vision Based Neuro Rehabilitation were somewhat shorter, however, it is likely that in a more traditional vision therapy practice model, where the therapist does therapy without the doctor's direct involvement at each session, would likely be closer to the textbook predicted time frames. As the model could be continuously trained, the outcome times could be more accurate the more data the model has about a particular practice. For this pilot, the model was not given any additional information other than the published text.

Chat GPT-5 was chosen for the LLM due to its ease of use, both on setting up a model that can only reference selected data, as well as being conversational and easy to understand. Other LLMs can reference select data sets but as their training is less broad, the conversational aspects of the responses are limited.

Chat GPT-5 is not without its limitations. It was not trained on any set number of patients as this is still in its early stages of development. If Chat GPT-5 is to be used further there will need to be significant time spent on fully training the model to reduce errors and hallucinations.

LLMs also cannot account for patient variability. Some patients are motivated to push through therapy sessions while others struggle with underlying depression or other metal health issues and are overwhelmed by simple therapy sessions. Finances can play a role in therapy regardless of motivation. The plan of treatment is always fluid and needs to be updated accordingly. Any LLM is not going to be able to predict human issues and therefore must remain as the assistant to the human clinician.

Chat GPT 5 can be praised for its ability to simplify complex medical terminology but can often get abbreviations incorrect and can misinterpret data.[12] The clinician must always challenge any LLM and verify the source of conclusions or recommendation. ChatGPT-5 is also known to come to plausible conclusions, but with further investigation may find the conclusion is incorrect.[13]

Chat GPT 5 was also able to recommend additional strategies that could easily be integrated into future cases to help move things along even quicker and more efficiently.

At the heart of therapy, the doctor alongside a well-trained vision therapist can make a huge impact in a patient's recovery along with the information recommended from a LLM. Good therapists are well trained to make subtle changes to the therapy protocols during each session such as:

- Increasing or decreasing depth of field
- Increase or decrease target size and speed
- Adding or removing central fixation targets
- Adding or reduce complexity of the targets used such as incorporating letters and numbers, capital or lower case, or changing the targets images to alter the clues to depth perception.
- Add in or remove the balance board depending on the active symptoms or presentation of the patient that day

We use a standard protocol at each session to evaluate all eye movements. We sometimes find eye movement deficiencies that were not noted in the initial evaluation. Once these are discovered, the therapy sessions are adjusted to address all areas of weakness.

In office sessions are scheduled weekly for 45-minute sessions where 30 minutes are face to face with the therapist and the 15 minutes are face to face with the treating optometrist. Exact time on each individual exercise is determined at the time by the therapist based on entering symptoms that day, motivation level, historical time to recover, etc. More time may be spent on more difficult areas for any particular patient with multiple different strategies per session. Homework is offered at times to reinforce tasks but is often unnecessary when recovery is moving along quickly. We have found that daily activity among a patient's already busy schedule of other therapy is often enough to reinforce the techniques worked on during the in-office sessions.

Clinical implications for using this AI assisted treatment program in a real-world clinic would need to factor in HIPAA compliance, the skill level of the therapist, and the equipment available in the clinic.

Limitations to this paper include a retrospective design, a small sample size and a single practice. The books uploaded to the model are limited to the contributing authors' real world experience, referencing material, as well as years of practice, but may differ from others' real world experience.

FUTURE IMPLICATIONS

The amount of literature that is uploaded into the model can further expand its capabilities. There is the potential to incorporate the AI model directly into EHR systems to keep information HIPAA compliant and streamline the recommendation process. Many EHR systems are beginning their journey to incorporate AI. These systems are in their infancy and need more work to incorporate AI generated suggestions that the human clinician may adapt or modify to plan out each case. Currently most EHRs are only focused on quicker

and more accurate data entry. AI models however are capable of much more.

Attempts have been made to integrate multiple offices and specialties to share patient data and progress notes. Most offices still rely on fax transmissions to share this data. Hopefully the AI systems will find a more robust way to integrate the data and be able to summarize it in a way that is understandable for different specialties. If the physical therapist can automatically generate an AI summary into the optometrist EHR and vice versa then a patient that is working on vestibular and vision therapy could have better complimenting therapy protocols to help in the overall recovery.

For the study, the next steps would be a prospective study, adding in clinic specific data such as the type of equipment available and the expertise level of the therapist as well as adding additional literature to the AI model. There is the concern of giving too much data and may be conflicting therefore confusing the model. A double-blind study could further solidify the power of the AI model to compare speed of recovery as well as resources needed to have excellent patient outcomes.

CONCLUSION

This AI model has the ability to point the doctor and therapy team in the right direction. This would include setting up a therapy protocol for each patient and predicting possible outcomes and timelines. This model could be used for an optometrist that already performs therapy in their office to help them think more thoroughly through their treatment plans. This could result in better outcomes for their patients. It could also be used for a therapist that is looking to expand their knowledge and process through more challenging cases. The model could also help an optometrist get their feet wet with therapy and build a therapy practice more quickly and efficiently than without it. The model could also be adjusted to the specifics of the therapy environment to increase or decrease projected timelines based on how a practice chooses to deploy their therapy. It could also consider the type of equipment available or what kind of multi-disciplinary team is available in the area the therapy is taking place.

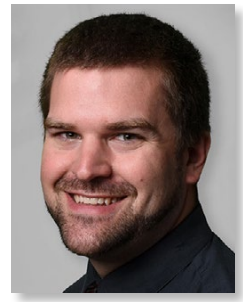
As good as any of these LLMs are at this time, they cannot be relied upon for clinical decision making. They are only a tool to assist a licensed doctor to consider, adapt and implement for any specific patient. LLMs focused on a good rehabilitation database can be an important tool to make a busy clinician think deeper about each case and bring up ideas that may have otherwise been overlooked. Often a team of doctors will sit down and go over cases together and bounce ideas off each other. This trained LLM can be a valuable assistant for the team and provide and interpret data without the time and resources that would otherwise be required.

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Dr. Chad Edmonds is an optometrist and practice owner in the Philadelphia area with a clinical focus on neuro-optometric rehabilitation, vision therapy, and primary eye care. He is a member of the staff at Magee Rehabilitation Hospital and Bryn Mawr Rehabilitation Hospital where he evaluates and manages visual dysfunction associated with concussion, traumatic brain injury, stroke, binocular vision disorders, and other neurologic conditions.

Dr. Edmonds is a 2017 Graduate of the Pennsylvania College of Optometry at Salus/Drexel University where he was recognized with awards for excellence in Primary Care and Low Vision.

Dr. Edmonds is actively involved in exploring the integration of artificial intelligence into neuro-optometric clinical decision-making. He has co-authored papers on the use of Large Language Models for Neuro-Rehabilitation and Vision Based Neuro-Rehabilitation.

AI output:
Below is a **personalized, vision-centered neuro-optometric rehabilitation plan and predicted outcome** for this patient, derived only from the uploaded clinical record and the Neuro-Optometric Rehabilitation reference files you provided, in alignment with your Concussion GPT rules.

