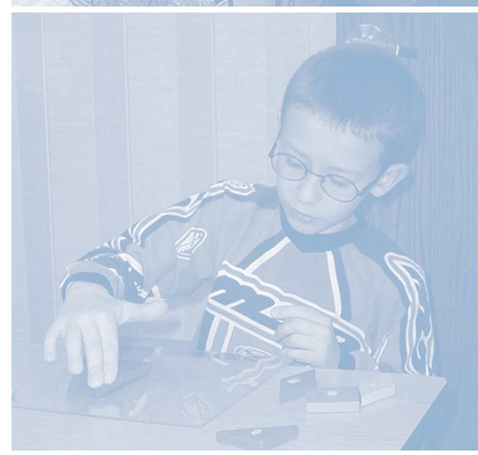
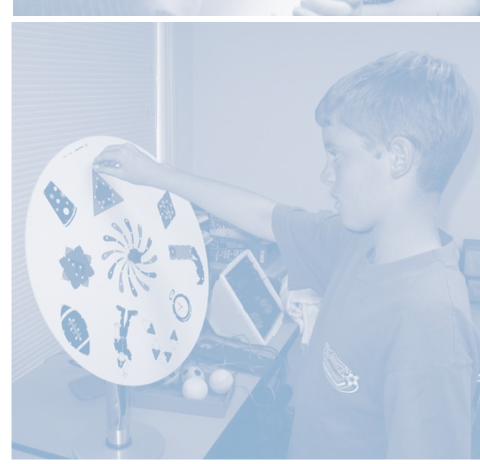
Vision & Development Rehabilitation

College of Optometrists in Vision Development
Prevention • Enhancement • Rehabilitation

ISSN 2374-6416 • Volume 5, Issue 4 • Journal of the College of Optometrists in Vision Development

TABLE OF CONTENTS





Vision Development & Rehabilitation

ISSN 2374-6416 • Volume 5, Issue 4

Journal of the College of Optometrists in Vision Development

Vision Development & Rehabilitation

ISSN 2374-6416 • Volume 5, Issue 4

Journal of the
College of Optometrists in Vision Development

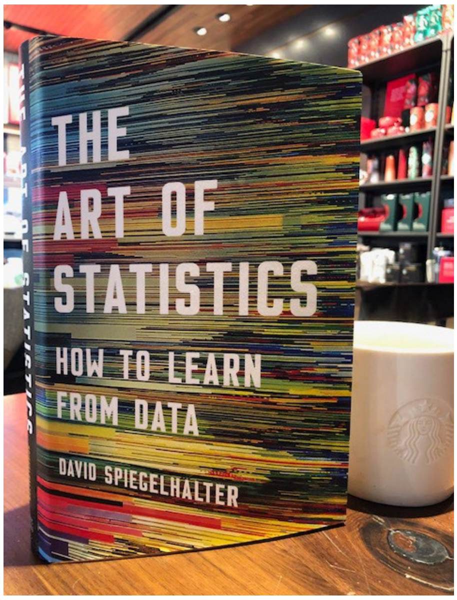
SUBMISSION OF MANUSCRIPTS

The entire manuscript submission and review process is conducted through Editorial Manager. All manuscripts are submitted at www.editorialmanager.com/vdr. A copy of Guidelines for Authors is available on the home page of the VDR Editorial Manager site or on the COVD website http://www.covd.org/?page=VDR. Editorial Manager will require you, as an Author and/or Reviewer, to create an account the first time you access the site. If you have questions with the site or the process please contact Managing Editor, Katie Kirschner at Katie@covd.org.

If access to Editorial Manager is not available please e-mail the Editor with your request for the Guidelines and submit your manuscripts to Editor@covd.org.

Learning from Data

Leonard J. Press, OD, FAAO, FCOVD, *Editor-in-Chief*



In the introduction to his new book The Art of Statistics: How to Learn From Data,[1] British statistician David Spiegelhalter quotes fellow statistician Nate Silver as saying: "The numbers have no way of speaking for themselves. We speak for them. We imbue them with meaning."

Later on, in a chapter titled "Learning from Experience the Bayesian Way", Spiegelhalter reinforces this notion: "Bayes' legacy is the fundamental insight that the data does not speak for itself – our external knowledge, and even our judgment, has a central role." A discussion about who Thomas Bayes was, and how his thinking factors into current research in health care is beyond our limitations of space here, but there is a concise primer on Bayesian inference and probability that you can access through Brown University.[2]

Caveats abound about inferences drawn from data, particularly with regard to studies that fail to find anticipated relationships. When you stop and think about it, this isn't entirely surprising. The enterprise of research is built around the deductive technique of seeking to falsify hypotheses. Citing the philosopher of science Karl Popper, A.C. Grayling notes: "We test the conjecture; a negative outcome refutes it, a positive outcome 'corroborates' but does not confirm it; it might still be refuted by further evidence."[3]

Writing in the October issue of Scientific American regarding the interpretation of statistical significance, contributing editor Lydia Denworth observes: "The same cannot be said for biomedical research, where the risk tends toward false negatives, with researchers reporting no statistical significance when effects exist. The absence of evidence is not the evidence of absence, just as the absence of a wedding ring on someone's hand is not proof that the person isn't married, only proof that the person isn't wearing a ring."[4]

One of Spiegelhalter's main themes is the manner in which the results of research are interpreted and communicated. I raise this issue because of the recent headline of a news release from the National Institutes of Health (NIH) on the Convergence Insufficiency Treatment Trial – Attention and Reading Trial-ART (CITT-ART) study.[5] The headline reads: "Treatment for common vision disorder does not improve children's reading skills". Looking at the data from the actual study, however, reveals that the subtitle of the release is a more accurate portrayal: "NIH-funded study finds therapy for convergence insufficiency is no better at improving reading than placebo."

Elsewhere I've blogged about the significance of this issue,[6,7] and it acquires added meaning because the design and methodology of the CITT-ART was originally described in the pages of this journal.[8] The aim of the study, as stated in that article, was that its results would contribute to a better understanding of the relationship between CI and reading and attention. Parents, eye care professionals, educators, and other health care providers would be better able to make informed decisions in caring for children with reading and attention problems.

The "X-Factor" in the study design may be the nature of the activities done by the children in the placebo group. It appears that these procedures involved more attention and processing than done by the children who were involved in the CI training group. While this is understandable since the criteria for the placebo group was that their activities did not elicit direct change in accommodation and vergence, it seems in retrospect that the placebo group experienced reading gains because their attention and processing activities enhanced reading readiness. The reason why so many parents report gains in their children's reading and academic behaviors in clinical practice may be due to the skill of the doctor or therapist in incorporating attention and processing factors into optometric vision therapy programs, whether or not the child has been diagnosed with convergence insufficiency.

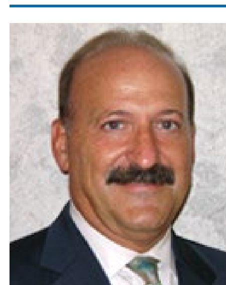
In hindsight, one might argue that a placebo arm in a study of this nature could be designed to more closely mirror what occurs in drug treatment trials. For example, the placebo group would receive various treatments consisting of ocular lubricants. These lubricants have no direct effect on accommodation and vergence, nor do they directly impact attention or processing skills. However, they might impact reading performance by improving visual comfort and visual resolution when the child is engaged in sustained reading. An elaborate protocol could be designed to have the child engage in their normal reading and homework activities, but with various forms of artificial tear supplementation, chemical composition, dosages, dosing schedules, and objective dry eye analyses.

A placebo group of this nature is not far-fetched. A number of items in the Convergence Insufficiency Symptom Survey (CISS) relate to visual comfort and the appearance of print. And in practice, there are children with convergence insufficiency whose symptoms are presumptively attributed to ocular surface disease. When these children present with chief concern regarding reading, there are practitioners who suggest that ocular lubricants be used as a primary treatment. Some will acknowledge that they expect the child to respond positively based on a placebo effect. It would therefore be reasonable to randomly assign patients to a CI treatment control group, or the type of placebo group utilized in practice.

In any event, buildup to the CITT-ART study has placed a spotlight on its data and its interpretation. No doubt the study chair, Dr. Mitchell Scheiman, will address these complexities in his continuing education presentation on the subject at the upcoming COVD annual meeting in Toronto.

REFERENCES

1. Spiegelhalter D. The Art of Statistics: How to Learn From Data. New York; Basic Books, 2019. http://bit.ly/2rVjFJ8
2. Seeing Theory: A Visual Introduction to Probability and Statistics. Brown University. Accessed November 21, 2019. http://bit.ly/2OPG6Zb
3. Grayling A.C. The History of Philosophy. New York: Penguin Random House, 2019. http://bit.ly/364uyXH
4. Denworth L. The significant problem of P values. Scientific American 2019; volume 321, issue 4. Accessed November 21, 2019. http://bit.ly/2LoflsA
5. National Institutes of Health. News Release. Accessed November 21, 2019. http://bit.ly/2P9NzRG
6. The VisionHelp Blog – Part 1. Accessed November 21, 2019. http://bit.ly/36alpNz
7. The VisionHelp Blog – Part 2. Accessed November 21, 2019. http://bit.ly/33Pdt2I
8. Scheiman M, Mitchell GL, Cotter SA, et al. Convergence Insufficiency Treatment Trial – Attention and Reading Trial (CITT-ART): Design and Methods. Vis Devel Rehab 2015;1(3):214-28. http://bit.ly/34X01eo



AUTHOR BIOGRAPHY:
Leonard J. Press, OD, FAAO, FCOVD
Lakewood, New Jersey

- OD 1977, Pennsylvania College of Optometry
- Principal Owner, Press Consulting, P.C.
- Adjunct Faculty, Southern College of Optometry
- Editor-in-Chief, *Vision Development & Rehabilitation*
- Past President, COVD

COVD 20/20 50th

ANNUAL MEETING TORONTO

APRIL 21-25, 2020

College of Optometrists in Vision Development

www.covd2020.org info@covd.org

BETA SIGMA KAPPA - COVD RESEARCH GRANT

MAUREEN PLAUMANN, OD, FAAO
The Ohio State University

Congratulations to Dr. Maureen Plaumann of The Ohio State University for being selected as the recipient of the 2019 BSK - COVD Research Grant.

Dr. Plaumann will receive a $4,000 grant for her project, *"Quantifying Suppression in Adults with Strabismus and Amblyopia"* and will be recognized during the 2020 COVD Annual Meeting.

Learn more about the BSK - COVD Research Grant online at covd.org/page/bsk_research_grant.

Biblia·Sunergoi·Koinonia

College of Optometrists in Vision Development
Prevention • Enhancement • Rehabilitation

You improve vision.
We improve your practice.

Improve efficiency office-wide with Nu Squared Vision Therapy EMR.

www.NuSquared.com

Come visit us in the COVD Annual Meeting exhibit hall!
www.NuSquared.com

EYEPORT® II Vision Training System

Clinically Proven, FDA Cleared, Doctor Recommended.

JLEP **$199.95** | 3+ **$184.95**

People around the world are doing daily eye exercises with the original EYEPORT®. Developed in the early 2000's, the EYEPORT® was the first FDA approved device for Vision Training. Unfortunately, the product was taken off the market in 2012 due to financial issues with the original parent company. After receiving a large number of requests from our customers, Bernell purchased the rights to EYEPORT® and have revived this great Vision Therapy training tool! The key training features and programs of the original model have not changed, however a new design plus added features have been incorporated into the new EYEPORT® II model from Bernell. Additionally, this device is now proudly made in the USA!

The EYEPORT® Home Vision Training System revolutionized the way people look at their vision. To read more comfortably, learn more easily, work less painfully, and play sports more effortlessly. The way the training system works is really quite simple. Your eyes' focusing muscles flex and relax when they follow the EYEPORT® II programmed series of alternating red and blue lights. Because the lights turn on in different directions, patterns and speeds, your eyes exercise through their full range of motion--horizontally, vertically, diagonally, near, and far. And you don't even work up a sweat. Since you often focus at close range while reading or working at a computer, your focusing system becomes stiff and cramped causing computer eye strain.

What makes the EYEPORT® II unique is its patented use of alternating red and blue lights. This makes it possible to achieve dramatic results, faster and more efficiently than with any other vision exercise product available today. Basically, your eyes react differently to red and blue lights due to a scientific phenomenon called chromatic aberration. The focusing system contracts when it sees red and relaxes looking at blue. By combining eye exercises with specific colored lights, the EYEPORT® II strengthens your vision skills in a revolutionary new way never previously done. And it's easy.

EYEPORT® II Features:

- Portable and easy to use
- Automatic shut-off
- Pre-programmed workouts
- Multiple speed settings
- Special glasses to train each eye individually
- Carrying case

After 3+ years of being off the market, Bernell has revived this revolutionary, FDA Approved VT training tool!

BERNELL
A Division of Vision Training Products, Inc.

Phone: 800-348-2225
E-mail: info@bernell.com
Online: www.bernell.com

Contact us today to order the EYEPORT® II for your practice.
Recommended resale price to patients is $269.95

PERSPECTIVE

Nutraceuticals for Visual Performance

Graham B. Erickson, OD, FAAO, FCOVD
Pacific University College of Optometry
Forest Grove, Oregon

ABSTRACT

Eye care professionals have several options to help athletes see their sport better. Recent placebo-controlled research has demonstrated that the carotenoids lutein (L) and zeaxanthin (Z) can improve visual performance factors in healthy eyes. The history, mechanisms of action, as well as their effects and potential benefits as shown in well-controlled trials are reviewed. A detailed description of how potential improvements in visual performance may apply to batting in baseball is provided. Recommendations for athletes with regard to modifications of diet to increase intake of carotenoids, or supplementation with purified forms of L and Z are discussed.

Correspondence regarding this article should be emailed to Graham B. Erickson, OD, FAAO, FCOVD, at graham@pacificu.edu. *https://doi.org/10.31707/VDR2019.5.4.p221.*

Erickson GB. Nutraceuticals for visual performance. Vision Dev & Rehab 2019;5(4):221-34.

Keywords: carotenoids, contrast sensitivity, glare disability, neural efficiency, photostress recovery, reaction time, sports, visual performance, visual processing speed

INTRODUCTION

Competitive athletes are continuously searching for ways to elevate their performance in their sport. The logical focus for performance improvements are methods to enhance physical factors such as strength, endurance and recovery. In addition to the strength and conditioning training regimens, biomechanical factors, rest and recovery programs, and psychological preparation, advances in nutritional support have produced dietary approaches to optimize physical performance. While these are certainly important considerations, options to improve visual performance can be a neglected element in the preparation for competition.

Table 1. Options to enhance visual performance

Refractive compensation for refractive errors • Methods for refractive compensation • Sports protective eyewear
Filters to enhance visibility of important features
Visual performance training
Nutrient intake of carotenoids that enhance visual performance

Eye care professionals have several options to help competitive athletes enhance visual performance (see Table 1). Visual performance is a broad term that can include basic vision factors as well as a more diverse array of visual information processing and visual motor response aspects that guide skilled motor performance. The purpose of this review is to highlight recent studies demonstrating the potential effects of nutrition on visual performance.

Visual Performance Overview

It is helpful to classify the areas of cognitive processing in order to better understand how these factors can affect performance. The modified Welford Processing Model[1] is useful for understanding how the critical sporting action output results from the successful execution of lower-level processes (see Fig. 1). Using this model, Table 2 provides an example

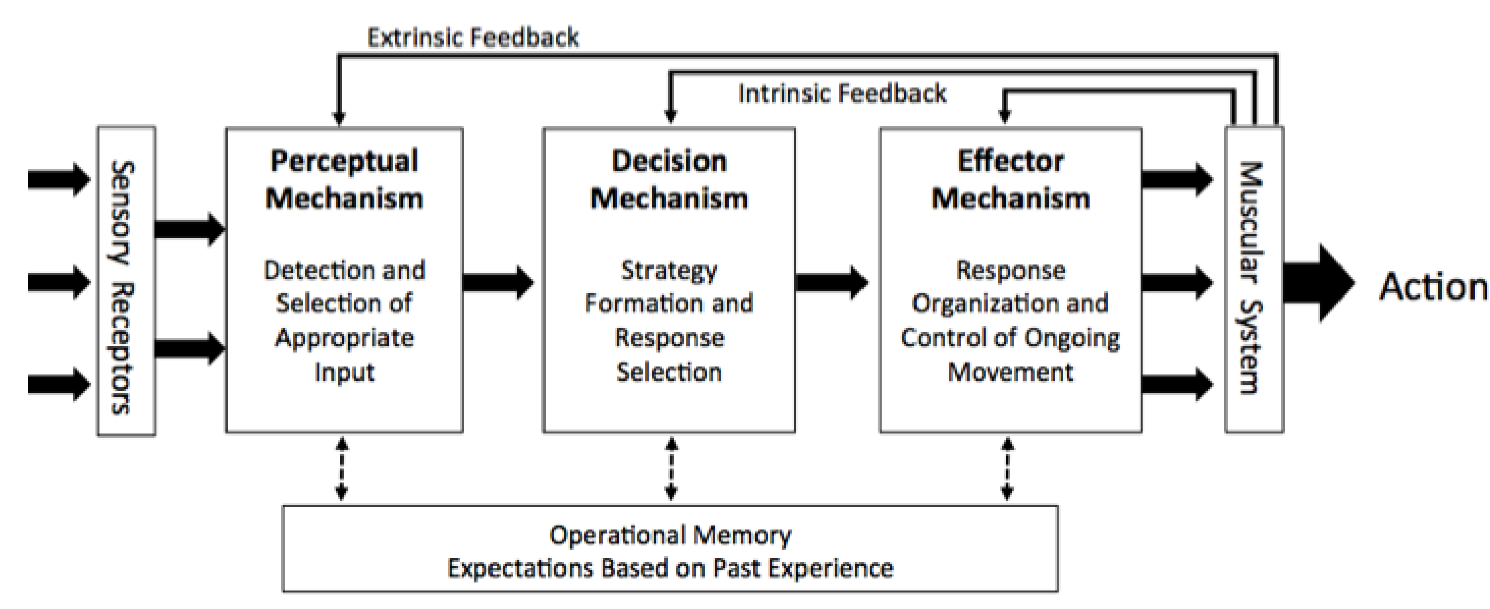


Figure 1: A modified information processing model of skilled performance first proposed by Welford.[1]

Table 2. Example of classifying assessment elements into relevant cognitive mechanisms from the modified Welford Processing Model

Perceptual Mechanism • Visual Acuity • Contrast Sensitivity • Dynamic Visual Acuity • Ocular Alignment • Stereopsis • Accommodative Function • Vergence Function • Oculomotor Function • Peripheral Vision
Decision Mechanism • Speed/Span of Recognition • Visual Attention/Visualization • Multiple Object Tracking
Effector Mechanism • Visual Motor Reaction Speed (Eye-Hand, Eye-Foot) • Vision & Balance • Peripheral Vision Response Speed • Coincidence-Anticipation

of classifying assessment elements into relevant cognitive mechanisms.