CASE 1: 14-Year-Old Male — Post-Concussion Visual Dysfunction
Dx:
- Concussion with brief LOC
- Convergence insufficiency
- Deficient saccadic eye movements
- Accommodative spasm
- Light sensitivity / autonomic instability

(Clinical findings summarized from the ABI evaluation)

CLINICAL PATTERN RECOGNITION
Primary Dysfunction Clusters
Based on testing and symptom surveys, this patient demonstrates a **classic post-concussion visual fatigue pattern:**

1. **Oculomotor Dysfunction**
- Jerky, inaccurate saccades with head movement
- Reduced vergence facility (unable to clear BO)
- Poor accommodative flexibility, especially with plus
- Visual fatigue worsening through the school day

(Consistent with eye movement dysfunction patterns described in)

2. **Binocular Stress / CI**
- Reduced BO ranges
- Diplopia at near under reduced illumination
- Near-point stress despite normal NPC

(Consistent with concussion-related CI mechanisms in)

3. **Autonomic / Sensory Overload**
- Light sensitivity (fluorescent & outdoor)
- Motion sensitivity
- Symptom escalation with cognitive-visual load
- Worse later in day → parasympathetic fatigue

(Consistent with autonomic dysregulation in)

4. **Functional Impact**
- Chromebook intolerance
- Reduced reading stamina
- Eye pain, headaches, visual fatigue
- Predictive symptom score >31 (high rehab responder likelihood)

PERSONALIZED TREATMENT PROTOCOL
Format: Office-based vision-centered neuro-rehabilitation
Frequency: 1-2× / week, 45-60 min
Estimated Duration: 12-16 weeks

PHASE 1: STABILIZATION & AUTONOMIC CALMING (Weeks 1-4)
Goals
- Reduce symptom volatility
- Improve visual comfort
- Establish parasympathetic dominance
- Prepare system for motor learning

Therapy Emphasis
- Large-field, low-frequency visual tasks
- Reduced demand, high success
- Controlled lighting environments

Procedures
- **Binocular anti-suppression at distance**
- **Gross vergence awareness without demand**
- **Monocular accommodative release**
- **Saccadic awareness without speed**
- **Visual-vestibular dissociation (yaw-limited)**

Home Support
- Light-filtered lenses as needed
- Scheduled visual breaks during Chromebook use
- Avoid end-range vergence stress early

(Phase structure consistent with concussion foundations framework)

PHASE 2: OCULOMOTOR & BINOCULAR INTEGRATION (Weeks 5-10)

Goals
- Normalize saccadic accuracy
- Improve BO facility
- Restore accommodative flexibility
- Increase visual stamina

Therapy Emphasis
- Precision before speed
- Binocular motor planning
- Reduced head substitution

Procedures
- Horizontal and vertical saccadic sequencing
- Vergence facility with graded BO demand
- Accommodative rock with controlled plus
- Near-to-far integration tasks
- Visual endurance drills with cognitive overlay

(Eye movement remediation principles from)

PHASE 3: FUNCTIONAL LOAD & SCHOOL SIMULATION (Weeks 11-16)

Goals
- Chromebook tolerance
- Sustained reading without symptom escalation
- Classroom visual resilience
- Return to full academic visual load

Therapy Emphasis
- Time-based endurance
- Multisensory integration
- Stress inoculation

Procedures
- Timed reading and tracking
- Complex saccade-vergence tasks
- Visual-cognitive dual tasking
- Lighting variability exposure
- Motion + visual integration drills

(Functional reintegration supported by)

PREDICTED PATIENT OUTCOME

Prognosis: GOOD → VERY GOOD

Positive Predictors
- Young age (high neuroplasticity)
- No prior concussion history
- Normal ocular health
- High symptom-test mismatch (responders)
- DEM speed intact (motor learning intact)
- NPC intact → good convergence potential

Expected Improvements

Domain	Expected Change
Reading stamina	↑ significantly
Chromebook tolerance	↑ to full school day
Eye pain/headaches	↓ markedly
Light sensitivity	↓ moderate to significant
Saccadic accuracy	Normalize
BO facility	Improve to functional levels
End-of-day fatigue	↓ substantially

Timeline
- **4-6 weeks:** noticeable symptom relief
- **8-10 weeks:** academic tolerance improves
- **12-16 weeks:** functional normalization for school

Residual Risk
- Temporary symptom flare with rapid academic load increases
- Requires coordination with school accommodations during rehab

SUMMARY

This patient demonstrates a **textbook post-concussion visual efficiency collapse,** not structural ocular pathology. Vision-centered neurorehabilitation is **indicated, necessary, and likely highly effective,** with expected return to age-appropriate visual performance when therapy is sequenced correctly.

If you want, I can:

- Convert this into a formal clinic-ready treatment plan
- Generate parent-friendly counseling language
- Create school accommodation recommendations
- Compare this case against poor-responder patterns

CASE 2: 26-Year-Old Male — Work-Related Concussion

Dx:

- Concussion without loss of consciousness
- Convergence insufficiency
- Accommodative spasm (bilateral)
- Deficient saccadic eye movements

(Clinical data derived from ABI/VT evaluation)

CLINICAL PATTERN RECOGNITION

Primary Dysfunction Clusters

This patient demonstrates a **moderate-severe post-concussion visual-vestibular integration failure** with binocular instability and autonomic overload.

1. Binocular Instability / CI Pattern

- Near exophoria with OS drifting in (confirmed by Occupational Therapy (OT))
- NPC significantly receded (18 cm break / 24 cm recovery)
- Vergence facility **0 CPM**, unable to clear BO
- Diplopia emerging in real-world environments (stadium, stores)
- Reduced stereoacuity (70 arc sec)

→ Indicates **poor fusional endurance,** not baseline strabismus.

2. Oculomotor Control Deficit

- Saccadic inaccuracy with head substitution
- Reduced DEM speed (FAIL/FAIL)
- Visual tracking breakdown under load
- Difficulty maintaining fixation in visually busy environments

(Consistent with concussion-related oculomotor inefficiency patterns described in the Eye Movements reference)

3. Autonomic/Sensory Overload

- Disorientation in loud environments
- Balance failure on Adult Developmental Eye Movement (ADEM)
- Difficulty in stores (classic visual motion sensitivity)
- Head pain escalation with sensory input

→ Suggests visual-vestibular mismatch and sympathetic dominance.