Development of visual and cognitive expertise is dependent on the demands of the sports experience of each athlete, since these demands can be quite variable across the vast array of sports. For example, stereopsis is an important vision factor for performance in many sports, but may have limited relevance for shooting sports where the athlete sights monocularly. Furthermore, stereopsis is commonly assessed at a near distance, but the stereopsis demands in most sporting situations are at relatively far distances. In addition, the testing of dynamic stereopsis may yield differential performance and discriminate sports-related visual abilities better than traditional static measures. These examples highlight the importance of a careful visual task analysis of the sport demands, and need for potential modification of the assessment to better simulate the visual demands important for specific sports applications.

The fundamental vision factors that seem to provide a foundation for most visual performance skills are visual acuity and contrast sensitivity. These factors are responsible for the visibility of important features that athletes must discern to perform optimally.[2] Additional influential vision factors can also degrade visibility, such as glare disability, discomfort and photostress recovery.[3] Compromised visual acuity and contrast sensitivity can negatively affect other areas of visual performance, and most options for improving visual performance directly or indirectly target these vision elements. In many sports situations, especially team sports, processing of information from the peripheral visual fields is also a beneficial element to successful sports performance, however this review will focus on the elements of central vision.

General Role of Carotenoids and Other Nutrients

Dietary nutrients accumulate in the tissues of the eye and visual pathway in a manner similar to other tissues in the body. There are several nutrients that are beneficial for eye health and function, including vitamins A, C, and E, omega-3 fatty acids, beta carotene, zinc, selenium, lutein and zeaxanthin. Vitamins A, C, and E, zinc, selenium and lutein provide protective effects for age-related changes to the crystalline lens and retina (eg, cataracts & AMD).[4-7] Nutrients containing omega-3 fatty acids can reduce dry eye effects,[8-14] and other nutrients protect the retina from sun damage (lutein, zeaxanthin and beta carotene).[4-7,15] These are naturally occurring nutrients that can be found in the foods listed in Table 3.

Table 3. Naturally occurring food sources of nutrients beneficial to eye health and function

- Green, leafy vegetables such as spinach, kale, collards
- Salmon, tuna, and other oily (cold-water) fish
- Eggs, nuts, beans, and other protein sources
- Oranges and other citrus fruits
- Colorful fruits and vegetables (bell peppers, berries)
- Whole grains such as quinoa, brown rice, whole oats
- Fish oil, flaxseed oil, black currant seed oil

There has been significant research into the effects of nutrients on visual performance in young, healthy people. Lutein (L) and zeaxanthin (Z) are plant-derived carotenoids that are found to be concentrated in the eye and brain, and were first noted in the macular pigment in 1985.[16] L and Z are the only two naturally occurring carotenoids found in the macula and concentrated within the inner layers of the primate fovea, specifically at Henle's fiber layer, and act as a filter for light.[15,17,18]

The dietary carotenoids L and Z have been shown to improve visual performance factors that may benefit athletes. There is a growing body of research that has shown empirically that increasing the intake of these carotenoids increases macular pigment density, which can improve contrast sensitivity, glare disability and discomfort, and photostress recovery. Since dietary intake of important nutrients is a modifiable factor, there is compelling evidence that increasing the intake of zeaxanthin and lutein may provide important improvements to visual function.

The challenge of increasing dietary intake is identifying the sources and quantity of L and Z, and determining whether it is reasonable to increase intake through dietary modification alone. Accurate assessment of carotenoid intake is difficult to measure, and can vary based on traditional diets. A systematic review of the data from the National Health and Nutrition Examination Survey (NHANES) 2003-2004 suggested that the average American diet consists of a 5:1 ratio of lutein to zeaxanthin, and the average American gets 1mg of zeaxanthin per day.[19] Lutein and zeaxanthin accumulate at a 1:2 ratio (L to Z) in the central part of the macula, suggesting that it is challenging to get enough zeaxanthin in a typical north American diet. All age groups, both sexes and all ethnicities show a greater intake of L than Z, and the relative intake ratio increases with age.[19] These findings suggest that supplementation with purified L and Z may provide performance benefits to those athletes who have difficulty ingesting enough of these nutrients through a regular diet.

Nutrition Effects on Visual Performance

The macular pigments have peak absorbance for short-wavelength light (400-500 nm), and filters light before it reaches the cone photoreceptors. The peak energy of both blue haze and sky light is 460 nm, which coincides with the peak absorbance of the macular pigments (similar to yellow-tinted filters).[20] It has long been noted that there is a ubiquity of intraocular yellow filters in most vertebrates, rather than filters targeting other wavelengths, and that these filters therefore must provide similar visual performance functions.[21-23] The visual performance effects of yellow filters have long been recognized as improving visual acuity by the reduction of chromatic aberration effects, enhancement of detail by the absorption of "blue haze," enhancement of contrast, and reduction of glare discomfort.[21-24]

Visibility

There are many internal and external sources of image degradation beyond refractive error. Wooten and Hammond proposed that the preponderance of short wavelength light in the atmosphere results in a bluish veiling luminance that degrades visibility.[25] Modeling suggests that macular pigment may improve vision through the atmosphere by preferentially absorbing the short wavelength energy produced by blue haze, which can enhance the figure-ground contrast of objects in the environment.[25] The difference between low and high levels of macular pigment can increase visibility by 30-40%.[25,26] This suggests that athletes with higher levels of macular pigment density can see details about 30% farther away than an athlete with low levels, which may afford a distinct advantage for successful performance in some sport situations.

The process of filtering some of the visible spectrum through the attenuation of the transmittance of the shorter wavelength colors (blue) decreases the chromatic aberration between the longer red wavelengths and the transmitted mid-range greens. The reduction in chromatic aberration leads to improved image clarity, and the selective transmission of yellow wavelength light concentrates the visible information at the most sensitive portion of the visible light spectrum. Therefore yellow-colored tints filter the glare produced by the short wavelength light while transmitting peak visible light information with reduced chromatic aberration. Studies have not been always been successful in quantifying the improvement in visual acuity and contrast sensitivity reported with yellow tints.[27] Yellow tints have been shown to improve depth perception, contour recognition, and reaction times.[28-40] Of note, many of these studies were conducted under artificial lighting conditions that are far below the intensity (in candelas per square meter) of natural sunlight, and this factor may obfuscate the enhancement effects.

Enhancement of visibility in outdoor daylight conditions would offer potential performance advances in many sports. Many athletes appreciate the improvement in contrast with yellow tints, especially in low light conditions such as twilight (dawn and dusk), fog, and heavy cloud cover. Yellow tints are popular with shooting and snow sports, and perform well in fog and low light conditions.[41] Mountaineers use yellow tints in whiteout conditions to enhance the contrast of environmental features, and these filters have been used for driving, boating or flying in low light conditions. A yellow tint may be helpful in sports where an object must be located and/or tracked against the background of a blue or overcast sky, such as tennis, baseball and soccer. These indications for external yellow tint use highlight the potential benefits of increasing the internal filter effects from higher levels of macular pigment density. Further, macular pigment does not degrade visibility in low light conditions, where external yellow tints may not perform as well in dim illumination.

Contrast Sensitivity

Contrast sensitivity measures the visual system's ability to process spatial or temporal information about objects and their backgrounds under varying lighting conditions. It has long been recognized that contrast sensitivity is an important visual factor, especially the application to edge detection in the natural environment. The retina uses lateral inhibition to accentuate edges and sends that enhanced signal to the visual cortex. Edge detection relies on luminance differences and chromatic contrast to detect and define edges.

Yellow tints, as described above, have long been known to play a role in edge detection for objects seen against a blue background, such as the sky.[42,43]

Macular pigment acts similar to a yellow tint, and appears to play a significant role in lateral inhibition and chromatic contrast. It has been shown that contrast enhancement is linearly related to macular pigment density in humans when viewing chromatic borders through simple filtration, especially against short-wavelength backgrounds.[44] Double-blind, placebo-controlled studies have shown that daily supplementation of L and Z produces statistically significant improvements in the lateral inhibitory process, chromatic contrast, and contrast sensitivity near the peak of human sensitivity (6 cycles per degree).[45-47] Of particular interest is that these improvements were found in young, healthy subjects.[45,47]

Measurement of contrast sensitivity has been recommended in athletes because many sports involve visual discrimination tasks in suboptimal lighting because of environmental variability.[48-50] Snellen-type visual acuity measurements may not be sensitive to the subtle visual discrimination tasks inherent in many sports because the acuity task is usually performed only under high-contrast conditions. Improvements in chromatic contrast offers potentially enhanced edge detection of important visual information in sports, and may also assist in seeing the rotation of fast moving objects, such as a ball. Performance of athletes on contrast sensitivity testing is significantly better than non-athletes across all spatial frequencies evaluated.[49,51,52] It is speculated that reduced contrast sensitivity may contribute to performance inconsistency due to the variable lighting conditions and figure-ground characteristics found in many sports.

A study of elderly drivers with mild to moderate macular degeneration found that supplementation with lutein and zeaxanthin produced macular re-pigmentation that improved contrast sensitivity, which translated to improvements in self-described driving ability.[53] Macular pigment also appears to enhance visual function in low light conditions in young, healthy subjects, and provides better dark adaptation efficiency.[54] These studies demonstrate that contrast sensitivity improvements from increased macular pigment density can translate to better visual performance in many situations in daily life as well as sports.

Glare Disability and Discomfort

When intense light enters the eyes, it can cause discomfort (photophobia) and disability glare due to photopigment bleaching. Photophobia is a subjective sensation of discomfort when exposed to intense light, and can interfere with performance in many activities. Several studies provide evidence that macular pigment density can reduce this type of visual discomfort, especially for light in the shorter wavelengths.[55-59] In conditions of glare, the intense light is scattered by the ocular media and can result in loss of image contrast and reduce visibility. Studies have demonstrated that higher levels of macular pigment density allow subjects to see a target under more intense light conditions than those with lower levels.[3,45,60] A study of young, healthy subjects also showed that supplementation with zeaxanthin and lutein produced a reduction in disability glare thresholds corresponding to increases in macular pigment density.[58] These studies suggest a filtering mechanism for macular pigment that can allow an average of 23% greater intensity of the glare source before losing sight of the target.

Visible light is responsible for glare that can cause significant interference with an athlete's ability to see the visual details critical for successful performance. For example, direct glare from the sun is evident in a blue sky because it affects the visibility of a lofted ball. For those athletes competing under artificial lighting for night games or indoors, the high intensity light sources used for these purposes can also cause a significant amount of direct glare when an athlete must look toward the light source to track a ball or other object. Reflected glare is exceptionally troubling for athletes when the sun is reflected off surfaces such as water, snow, pavement, and sand. These surfaces reflect horizontally polarized light that can produce substantial glare, particularly water surfaces that are constantly moving. Glare sensitivity and slow glare recovery may contribute to errors in certain sport conditions. Athletes may benefit from higher macular pigment density by having less glare discomfort and better visibility under glare conditions.

Photostress Recovery

Intense light or glare can also result in bleaching of the photoreceptors. On a bright sunny day, illuminance ranges from 1000 to 10,000 foot-lamberts, saturating the retina and reducing finer levels of contrast sensitivity.[61] Dark sunglasses aid in recovery of contrast sensitivity and dark adaptation after photoreceptor saturation.[27] Macular pigment density has also been shown to reduce photostress recovery time by absorbing intense light before it reaches the photoreceptors; recovery time is faster for those with higher macular pigment density.[3,58,60] Further, supplementation with L and Z has been shown to significantly improve photostress recovery,[3,45] with an average improvement of almost 9 seconds measured from 1 year of supplementation in a double-blind, placebo-controlled study.[45] Macular pigment density may also enhance visual function in low light levels by enhancing dark adaptation kinetics, thereby improving the efficiency of photopigment regeneration.[54]

There are many sports situations that involve intense glare, and requires the athlete to recover visual function quickly from photopigment saturation. Slow visual recovery after looking toward the sun or arena lighting, or reflected from surfaces, can result in performance errors. These errors may put the athlete at risk of injury, such as in cycling sports or downhill skiing. Some sports situations require the athlete to move quickly between areas of bright sunlight and shadow, and quicker visual recovery offers a potential performance advantage. Higher levels of macular pigment may improve a driver's ability to see when moving into and out of tunnels, when driving toward the sun when it is low in the sky, or recovering from the glare of oncoming headlights when driving at night.[53]

Nutrition Effects on Neural Performance

While the presence of L and Z in the macula is well recognized, these carotenoids also concentrate in the brain. Randomized, double-masked, placebo-controlled trials of young healthy subjects have shown that macular pigment density is linked to L and Z levels in the brain,[62-64] and the level present is related to functions such as cognition, reaction time, and temporal visual processing.[65-73] Neuroimaging to measure the relation of L and Z to brain structure in vivo has confirmed that L and Z influences white matter integrity, particularly in regions vulnerable to age-related decline.[74] L and Z are incorporated in cell membranes and axonal projections, which serve to enhance interneuronal and neural-glial communication.[75] Recently, supplementation with carotenoids has been shown to increase critical flicker frequency thresholds, visual motor reaction time, and temporal contrast sensitivity function compared to a placebo control group in young, healthy subjects, improving processing speed by an average of 10% to 20%.[71,72] Specifically, studies show a highly significant change in CFF thresholds for the supplementation group compared to the placebo group (average 12% increase), an average of 10% improvement in variable visual motor reaction time, and an average improvement of 20% in temporal contrast sensitivity. CFF and reaction time are well-established methods for gauging neural efficiency. Further evidence of cognitive improvements with supplementation of lutein and zeaxanthin in young healthy subjects have been found with composite memory (an overall metric of memory performance), verbal memory, sustained attention, psychomotor speed, and processing speed.[76]

In dynamic, reactive sports situations, improved cortical efficiency and visual processing speed may enhance the ability to evaluate critical visual information faster. For example, more rapid visual processing allows a baseball batter to process more visual information regarding the judgment of the speed and trajectory of a pitched ball. In many sports, improved visual motor reaction time provides a well-recognized performance advantage. While caffeine also increases cortical processing speed, its effect is notably transient compared to the structural effects of carotenoids. These cortical effects may offer advantages in daily activities such as driving, and also be protective of age-related changes.

Application of Performance Benefits to Baseball Batting

Hitting a baseball is one of the more difficult sports tasks due to the speed and complexity of visual and motor demands involved. The time frame between the release of the ball by the pitcher and when it reaches the batter must be considered. Simplified calculations have suggested that a 90 mile-per-hour (mph) fastball pitch reaches the bat approximately 400ms after release, and a 75 mph curveball arrives in approximately 480ms.[77] The type of pitch thrown will have significant consequences on the flight trajectory of the ball due to properties of aerodynamics. The batter will have access to visual and cognitive cues to help anticipate the most likely type of pitch to expect, but time must be allocated for completing the mechanics of the swing. While batters typically complete the swing in 150ms, some can perform this feat faster. It is important to note that fielding in baseball also presents significant visual challenges for catching fly balls or fielding grounders, however this review will focus on visual performance factors in batting.

Judging the Pitch Release and Trajectory

The successful batter uses efficient and effective visual search patterns during the pitching motion to analyze any advance cues to the pitch type,[78-80] hopefully narrowing the trajectory probabilities that will need to be considered. At the moment of release, the batter initiates a saccadic eye movement to move fixation to the release point[78] or pitcher's elbow.[79] Basic physiology indicates that retinal cell information must be encoded and assembled, a process that takes approximately 25ms. This retinal information must be conducted to the visual cortex, requiring another 20ms. The visual cortex must process the retinal information to construct the image; since there is a substantial amount of memory available, this process takes approximately 30ms. Therefore, the visual information contained in the pitch release takes approximately 75ms to process, and the ball is now one-fifth of the distance to the plate. The visual images from tracking the ball can be processed continuously with only the 25ms conduction delay providing that the batter maintain pursuit eye movements.[81]

How good is the visual information being received? Studies presented previously have demonstrated superior visual resolution skills, contrast sensitivity, and dynamic visual acuity in baseball players.[82-87] While enhancing the seams of the baseball has been shown to improve curveball hitting, the sport demands that the visual system use the subtle cues of a traditional ball.[88] Some measures of stereopsis have been found to be superior in baseball players,[83,89,90] as have the visual field size of female softball players.[91] A recent study using a large sample of professional baseball players found that some sensorimotor abilities predict on-base percentage, walk rate, and strikeout rate, but not slugging percentage or fielder-independent pitching.[92] Specifically, the factors found to contribute to better batting performance were contrast sensitivity, depth perception, dynamic visual acuity, near-far accommodative-vergence facility, perception span, eye-hand coordination, and reaction time. The interaction effect of the dominant eye and dominant hand on batting in baseball has also received considerable scrutiny. Although reports have been somewhat contradictory, the preponderance of evidence indicates no relationship between eye dominance patterns and batting performance.[93-100]

Based on the extensive research demonstrating the improvements in visibility, contrast sensitivity and glare disability with increased macular pigment density, carotenoid intake appears to be an important factor for baseball athletes to consider. Enhancement of these visual performance factors provide the opportunity to better see the release of the ball, and make judgments regarding the pitch speed and trajectory.