(Aligned with autonomic dysfunction patterns in)

4. Functional Impact

- Daily headaches
- Diplopia in public environments
- Work intolerance
- Reliance on OT/Physical Therapy (PT) without visual system normalization

PERSONALIZED TREATMENT PROTOCOL

Format: Vision-centered neuro-optometric rehabilitation (office-based)
Frequency: 1-2× / week, 45-60 minutes
Estimated Duration: 16-20 weeks (severity-adjusted)

PHASE 1: STABILIZATION & BINOCULAR CALMING (Weeks 1-4)

Goals

- Reduce diplopia frequency
- Establish binocular awareness without demand
- Calm autonomic overactivation
- Reduce head movement compensation

Therapy Emphasis
- Low-load, high-success binocular tasks
- Controlled sensory environment
- Avoidance of end-range vergence stress

Procedures
- **Large-field fusion awareness**
- **Monocular accommodative release**
- **Anti-suppression with distance emphasis**
- **Slow, predictable saccadic initiation**
- **Visual-vestibular dissociation (yaw-limited)**

(Home support coordinated with OT to prevent overloading)
(Foundational sequencing supported by)

PHASE 2: BINOCULAR & OCULOMOTOR INTEGRATION (Weeks 5-12)
Goals
- Restore BO vergence control
- Improve fusional stamina
- Normalize saccadic accuracy
- Reduce diplopia under motion

Therapy Emphasis
- Precision before speed
- Binocular motor planning
- Gradual sensory complexity

Procedures
- **Stepwise BO vergence training**
- **Vergence facility with extended recovery time**
- **Horizontal & vertical saccadic sequencing**
- **Fixation stability under motion**
- **Near-far integration drills**

(Protocols aligned with concussion rehab framework)

PHASE 3: FUNCTIONAL LOAD & WORK SIMULATION (Weeks 13-20)
Goals
- Tolerance of visually complex environments
- Eliminate real-world diplopia
- Restore balance confidence
- Return to work without symptom escalation

Therapy Emphasis
- Time-based endurance
- Multisensory integration
- Stress inoculation

Procedures
- **Store-aisle simulation tasks**
- **Visual motion desensitization**
- **Dual-task visual-vestibular drills**
- **Noise + visual integration**
- **Work-specific visual demands**

PREDICTED PATIENT OUTCOME
Prognosis: FAIR → GOOD

Positive Predictors
- No prior concussion history
- Intact ocular health
- Actively engaged in multidisciplinary rehab
- Strong accommodative amplitude baseline

Limiting Factors
- Significant vestibular involvement
- Balance failure on ADEM
- Diplopia emerging under real-world load
- Autonomic instability

Expected Improvements

Domain	Expected Change
Diplopia	↓ significant
Store tolerance	↑ moderate to high
Headaches	↓ frequency & intensity
Balance confidence	↑
Vergence facility	Improve from 0 CPM to functional
Work endurance	Gradual restoration

Timeline
- **4-6 weeks:** reduced diplopia frequency
- **8-12 weeks:** improved store/work tolerance
- **16-20 weeks:** functional binocular stability

SUMMARY

This patient exhibits a **true binocular-vestibular concussion profile,** not a refractive or structural issue. Vision-centered neuro-rehabilitation is **essential** for resolution, as OT/PT alone cannot normalize binocular control. With appropriately sequenced therapy, return to functional work performance is expected.

When you're ready, say **"next case"**, and I'll proceed with the following patient in the **same exact format**.

CASE 3: 27-Year-Old Female — Motor Vehicle Accident with Prior Concussion

Dx:

- Concussion without loss of consciousness
- Convergence insufficiency
- Deficient saccadic eye movements
- Congenital retinal finding (CHRPE OS — incidental, monitored)

(Clinical data derived from ABI/VT evaluation)

CLINICAL PATTERN RECOGNITION

Primary Dysfunction Clusters

This patient demonstrates a **recurrent concussion visual efficiency collapse** with preserved basic oculomotor mechanics but **poor endurance, speed, and sensory tolerance** under load.

1. Binocular Stress / CI with Endurance Failure

- Near exophoria with reduced BI facility (fails BI)
- BO ranges adequate in isolation but degrade with sustained demand
- NPC to nose intact → **capacity present, stamina deficient**
- Alternating suppression on Worth 4 Dot (distance/near)

→Indicates **binocular instability under fatigue,** not fixed alignment loss.

2. Oculomotor Endurance & Speed Deficit

- NSUCO largely passing except **saccadic body control**
- Developmental Eye Movement (DEM): **Speed FAIL** despite otherwise organized eye movements
- Patient-reported difficulty with reading, screen time, and fatigue

→ Classic **post-concussion oculomotor inefficiency,** not primary eye movement paresis.

(Consistent with concussion eye-movement fatigue patterns in)

3. Autonomic/Sensory Overload

- Visual distortions under fluorescent lighting ("squiggly lines")
- End-of-day headaches (4/7 days)
- Sensory fatigue in busy environments
- Balance complaints (stairs), though ADEM balance passes

→ Reflects **sensory integration overload** rather than vestibular failure.

(Aligned with autonomic dysregulation described in)

4. Functional Impact

- Screen intolerance
- Reduced reading stamina
- Visual fatigue limiting OT progression
- History of **multiple concussions → lower reserve**

PERSONALIZED TREATMENT PROTOCOL

Format: Vision-centered neuro-optometric rehabilitation (office-based)

Frequency: 1× / week initially → progress to 2×/week as tolerated

Estimated Duration: 12-16 weeks

PHASE 1: VISUAL EFFICIENCY STABILIZATION (Weeks 1-4)

Goals

- Reduce visual fatigue
- Improve binocular comfort
- Normalize autonomic tone
- Prevent over-fatigue during therapy

Therapy Emphasis

- Endurance-based tasks at sub-symptom threshold
- Controlled lighting
- High success, low variability

Procedures

- Sustained fixation with controlled breaks
- Low-demand binocular fusion tasks
- Monocular accommodative flexibility
- Slow saccadic initiation without speed demand
- Visual calming techniques

(Sequencing consistent with concussion foundations framework)

PHASE 2: BINOCULAR & OCULOMOTOR ENDURANCE (Weeks 5-10)

Goals

- Improve BI facility
- Increase reading and screen stamina
- Normalize saccadic speed without body substitution

Therapy Emphasis

- Time-on-task pr ogression
- Precision before speed
- Gradual sensory complexity

Procedures

- BI vergence stamina training
- Vergence facility with fatigue monitoring
- Saccadic sequencing with postural control
- Near-far integration
- Cognitive-visual dual tasks

(Protocols supported by concussion treatment framework)

PHASE 3: FUNCTIONAL INTEGRATION & RETURN TO LOAD (Weeks 11-16)

Goals

- Restore screen tolerance
- Reduce end-of-day headaches
- Improve performance in visually busy environments
- Support full participation in OT and daily activities

Therapy Emphasis

- Endurance under realistic conditions
- Sensory stress inoculation
- Task-specific carryover

Procedures

- **Extended reading and screen simulations**
- **Fluorescent lighting exposure drills**
- **Dual-task visual-cognitive activities**
- **Stair-navigation visual tasks**
- **Fatigue recovery training**

PREDICTED PATIENT OUTCOME

Prognosis: GOOD

Positive Predictors

- Young age
- Intact basic oculomotor mechanics
- NPC intact
- Good stereoacuity
- Actively engaged in multidisciplinary care

Limiting Factors

- History of prior concussion
- Sensory overload susceptibility
- Endurance-based symptom provocation

Expected Improvements

Domain	Expected Change
Screen tolerance	↑ significant
Reading stamina	↑
End-of-day headaches	↓ frequency
Visual fatigue	↓
Saccadic speed	Normalize
Sensory tolerance	↑ moderate

Timeline

- **3-5 weeks:** noticeable reduction in visual fatigue
- **8-10 weeks:** improved screen and reading tolerance
- **12-16 weeks:** functional normalization for daily activities

SUMMARY

This patient represents a high-capacity, low-endurance post-concussion profile compounded by repeat injury. Vision-centered neuro-rehabilitation is strongly indicated and is expected to significantly reduce fatigue-based symptoms, enabling more effective participation in OT and daily life.