Decision Making in Batting

Since the swing will take approximately 150ms to initiate to the point of contact, the decision of where and when to swing must be made by 250ms after the release. There are many models proposed to explain how cognition occurs in situations like batting in baseball,[101] but a minimum of 50ms is needed to select the appropriate response and send it to the motor processing areas of the brain to begin the action. This means the batter has approximately 200ms to process the visual information from the pitch in order to make an accurate decision; this timeframe is not much longer than measurements of simple reaction time in humans (approximately 150ms),[102] and is much shorter than the reaction times to complex choice conditions as demonstrated by Hicks.[103] The batter has many issues of spatial and temporal uncertainty to resolve due to the aerodynamics produced by the seam and texture of a baseball, and these must be resolved rather quickly. Considering the positive cortical benefits of increased macular pigment density on visual processing speed, temporal contrast sensitivity and reaction time, carotenoid intake may provide benefits with this stage of batting.

The Swing of the Bat

The swing mechanics takes approximately 150ms, but further visual processing can provide feedback for adjustments. After the first 50ms of the swing, the bat is moving at approximately 30% of its final velocity, and the swing can be changed substantially (or checked) based on continued visual information processing.[81] By 100ms into the swing, the bat is moving at approximately 75% of its final speed and can't be changed anymore due to the time factors to the muscles.[81] There are some who can execute the swing more rapidly than the time course described, which may allow more time for visual information processing and decision making, however studies have not found correlation between simple motor reaction time and batting skill,[104] or seen a difference during play in cricket batting.[105] Again, the improvements in reaction time found with higher macular pigment density may provide an advantage in swing performance.

Hammond and Fletcher proposed that the visual and neural factors influenced by L and Z accumulation may provide significant benefits for performance in baseball.[106] When considering the daylight variations and high intensity lighting for night games, the improvements in visibility, contrast sensitivity and glare disability with higher macular pigment density could provide substantial advantages. The very short time frame of the pitch and timing needed for the swing suggests there are also additional gains from improved neural processing speed. Baseball players who have diets that are low in carotenoid-rich fruits and vegetables, and who have relatively low macular pigment density, can be expected to reap considerable improvements by increasing their intake of carotenoids.

Efficacy of Supplementation of Macular Pigment

To help athletes achieve optimal visual performance, recommendations should include modifications to diet to increase intake of carotenoids, and possibly supplementation with purified forms of L and Z. It can be difficult for athletes to modify and sustain a diet rich in carotenoids, especially with the intake level needed to make a substantial change in macular pigment density. Placebo-controlled studies have found that macular pigment density can be increased an average of about 20% with supplementation.[3,60] This is consistent with numerous studies globally, although a small percentage of individuals do not appear to respond to supplementation. Studies find a non-responder rate ranging from 5-30%, depending on the supplement used in the study.[53,107-109] Recent studies have used 20 mg of dietary zeaxanthin in the supplements for those that are young and healthy, compared to lower concentrations for the aging population (the AREDS 2 formula has only 2 mg of zeaxanthin).[45,71,72]

For competitive athletes, care should be taken to recommend supplements that have been certified for content, including for substances banned in sports. Currently the only available supplements that are formulated specifically for athletes are Eyepromise Vizual Edge products (www.eyepromise.com) and Vizion Edge (https://vizionedge.com), which are National Science Foundation (NSF) certified for sports.

In addition to the visual performance improvements found with supplementation, evidence suggests that L and Z have protective effects for the retina from photo-oxidative damage. In addition to the protective effects from accumulated sun exposure over time in athletes, many also spend a considerable amount of time gaming. The protective effects of macular pigment density may also help with "blue light" exposure from extensive "screen time."

For those athletes who experience difficulties with glare, photostress and contrast judgment, increasing macular pigment density offers a potential method to improve these functions by enriching natural physiology. Some athletes do not see a benefit from sunglass filter recommendations to help with glare disability, and filters can be cumbersome to change when moving between bright light and shadow. It may be that improvement in L and Z concentrations in the macula can provide enhanced visual function without the reduction in overall luminance that occurs with external filter use. Further, common symptoms following traumatic brain injury include increased sensitivity to bright light (glare discomfort), glare disability, and slowed photostress recovery. Supplementation with L and Z may provide similar benefits to filter application in athletes who have concussion-related symptoms, and may provide more long term relief.

Athletes should be counseled on the time course for the effects of supplementation. The protective effects of L & Z are fairly immediate, but the visual and neural benefits will take longer for the macular pigment to accumulate. As a general guide, the visual benefits will take one to two months, and the neural benefits will need three to four months to become noticeable.

A third component of macular pigment is meso-zeaxanthin. Meso-zeaxanthin is a stereoisomer of zeaxanthin, and is the result of the human body's enzymatic conversion of dietary lutein. Importantly, L and Z are found naturally in many food sources, whereas meso-zeaxanthin is not found in any conventional food source.[110,111] Rather, meso-zeaxanthin is commercially produced from lutein by a synthetic chemical process. Another major difference is that there is wide anatomical distribution of L and Z,[18,112] but meso-zeaxanthin is only found naturally in the retina.[111,113] While there is evidence that meso-zeaxanthin is a potent anti-oxidant,[114] supplementation with lutein provides a natural source of the nutrient necessary for the production of meso-zeaxanthin without the need for a synthetic source. While there is some debate regarding the role of supplementation with a product that contains synthetic meso-zeaxanthin, it is important to note that all of the supplementation studies with young, healthy subjects have been conducted with dietary L and Z, and not with products containing meso-zeaxanthin.

Macular pigment optical density (MPOD) can be measured with several methods, including heterochromatic flicker photometry, autofluorescence imaging, and resonance Raman spectroscopy.[115-122] Use of clinical devices to measure MPOD help to identify which athletes may benefit the most from increasing the intake of L and Z. Examples of commercially available devices to measure MPOD include the macular densitometer (Macular Metrics; Rehoboth, MA), and Quantifeye Macular Pigment Screener (Zeavision, Inc., Chesterfield, MO). These devices are also useful for determining the effects of supplementation or changes in diet.

There are several challenges when conducting clinical trials with nutrient supplementation that are different than pharmaceutical trials. Since L and Z are naturally occurring nutrients, all subjects in any study have been exposed to the nutrients. This makes a true placebo group difficult since all subjects have some level of L and Z intake, and it is extremely difficult to control daily nutrient intake to limit the effects of changes or variations in diet. Additionally, nutrients are pan-systemic rather than targeted to a specific system, which is how most synthetic pharmaceutical agents are designed. These factors necessitate very large sample sizes in order to show significant effects from nutritional supplementation. The robust literature supporting the positive effects of carotenoid supplements is impressive in light of these challenges.

SUMMARY

There is ample evidence that increasing macular pigment density in young healthy subjects produces significant improvements in visibility, contrast sensitivity, glare disability and discomfort, photostress recovery, visual processing speed and reaction time. The current studies have not used competitive athletes as subjects, but it is reasonable to expect similar results to those found in young healthy subjects. For competitive athletes, the potential benefits from higher macular pigment density is an important area for consideration. It is likely that many athletes have not received information about the potential benefits of increasing carotenoid intake or supplementation. Optometrists can provide this information to help patients understand the role these nutrients play in the health and performance of their eyes, as well as guidance on effective use of supplements.

This article has focused on the role of carotenoids in improving visual performance in athletes, however these improvements can be applied to many other patients. The visual performance benefits would clearly be advantageous to daily activities such as driving or gaming. There are potential benefits for those in occupations such as law enforcement, firefighting, pilots and the military. Certainly, dietary improvements or supplementation provide protective effects from age-related changes. Therefore, it is logical to expand this topic beyond athletes, and realize that many patients may be interested in the potential benefits of increasing macular pigment density.

DISCLOSURE

Graham Erickson is a consultant and member of the Scientific Advisory Board for ZeaVision.

REFERENCES

1. Welford AT. The measurement of sensory-motor performance: survey and reappraisal of twelve years progress. Ergonomics 1960; 3:1,89-230.

2. Wooten BR, Hammond BR. Macular pigment: influences on visual acuity and visibility. Prog Retina Eye Res 2002; 21:225-40.
3. Stringham JM, Hammond BR. The glare hypothesis of macular pigment function. Optom Vis Sci 2007; 84:859-64.
4. Age-Related Eye Disease Study Research Group, SanGiovanni JP, Chew EY, Clemons TE, et al. The relationship of dietary carotenoid and vitamin A, E and C intake with age-related macular degeneration in a case-control study: AREDS report no. 22. Arch Ophthalmol 2007; 125:1225-32.
5. Age-Related Eye Disease Study 2 Research Group. Lutein + zeaxanthin and omega-3 fatty acids for age-related macular degeneration: the Age-Related Eye Disease Study 2 (AREDS2) randomized clinical trial. JAMA 2013; 309:2005-15.
6. Age-Related Eye Disease Study 2 Research Group, Chew EY, Clemons TE, SanGiovanni JP, et al. Secondary analyses of the effects of lutein/zeaxanthin on age-related macular degeneration progression: AREDS2 report no. 3. JAMA Ophthalmol 2014; 132:142-9.
7. Age-Related Eye Disease Study 2 Research Group, Chew EY, SanGiovanni JP, Ferris FL, et al. Lutein/zeaxanthin for the treatment of age-related cataract: AREDS2 randomized trial report no. 4. JAMA Ophthalmol 2013; 131:843-50.
8. Deinema LA, Vingrys AJ, Wong CY, et al. A randomized, double-masked, placebo-controlled clinical trial of two forms of omega-3 supplements for treating dry eye disease. Ophthalmol 2017; 124:43-52.
9. Epitropoulos AT, Donnenfeld ED, Shah ZA, et al. Effect of oral re-esterified omega-3 nutritional supplementation on dry eyes. Cornea 2016; 35:1185-91.
10. Kangari H, Eftekhari MH, Sardari S, et al. Short-term consumption of oral omega-3 and dry eye syndrome. Ophthalmol 2013; 120:2191-6.
11. Kawakita T, Kawabata F, Tsuji T, Kawashima M, Shimmura S, Tsubota K. Effects of dietary supplementation with fish oil on dry eye syndrome subjects: randomized controlled trial. Biomed Res 2013; 34:215-20.
12. Malhotra C, Singh S, Chakma P, et al. Effect of oral omega-3 fatty acid supplementation on contrast sensitivity in patients with moderate meibomian gland dysfunction: a prospective placebo-controlled study. Cornea 2015; 34:637-43.
13. Oleñik A, Jiménez-Alfaro I, Alejandre-Alba N, et al. A randomized, double-masked study to evaluate the effect of omega-3 fatty acids supplementation in meibomian gland dysfunction. Clin Interv Aging 2013; 8:1133-8.
14. Wojtowicz JC, Butovich I, Uchiyama E, et al. Pilot, prospective, randomized, double-masked, placebo-controlled clinical trial of an omega-3 supplement for dry eye. Cornea 2011; 30:308-14.
15. Krinsky, NI, Landrum JT, Bone RA. Biologic mechanisms of the protective role of lutein and zeaxanthin in the eye. Annual Rev Nutrition. 2003; 23:171–201.
16. Bone RA, Landrum JT, Tarsis SL. Preliminary identification of the human macular pigment. Vis Res 1985; 25:1531-5.
17. Krachik F, Beecher GR, Goli MB et al. Separation and identification of carotenoids and their oxidation products in the extracts of human plasma. Anal Chem 1992; 64:2111-22.
18. Landrum JT, Bone RA. Lutein, zeaxanthin, and the macular pigment. Archives of Biochemistry and Biophysics. 2001; 385:28–40.
19. Johnson EJ, Maras JE, Rasmussen HM, Tucker KL. Intake of lutein and zeaxanthin differ with age, sex, and ethnicity. J Am Diet Assoc 2010; 110:1357-62.
20. Bone RA, Landrum JT, Cains A. Optical density spectra of the macular pigment in vivo and in vitro. Vision Research. 1992; 32:105-110.
21. Walls GL, Judd HD. The intra-ocular colour-filters of vertebrates. Br J Ophthalmol 1933; 17:641-75.
22. Douglas RH, Marshall J. A review of vertebrate and invertebrate ocular filters. In SN Archer, MBA Djamgoz, ER Loew, JC Partridge & S Vallerga (Eds.), Adaptive Mechanisms in the Ecology of Vision. UK 1999: Kluwer Academic Publishers, pp 95-162.
23. Hammond BR. The visual effects of intraocular colored filters. Scientifica (Cairo) 2012; 2012: 424965. doi: 10.6064/2012/424965
24. Nussbaum JJ, Pruett RC, Delori FC. Macular yellow pigment. The first 200 years. Retina 1981; 1:296-310.
25. Wooten BR, Hammond BR. Macular pigment: influences on visual acuity and visibility. Prog Retin Eye Res 2002; 21:225-40.
26. Wong JC, Engles M, Wooten BR, Hammond BR. Testing the visibility hypothesis of macular pigment using a filter that simulates changes in macular pigment density. ARVO Abstract Invest Ophthalmol Vis Sci 2009:1704.
27. Clark BAJ. Color in sunglass lenses. Am J Optom Arch Am Acad Optom 1969; 46:825-40.
28. Kinney JS, Schlichting CL, Neri D, et al. Various measures of the effectiveness of yellow goggles. Naval Submarine Medical Research Laboratory, Report No. 941. Naval Medical Research and Development Command, RWU-MF58.524.0.13-1039. Groton: Naval Submarine Medical Research Laboratory, 1980.
29. Kinney JS, Schlichting CL, Neri DF, et al. Reaction time to spatial frequencies using yellow and luminance-matched neutral goggles. Am J Optom Physiol Optics 1983; 60:132-8.
30. Kinney JAS, Luria SM, Schlichting CL, Neri DF. The perception of depth contours with yellow goggles. Perception 1983; 12:363-6.
31. Kelly SA, Goldberg SE, Banton TA. Effect of yellow-tinted lenses on contrast sensitivity. Am J Optom Physiol Optics 1984; 61:657-62.
32. Yap M. The effect of a yellow filter on contrast sensitivity. Ophthalmol Physiol Opt 1984; 4:227-232.

33. Corth R. The perception of depth contours with yellow goggles – An alternative explanation. Perception 1985; 14:377-8.
34. Kinney JAS. The perception of depth contours with yellow goggles – comments on letter by Richard Corth. Perception 1985; 14:378-9.
35. Kelly SA. Effect of yellow-tinted lenses on brightness. J Opt Soc Am A 1990; A7:1905-11.
36. Zigman S. Vision enhancement using a short wavelength light-absorbing filter. Optom Vis Sci 1990; 67:100-4.
37. Rieger G. Improvement of contrast sensitivity with yellow filter glasses. Can J Ophthalmol 1992; 27:137-8.
38. Zigman S. Light filters to improve vision. Optom Vis Sci 1992; 69:325-8.
39. Rabin J, Wiley R. Differences in apparent contrast in yellow and white light. Ophthalmol Physiol Opt 1996; 16:68-72.
40. Lee JE, Stein JJ, Prevor MB, Seiple WH, Holopigian K, Greenstein VC, Stenson SM. Effect of variable tinted spectacle lenses on visual performance in controlled subjects. CLAO J 2002; 28:80-2.
41. Jendrusch G, Senner V, Schaff P, et al. Vision – an essential factor for safety in skiing: visual acuity, stereoscopic depth perception, effect of colored lenses. In: Johnson RJ, ed. Skiing Trauma and Safety, Twelfth Volume. West Conshohocken, PA: American Society for Testing and Materials, 1999:23-34
42. Luria SM. Vision and chromatic filters. Am J Optom Arch Am Acad Optom 1972; 49:818-29.
43. Wolffsohn JS, Cochrane AL, Khoo H et al. Contrast is enhanced by yellow lenses because of selective reduction of short-wavelength light. Optom Vis Sci 2000; 77:73-81.
44. Renzi LM, Hammond BR. The effect of macular pigment on heterochromatic luminance contrast. Exp Eye Res 2010; 91:896-900.
45. Hammond BR, Fletcher L, Roos F, et al. A double-blind, placebo-controlled study on the effects of lutein and zeaxanthin on photostress recovery, glare disability and chromatic contrast. Invest Ophthalmol Vis Sci. 2014; 55:8583-9.
46. Nolan JM, Power R, Stringham J, et al. Enrichment of macular pigment enhances contrast sensitivity in subjects free of retinal disease: Central retinal enrichment supplementation trials – Report 1. Invest Ophthalmol Vis Sci 2016; 57:3429-39.
47. Stringham JM, O'Brien KJ, Stringham NT. Contrast sensitivity and lateral inhibition are enhanced with macular carotenoid supplementation. Invest Ophthalmol Vis Sci 2017; 58:2291-5.
48. Hitzeman SA, Beckerman SA. What the literature says about sports vision. Optom Clin 1993; 3(1):145-69.
49. Coffey B, Reichow AW. Optometric evaluation of the elite athlete. Problems in Optometry 1990; 2(1):32-59.
50. Gardner JJ, Sherman A. Vision requirements in sport. In: Loran DFC, MacEwen CJ, eds. Sports Vision. Oxford: Butterworth-Heinemann 1995.
51. Kluka DA, Love PA, Sanet R, et al. Contrast sensitivity function profiling: By sport and sport ability level. Int J Sports Vision 1995; 2:5-16.
52. Love PA, Kluka DA. Contrast sensitivity function in elite women and men softball players. Int J Sports Vision 1993; 1:25-30.
53. Richer S, Park D-W, Epstein R, et al. Macular re-pigmentation enhances driving vision in elderly adult males with macular degeneration. J CLin Exp Ophthalmol 2012; 3:217.
54. Stringham JM, Garcia PV, Smith PA, et al. Macular pigment and visual performance in low-light conditions. Invest Ophthalmol Vis Sci 2015; 56:2459-68.
55. Stringham JM, Fuld K, Wenzel AJ. Action spectrum for photophobia. J Opt Soc Am A 2003; 20:1852-8.
56. Stringham JM, Fuld K, Wenzel AJ. Spatial properties of photophobia. Invest Ophthalmol Vis Sci 2004; 45:3838-48.
57. Wenzel AJ, Fuld K, Stringham JM, et al. Macular pigment optical density and photophobia light threshold. Vision Res 2006; 46:4615-22.
58. Stringham JM, Garcia PV, Smith PA, et al. Macular pigment and visual performance in glare: benefits for photostress recovery, disability glare, and visual discomfort. Invest Ophthalmol Vis Sci 2011; 52:7406-15.
59. Stringham JM, Snodderly DM. Enhancing performance while avoiding damage: a contribution of macular pigment. Invest Ophthalmol Vis Sci 2013; 54:6298-6306.
60. Stringham JM, Hammond BR. Macular pigment and visual performance under glare conditions. Optom Vis Sci 2008; 85:82-8.
61. Michaels, D. Visual Optics and Refraction: A Clinical Approach. Third edition, St. Louis: Mosby, 1985:12-13.
62. Craft NE, Haitema TB, Garnett KM, et al. Carotenoid, tocopherol, and retinol concentrations in elderly human brain. J Nutr Health Aging 2004; 8:156-62.
63. Vishwanathan R, Neuringer M, Schalch W, et al. Lutein (L) and zeaxanthin (Z) levels in retina are related to levels in the brain. FASEB J 2011; 25: 1_supplement, 344.1-344.
64. Vishwanathan R, Neuringer M, Snodderly DM, et al. Macular lutein and zeaxanthin are related to brain lutein and zeaxanthin in primates. Nutr Neurosci 2013; 16:21–29.
65. Hammond BR, Wooten BR. CFF thresholds: relation to macular pigment optical density. Ophthalmic Physiol Opt 2005; 25; 315-9.
66. Johnson EJ, McDonald K, Caldarella SM, et al. Cognitive findings of an exploratory trial of docosahexaenoic acid and lutein supplementation in older women. Nutr Neurosci 2008; 11:75-83.
67. Renzi LM, Hammond BR. The relationship between macular carotenoids, lutein and zeaxanthin, and temporal vision. Ophthalmic Physiol Opt 2010; 30:351-7.
68. Feeney J, Finucane C, Savva GM, et al. Low macular pigment optical density is associated with lower cognitive performance in a large, population-based sample of older adults. Neurobiol Aging 2013; 34:2449-56.