Say **"next case"** when ready, and I'll continue with the following patient in the **same exact format.**

CASE 4: 55-Year-Old Female — Assault-Related Concussion with Severe Visual Disruption

Dx:

- Concussion with loss of consciousness (<30 minutes)
- Convergence insufficiency
- Deficient saccadic eye movements
- Deficient smooth pursuit eye movements
- Suspected visual field disruption (pending automated fields)

(Clinical data derived from ABI/VT evaluation)

CLINICAL PATTERN RECOGNITION

Primary Dysfunction Clusters

This patient presents with a **severe, multi-system post-concussion visual breakdown** characterized by binocular collapse, oculomotor instability, sensory overload, and balance failure.

1. Severe Binocular Dysfunction/Diplopia Dominant Profile

- Markedly receded NPC (blur 42 cm; break 35 cm)
- Extremely reduced vergence ranges and vergence facility 0 CPM (fails BI & BO)
- Constant diplopia, worse at near; frequent eye closure for clarity
- Poor stereoacuity (200 arc sec; 2/10 Randot)
- Indicates loss of fusional control, not simple CI.

2. Global Oculomotor Instability

- Jerky pursuits and saccades with undershoots
- NSUCO scores reduced across ability, accuracy, and head control
- DEM: Speed FAIL / Balance FAIL
- Difficulty maintaining fixation and tracking

→ Reflects diffuse oculomotor network disruption, consistent with moderate TBI visual sequelae.

(*Consistent with eye-movement pathology described in*)

3. Autonomic & Sensory Overload

- Severe light sensitivity (fluorescent and outdoor)
- Visual motion intolerance; avoidance of busy environments
- Balance failure on ADEM
- Headaches provoked by visual use

→ Suggests significant autonomic dysregulation with visual-vestibular mismatch.

(Aligned with autonomic mechanisms outlined in)

4. Functional Impact

- Constant diplopia
- Reading nearly impossible
- Severe depth perception disturbance
- Loss of confidence walking and navigating space
- High symptom burden (BIVSS 93/112; strong rehab responder)

PERSONALIZED TREATMENT PROTOCOL

Format: Vision-centered neuro-optometric rehabilitation (office-based)
Frequency: 2× / week (severity-driven)
Estimated Duration: 20-24 weeks

PHASE 1: NEUROVISUAL STABILIZATION & SAFETY (Weeks 1-6)

Goals

- Reduce diplopia intensity
- Improve visual comfort and safety
- Establish basic binocular awareness
- Decrease autonomic overactivation

Therapy Emphasis

- Very low demand
- Large-field stimuli
- High success, short duration tasks

Procedures

- **Monocular control with gradual binocular introduction**
- **Large, slow pursuit awareness**
- **Anti-suppression without vergence demand**
- **Visual midline and spatial orientation tasks**
- **Environmental light control strategies**

(Foundational sequencing per concussion rehab principles)

PHASE 2: BINOCULAR RE-ESTABLISHMENT & OCULOMOTOR CONTROL (Weeks 7-16)

Goals

- Rebuild fusional reserves
- Improve pursuit and saccadic accuracy
- Increase tolerance to near tasks
- Reduce reliance on eye closure

Therapy Emphasis

- Precision before endurance
- One parameter at a time
- Strict symptom threshold monitoring

Procedures

- Stepwise vergence re-training (short ranges)
- Vergence facility with extended recovery
- Guided pursuit tracking
- Saccadic accuracy drills without speed
- Near-far integration in controlled posture

(Protocols consistent with concussion treatment framework)

PHASE 3: FUNCTIONAL INTEGRATION & MOBILITY CONFIDENCE (Weeks 17-24)

Goals

- Improve depth perception
- Reduce diplopia in real-world settings
- Restore walking and navigation confidence
- Improve reading tolerance

Therapy Emphasis

- Functional carryover
- Visual-vestibular reintegration
- Gradual sensory complexity

Procedures

- **Depth judgment tasks**
- **Mobility-based visual drills**
- **Dual-task balance + vision activities**
- **Graduated exposure to busy environments**
- **Extended reading with pacing strategies**

PREDICTED PATIENT OUTCOME

Prognosis: GUARDED → FAIR

Positive Predictors

- No prior concussion history
- Strong motivation and compliance
- Clear symptom-test correlation (high responder score)

Limiting Factors

- Age
- LOC with assault mechanism
- Severe baseline diplopia
- Balance failure and sensory overload

Expected Improvements

Domain	Expected Change
Diplopia	↓ meaningful but may persist intermittently
Reading tolerance	↑ from minimal to functional
Light sensitivity	↓ moderate
Depth perception	↑
Balance confidence	↑
Overall visual comfort	↑

Timeline

- **6-8 weeks:** reduced visual distress, safer mobility
- **12-16 weeks:** improved near task tolerance
- **20-24 weeks:** functional gains with possible residual symptoms

SUMMARY

This patient represents a **severe post-concussion visual integration failure** involving binocular vision, oculomotor control, and autonomic regulation. Vision-centered neuro-rehabilitation is medically necessary, safety-oriented early, and expected to yield meaningful functional improvement, though some residual symptoms may remain.

Say **"next case"** when ready, and I'll proceed with the following patient in the **same exact format.**

CASE 5: 48-Year-Old Female — Motor Vehicle Accident with Visual Fatigue & Reading Dysfunction

Dx:

- Concussion without loss of consciousness
- Convergence insufficiency
- Deficient saccadic eye movements

(Clinical data derived from ABI/VT evaluation)

CLINICAL PATTERN RECOGNITION

Primary Dysfunction Clusters

This patient demonstrates a **moderate post-concussion visual efficiency and endurance collapse** with preserved alignment and eye movement mechanics, but **poor tolerance to sustained visual and cognitive load,** particularly with screens and reading.

1. Binocular Stress/CI with Fatigue Component

- Near vergence breakdown with failure to clear BO on vergence facility
- NPC blur at 24 cm despite ability to converge to nose
- Stereoacuity intact (20 arc sec) → **binocular potential preserved**
- Worth 4 Dot stable → no constant suppression or diplopia

→ Indicates **binocular stamina failure**, not structural binocular loss.

2. Oculomotor Endurance & Speed Deficit

- Pursuits and saccades smooth on qualitative testing
- NSUCO scores all passing
- DEM: **Speed FAIL** despite normal balance

→ Reflects **reduced processing speed and visual sequencing efficiency,** common in concussion without focal eye movement paresis.