69. Renzi LM, Dengler MJ, Puente A, et al. Relations between macular pigment optical density and cognitive function in unimpaired and mildly cognitively impaired older adults. Neurobiol Aging 2014; 35:1695-9.

70. Vishwanathan R, Iannaccone A, Scott TM, et al. Macular pigment optical density is related to cognitive function in older people. Age Ageing 2014; 43:271-5.

71. Bovier ER, Renzi LM, Hammond BR. A double-blind, placebo-controlled study on the effects of lutein and zeaxanthin on neural processing speed and efficiency. PLoS ONE 9 2014; 9:e108178.

72. Bovier ER, Hammond BR. A randomized placebo-controlled study on the effects of lutein and zeaxanthin on visual processing speed in young healthy subjects. Arch Biochem Biophys. 2015;15(572):54-57.

73. Renzi-Hammond LM, Bovier ER, Fletcher LM, et al. Effects of a lutein and zeaxanthin intervention on cognitive function: a randomized, double-masked, placebo-controlled trial of younger healthy adults. Nutrients 2017; 9:1246-59.

74. Mewborn CM, Terry DP, Renzi-Hammond LM, et al. Relation of retinal and serum lutein and zeaxanthin to white matter integrity in older adults: a diffusion tensor imaging study. Arch Clin Neuropsychology 2017; 33:1–14.

75. Stahl W, Sies H. Effects of carotenoids and retinoids on gap junctional communication. Biofactors 2001;15:95–8.

76. Stringham NT, Holmes PV, Stringham JM. Effects of macular xanthophyll supplementation on brain-derived neurotrophic factor, pro-inflammatory cytokines, and cognitive performance. Physiol Behav 2019; 211:112650.

77. Bahill AT, Karnavas WJ. The perception of baseball's rising fastball and breaking curveball. J Exp Psychol: Hum Percept Perf 1993; 19:3-14.

78. Shank MD, Haywood KM. Eye movements while viewing a baseball pitch. Percept Mot Skills 1987; 64:1191-7.

79. Kato T, Fukuda T. Visual search strategies of baseball batters: eye movements during the preparatory phase of batting. Percept Mot Skills 2002; 380-6.

80. Uchida Y, Kudoh D, Higuchi T, et al. Dynamic visual acuity in baseball players is due to superior trackining abilities. Med Sci Sports Exerc 2013; 45:319–25.

81. Adair RK. The Physics of Baseball. New York: Perennial, 2002.

82. Winograd S. The relationship of timing and vision to baseball performance. Res Q Am Assoc Health Phys Educ 1942; 13:481-93.

83. Laby DM, Rosenbaum AL, Kirschen DG, et al. The visual function of professional baseball players. Am J Ophthamol 1996; 122:476-85.

84. Rouse MW, DeLand P, Christian R, et al. A comparison study of dynamic visual acuity between athletes and nonathletes. J Am Optom Assoc 1988; 59:946-50.

85. Ishigaki H, Miyao M. Differences in dynamic visual acuity between athletes and nonathletes. Percept Mot Skills 1993; 77:835-9.

86. Hoffman LG, Polan G, Powell J. The relationship of contrast sensitivity functions to sports vision. J Am Optom Assoc 1984; 55:747-52.

87. Klemish D, Ramger B, Vittetoe K, et al. Visual abilities distinguish pitchers from hitters in professional baseball. J Sports Sciences 2017; 36:171-9.

88. Osborne K, Rudrud E, Zezoney F. Improved curveball hitting through the enhancement of visual cues. J Applied Behav Analysis 1990; 23:371-7.

89. Soloman H, Zinn HJ, Vacroux R. Dynamic stereoacuity: a test for hitting a baseball? J Am Optom Assoc 1988; 59:522-6.

90. Hofeldt AJ, Hoefle FB. Stereophotometric testing for Pulfrich's phenomenon in professional baseball players. Percept Mot Skills 1993; 77:407-16.

91. Berg WP, Killian SM. Size of the visual field in collegiate fast-pitch softball players and nonathletes. Percept Mot Skills 1995; 81:1307-12.

92. Burris K, Vittetoe K, Ramger B, et al. Sensorimotor abilities predict onfield performance in professional baseball. Scientific Reports 2018; 8:116-24.

93. Adams GL. Effect of eye dominance in baseball batting. Res Q Am Assoc Health Phys Educ 1965; 36:3-9.

94. Falkowitz C, Mendel H. The role of visual skills in batting averages. Optom Weekly 1977; 68(20):33-6.

95. Portal JM, Romano PE. Patterns of eye-hand dominance in baseball players. New Eng J Med 1988; 319:655-6.

96. Dunham P. Coincidence-anticipation performance of adolescent baseball players and nonplayers. Percept Mot Skills 1989; 68:1151-6.

97. Milne C, Buckolz E, Cardenas M. Relationship of eye dominance and batting performance in baseball players. Int J Sports Vis 1995; 2:17-21.

98. Classe JG, Daum KM, Semes LP, et al. Association between eye and hand dominance and hitting, fielding and pitching skill among players of the Southern Baseball League. J Am Optometric Assoc 1996; 67:81-6.

99. Goss DA. The relationship of eye dominance and baseball batting: a critical literature review. J Behav Optom 1998; 9(4):87-91.

100. Laby DM, Kirschen DG, Rosenbaum AL, et al. The effect of ocular dominance on the performance of professional baseball players. Ophthalmol 1998; 105:864-6.

101. Paull G, Glencross D. Expert perception and decision making in baseball. Int J of Sport Psych 1997; 28:35-56.

102. Glencross DJ, Cibich BJ. A decision analysis of games skills. Austr J Sports Med 1977; 9:72-5.

103. Hicks WE. On the rate of gain of information. Q J Exp Psychol 1952; 4:11-26.

104. Classe JG, Semes LP, Daum KM, et al. Association between visual reaction time and batting, fielding, and earned run averages among players of the Southern Baseball League. J Am Optom Assoc 1997; 68:43-9.

105. McLeod P. Visual reaction time and high-speed ball games. Perception 1997; 16:49-59.

106. Hammond BR, Fletcher LM. Influence of the dietary carotenoids lutein and zeaxanthin on visual performance: application to baseball. Am J Clin Nutr 2012; 96:1207S-1213S.

107. Hammond BR, Johnson EJ, Russell RM, et al. Dietary modification of human macular pigment density. Invest Ophthalmol Vis Sci 1997; 38:1795-801.

108. Richer S, Devenport J, Lang JC. LAST II: Differential temporal responses of macular pigment optical density in patients with atrophic age-related macular degeneration to dietary supplementation with xanthophylls. Optometry 2007, 78:213-9.

109. Trieschmann M, Beatty S, Nolan JM, et al. Changes in macular pigment optical density and serum concentrations of its constituent carotenoids following supplemental lutein and zeaxanthin: the LUNA study. Exp Eye Res 2007; 84:718-28.

110. Maoka T, Arai A, Shimizu M, et al. The first isolation of enantiomeric and meso-zeaxanthin in nature. Comp Biochem Physiol B 1986; 83:121-4.

111. Rasmussen HM, Muzhingi T, Eggert EMR, et al. Lutein, zeaxanthin, meso-zeaxanthin content in egg yolk and their absence in fish and seafood. J Food Com Anal 2012; 27:139-44.

112. Peng YM, Peng YS, Lin Y. A nonsaponification method for the determination of carotenoids, retinoids, and tocopherols in solid human tissues. Cancer Epidemiol Biomarkers Prev 1993; 2:139-44.

113. Johnson EJ, Neuringer M, Russell RM, et al. Nutritional manipulation of primate retinas, III: effects of lutein or zeaxanthin supplementation on adipose tissue and retina of xanthophyll-free monkeys. Invest Ophthalmol Vis Sci 2005; 46:692-702.

114. Li B, Ahmed F, Bernstein PS. Studies on the singlet oxygen scavenging mechanism of human macular pigment. Arch Biochem Biophys 2010; 504:56-60.

115. Wooten BR, Hammond BR, Land R et al. A practical method of measuring macular pigment optical density. Invest Ophthalmol Vis Sci 1999; 40:2481-9.

116. Hammond BR, Wooten BR. Noninvasive assessment of the macular carotenoids. In: Retina and Optic Nerve Imaging, Ciulla TA, Regillo CD, Harris A, eds. Delaware: Lippincott, Williams & Wilkins, 2003:231-43.

117. Wooten BR, Hammond BR, Smollon B. Assessment of the validity of heterochromatic flicker photometry for measuring macular pigment optical density in normal subjects. Optom Vis Sci 2005; 82:378-86.

118. Makridaki M, Carden D, Murray IJ. Macular pigment measurement in clinics: controlling the effect of the ageing media. Ophthal Physiol Opt 2009; 29:1-7.

119. Van der Veen RLP, Berendschot TJM, Hendrikse F, et al. A new desktop instrument for measuring macular pigment optical density based on a novel technique for setting flicker thresholds. Ophthal Physiol Opt 2009; 29:127-37.

120. Murray IJ, Carden D, Makridaki M. The repeatability of the MPS 9000 macular pigment screener. Br J Ophthalmol 2011; 95:431-2.

121. Raman R, Biswas S, Gupta A, et al. Association of macular pigment optical density with risk factors for wet age-related macular degeneration in the Indian population. Eye (London) 2012; 26:950-7.

122. Dennison JL, Stack J, Beatty S, et al. Concordance of macular pigment measurements obtained using customized heterochromatic flicker photometry, dual-wavelength autofluorescence, and single-wavelength reflectance. Exp Eye Res 2013; 116:190-8.

CORRESPONDING AUTHOR BIOGRAPHY:

Graham B. Erickson, OD, FAAO, FCOVD
Forest Grove, Oregon

Dr. Graham Erickson is a professor at Pacific University College of Optometry, and currently teaches the Vision Therapy, Strabismus/Amblyopia and Sports Vision courses. He is the coordinator of the Vision Therapy/Rehabilitation and Pediatric Optometry residency at Pacific University. He has worked with collegiate, Olympic and professional sports athletes and teams for more than 25 years. Dr. Erickson has authored the text *Sports Vision: Vision Care for the Enhancement of Sports Performance*, as well as co-authoring the text *Optometric Management of Reading Dysfunction*, and published chapters and articles in various professional journals. He lectures internationally on the topics of sports vision, pediatrics, and binocular vision.

ARTICLE

Neuro-Optometric Rehabilitation Using a Multisensory-Based Bottom-Up to Top-Down Paradigm for Post-Concussion Syndrome – A Retrospective Case Series Study

Steven J. Curtis OD, FCOVD, FNORA

ABSTRACT

Background

Optometrists are becoming increasingly involved in the rehabilitation management of concussion patients who experience prolonged recovery of symptoms, often referred to as post-concussion syndrome (PCS). Literature pertaining to this population provides ample evidence that neuro-optometric rehabilitation is effective for oculomotor (OM) vision dysfunctions, but lacks the same for non-oculomotor vision (NOM) dysfunctions. Many PCS patients suffer NOM vision dysfunctions whereby they are foundationally too sensory-fragile to tolerate a traditional top-down therapy approach early in their treatment process. This study provides a review of PCS patients receiving a multisensory-based bottom-up to top-down neuro-optometric rehabilitation paradigm that targets NOM, as well as, OM vision dysfunctions.

Methods

The participants are mild traumatic brain injury (mTBI) patients with persistent symptoms. The design is a retrospective chart review of clinical measures one week before and an average of 38 days after initiation of neuro-optometric rehabilitation. The main measures reviewed are the Sports Concussion Assessment Tool 5 (SCAT5) symptom survey, the Dizziness Handicap Index (DHI), the Test of Information Processing Skills (TIPS), and the FCFTester kinetic color visual fields. A secondary measure is near point of convergence (NPC).

Results

The patient group averaged an improvement of 50% in SCAT5 symptoms survey score and a 48% improvement in the DHI total score. The TIPS revealed a group average improvement in visual modality, auditory modality, and delayed recall of 231%, 123%, 172%, respectively. Kinetic color visual fields increased an average of: green OD 51%, OS 47%, red OD 27%, OS 26%, and blue OD 21%, OS 23%.

Conclusion

The clinical findings and treatment of 56 PCS patients are described. Neuro-optometric rehabilitation utilizing a specific paradigm provided substantial improvement in both OM and NOM vision measures and symptoms for the group in 38 days of therapy. The paradigm began with a multisensory-based passive, input-only, bottom-up therapy accompanied by gradual addition of active, output-based, top-down techniques.

Correspondence regarding this article should be emailed to Steven J. Curtis OD, FCOVD, FNORA, at curtisten@sbcglobal.net. *https://doi.org/10.31707/VDR2019.5.4.p235.*

Curtis SJ. Neuro-Optometric Rehabilitation using a multi-sensory-based bottom-up to top-down paradigm for post-concussion syndrome – A retrospective case series study. Vision Dev & Rehab 2019;5(4):235-48.

Keywords: bottom-up therapy, multisensory processing, neuro-optometric rehabilitation, non-oculomotor based vision problems, oculomotor-based vision problems, optometric phototherapy, post-concussion syndrome, top-down therapy

BACKGROUND

Neuro-optometric rehabilitation (NOR) is a therapy service provided by specially trained optometrists which utilizes therapeutic prisms, lenses, filters, occlusion, and vision therapy to help stimulate and reorganize visual pathways of the brain which are not functioning properly due to brain injury. NOR differentiates itself from other optometric specializations through extensive collaboration with other professions. This is particularly seen in management of concussion, also referred to as mild traumatic brain injury (mTBI). According to the American Association of Neurological Surgeons, the formal medical definition of concussion is a clinical syndrome characterized by immediate and transient alteration in brain function, including alteration of mental status and level of consciousness, resulting from mechanical force or trauma.[1] In many cases, there are no external signs of head trauma nor loss of consciousness. However, a wide constellation of symptoms can result including vision disturbances. Literature reveals that more than 70 percent of concussion patients suffer related vision dysfunctions.[2] Since no system provides more neurosensory input to the brain than vision, optometry is best equipped and has an obligation to provide rehabilitation of vision to this patient population.

Incidence rates for concussion range widely from a conservative 300,000 per year to a more liberal and recent estimate of 3.8 million cases in the United States annually.[3] A recently signed bill, The Traumatic Brain Injury Program Reauthorization Act of 2018, has directed the CDC to implement a National Concussion Surveillance System. This will allow for more accurate determination of the incidence and cause of concussions (children and adults).[4]

Symptoms of a concussion include, but are not limited to, blurred vision, fatigue, dizziness, headache, irritability, insomnia, inattention, photophobia, vertigo, and cognitive difficulties.[5-9] Symptoms largely reflect a functional disturbance rather than a structural injury and, as such, no abnormality is seen on standard structural neuroimaging (CT scans and MRI). However, several studies have revealed existence of brain damage in mTBI's using functional imaging techniques such as Diffusion Tensor Imaging, a more advanced variation of MRI.[10] Unfortunately, this imaging technology remains expensive and is often reserved for research purposes rather than clinical patient evaluation. Therefore, a patient with an isolated concussion commonly receives a CT scan and if negative, emergency room physicians are advised that the patient may be safely discharged. Although most patients do recover from a concussion within three weeks,[11] others suffer persistent symptoms leading to a diagnosis of post-concussion syndrome (PCS).

While a number of definitions for PCS have been proposed in the literature, generally it refers to concussion symptoms that persist for weeks, months, or more than a year after a concussion.[12,13] The incidence of PCS varies, but most studies report that about 15% of individuals with a history of a single concussion develop persistent symptoms associated with the injury. Premorbid risk factors for PCS include prior concussions, mental health problems, physician-diagnosed migraine, vestibulo-ocular dysfunction, hyperacusis, and ADHD/learning disabilities.[14-17]

There remains a lack of evidence-based treatment strategies for PCS, however, some individuals benefit from several interventions depending on the particular presenting signs and symptoms. The most common treatment options that are considered to be effective consist of medications, early education,[18,19] cognitive behavioral therapy,[20] aerobic exercise therapy,[21] and physical therapy.

Many of the symptoms of PCS are, in part, a result of compromised processing of sensory inputs, including visual.[22] Research has shown that optometric vision therapy should be included in the overall treatment as it provides improvement in post-concussion vision problems.[23] However, optimal human perception is dependent upon the combined processing of multiple sensory inputs more so than unisensory processing. Neuroscience research has long-established that there is widespread distribution of brain pathways dedicated to multisensory integration and processing. And multisensory plasticity has strong implications for successful rehabilitation of mTBI patients. This case series provides a retrospective review of PCS patients receiving a neuro-optometric rehabilitation paradigm with multisensory processing training at its core with adjunctive optometric vision therapy. This author considers this a bottom-up to top-down therapeutic approach and has experienced it to be uniquely effective when non-oculomotor-based vision dysfunctions are diagnosed at the patient's initial evaluation.

METHOD

This study was a retrospective chart review of 66 consecutive PCS patients who presented with both OM and NOM vision dysfunctions to the author's clinic during the period of August 2017 to June 2019. Reviewed were clinical measures performed one week before and an average of 38 days after initiation of treatment. Six patients were excluded due to their concussion occurring greater than three years prior to evaluation. Controversy in literature proposes the further out a patient is from his/her injury, persistent symptoms could be strongly associated with conditions other than the head injury.[24] Four patients were excluded due to already receiving optometric vision therapy elsewhere. Patient exposure to all other prior interventions was included. This resulted in an "N" of 56. Each patient was de-identified via meeting safe harbor requirements under section 164.514(a) of the HIPAA Privacy Rule.[25] Patient ages ranged from 15 to 73 with a median age of 45. The time between most recent concussion and initiation of treatment ranged from 48 to 1010 days with a median time of 134 days. Fifteen were men and 41 were women. The number of previous concussions ranged from zero to four. No patients were excluded based on socioeconomics nor method of payment (i.e. insurance, private pay, etc.) factors. Since physical therapy (PT) is commonly considered a standard of care recommendation for post-concussion, the study made note that thirty-seven patients had received (PT) at some point since their injury and before receiving our treatment.

The neuro-optometric rehabilitation protocol utilized consisted of 12 consecutive days of in-office visits each lasting 75 minutes followed by 18 days of home therapy. The treatment consisted of a blending of 1) multi-sensory integration via simultaneous application of optometric phototherapy, vestibular stimulation, auditory stimulation, and 2) gradually applied sensorimotor output activities such as versional and vergence oculomotor therapy and balance tasks. See Figure 1 for an example. Many patients were also provided weighted proprioceptive sensory support (e.g. weighted blankets/toys, ankle weights) per their gravity-based sensitivity.

Optometric Phototherapy, also known as Syntonic Optometry, is the application of light through the pupil to the retinal blood supply and to retinal photoreceptors. It is a method of neuromodulation using photo-transduction – photons of light activating a graded change in membrane potential and a corresponding change in the rate of transmitter release onto postsynaptic neurons.[26] It is a noninvasive use of prescribed light frequencies to treat visual dysfunction, brain injury, and imbalanced autonomic nervous systems.[27,28] As the photonic energy of the light stimulates the retinal ganglion cells of the retina, it can subsequently re-energize many neural structures including, but not limited to, the thalamus, hypothalamus, superior colliculus, pineal gland, and the pituitary gland. The optometric phototherapy in this treatment protocol consisted of 12 days of a progression through magenta, ruby, yellow-green, yellow-

Day	Bottom-up Passive therapy (in-office)	Bottom-up Passive therapy (in-office)
1	Optometric phototherapy (OP) using one color, lateral canal vestibular stimulation, auditory training	monocular and binocular horizontal pursuits
2	OP using three colors, posterior and anterior canal vestibular stimulation, auditory training	monocular and binocular vertical pursuits
3	OP using three, lateral canal vestibular stimulation, auditory training	monocular and binocular horizontal pursuits
4	OP using three colors, posterior and anterior canal vestibular stimulation, auditory training	monocular and binocular vertical pursuits monocular 4-corner wall saccades, clock dial saccades, Percon mazes level 1
5	OP using four colors, lateral canal vestibular stimulation, auditory training	monocular horizontal pursuits monocular arrow wall saccades, clock dial saccades, Percon mazes level 1
6	OP using four, posterior and anterior canal vestibular stimulation, auditory training	monocular and binocular vertical pursuits, monocular 4-corner wall saccades, clock dial saccades, Percon mazes level 1
7	OP using six colors, lateral canal vestibular stimulation, auditory training	binocular horizontal pursuits binocular arrow wall saccades, clock dial saccades and peripheral awareness, Percon mazes level 1
8	OP using six colors, posterior and anterior canal vestibular stimulation, auditory training	binocular vertical pursuits binocular 4-corner wall saccades, clock dial saccades and peripheral awareness, Percon mazes level 2 +/-.25 lens flipper at near 2 BO and BI loose prism fusion at 15 ft
9	OP using six colors, lateral canal vestibular stimulation, auditory training	binocular horizontal pursuits binocular arrow wall saccades with tandem stance, clock dial saccades and peripheral awareness, Percon mazes level 2 +/-.50 lens flipper at near 4 BO and 2 BI loose prism fusion at 15 ft
10	OP using six colors, posterior and anterior canal vestibular stimulation, auditory training	binocular vertical pursuits binocular 4-corner wall saccades with tandem stance, clock dial saccades and peripheral awareness, Percon mazes level 2 +/-.50 lens flipper 6 BO and 2 BI loose prism fusion at 15 ft
11	OP using six colors, lateral canal vestibular stimulation, auditory training	binocular horizontal pursuits binocular arrow wall saccades balancing on one foot, clock dial saccades and peripheral awareness, Percon mazes level 2 +/-1.00 lens flipper 8 BO and 4 BI loose prism fusion
12	OP using six colors, posterior and anterior canal vestibular stimulation, auditory training	

HOME THERAPY ONLY BEGINS

13-30	home OP twice daily using one color; NO further vestibular or auditory training	head rotation pursuits and 4-corner saccades

Figure 1: Example of the OMST protocol with adjunctive active vision therapy. The left column represents the passive, bottom-up therapy and the right column represents the active, top-down therapy. Note, many PCS patients will not tolerate the above "rapid" active schedule in this example and modification to this would be left up to the practitioner's professional judgement. The optometrist well trained in syntonics is able to make modifications to the left column on a daily basis but within the boundaries of the above protocol.

blue, violet, and magenta again. The order, exposure time, and combination of filters was determined based on integration of principles taught by the College of Syntonic Optometry, the Sensory Learning Institute, and readings from the works of Dr. Edwin Babbit and Dr. Dinshah Ghadiali, 19th and 20th century pioneers in the use of light therapeutically.[29]

Vestibular stimulation is provided by movement of the patient whereby the vestibular system in the inner ear registers motion, both linear and rotational, and sends this sensory information via the eighth cranial nerve to the vestibular nuclei of the brainstem. Here it engages with sensory inputs from the visual, somatosensory, and auditory systems. Efferent fibers proceed from here to provide motor output to extraocular muscles for appropriate oculomotor response and the spinal cord for balance. The vestibular nuclei also send sensory information to the cerebellum so it can modify and further control the motor responses.[30] The vestibular stimulation in this treatment protocol was achieved by slow and gentle 7" circular rotation of the patient in supine position on a trochoidal motion table. The supine position provides favorable neck support, gravity-induced somatosensory/proprioceptive input, and comprehensive stimulation of the various vestibular apparatuses. The patient's horizontal position is alternated daily so that stimulation of both the anterior/posterior and the lateral semicircular canals is achieved.

The auditory stimulation was comprised of tracks of unfamiliar music that is randomly attenuated and has pre-determined frequencies filtered out. The specific program utilized is The Sensory Learning Acoustic Training program created by Mary Bolles based on the work of French physicians Alfred Tomatis and Guy Berard.

OMST Active Therapy Log

Day	Date	PP	FS	4CS	AS	CDS	PM1	PM2	CDPA	25LFN	50LFN	100LFN	11FPFD	21LPFD	42LPFD	MD	ATS	A1F	AV	MD2
1																				
2																				
3																				
4																				
5																				
6																				
7																				
8																				
9																				
10																				
11																				
12																				

PP – Pencil Pursuits
FS – Figurine Saccades
4CS – 4 Corner Saccades
CDS – Clock Dial Saccades
PM1 – Percon Mazes, Level 1
PM2 – Percon Mazes, Level 2
CDPA – Clock Dial Peripheral Awareness
AS – Arrow Saccades

25LFN – +/- .25 Lens Flipper @ Near (M or B)
50LFN – +/- .50 Lens Flipper @ Near (M or B)
100LFN – +/- 1.0 Lens Flipper @ Near (M or B)
11FPFN – 2 BO/ 2 BI Flip Prism Fusion @ 10 ft
21LPFD – 4 BO/2 BI Flip Prism Fusion @ 10 ft
42LPFD – 8 BO/4BI Flip Prism Fusion @ 10 ft
MD – Modified ___________________

ATS – Add Tandom Stance
A1F – Add 1 Foot Balance
AV – Add Visualization
MD2 - Modified ___________________

Notes:

M= Monocular B = Binocular

Figure 2: Log sheet for administering and tracking of the active, top-down therapy that is adjunctive to the passive, bottom-up therapy.

Oculomotor therapy is commonly prescribed for remediation of oculomotor (eye movement) deficits prevalent in brain injured patients. It has also been proven to improve visual attention.[31] The patient is guided through saccadic and pursuit eye movement tasks to improve visual fixation accuracy and smoothness. It also includes vergence training to enhance efficiency and stamina of maintaining clear and single binocular fixation. The oculomotor therapy in this treatment protocol was initially minimal, consisting of five to ten minutes of monocular (progressing to binocular) saccadic and pursuit activities. Then convergence therapy was gradually introduced as tolerated without aggravating patient symptoms. These activities increased in difficulty during the in-office phase of treatment. Balance and other sensorimotor output activities were gradually added to further rehabilitate integration of sensorimotor pathways. See Figure 2.

The primary clinical measures reviewed were the symptom survey of the Sports Concussion Assessment Tool 5 (assessment of both OM and NOM), the Dizziness Handicap Index (assessment of NOM), FCFTester kinetic visual fields (assessment of NOM), and the Test of Information Processing Skills (assessment of NOM). A secondary measure was near point of convergence (assessment of OM). These measures were administered one week prior to the initiation of the therapy and an average of 38 days post-initiation of the therapy.

The Sports Concussion Assessment Tool, 5th edition (SCAT5) symptom survey, measures a global range of self-reported concussion symptoms. The patient rates 22 symptoms on a scale from zero to six.[32] According to the Consensus statement on concussion in sport—the 5th international conference on concussion in sport held in Berlin, October 2016, the SCAT5 symptom survey demonstrates clinical utility in tracking recovery.

The Dizziness Handicap Inventory (DHI) measures the self-perceived level of handicap associated with the symptom of dizziness with high test-retest reliability and internal consistency.[33] The DHI has 25 items with 3 response levels, sub-grouped into three domains: functional, emotional, and physical. The patient has a response option for each symptom of 0, 2, or 4. The total score is the sum of the responses. Possible total score range is 0–100; a higher score indicates a worse handicap. Whitney et al. propose that a total score of 0–30 indicates mild, 31–60 moderate, and 61–100 severe handicap, and that scores correlate well to levels of functional balance impairment.[34]

The kinetic color fields (green, red, and blue) were measured monocularly utilizing the FCFTester computerized program. The parameters for each patient were the following: 1.6-2.2 mm diameter target size; target presentation speed of 24-36mm/second; target brightness setting for each color was 176 (no unit); random order of presentation of the three colors; each target presentation began at 30 degrees from center; twelve meridians were tested at 30-degree intervals; central fixation target was a single digit that randomly changed and flashed at a frequency of once per 1500ms. Although there was a range of target size and target presentation speed throughout the group, the same settings were repeated at follow-up testing for each patient.

The Test of Information Processing Skills (TIPS) represents an assessment of attentive cortical (cognitive/executive function) processing. The TIPS is a norm-referenced test developed by neuropsychologist, Dr. Raymond Webster, that assesses information processing skills in children and adults between age 5 and 90. Performance on the TIPS represents a top-down assessment since it measures cortically based visual processing, auditory processing, executive functioning, working memory, and delayed recall.

Near point of convergence (NPC) is a clinical measure utilized to assess a patient's ability to maintain alignment of the eyes on a near object. Inability to sustain convergence

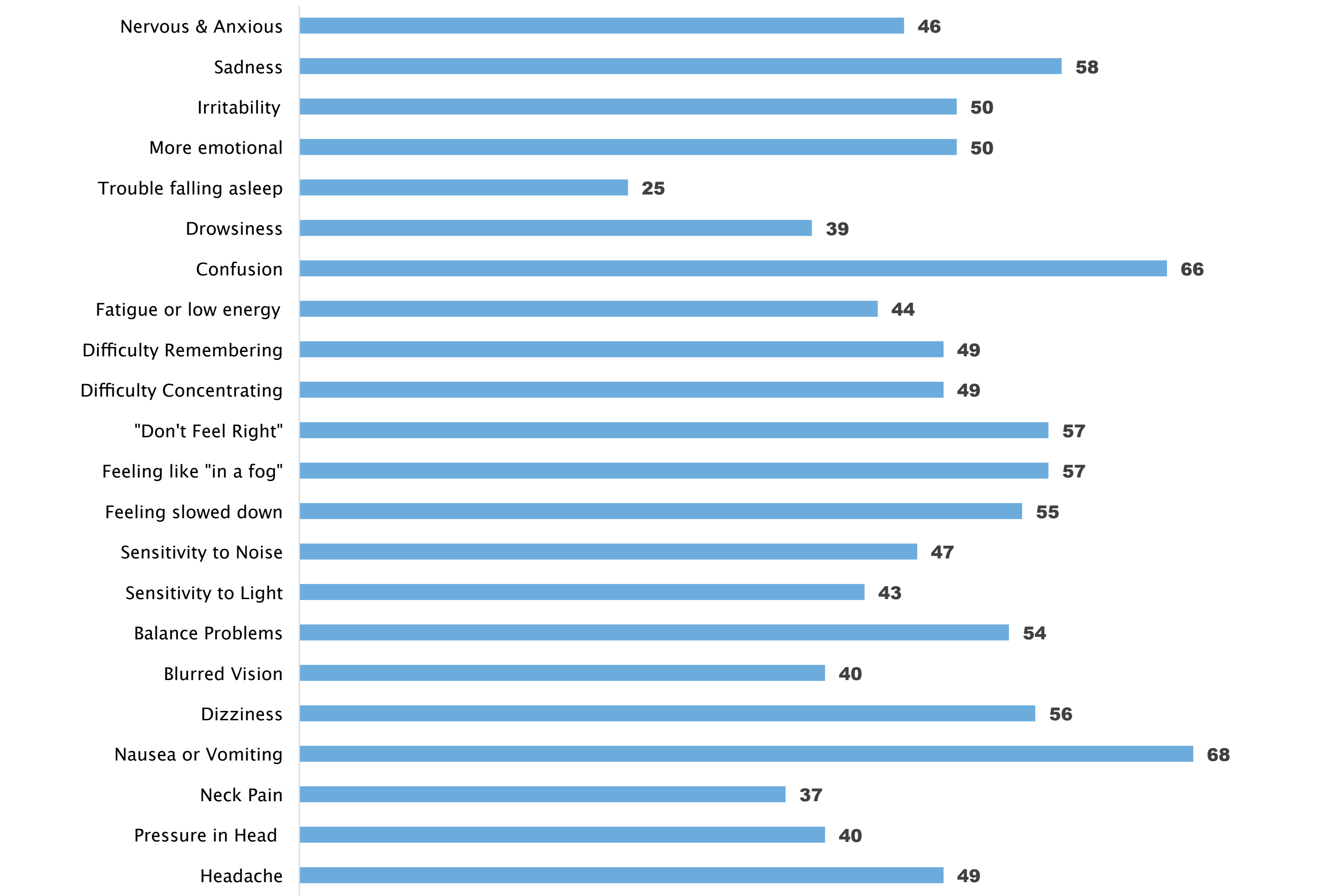


Figure 3: Percent improvement of the group for each SCAT5 symptom at the 38 day follow up.

at near may cause a person to fixate with one eye at a time, or to experience diplopia. A reduced NPC is commonly found in post TBI patients and has been found to be a good objective marker in acute mTBI.[35]

RESULTS

The patient group averaged an improvement of 50% in SCAT5 symptom survey total score. See Figure 3 for improvement averages specific to each symptom of the SCAT5 survey. The majority of patients (30/56) reported greater than 50% improvement. See Figure 4. All 56 patients completed the survey before and after therapy. The group averaged an improvement of 48% in their DHI total score. All patients completed the survey before and after therapy. Ninety-one percent of patients had an improved score and 9% had a worsened score. Kinetic functional color visual fields increased

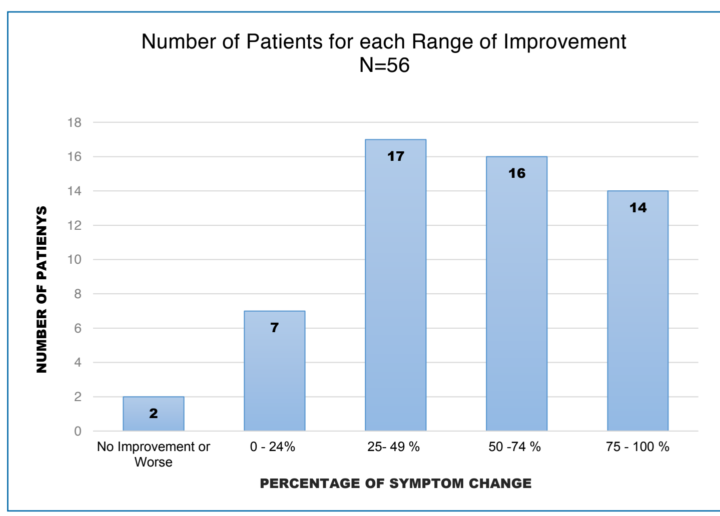


Figure 4: Number of patients in each range of improvement in SCAT5 symptom survey. Note: the majority of patients (30) reported greater than 50% improvement at the 38-day follow up evaluation.

an average of: green OD 51%, OS 47%, red OD 27%, OS 26%, and blue OD 21%, OS 23%. All patients performed the test. The TIPS revealed a group average improvement in visual

Table 1. Summary of clinical measure results at the 38-day follow up.