(Consistent with post-concussion eye movement inefficiency described in)

3. Autonomic/Sensory Load Sensitivity

- Eye strain and headaches provoked by screen use
- Sound sensitivity and visually busy environment intolerance
- Driving anxiety (better as passenger)
- End-of-day fatigue pattern

→ Suggests **visual-autonomic overload,** rather than vestibular failure (ADEM balance PASS).

(Aligned with autonomic dysregulation patterns in)

4. Functional Impact

- Reading difficulty and slow comprehension
- Screen intolerance
- Reduced confidence in driving
- Visual fatigue limiting daily activities

PERSONALIZED TREATMENT PROTOCOL

Format: Vision-centered neuro-optometric rehabilitation (office-based)

Frequency: 1× / week (progress to 2× / week if tolerated)

Estimated Duration: 12-14 weeks

PHASE 1: VISUAL COMFORT & STABILITY (Weeks 1-4)

Goals
- Reduce visual strain and headaches
- Improve comfort with near work
- Normalize autonomic tone during visual tasks

Therapy Emphasis
- Low variability
- Controlled lighting
- Short-duration, high-success tasks

Procedures
- **Sustained fixation with pacing**
- **Low-demand binocular fusion tasks**
- **Monocular accommodative balance**
- **Slow saccadic initiation**
- **Visual rest-recovery training**

(Sequencing aligned with concussion foundations framework)

PHASE 2: BINOCULAR & OCULOMOTOR ENDURANCE (Weeks 5-9)

Goals
- Improve BO vergence stamina
- Increase reading endurance
- Normalize visual processing speed

Therapy Emphasis
- Time-on-task progression
- Precision before speed
- Symptom-threshold monitoring

Procedures
- **BO vergence stamina training**
- **Vergence facility with extended recovery**
- **Saccadic sequencing drills**
- **Near-far integration**
- **Reading efficiency tasks**

(Protocols consistent with concussion rehab strategies)

PHASE 3: FUNCTIONAL INTEGRATION & DRIVING CONFIDENCE (Weeks 10-14)

Goals
- Restore screen tolerance
- Improve reading speed and comprehension
- Reduce visual fatigue during driving and busy environments

Therapy Emphasis
- Real-world simulation
- Cognitive-visual integration
- Endurance under realistic conditions

Procedures
- **Extended screen simulation**
- **Dual-task visual-cognitive drills**
- **Visual scanning for driving**
- **Environmental complexity exposure**
- **Fatigue recovery strategies**

PREDICTED PATIENT OUTCOME

Prognosis: GOOD

Positive Predictors
- No LOC
- No prior concussions
- Intact stereoacuity and eye movements
- ADEM balance intact
- Moderate symptom profile (predictive score >31)

Limiting Factors
- High daily screen exposure
- Sensory sensitivity

Expected Improvements

Domain	Expected Change
Reading stamina	↑ significant
Screen tolerance	↑
Headaches	↓ frequency
Eye strain	↓
Visual processing speed	Normalize
Driving confidence	↑

Timeline

- **3-4 weeks:** reduced eye strain and headaches
- **6-8 weeks:** improved reading and screen tolerance
- **12-14 weeks:** return to normal daily visual demands

SUMMARY

This patient represents a visual endurance-dominant concussion profile with preserved mechanics but impaired stamina and processing speed. Vision-centered neuro-rehabilitation is expected to be highly effective, with strong likelihood of full functional recovery.

Say **"next case"** when ready, and I'll continue with the following patient in the **same exact format.**

CASE 6: 49-Year-Old Female — Motor Vehicle Accident with Headache-Dominant Visual Instability

Dx:

- Concussion with brief loss of consciousness (<1 minute)
- Convergence insufficiency
- Deficient saccadic eye movements

(Clinical data derived from ABI/VT evaluation)

CLINICAL PATTERN RECOGNITION

Primary Dysfunction Clusters

This patient presents with a **headache- and dis-orientation-dominant post-concussion visual profile,** where binocular instability and visual overload are major drivers of symptoms rather than gross oculomotor paresis.

1. **Binocular Instability with Headache Provocation**
   - Esophoric tendency on OD in vertical positions at near
   - Reduced BO ranges with **vergence facility 0 CPM (fails BO)**
   - NPC blur at 33 cm despite ability to converge to nose
   - Closing the **right eye reduces headache and disorientation**

   → Indicates **binocular stress-driven headache,** not primary ocular disease.

2. **Oculomotor Mechanics Intact, Efficiency Reduced**
   - Qualitative pursuits and saccades smooth
   - NSUCO scores passing across domains
   - DEM: **Speed FAIL** with preserved balance

   → Suggests **processing-speed and endurance inefficiency,** common after concussion with brief LOC.

(Consistent with concussion-related eye-movement inefficiency described in)

3. **Autonomic/Sensory Overload**
   - Headaches all day (1-7/10), worsened by screens
   - Disorientation with visual strain
   - Photo-sensitivity intermittent
   - Postural instability when both eyes closed

   → Reflects visual-autonomic imbalance and poor visual contribution to postural control.

(Aligned with autonomic dysregulation mechanisms in)

4. **Functional Impact**
   - Inability to tolerate dual screens or multiple tabs
   - Reading intolerance
   - Screen time precipitates headache and dizziness
   - Reliance on eye closure to manage symptoms

PERSONALIZED TREATMENT PROTOCOL

Format: Vision-centered neuro-optometric rehabilitation (office-based)
Frequency: 1-2× / week (symptom-driven)
Estimated Duration: 14-18 weeks

PHASE 1: HEADACHE REDUCTION & BINOCULAR CALMING (Weeks 1-5)

Goals
- Reduce headache frequency and intensity
- Improve binocular comfort
- Decrease visual-driven disorientation
- Establish stable visual input for posture

Therapy Emphasis
- Low-load binocular work
- Controlled lighting
- Strict symptom-threshold pacing

Procedures
- **Monocular-binocular balance tasks**
- **Large-field fusion awareness**
- **Accommodative release techniques**
- **Slow, predictable saccadic initiation**
- **Visual midline and posture stabilization tasks**

(Foundational sequencing supported by)

PHASE 2: BINOCULAR STAMINA & OCULOMOTOR EFFICIENCY (Weeks 6-12)

Goals
- Improve BO vergence endurance
- Reduce reliance on eye closure
- Normalize visual processing speed
- Improve tolerance to reading and screens

Therapy Emphasis
- Precision before endurance
- Gradual load increases
- Recovery-based pacing

Procedures
- **Stepwise BO vergence training**
- **Vergence facility with extended recovery**
- **Saccadic sequencing with postural control**
- **Near-far integration drills**
- **Reading efficiency training**

(Protocols consistent with concussion rehabilitation framework)

PHASE 3: FUNCTIONAL LOAD & SCREEN REINTEGRATION (Weeks 13-18)

Goals
- Tolerate sustained screen use
- Reduce headache provocation from visual tasks
- Restore confidence with multitasking
- Improve daily functional endurance