	SCAT5 Survey	Dizziness Handicap Index	Kinetic Fields Green OD/OS	Kinetic Fields Red OD/OS	Kinetic Fields Blue OD/OS	TIPS Visual Modality	TIPS Delayed Recall	NPC
Average Percent improvement of the group	50	48	51/47	27/26	21/23	231	172	21.6
Percentage of subjects that improved	95	91	71/64	71/75	79/74	56	79	84
Percentage of subjects that worsened	3.5	9	29/36	29/25	21/26	38	10	9
Percentage of Patients with no change	1.7	0	0/0	0/0	0/0	6	11	6

modality, auditory modality, and delayed recall of 231%, 123%, 172%, respectively. Five of the 56 patients did not complete the TIPS due to being too symptomatic to finish it. The group averaged an improvement of 21.6% in NPC measurements. Thirty-two of the 56 patients were included in the NPC data results. Patients not included were those with normal (less than 8cm) baseline measures (18/56) and five patients who did not receive the test measures at one or both visits. See Table 1 for summary.

No appreciable difference in SCAT5 symptom survey and DHI measures were found when taking into consideration whether or not PT occurred prior to the neuro-optometric rehabilitation. The group that received PT prior to our treatment had an average improvement of 51.0% and 48.8% in SCAT5 and DHI scores, respectively. The group that did NOT receive PT prior to our treatment had an average improvement of 49.2% and 47.05% in SCAT5 and DHI scores, respectively.

DISCUSSION

The PCS patients in this case series received neuro-optometric vision rehabilitation that was based on a bottom-up to top-down therapeutic protocol. All included patients presented with both non-oculomotor based and oculomotor based vision disturbances. Non-oculomotor (NOM) based vision disturbances include motion sensitivity, mental fogginess, photophobia, dizziness, nausea, and visual information processing deficits,[36] whereas, oculomotor (OM) based vision disturbances refer to vergence, accommodative, saccadic, and pursuit dysfunctions. The literature has a significant amount of evidence that optometric rehabilitation is effective for OM based vision dysfunctions, but lacks the same for NOM based vision dysfunctions. The outcome of this study suggests that PCS patients who present with NOM based vision symptoms recover efficiently and globally when a bottom-up to top-down therapeutic approach is employed. Therefore, this protocol is appropriate to consider. Concussed patients who present with primarily OM based vision dysfunctions should continue to be provided with a traditional top-down model as described by D'Angelo and Tannen.[37]

Top-down therapy requires conscious and intentional mental processing at the level of the cerebral cortex.[38] It employs an attentional mechanism of voluntarily orienting toward relevant stimuli, objects, events, or locations based on one's goals, motivation, or expectation. In contrast, a bottom-up therapy approach more likely targets automatic, pre-attentive[39,40] neural mechanisms that are based on responding to stimuli and environmental events which may be salient, counter to, or irrelevant to the individual's

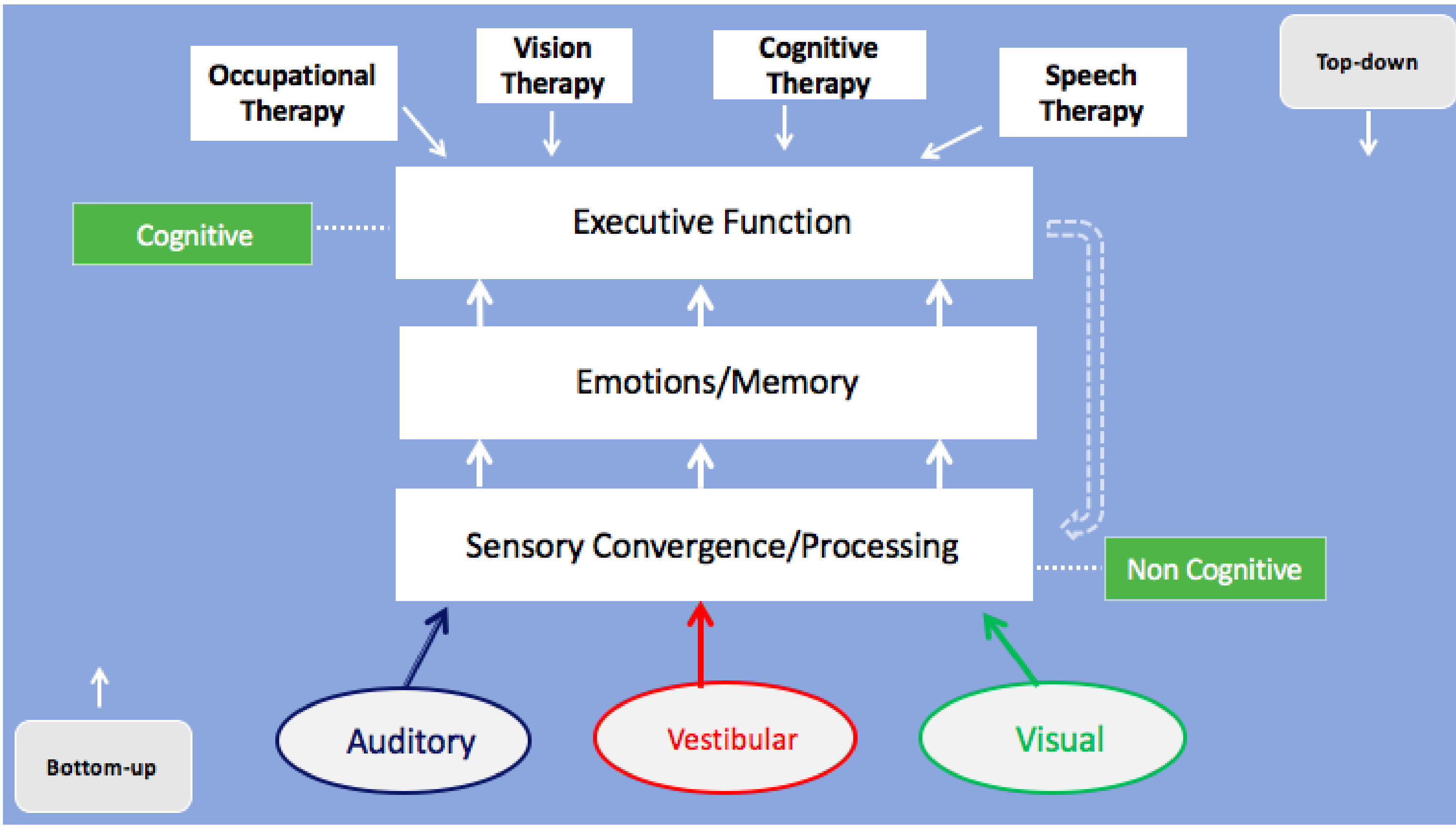


Figure 5: Sensory convergence and processing begins in the brainstem/midbrain (evolutionarily, embryonically, and developmentally after birth), and ideally becomes an efficient process on "autopilot". This provides momentum for higher "new" (cognitive/cortical) processing skills to be learned, which can then further enhance brainstem processing. Head injury can cause dysfunction at brainstem/midbrain areas (especially whiplash) causing information processing to get stuck in "first gear". This results in difficulty with cognitive perception (mental slowness, foggy headedness, sequencing difficulties, etc). Therefore, many PCS patients benefit from therapeutic interventions that begin with a passive, bottom-up approach such as OMST that is initially targeted at the brainstem/midbrain areas, before or adjunctive to top-down therapeutic approaches.

goals or expectations.[41] Despite the existence of these two "directions of processing", there is evidence that they influence each other and synergistically interact to provide optimal perception.[42]

Bottom-up therapy mechanisms influence central neural processing activities via ascending pathways from the periphery to the brainstem and then the cerebral cortex.[38] Bottom-up processing is reactive and involves the brain's initial reception of information from sensory inputs, whereas, top-down processing is strategic and planned.[43] See Figure 5 for this author's diagrammatic representation.

Peachy and Peachy refer to bottom-up and top-down visual pathways as subconscious/subcortical and purposeful/cortical, respectively. They state that initially, vision rehabilitation in traumatic brain injured patients may need to address dysfunctional subcortical colliculi and multisensory pathways. Then oculomotor deficits can benefit from therapeutic procedures that require visual direction, followed by perceptual accuracy treatment.[44]

As most optometrists have experienced, PCS patients are sometimes too sensitive to tolerate an approach that begins with attention driven techniques (i.e. active/output based, high complex vision therapy). This top-down therapy approach assumes the patient is ready for motor output demands such as saccades, pursuits, vergences, and accommodation. Often the patient with PCS is too sensory-input "fragile" and "pulls back", contorts their face, breaks into a cold sweat, alters their breathing, or completely resists the sensory-motor activity. This type of patient needs to first be provided with an opportunity to establish improved automaticity and efficiency in pre-attentive processing, particularly, multisensory integration processing. This can then provide sensory regulation that is foundationally essential before attempting top-down therapy such as instructional oculomotor skill activities.

Multisensory processing describes the ability of an intact, well-developed brain to receive information from multiple sensory pathways and modulate these inputs for optimal identification of and reactivity to environmental events. This is a biological example of the "whole being greater than the sum of its parts".[45] This ability of the brain to simultaneously process information from multiple independent sensory channels has significantly contributed to our survival. Studies including the midbrain have shown that the development of this process is not predetermined.[46] Neurons in a newborn's brain are not capable of multisensory integration, but as their brains are given the cross-modal experiences, their brains learn and engage this strategy at multiple levels of the neuraxis.[47]

Most researchers believe multisensory processing begins in the thalamus[48] and midbrain regions. For example, the superior colliculus houses the initial regions that are involved in receiving converged sensory inputs (structural) from numerous ascending and descending unisensory afferent sources[49] before multisensory processing (functional effect) even occurs.[50] Information from here projects to the multisensory region of the thalamus, namely the pulvinar. This complex of nuclei has extensive connections to cortical areas, see Figure 6.

Correspondingly, several cortical areas contribute to multisensory integration. For instance, Cappe, Morel, Barone, and Rouiller explain that the premotor cortex is a polymodal integration area, with convergence of visual, auditory, and somatosensory inputs, creating intention used for preparation of voluntary movements. Extensive multisensory interplay also occurs from many other cortical sources of inputs, such as multisensory parietal areas.[51] The success of these cortical influences on perception relies on efficient multisensory convergence and integration that originates in the thalamus and brainstem. Any or all of these pathways are susceptible to the diffuse damage a concussion can cause.

The patients in this study received a bottom-up to top-down protocol that is foundationally built on early efforts to establish enhanced multisensory processing. This author refers to it as optometric multisensory training (OMST). It involves simultaneous application of optometric phototherapy, vestibular stimulation, auditory stimulation, proprioceptive input as needed, and gradually introduced and applied oculomotor therapy as tolerated. This multisensory training encourages the neural activity of one sensory input to influence that of others. It spreads the therapeutic effect amongst several sensory systems creating opportunity for the stronger systems to support the weaker systems until

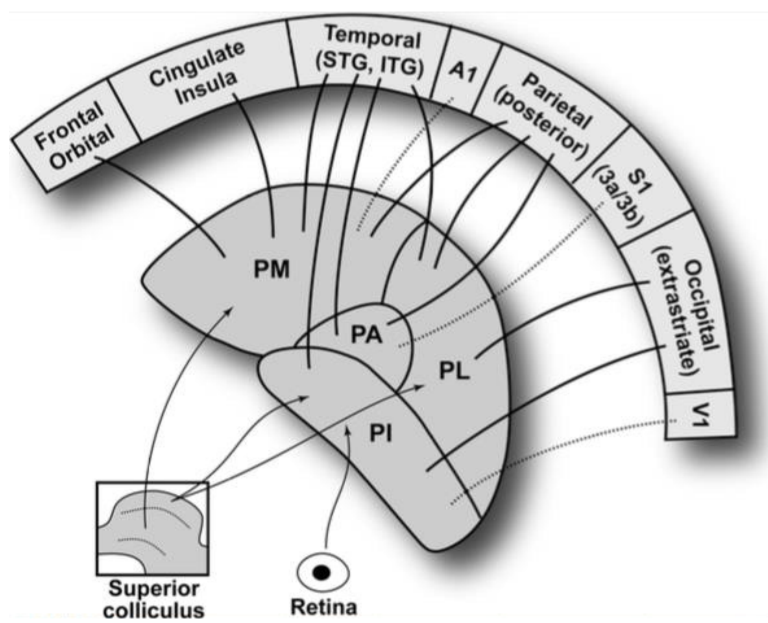


Figure 6: A topographical representation of connections to cortical areas from subcortical inputs (pulvinar of the thalamus) arising from multiple sensory inputs that the superior colliculus receives, including non-lateral geniculate vision paths. The retina component in the figure is small representing the retinal ganglion cell input that directly goes to the LGN. *Stein, Barry E., The New Handbook of Multisensory Processing, figure 3.2, page 53, copyright 2012 Massachusetts Institute of Technology, by permission of The MIT Press.*

all reach the balanced and synergistic status that ideally existed before the brain injury. This study supports that this bottom-up to top-down neuro-optometric rehabilitation approach using OMST provided patients with relatively quick and comprehensive gains. It therefore prepared them for higher complex neuro-optometric rehabilitation activities that would follow in a more traditional clinical manner.

This author hypothesizes that the primary catalyst for the efficacious improvements that this protocol provides is the influence of the optometric phototherapy on the multisensory processing. Retinal ganglion cells (RGC) are the primary driver of the LGN of the thalamus.[52] And recent studies of mammalian retina circuitry confirms that a number of RGC types innervate the superior colliculus independent of these thalamic tracts.[53] Therefore, optometric phototherapy naturally has a great effect on both thalamic and superior colliculus activity. As a result, the optometric phototherapy energizes transmission along visual-based pathways at the same time that energized oculomotor, auditory, and vestibular inputs arrive. The optometric phototherapy also likely improves the flow of neural energy through the magnocellular pathway and dorsal stream enhancing parietal lobe function. This poly-regional access of the optometric phototherapy potentiates improved spatial awareness of and anchoring in the environment. This subsequently prepares the brain to attend to cortical visual processing and thereby receive top-down therapy.

Moreover, as the multisensory networks of the brain repetitively receive this energized information while simultaneously receiving the oculomotor and vestibular input, the brain has an opportunity to be retrained in the modulation and integration of multiple sensory signals while doing this in a safe and controlled clinical situation. This provides a foundation for development of more accurate and efficient production of motor output including posture, balance,[54] and eye movements thereby reducing related PCS symptoms, both oculomotor-based and non-oculomotor-based.

CONCLUSION

This case series presents a paradigm for neuro-optometric rehabilitation when NOM vision dysfunctions are included in the PCS patient presentation; the initial phase of therapy is heavily weighted with a multisensory based bottom-up technique. It is accompanied by a gradual addition of top-down based techniques as tolerated such as oculomotor, accommodative, and vergence activities. There is an initial emphasis on rehabilitation of pre-attentive multisensory processing to better prepare the patient for optimal engagement in attentive output activities. The results indicate that this paradigm provided the PCS patients broad resolution of symptoms in a relatively short period of time.

The majority of the patients in this study continued further neuro-optometric rehabilitation after the 38 days that shifted into a more traditional approach (weekly one-hour visits) with top-down emphasis yet further bottom-up support as needed such as yoked prism, binasal occlusion, filters, plus-lens therapy, isolated optometric phototherapy, and if indicated, new spectacle lenses. This author frequently delays prescribing of glasses for PCS patients who present with NOM based vision issues until the 38 day follow up evaluation. This is because they are often too sensory "fragile" to refract reliably and adapt easily to the refractive change initially. Moreover, blurred vision is the lowest rated presenting symptom on the SCAT5 symptom survey in this study. See Table 2. Further noteworthy is the significant improvement in blurred vision at the 38 day follow up likely due to the improved visual-vestibular integration achieved from this paradigm. Additionally, determination of the appropriate lens filter/tint for photophobia to be included is more accurate at the 38 day follow up due

Table 2. Self-rating scores (average of the group) 1 week prior and at 38-day follow up in order of highest to lowest severity rating. Note: blurred vision is not a highly rated symptom, therefore a general eye exam may not be adequate for the PCS patient population. Note: blurred vision symptom improved 41% in absence of prescribing spectacle lens changes.

Symptom	Pre-treatment self rating	38-day self rating	Percent change
1: Don't Feel Right	3.7	1.6	57
2: Fatigued or low energy	3.6	1.8	50
3: Feeling slowed down	3.4	1.4	59
4: Feeling in a fog	3.3	1.6	52
5: Difficulty Concentrating	3.1	1.4	55
6: Nervous or Anxious	3.0	1.6	47
7: More Emotional	2.9	1.4	52
8: Sensitivity to the Light	2.9	1.5	48
9: Difficulty Remembering	2.9	1.6	45
10: Sensitivity to Noise	2.8	1.4	50
11: Irritability	2.7	1.4	48
12: Trouble Falling Asleep	2.6	1.5	42
13: Drowsiness	2.6	1.3	50
14: Sadness	2.5	1.2	52
15: Pressure in the head	2.5	1.4	44
16: Headache	2.5	1.3	48
17: Neck Pain	2.3	1.6	30
18: Balance Problems	2.2	1.0	55
19: Confusion	2.1	0.8	62
20: Dizziness	1.9	0.9	53
21: Blurred Vision	1.7	1.0	41
22: Nausea or Vomiting	1.1	0.4	64

to change in status of photophobia that often occurs from the OMST.