Therapy Emphasis
- Real-world simulation
- Cognitive-visual dual tasking
- Endurance under realistic conditions

Procedures
- **Single- to dual-screen simulation**
- **Multi-tab visual scanning tasks**
- **Extended reading with pacing**
- **Visual-cognitive multitasking**
- **Fatigue recovery strategies**

PREDICTED PATIENT OUTCOME
Prognosis: GOOD

Positive Predictors
- Brief LOC only
- No prior concussion history
- Intact oculomotor mechanics
- Good engagement in multidisciplinary care

Limiting Factors
- Daily headache burden
- Visual-driven disorientation
- Autonomic sensitivity

Expected Improvements

Domain	Expected Change
Headache frequency/intensity	↓ significant
Screen tolerance	↑
Reading stamina	↑
Visual-induced dizziness	↓
Vergence stamina	Normalize
Daily functional endurance	↑

Timeline

- **4-6 weeks**: reduction in headache severity
- **8-12 weeks:** improved reading and screen tolerance
- **14-18 weeks:** functional return to daily visual demands

SUMMARY

This patient exhibits a **binocular stress-driven, headache-dominant concussion profile.** Vision-centered neuro-rehabilitation is strongly indicated and expected to significantly reduce headaches and visual-provoked disorientation, enabling return to normal screen and reading activities.

Say **"next case"** when ready, and I'll proceed with the following patient in the **same exact format.**

CASE 7: 49-Year-Old Female — Motor Vehicle Accident with Photophobia & Visual Fatigue

Dx:

- Concussion without loss of consciousness
- Convergence insufficiency
- Deficient saccadic eye movements
- Deficient smooth pursuit eye movements

(Clinical data derived from ABI/VT evaluation)

CLINICAL PATTERN RECOGNITION

Primary Dysfunction Clusters

This patient demonstrates a **light-sensitivity-dominant post-concussion visual profile** with binocular inefficiency, oculomotor fatigue, and autonomic visual overload, compounded by **high visual demand occupational exposure** (teaching).

1. Photophobia & Autonomic Visual Overload

- Severe light sensitivity: indoor glare, fluorescent lights, outdoor light **always problematic**
- Requires environmental light reduction at work
- Visual discomfort worsens as day progresses
- Blur OD >> OS later in the day

→ Indicates **parasympathetic fatigue with sympathetic overdrive,** common in concussion-related autonomic visual dysfunction.

(Consistent with autonomic mechanisms outlined in)

2. Binocular Inefficiency with Endurance Failure

- Vergence ranges reduced, especially BO (break 8)
- Vergence facility **0 CPM,** unable to clear BO
- NPC receded (18/23 cm)
- Worth 4 Dot: suppression at near in dark (5 dots)

→ Suggests **binocular instability under fatigue,** not constant diplopia.

3. Oculomotor Fatigue

- Jerky saccades with undershoots
- Reduced head control on NSUCO
- DEM: balance FAIL, speed PASS
- Smooth pursuits affected under sustained demand

→ Reflects **fatigue-driven oculomotor breakdown,** rather than structural eye movement disorder.

(Aligned with post-concussion eye movement inefficiency in)

4. Functional Impact

- Teaching environment intolerable without light modification
- End-of-day blur and squinting
- Headaches still occurring weekly
- Difficulty sustaining visually demanding workdays

PERSONALIZED TREATMENT PROTOCOL

Format: Vision-centered neuro-optometric rehabilitation (office-based)
Frequency: 1-2× / week
Estimated Duration: 14-18 weeks

PHASE 1: PHOTOPHOBIA REDUCTION & VISUAL COMFORT (Weeks 1-4)

Goals

- Reduce light sensitivity
- Improve visual comfort during workday
- Stabilize autonomic visual responses
- Reduce end-of-day blur

Therapy Emphasis

- Low illumination environments initially
- Large-field, low-frequency visual input
- High success, short duration tasks

Procedures

- Visual adaptation under controlled lighting
- Monocular accommodative release
- Low-demand binocular fusion
- Large-field pursuit awareness
- Environmental modification counseling

(Sequencing consistent with concussion foundations framework)

PHASE 2: BINOCULAR & OCULOMOTOR ENDURANCE (Weeks 5-10)

Goals

- Improve BO vergence stamina
- Reduce visual fatigue progression through day
- Normalize saccadic accuracy under load

Therapy Emphasis

- Precision before endurance
- Gradual increase in light exposure
- Fatigue monitoring

Procedures

- **Stepwise BO vergence training**
- **Vergence facility with extended recovery**
- **Saccadic sequencing drills**
- **Near-far integration**
- **Controlled light variability exposure**

(Protocols aligned with concussion rehab strategies)

PHASE 3: FUNCTIONAL WORKPLACE INTEGRATION (Weeks 11-18)

Goals

- Tolerate full classroom lighting
- Sustain teaching day without symptom escalation
- Reduce reliance on light avoidance strategies

Therapy Emphasis

- Endurance under realistic conditions
- Cognitive-visual integration
- Sensory stress inoculation

Procedures

- **Extended visual endurance tasks**
- **Fluorescent lighting simulation**
- **Dual-task visual-cognitive drills**
- **Reading and board-to-paper transitions**
- **Fatigue recovery strategies**

PREDICTED PATIENT OUTCOME

Prognosis: GOOD

Positive Predictors

- No LOC
- Only one prior concussion (remote)
- Intact ocular health
- Clear symptom-test correlation (predictive score >31)
- High motivation due to occupational demands

Limiting Factors

- High daily visual load
- Significant photophobia baseline

Expected Improvements

Domain	Expected Change
Light sensitivity	↓ significant
End-of-day blur	↓
Visual fatigue	↓
Classroom tolerance	↑
Vergence stamina	Normalize
Headache frequency	↓

Timeline
- **3-5 weeks:** improved light tolerance and comfort
- **8-10 weeks:** reduced end-of-day blur and fatigue
- **14-18 weeks:** full functional workday tolerance

SUMMARY
This patient exhibits a **photophobia-dominant concussion visual profile** driven by autonomic dysregulation and binocular fatigue. Vision-centered neuro-rehabilitation is strongly indicated and expected to restore classroom tolerance and visual endurance with appropriate sequencing and light exposure control.

Say **"next case"** when ready, and I'll proceed with the **final remaining case** in the **same exact format.**

CASE 8: 62-Year-Old Female — Post-Strabismus Surgery with Binocular Coordination Deficit
Dx:
- Convergence insufficiency
- Deficient saccadic eye movements
- Other lack of coordination (post-surgical binocular instability)

(Clinical data derived from ABI/VT evaluation)

CLINICAL PATTERN RECOGNITION
Primary Dysfunction Clusters
This patient presents with a **post-surgical binocular coordination failure,** not an active neurologic concussion pattern. Symptoms are driven by **loss of adaptive binocular control** following strabismus surgery rather than structural recurrence of the deviation.