This case series study substantiates pursuit of higher evidence-based, prospective studies. Future studies should include follow up for several months to assess whether the reported improvements were retained by the patient. However, an advantage of reporting clinical changes in only 38 days is that it lessens the argument that spontaneous recovery was heavily involved.

Future studies should include additional optometric clinical measures that have been supported in literature for post-concussion monitoring. For example, this author finds value in utilizing the Distance Fusional Facility Test, developed by Barry Tannen, OD,[55] and the King Devick Test at the initial evaluation, but due to time constraints at the 38-day follow up, often defers retesting of these until the three month follow up evaluation.

Finally, results of this study indicate that neuro-optometric rehabilitation using this paradigm is effective for PCS patients regardless of whether or not the patient has already received PT. Therefore, physicians who manage PCS patients should be encouraged to also include referral for neuro-optometric rehabilitation in their patient plans. Future studies comparing outcomes between PCS groups receiving neuro-optometric rehabilitation versus physical therapy, versus both, would be informative for future patient care.

DISCLOSURE OF FUNDING

The author has contributed to upgrading the optometric phototherapy instruments utilized in the equipment described in this study.

REFERENCES

1. American Association of Neurological Surgeons.org. Illinois: Concussion; cited 2019 September 15. Available from: http://bit.ly/34Ykxeu
2. Goodrich GL, Kirby J, Cockerham G, Ingalla S. P., Lew H. L. Visual function in patients of a polytrauma rehabilitation center: a descriptive study. J Rehabil Res Dev 2007; 44: 929-936. doi:10.1682/jrrd.2007.01.0003
3. Langlois J, Rutland-Brown W, Waldo M. The epidemiology and impact of traumatic brain injury: a brief overview. J Head Trauma Rehabil. 2006;21(5):375–378.
4. Center for Disease Control and Prevention, National Center for Health Science. National Concussion Surveillance System. Cited 2019 September 15. Available from: http://bit.ly/2rX67gd.
5. Hoge CW, McGurk D,Thomas JL,CoxAL, Engel CC, Castro CA. Mild traumatic brain injury in U.S. soldiers returning from Iraq. N Engl J Med. 2008;358(5):453-463. doi:10.1056/NEJMoa072972
6. Jaffee MS, Winter WC, Jones CC, Ling G. Sleep disturbances in athletic concussion. Brain Inj. 2015; 29(2):221-227. doi:10.3109/02699052.2014.983978
7. Master CL, Maye rAR, Quinn D, Grady MF. Concussion. Ann Intern Med. 2018;169(1):ITC1-ITC16. doi:10.7326/AITC201807030

8. Carroll LJ, Cassidy JD, Cancelliere C, et al. Systematic review of the prognosis after mild traumatic brain injury in adults: cognitive, psychiatric, and mortality outcomes: results of the International Collaboration on Mild Traumatic Brain Injury Prognosis. Arch Phys Med Rehabil. 2014;95 (3)(suppl):S152-S173. doi:10.1016/j.apmr.2013.08.300

9. McInnes K, Friesen CL, MacKenzie DE, Westwood DA, Boe SG. Mild traumatic brain injury (mTBI) and chronic cognitive impairment: a scoping review. PLoS One. 2017;12(4):e0174847. doi:10.1371/journal.pone.0174847

10. Dean PJA, Sato JR, Vieira G, et al. Long-term structural changes after mTBI and their relation to post-concussion symptoms, Brain Injury 2015 Sep 29(10):1211-1218, doi:10.3109/02699052.2015.1035334

11. Guskiewicz KM, McCrea M, Marshall SW, et al. Cumulative Effects Associated With Recurrent Concussion in Collegiate Football Players: The NCAA Concussion Study. JAMA. 2003;290(19):2549–2555. doi:10.1001/jama.290.19.2549

12. Bigler ED. Neuropsychology and clinical neuroscience of persistent post-concussive syndrome. Journal of Intemational Neuropsychological Society 14(1): 1-22. http://bit.ly/35sp9tQ

13. Alves W, Macciocchi S, Barth J; Postconcussive symptoms after uncomplicated mild head i~ury. J Head Trauma Rehabilitation 1993;8(3): 48-59.

14. Vander Naalt J, Timmerman ME, de Koning ME, et al. Early predictors of outcome after mild traumatic brain injury (UPFRONT): an observational cohort study. Lancet Neurol2017;16(7):532-540. doi:10.1016/S1474-4422(17)30117-5

15. Zemek R, Barrowman N, Freedman SB, et al. Clinical Risk Score for Persistent Postconcussion Symptoms Among Children With Acute Concussion in the ED. JAMA. 2016;315(10):1014–1025. doi:10.1001/jama.2016.1203

16. Master CL, Master SR, Wiebe DJ, Storey EP, Lockyer JE, Podolak OE, and Grady MF. Vision and vestibular system dysfunction predicts prolonged concussion recovery in children. Clin J Sport Med 2018; 28 (2): 139-145.

17. Ellis M, Cordingley D, Vis S, Reimer K, Leiter J, Russell K. Clinical predictors ofvestibulo-ocular dysfunction in pediatric sports-related concussion. J Neurosurg Pediatr 20 17; Jan 19 (1):38-45.

18. Mittenberg W, Canyock E, Condit D, Patton C. Treatment of post-concussion syndrome following mild head injury. J Clin Exp Neuropsychol. 2001;23(6):829-836.

19. Wade D, King N, Wenden F, Crawford S, Caldwell F. Routine Follow Up After Head Injury: a Second Randomized Controlled Trial. J Neurol Neurosurgery Psychiatry. 1998; 65 (2): 177-183.

20. Al Sayegh A, Sandford D, Carson AJ. Psychological approaches to treatment of postconcussion syndrome: a systematic review. J Neurol Neurosurg Psychiatry. 2010;81(10):1128-1134.

21. Leddy J, Kozlowski K, Donnelly J, Pendergast D, Epstein L, Willer B. A Preliminary Study of Subsystem Threshold Exercise Training for Refractory Post-concussion Syndrome. Clin J Sport Med. 2010;20(1):21-27.

22. Brosseau-Lachaine O, Gagnon I, Forget R, Faubert J. Mild traumatic brain injury induces prolonged visual processing deficits in children. Brain Inj. 2008;22(9):657–668.

23. Gallaway M, Scheiman M, Mitchell GL. Vision Therapy for Post-Concussion Vision Disorders. Optom Vis Sci. 2017 Jan;94(1):68-73.

24. Evans RW. Persistent post-traumatic headache, postconcussion syndrome, and whiplash injuries: the evidence for a non-traumatic basis with an historical review. Headache. (2010) 50:716–24. doi:10.1111/j.1526-4610.2010.01645.x

25. Department of Health and Human Services. Washington, DC. Guidance Regarding Methods for De-identification of Protected Health Information in Accordance with the Health Insurance Portability and Accountability Act Privacy Rule. Available from: http://bit.ly/36cLImm.

26. Purves D, Augustine G, Fitzpatrick D, et al., editors. Neuroscience. 2nd edition. Sunderland (MA): Sinauer Associates; 2001.

27. Gottlieb R, Wallace L. Syntonic Phototherapy; Photomedicine and Laser Surgery, vol 28 #4, 2010; copyright Mary Ann Liebert, Inc

28. Wallace L. Syntonics: Optometric Color Therapy for the Treatment of Acquired Brain Injuries; Journal of Optometric Phototherapy, April 2001

29. Azeemi STY, Raza SM. A Critical Analysis of Chromotherapy and Its Scientific Evolution. Evidence-based Complementary and Alternative Medicine. 2005;2(4):481-488. doi:10.1093/ecam/neh137.

30. Suchoffl, Ciuffreda K, Kapoor N. Visual & Vestibular Consequences of Acquired Brain Injury. Optometric Extension Program Foundation; 2001.

31. Yadav N, Thiagarajan P, Ciuffreda K. Effect of Oculomotor Vision Rehabilitation on Visual Evoked Potential; Brain Inj. 2014;28(7):922-9. doi:10.3109/02699052.2014.887227. Epub 2014 Feb 24.

32. British Journal of Sports Medicine.com: first published on 26 April 2017; cited 2019 September 14. Available from: http://bjsm.bmj.com

33. Jacobson GP, Newman CW. The Development of the Dizziness Handicap Inventory. Arch Otolaryngol Head Neck Surg. (1990) 116:424–7. doi:10.1001/archotol.1990.01870040046011

34. Whitney SL, Wrisley DM, Brown KE, et al. Is perception of handicap related to functional performance in persons with vestibular dysfunction. OtolNeurotol. 2004;25(2):139.

35. José E Capó-Aponte, Thomas A Beltran, David V Walsh, Wesley R Cole, Joseph Y Dumayas, Validation of Visual Objective Biomarkers for Acute Concussion, Military Medicine, Volume 183, Issue suppl_1, March-April 2018, Pages 9-17.

36. Ciuffreda K, Ludlam D. Conceptual Model of Optometric Vision Care in Mild Traumatic Brain Injury. J Behav Optom 22;10-12.
37. D'Angelo M, Tannen B. The optometric care of vision problems after concussion: A clinical guide. Optom Vis Perf 2015; 3(6):298-306.
38. Taylor A, Goehler L, Galper D, et al. Top-Down and Bottom-Up Mechanisms in Mind-Body Medicine: Development of an Integrative Framework for Psychophysiological Research Explore: The Journal of Science and Healing , Volume 6 , Issue 1, 29-41.
39. de Gelder, B., & Bertelson, P. (2003). Multisensory integration, perception and ecological validity. Trends in Cognitive Sciences, 7, 460-467.
40. Wikipedia contributors. Pre-attentive processing. Wikipedia, The Free Encyclopedia. May 21, 2019, 15:24 UTC. Available at: http://bit.ly/2sSKnTd. Accessed June 20, 2019.
41. Serences J., Boynton G. Feature-based attentional modulations in the absence of direct visual stimulation. Neuron. 2007;55:301–312. doi:10.1016/j.neuron.2007.06.015.
42. Senkowski D, Talsma D, Herrmann CS, Woldorff MG. Multisensory processing and oscillatory gamma responses: effects of spatial selective attention. Exp. Brain Res. 2005;166:411–426. doi:10.1007/s00221-005-2381-z.
43. Chang A, Cohen A, Kapoor N. Top-down visual framework for optometric vision therapy for those with traumatic brain injury. Optom Vis Perf 2013;1(2):48-53.
44. Peachey GT, Peachey P. Optometric vision therapy for visual deficits and dysfunctions: A suggested model for evidence-based practice. Vision Dev & Rehab 2015;1(4):290-336.
45. Stein B, The New Handbook of Multisensory Processing. p xiii. Cambridge, MA; MIT Press. 2012
46. Stein BE, Stanford TR, Rowland BA. Development of multisensory integration from the perspective of the individual neuron. Nat Rev Neurosci. 2014;15(8):520–535.
47. Calvert G, Spence C, Stein B. The Handbook of Multisensory Processes. Cambridge, MA: MIT Press; 2004.
48. Stein B, The New Handbook of Multisensory Processing. p 49. Cambridge, MA; MIT Press. 2012
49. McHaffie, John & Fuentes-Santamaria, Veronica & Alvarado, Juan & Fuentes-Farías, AL & Gutiérrez-Ospina, Gabriel & Stein, BE. (2012). Anatomical features of the intrinsic circuitry underlying multisensory integration in the superior colliculus.
50. Stein B, Stanford T, Rowland B. Development of multisensory integration from the perspective of the individual neuron. Nature reviews Neuroscience. 2014;15(8): p523.
51. Cappe C, Morel A, Barone P, Rouiller EM. The thalamocortical projection systems in primate: an anatomical support for multisensory and sensorimotor interplay. Cereb Cortex. 2009;19(9):2025–2037. doi:10.1093/cercor/bhn228
52. Usrey WM, Alitto HJ. Visual Functions of the Thalamus. Annu Rev Vis Sci. 2015;1:351–371. doi:10.1146/annurev-vision-082114-035920
53. Ellis EM, Gauvain G, Sivyer B, Murphy GJ. Shared and distinct retinal input to the mouse superior colliculus and dorsal lateral geniculate nucleus. J Neurophysiol. 2016;116(2):602–610. doi:10.1152/jn.00227.2016
54. Curtis J, Association of Functional Color Field Changes with Imbalance, Spatial Misjudgment, and Nausea in the Treatment of Post-Concussion Syndrome Patients. J Optom Phototherapy, April 2018, 14-19.
55. Tannen B, Rogers J, Ciuffreda K, Lyon E, Shelley- Tremblay J. Distance horizontal fusional facility (DFF): A proposed new diagnostic test for concussion patients. Vision Dev & Rehab 2016;2(3):170-75

AFFILIATIONS

Riverview Eye Associates, Ohio Health Rehabilitation Hospital, Columbus Crew SC.

CORRESPONDING AUTHOR BIOGRAPHY:

Steven J. Curtis OD, FCOVD, FNORA
Columbus, Ohio

Dr. Steven J. Curtis is in private practice in Columbus Ohio where he provides general, developmental, and neuro-optometric vision services. He is also on the medical staff of the OhioHealth Rehabilitation Hospital and the professional soccer team, Columbus Crew. Dr. Curtis is a fellow of COVD and NORA. He lectures to area physicians and therapists on neuro-optometric involvement in rehabilitation. Dr. Curtis works extensively with patients who suffer persistent vision disturbances after concussion.

ARTICLE

Pediatric Visual Snow Syndrome (VSS): A Case Series

Kenneth J. Ciuffreda, OD, PhD[1]
M.H. Esther Han, OD
Barry Tannen, OD
[1]*SUNY/College of Optometry*
Brain Injury Clinical Research Unit
New York, New York

ABSTRACT

Visual snow syndrome (VSS) is a relatively rare, unusual, and disturbing abnormal visual condition. The individual perceives "visual snow" (VS) throughout the entire visual field, as well as other abnormal visual phenomena (e.g., photopsia). Only relatively recently has treatment been proposed (e.g., chromatic filters) in adults with VSS, but rarely in the pediatric VSS population (i.e., medications). In this paper, we present three well-documented cases of VSS in children, including their successful neuro-optometric therapeutic interventions (i.e., chromatic filters and saccadic-based vision therapy).

Correspondence regarding this article should be emailed to Kenneth J. Ciuffreda, OD, PhD, at kciuffreda@sunyopt.edu. *https://doi.org/10.31707/VDR2019.5.4.p249.*

Ciuffreda KJ, Han MH, Tannen B. Pediatric Visual Snow Syndrome (VSS): A case series. Vision Dev & Rehab 2019;5(4):249-54.

Keywords: neuro-optometry, vision, visual intervention, visual perception, visual snow, visual snow syndrome

INTRODUCTION

The clinical condition of "visual snow syndrome" (VSS) is reported to be a relatively rare and unique, cortically-based entity primarily described in the medical literature.[1-15] Its hallmark characterization is the perception of "visual snow" (VS) appearing in a single plane in front of and throughout the entire visual field (Figure 1). VS has been described by some as appearing like the electronic "snow" on a television set when the primary video signal is either reduced or absent, as well as pixelated fuzz and bubbles.[1-15]

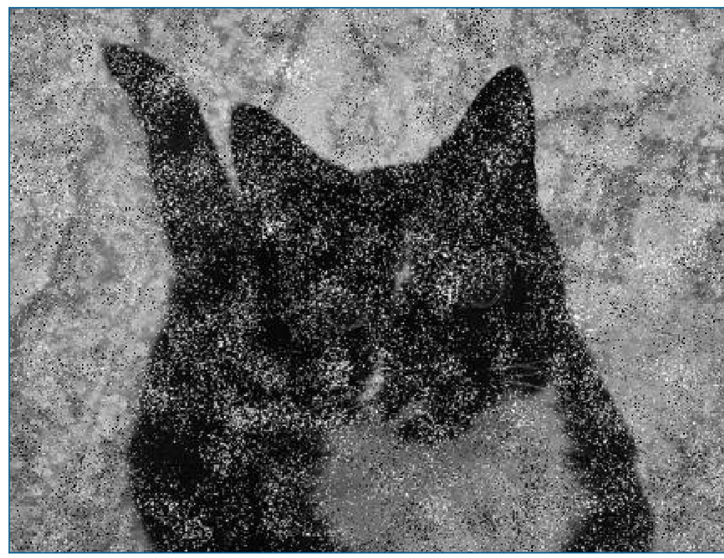


Figure 1: Cat with overlay of visual snow as depicted by a patient.