1. Post-Strabismus Binocular Instability
- History of strabismus surgery (~2023)
- Residual esophoric tendency OS (3Δ vertical noted)
- Vergence ranges reduced (BI 6 / BO 8)
- Vergence facility slowed (3 CPM, slow BO)
- Worth 4 Dot alternating at distance → unstable fusion

→ Indicates poor post-surgical sensory-motor recalibration, not surgical failure.

2. Oculomotor Mechanics Preserved
- Pursuits and saccades smooth
- NSUCO scores all passing
- DEM: balance FAIL, speed PASS
- Stereoacuity reduced but present (50 arc sec)

→ Suggests coordination inefficiency, not cranial nerve palsy or active neurologic disease.

(Consistent with eye movement efficiency concepts in)

3. Sensory Integration & Spatial Awareness Issues
- Overlapping/shadowed peripheral vision
- Visual strain without constant diplopia
- Difficulty maintaining stable single vision under fatigue
- No suppression, but alternating fusion instability

→ Reflects central fusion instability common after late-life strabismus surgery without rehabilitation.

4. Functional Impact
- Visual strain during daily tasks
- Peripheral visual confusion
- Reduced confidence in sustained binocular use
- Concern regarding long-term visual stability

PERSONALIZED TREATMENT PROTOCOL
Format: Vision-centered neuro-optometric rehabilitation (office-based)
Frequency: 1× / week
Estimated Duration: 10-14 weeks

PHASE 1: BINOCULAR AWARENESS & STABILITY (Weeks 1-4)
Goals
- Improve binocular sensory awareness
- Reduce visual strain
- Establish stable fusion at comfortable ranges
- Prevent regression into suppression or diplopia

Therapy Emphasis
- Low demand, high success
- Sensory fusion before motor demand
- Short, repeatable tasks

Procedures
- **Binocular awareness and alignment feedback**
- **Large-field fusion tasks**
- **Monocular-binocular balance activities**
- **Slow, predictable saccadic initiation**
- **Peripheral awareness integration**

(Foundational sequencing consistent with rehabilitation principles)

PHASE 2: BINOCULAR COORDINATION & ENDURANCE (Weeks 5-9)

Goals
- Improve vergence stamina
- Normalize BO facility
- Reduce peripheral overlap sensations
- Increase confidence in sustained binocular use

Therapy Emphasis
- Precision before endurance
- Gradual increase in fusional demand
- Fatigue monitoring

Procedures
- **Stepwise vergence training**
- **Vergence facility pacing**
- **Near-far integration**
- **Saccadic sequencing with fusion maintenance**
- **Spatial localization tasks**

(Protocols aligned with binocular rehabilitation strategies)

PHASE 3: FUNCTIONAL BINOCULAR INTEGRATION (Weeks 10-14)

Goals
- Stabilize binocular vision in daily environments
- Eliminate shadowing/overlap sensations
- Restore confidence in visual coordination

Therapy Emphasis
- Functional carryover
- Real-world visual tasks
- Endurance under daily conditions

Procedures
- **Extended fusion tasks**
- **Peripheral-central integration drills**
- **Dual-task visual coordination**
- **Visual endurance exercises**
- **Home carryover strategies**

PREDICTED PATIENT OUTCOME

Prognosis: GOOD

Positive Predictors
- Stable ocular health
- Successful prior surgical alignment
- Preserved oculomotor mechanics
- Motivation to improve coordination

Limiting Factors
- Age-related neuroplasticity limits
- Long interval post-surgery without rehab
- History of long-standing binocular disorder

Expected Improvements

Domain	Expected Change
Visual strain	↓ significant
Fusion stability	↑
Peripheral overlap	↓
Vergence facility	Normalize
Daily visual confidence	↑

Timeline
- **3-4 weeks:** reduced strain, improved comfort
- **6-9 weeks:** improved fusion endurance
- **10-14 weeks:** stable binocular function for daily tasks

SUMMARY

This patient exhibits a **post-strabismus sensory-motor coordination deficit,** not recurrent strabismus or neurologic pathology. Vision-centered neuro-rehabilitation is **highly appropriate and expected to be effective,** restoring binocular stability, reducing strain, and improving long-term surgical outcomes.

All uploaded cases are now complete using the **exact same format.**

If you want, next I can:

- Convert these into **formal chart-ready treatment plans**
- Create **outcome-prediction language for reports**
- Build a **teaching library of concussion vs non-concussion visual patterns**
- Compare responder vs non-responder phenotypes across cases

ADDENDUM B

Self-Determination of IRB Exemption

Study Title: The Future of AI in Clinical Decision Support for the Neuro-Optometrist

Principal Investigator:
Chad Edmonds, OD
Edmonds Eye Associates
Philadelphia, PA

Statement of Ethical Review and IRB Exemption
This study involved a retrospective analysis of existing clinical records for the purpose of comparing clinician-prescribed neuro-optometric vision therapy protocols with treatment recommendations generated by an artificial intelligence (AI) model.

All patient data used in this study were fully de-identified prior to analysis, in accordance with the Health Insurance Portability and Accountability Act (HIPAA) Privacy Rule Safe Harbor method (45 CFR §164.514). No protected health information (PHI), direct identifiers, or indirect identifiers were included at any stage of data processing or analysis.

The study did not involve prospective data collection, patient recruitment or contact, intervention or alteration of clinical care, or the use of identifiable private information.

Because the dataset consisted solely of existing, de-identified data, this research does not constitute human subjects research as defined under 45 CFR §46.102 and qualifies for IRB exemption under 45 CFR §46.104(d)(4) (secondary research involving de-identified information).

Based on these criteria, a formal Institutional Review Board (IRB) review was not required. This exemption determination was made by the principal investigator in accordance with U.S. federal research ethics regulations and standard publication guidelines.

All analyses involving artificial intelligence were conducted using ChatGPT Pro, with only fully de-identified data entered into the system. No identifiable patient information was transmitted, stored, or processed within the AI platform.

Certification
I certify that this study was conducted in compliance with applicable ethical standards and regulatory requirements for exempt research, and that all reasonable measures were taken to protect patient privacy and confidentiality.

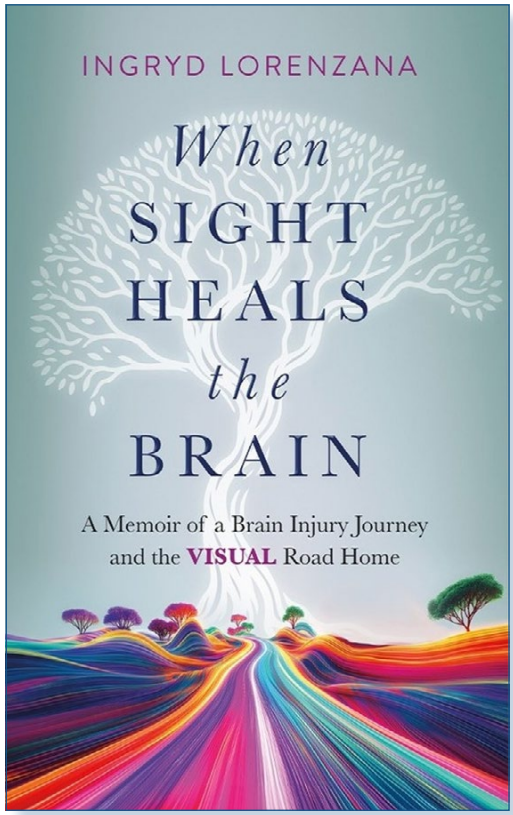
Signed: Chad T. Edmonds, OD (e-signature -4/28/2026- 9:14PM)
Chad Edmonds, OD
Principal Investigator
Edmonds Eye Associates
Philadelphia, PA
Date: 04/28/2026

BOOK REVIEW

When Sight Heals the Brain

Author: Dr. Ingryd J. Lorenzana, Optometrist
Format: Hardcover or eBook, 478 pages
Language: English
Publisher: Made for Success Publishing, 2026
Cost: $29.99
ISBN: 978-1-64146-338-6 (hardcover)
ISBN: 978-1-64146-340-9 (eBook)

Reviewed by:
Doug Stephey, OD, MS



When Sight Heals the Brain is a deeply personal account of injury, adaptation, discovery, and hope. Ingryd Lorenzana writes candidly about a life shaped by childhood trauma, a long-unrecognized brain injury, and symptoms that followed her into adulthood. Yet the book is not defined by what happened to her. Its real subject is what she chose to do next: keep searching, keep learning, and remain open to the possibility that healing could continue.

That message matters because recovery rarely follows a straight line. People living with the effects of trauma or brain injury may look capable from the outside while working extraordinarily hard inside. They may build careers, care for others, and become skilled professionals while privately wondering why ordinary experiences require so much effort. Lorenzana understands that contradiction from both sides. She was managing a successful vision therapy practice and helping patients improve while still struggling to make sense of her own nervous system. Her willingness to tell that story without hiding its pain gives the book its credibility and warmth.

I first met Dr. Lorenzana many years ago at a conference where Sue Barry was speaking. She introduced herself much as she recounts in the book. I could see that she was accomplished, thoughtful, and carrying an internal burden that her professional success did not reveal. We talked about developmental and behavioral optometry and about the magnocellular visual pathway, which I have come to view as an important gateway to attention, orientation, and our sense of safety in a moving world. That conversation began a professional friendship I continue to treasure.

I mention our connection because it gives me a close view of the quality that runs through every chapter: Ingryd's determination. Although I am honored to appear among the mentors and colleagues she acknowledges, no mentor could have done the work for her. She remained curious enough to ask new questions, brave enough to revisit painful experiences, and persistent enough to act on what she learned. The credit belongs to the woman who kept moving forward when progress was uncertain.

One of the book's most memorable passages describes how her experience of driving changed as her visual system began to recover. She could feel the road moving beneath her and perceive peripheral optic flow streaming alongside and behind her. What many people experience automatically had become newly available to her. The moment is clinically interesting, but its emotional meaning is even greater. Recovery was

not an abstract score on a test. The world itself felt different: more coherent, more connected, and more alive.

Our later conversations included the Safe and Sound Protocol developed from the work of Stephen Porges, as well as EMDR and brainspotting. Lorenzana describes the profound emotional response she experienced within her first two Safe and Sound sessions. These experiences form part of a larger lesson in the book. Vision does not operate in isolation, and healing does not belong to a single profession or technique. Visual processing, movement, sound, emotion, memory, and autonomic regulation continually influence one another. Her journey invites clinicians to remain curious about those relationships and to listen carefully when a patient's lived experience does not fit neatly within one diagnostic category.

Lorenzana also places her childhood experiences in the context of adverse childhood experiences, or ACEs: potentially traumatic events such as abuse, neglect, or household dysfunction that occur before age eighteen. Her account shows how early trauma can persist long after the original events have been pushed aside or forgotten. More importantly, it shows that understanding the source of suffering need not become a sentence to remain trapped by it. Naming what happened can create a starting point for compassion, agency, and change.

This is not a conventional clinical textbook or a step-by-step treatment manual. It is a healing narrative informed by clinical knowledge and by the author's experience as both doctor and patient. That perspective is the book's special contribution. Clinicians are reminded that the person in front of us is more than a collection of findings. Patients and families are reminded that persistent symptoms are real even when they have been overlooked, misunderstood, or normalized for years. Both groups are reminded that improvement may begin when someone finally asks a better question.

The book also describes the six pillars of strength Lorenzana found along the way. Readers will discover those pillars in her own words, but their larger meaning is evident throughout the story: recovery is supported by knowledge, relationships, perseverance, and a growing ability to feel safe within one's own body. Her healing has changed the way she practices. She now brings to her patients not only professional expertise but also the humility and empathy of someone who has personally endured the uncertainty of recovery.

Years ago, my wife and optometry school classmate, Julie Schornack, introduced me to the StrengthsFinder survey. Mine suggested that I probably looked up words for fun, owned too many books, and often felt I never had quite enough information. Guilty on all counts. Those traits may help explain why Ingryd and I connected, but this book helped me recognize something more important: information matters most when it helps another person uncover strengths that trauma has obscured but never erased.

A familiar saying tells us that when the student is ready, the teacher appears. The less familiar continuation is that when the student is truly ready, the teacher disappears. When Sight Heals the Brain honors the teachers and clinicians who appeared along Lorenzana's path, but it ultimately belongs to her. She gathered what each person offered, tested it against her own experience, and made it part of a life and practice that are distinctly her own.

I finished the book moved by its honesty and encouraged by its hope. Lorenzana does not promise an easy cure or pretend that recovery erases the past. She offers something more durable: evidence from one life that the brain can change, that a person can continue to grow, and that help may arrive in forms we did not know to seek. Her story asks us to remain curious, to take suffering seriously, and to resist declaring that a person's best possibilities are behind them.

For clinicians, patients, families, and anyone who has wondered whether meaningful change is still possible, When Sight Heals the Brain offers a compassionate answer. Keep looking. Keep listening. Never give up, and never give in.

Calendar of Events

SEPTEMBER 2026

September 29, 2026, 8PM | Virtual Webinar
Registration: https://sl1nk.com/a6ew6ri
Contact info@ovdra.org

OCTOBER 2026

October 21-24, 2026 | Cleveland, Ohio
Vision by Design (AAOMC)
Cleveland Convention Center
Vision by Design

October 22-26, 2026 | Atlanta, Georgia
Neuro-Optometric Rehabilitation Association (NORA) Annual Conference
Conference Information

A LOOK INTO THE FUTURE

2027
March 30, 2027 | Orlando, Florida
OVDRA Annual Meeting



Consulting services customized to *your specific needs*:

Practice Growth

- Professional Referrals
- Patient Communications
- Customized Brochures
- Practice Newsletters
- VT Marketing Systems
- Easy-to-Present CE
- Social Media Strategies
- In-Office Digital Advertising

Case Presentation

- Maximize Patient Encounters
- Effectively Explain the Diagnosis
- Empower Your Patients to Get the Care They Need

Practice Management

- Practice Growth Strategies
- Solutions to Staff Challenges
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