In addition to the presence of VS, and based on currently evolving criteria,[2-15] the individual must also report two or more of the following to be formally diagnosed with VSS: palinopsia, enhanced entoptic imagery, "nyctalopia", and photophobia/photosensitivity. These individuals also frequently report having migraine, photopsia, phonophobia, hyperacusis, cutaneous allodynia, tinnitus, balance problems, and tremor.[2-15]

Due to its presumed extremely low prevalence, there is a relative paucity of reports in the literature on VSS in adults dating back to 1944,[1-12] and only three case reports in children and adolescents ages 11-14 years over the past few years.[13-15] In

Table 1. Case Summaries

	Case 1	Case 2	Case 3
DEMOGRAPHICS			
Current age (in years)	10	10	15
Gender (M,F)	M	F	F
Age first noticed snow (in years)	10	8	11
Is visual snow constant (C) or transient (T)?	C	C	T
Is visual snow chromatic (C) or monochromatic (M)?	C, M	C, M	C
Provocative environments?	Close objects	None	Bright lights; Computers
Possible etiology?	Pneumonia	Brain cyst removal	Migraine
Possible co-morbid conditions?	None	None	Migraine
Treatment(s)?	Tint	Tint	Tint & VT
Medications?	None	Nortriptyline	None

PRIMARY VISUAL SYMPTOMS			
Palinopsia		√	√
Enhanced entoptic imagery	√		√
Photosensitivity	√	√	√
"Nyctalopia" (impaired night vision)			

SECONDARY VISUAL/NONPVISUAL SYMPTOMS			
Photopsia		√	√
Migraine			√
Phonophobia			
Hyperacusis			
Cutaneous allodynia	√		
Tinnitus			
Balance problems	√	√	
Tremor			

these three cases, all conventional medical diagnostic testing was negative (e.g., brain imaging[15]), and all attempted drug treatments (e.g., sumatriptan[15]) were unsuccessful. None of the reports in the pediatric literature noted any successful treatments such as chromatic filters or vision therapy. Neither were there any successful medications directed solely at VS, but rather only medications toward migraine symptoms or other coincident symptoms.

Thus, we present a neuro-optometrically-based, pediatric case series (ages 10-15 years) of three patients with well-documented VSS. This report includes both valuable diagnostic and therapeutic aspects for the clinician.

CASE PRESENTATIONS

All received a comprehensive optometric vision examination, including refractive, binocular, and ocular health status. The case descriptions will be restricted to the relevant VSS details, as well as the other primary visual diagnoses. The key VSS findings are presented in Table 1 for each patient.[12]

Case One

The patient was a 10-year-old male who first noticed VS soon after an extended bout (~4 weeks) of pneumonia for which he was successfully treated five months prior to his sensorimotor evaluation. The perception of VS was constant and typically monochromatic in nature. His primary VSS-related symptoms included photosensitivity and enhanced entoptic imagery, whereas his secondary VSS-related symptoms included balance problems and cutaneous allodynia. In addition, his other symptoms included intermittent diplopia at near, dizziness, positional vertigo, and general fatigue. Interestingly, all of these symptoms worsened following remission of the pneumonia, and thus he was referred for additional medical care. The MRI and ENT results were normal. Moreover, the Lyme titer was clear. There was no history of head trauma. He was not taking any medications.

The patient was then referred by a local optometrist to the SUNY/University Eye Center for a vision therapy evaluation. He was diagnosed with low hyperopia, binocular instability, and a receded near point of convergence, and he is scheduled for vision therapy in the near future. At the same time, he was referred for a Cerium-based (Intuitive Colorimeter), colorimetric examination.[16] He preferred a 40% BPI-IR blue tint, which was prescribed in spectacle form (CR-39). This tint reduced most of his visual symptoms, especially the VS and photosensitivity, thus resulting in overall "improved visual comfort". Lastly, he had a neutral response to a session of optometric phototherapy (i.e., syntonics),[17] so it was not pursued at this time.

Case Two

The patient was a 10-year-old female who first noticed VS within a day following surgical removal of a brain cyst two years prior to her optometric evaluation. The cyst was first discovered at age 3 years. The perception of VS was constant and typically monochromatic, but also purple with a green background at times (e.g., after yawning). Her primary VSS-related visual symptoms included photosensitivity and palinopsia (especially when reading), whereas her secondary VSS-related symptoms included photopsia and balance problems. In addition, her other symptoms included blurred vision, headaches, and loss of place, all when reading, as well as the sensation of dizziness and not feeling "grounded". Prior visual fields and dilated fundus examinations by two ophthalmologists were normal. She was taking nortriptyline (25 mg, 3 times per day) for depression and leg pain.

The patient was referred by her neuro-ophthalmologist to the SUNY/University Eye Center, Vision Rehabilitation Service specifically for a Cerium colorimetric evaluation. The basic vision examination revealed esophoria at distance, low hyperopia, and saccadic dysfunction, for which she received successful oculomotor-based vision therapy; that is, symptoms reduced, and signs improved. No refractive correction was prescribed at that time. Then, the Cerium-based (Intuitive Colorimeter),[16] colorimetric testing was performed. She required two different spectacle tints. One was for lessening the VS, which reduced it by approximately 75% (pink-purple, hue = 320, saturation = 25, Purple A6, Purple Rose A6, Rose C4, 45% overall transmission). The other was for reducing the palinopsia duration when reading, which decreased the visual persistence from 17 seconds to 5 seconds, and thus it improved her reading comfort (turquoise/green, hue=180, saturation=25, turquoise C3, green C3, 47% overall transmission).

Case Three

The patient was a 15-year-old female who first noticed VS at the age of 11 years, after experiencing her first ocular migraine attack. The perception of VS was transient and chromatic. It could be precipitated by bright lights, fatigue, and computer screen viewing. Her primary VSS-related symptoms included palinopsia with trailing a few times per day, enhanced chromatic entoptic imagery once per day, and constant photosensitivity, whereas her VSS-related secondary symptom was intermittent photopsia. The VS and palinopsia occurred independently. She also reported two other interesting perceptual phenomena. First, at times, hallways were perceived to be longer, and objects appeared to be either closer (i.e., pelopsia) or farther (i.e., teleopsia) than their physical presence, with such size and distance distortions suggesting Alice in Wonderland Syndrome.[18] Second, at times, she noticed a strobe/pulse effect surrounding letters during reading. She had no history of concussion and was not taking any medications.

The patient was self-referred to a private practice, neuro-optometrist for a comprehensive vision evaluation. She was diagnosed with accommodative insufficiency, convergence excess, saccadic oculomotor dysfunction, visual-directional problems, and visual-memory deficits. She was successfully treated with oculomotor-based vision therapy and visual perceptual therapy; that is, symptoms reduced, and signs improved. The saccadic therapy reduced the palinopsia persistence and related trailing,[12,19] presumably by reestablishing a normal saccadic suppression inhibitory level.[12] In addition, an FL-41 tint was prescribed in spectacle form, which reduced the perception of VS.

DISCUSSION

VSS represents a new and exciting challenge to the neuro-optometrist, and others (e.g., the ophthalmologist, neurologist). This syndrome has been a long misunderstood,[9,18] somewhat perplexing, unique, and even questioned (especially in young children) diagnosis among the medical community, at least since 1944 when visual snow was reported by some adult patients taking digitalis for heart problems.[1]

The neuro-optometrist has expertise in general vision function/dysfunction, with specialty knowledge in optics (e.g., tint spectra, lens design), human visual perception (e.g., motion, saccadic suppression, reading), and vision therapy/neurorehabilitation (e.g., perceptual and motor learning, oculomotor control). Thus, the neuro-optometrist can provide the comprehensive, basic vision examination (i.e., refractive binocular, and ocular health), as well as the vision therapy, and also both the general (e.g., neutral gray) and more advanced (e.g., FL-41, BPI Omega) tint prescribing. In addition, an ever increasing number of optometrists have knowledge and experience in specialized colorimetric testing (i.e., Cerium Intuitive Colorimeter)[16] to prescribe the optimal filter combination for maximum visual comfort and benefit (e.g., reading efficiency) in the patient with VSS. This would especially include the visual symptoms of VS, palinopsia, and photosensitivity/photophobia. Lastly, others may be assistive in the process, such as the occupational therapist and ophthalmologist, to assess vision and related gains with the therapeutic lenses within the context of their own discipline.

Treatment for VSS is important for all individuals, but perhaps even more so in young children/adolescents, for several reasons. First, there is the possible psychological effect of having such unusual and abnormal, undiagnosed, visual percepts. Some adult patients with VSS indicated their initial childhood reservation about telling this to a friend or family member, for fear of disbelief and even possible ridicule. Therefore, a detailed, probing case history may be required to elicit the presence of VS in some patients, especially in those conditions with a relatively high probability of occurrence such as concussion, brain surgery, and taking a new medication.[12] Second, those manifesting palinopsia, which seems to be the majority,[12] frequently find presence of their sustained and superimposed afterimage, especially with trailing, to be both distracting and disruptive when reading, as expected, thus likely leading to reduced reading rate, reduced visual comfort, and poorer comprehension. And lastly, as they become of driving age, the presence of VSS, palinopsia, and/or enhanced entoptic imagery, especially at night, may impact adversely on their driving ability and safety due to impaired visual perception and attentional distraction. If so, they should be advised to obtain counselling from a professional driving trainer with expertise in those having visual impairment.[20]

In an earlier paper on this topic,[12] we had proposed a "unifying hypothesis", or mechanism, to understand many/all aspects of VSS, namely neural "disinhibition". Others had proposed the mechanism to be a "hypersensitivity phenomenon".[8] At first blush, this appeared to be descriptive. While not necessarily incorrect, it seems to emphasize the wrong aspect. These patients are not hypersensitive per se, but rather appear to exhibit abnormally-reduced levels of naturally-occurring neural inhibition/have less suppression. This idea is supported by recent laboratory findings.[21,22] Thus, the patient with VSS sees/experiences what others do not see (e.g., palinopsia) or feel (e.g., cutaneous allodynia).

In the future, a randomized clinical trial (RCT) is warranted to assess efficacy of the two current neuro-optometric, therapeutic interventions, namely tinted spectacles and oculomotor-based vision therapy, separately and combined, in conjunction with a placebo condition.[23] However, a cross-over, interventional experimental design would also be both acceptable and more efficient.[21] If proven to provide symptomatic relief (e.g., per the VSS questionnaire)[12] and/or increased visual/reading efficiency (e.g., per the objective Visagraph reading testing),[24] additional studies would be helpful to determine retention of the long-term benefits and any visual adaptation effects.

REFERENCES

1. Carroll F. Visual symptoms caused by digitalis. Trans Amer Ophthal Soc. 1944; 42:243-249.
2. Puledda F, Schankin C, Digre K, Goadsby PJ. Visual snow syndrome: what we know so far. Curr Opin Neurol 2018; 31: 52-58.
3. White OB, Clough M, McKendrick AM, Fielding J. Visual snow: visual misperception. J Neuro-Ophthalmol 2018; 38: 514-521.
4. Schankin C, Maniyar FH, Digre KB, Goadsly PJ. "Visual snow"- a disorder distinct from persistent migraine aura. Brain 2014; 137: 1419-1428.
5. Liu GT, Svatz NJ, Galetta SL, Volpe NJ, Skobieranda F, Komorsky GS. Persistent positive visual phenomena in migraine. Neurol 1995; 45: 664-668.
6. Santos-Bueso E, Sastre-Ibanez M, Saenz-Frances F et al. Visual snow: from a symptom to a syndrome? Arch Soc Esp Oftalmol 2015; 9:47-52.
7. Fraser CL, White OB. There's something in the air. Sur Ophthal 2018; 63: 1-5.
8. Ghannam B, Pelak VS. Visual snow: a potential cortical hyperexcitability syndrome. Curr Treat Options Neurol 2017; 19: 8-20.
9. Schankin CJ, Maniyar FH, Sprenger T, et al. The relation between migraine, typical migraine aura, and "visual snow". Headache 2014; 54: 957-966.
10. Laushke JL, Plant GL, Fraser CL. Visual snow: a thalamocortical dysrhythmia of the visual pathway? J Clin Neurosci 2016; 28: 123-127.
11. Metzler AI, Robertson CE. Visual snow syndrome: proposed criteria, clinical implications, and pathophysiology. Curr Neurol Neurosci Rep 2018; 18:52. https://doi.org/10.1007/s11910-018-0854-2
12. Ciuffreda KJ, Tannen B, Han ME. Visual snow syndrome (VSS): an evolving neuro-optometric perspective. Vis Dev Rehab 2019; 5: 75-82.
13. Bruen R, Peng SL, Perreault S, Major P, Ospina LH. Persistent migraine aura in an adolescent girl. J AAPOS 2013; 17: 426-427.
14. Santos-Bueso E, Munoz-Hernandez N, Avalos-Franco N, Garcia-Saenz S, Saenz-Frances F. Porta-Etessom J. Visual snow in a pediatric patient. Arch Soc Esp Oftalmol. 2017; 92: 602-604.
15. Simpson JC, Goadsby PJ, Prabhakar P. Positive, persistent visual symptoms (visual snow) presenting as a migraine variant in a 12-year-old girl. Pediatr Neurol 2013; 49:361-363.
16. Willeford KT, Fimreite V, Ciuffreda KJ. The effect of spectral filters on VEP and alpha-wave responses. J Optom 2016; 9:110-117.
17. Collier S. In syntony. Herstellung and Verlag, Norderstedr, Germany, 2011.
18. Rastogi RG, VanderPluym J, Lewis KS. Migrainous aura, visual snow, and "Alice in Wonderland" Syndrome in childhood. Sem Pediatr Neurol 2016; 23:14-17.
19. Ciuffreda KJ, Ludlam DP, Kapoor N. Clinical oculomotor training in traumatic brain injury. Optom Vis Dev 2009; 40: 16-23.
20. Gianutsos R, Campbell A, Beattie A, Mandriota F. The driving advisement system: a computer augmented quasi-simulation of the cognitive prerequisites for resumption of driving after brain injury. Assistive Tech. 1992; 4: 70-86.
21. Mc Kendrick AM, Chan YM, Tien M, et al. Behavioral measures of cortical hyperexcitability assessed in people who experience visual snow. Neurol 2017; 88; 1243-1249.
22. Yildiz FG, Turkyilmaz U, Unal-Cevik I. The clinical characteristics and neurophysiological assessments of the occipital cortex in visual snow syndrome with or without migraine. Headache 2019;59(4):484-494.
23. Hatch SW. Ophthalmic research and epidemiology. Butterworth-Heinemann, Boston MA, 1998, pp. 61-110.
24. Ciuffreda KJ, Tannen B. Eye movement basics for the clinician. St. Louis: Mosby Year Book, 1995.

CORRESPONDING AUTHOR BIOGRAPHY:

Kenneth J. Ciuffreda, OD, PhD
New York, New York

Kenneth J. Ciuffreda received his B.S in biology from Seton Hall University in 1969, his O.D. from the Massachusetts College of Optometry in 1973, and his Ph.D. degree in physiological optics from the University of California/School Optometry at Berkeley in 1977. He has been a faculty member at the SUNY/State College of Optometry in New York City since 1979, where he is presently a Distinguished Teaching Professor. He has also had adjunct appointments for many years at Rutgers/The State University of New Jersey, as well as at the New Jersey Institute of Technology, both in the department of biomedical engineering. He also helped establish a school of optometry in Harbin, China. He has conducted research in many areas: amblyopia, strabismus, reading, myopia, eye movements, accommodation, bioengineering applications to optometry, and more recently with an emphasis in the area of acquired brain injury, both the diagnostic and therapeutic aspects. His goal has been the use of objective recording techniques in the diagnosis and treatment of neurological and ocular conditions. He holds two patents, and has received many awards and honors from the AAO, AOA, NORA, COVD, and various state optometric associations and colleges. He has authored over 400 research papers/chapters, and 10 books. His hobbies are playing jazz guitar and enjoying the visual aspects of art.

ARTICLE

Incorporating Behavior Modifications, Strategies, and Supports to Maximize the Effectiveness of Vision Therapy in the Autism Spectrum Disorder Population

Jennifer Fisher, OD, FAAO
Assistant Clinical Professor
UC Berkeley School of Optometry
Berkeley, California

Tyler Phan, OD, FAAO
Director of Vision Therapy
Union Community Health Center

SBH Health System,
Department of Ophthalmology
Bronx, New York

ABSTRACT

The prevalence of autism spectrum disorders has increased almost two-fold since 2007. This increase has facilitated a need for a new approach in vision therapy when treating deficits in accommodation, binocularity, oculomotor, and visual processing in children with autism. The clinician may face challenges when treating this population because children with autism may have difficulty engaging in reciprocal conversation, have poor attention due to sensory overstimulation, be resistant to change, or be non-verbal. This paper seeks to compile effective behavior modifications, strategies, and supports used by other professionals, such as teachers, and physical, occupational, and behavioral therapists, that may be incorporated into optometric vision therapy to maximize visual outcomes for this patient population.

Fisher J, Phan T. Incorporating Behavior Modifications, Strategies, and Supports to Maximize the Effectiveness of Vision Therapy in the Autism Spectrum Disorder Population. Vision Dev & Rehab 2019;5(4):255-67.

Keywords: accommodations, autism, autism spectrum disorder, behavior modification, vision therapy

INTRODUCTION

The prevalence of autism spectrum disorder (ASD) has increased by almost double since 2007 to 1 in every 50 American children ages 6-17.[1] This increase has facilitated a need for a specific approach in treating visual dysfunctions and visual processing disorders in this patient population. Table 1 and 2 summarize common visual deficits and common signs of visual problems in children with ASD. ASD is a neurological disorder characterized by specific characteristics, such as difficulties with social interaction, communication, and repetitive, stereotyped behaviors.[2] Patients with autism may have decreased eye contact, lack facial expression, and have difficulty developing relationships with others. They may also have difficulty carrying a conversation, use repetitive, meaningless language, and are unable to participate in imaginative play. Repetitive behaviors can include repetitive and obsessive interest to specific parts of an object.[2] Further research is needed to understand the underlying mechanism for these behaviors. However, it is important for the clinician to be aware of these behaviors in order to effectively treat this special population.

Due to the difficulty with social interaction, communication, and repetitive behaviors, often children with ASD require an inter-professional team of speech, occupational, physical, and behavioral therapies to encourage meaningful communication and lessen anxiety in multiply-stimulating settings. In conjunction with these aforementioned therapies, vision therapy should also be included to improve visual coordination, visual motor integration, and visual information processing. Vision deficits can contribute to reduced social interactions, as well as to academic difficulties. According to a study by Milne et al. in 2009, there was an increased prevalence of reduced convergence in children with ASD.[3] Children with autism also have visuo-spatial processing deficits that manifest as difficulty relating their own bodies in space. In order to improve body awareness, ASD patients rely on proprioceptive behaviors, such as toe walking and hand flapping near their faces.[2] Other common vision deficits in ASD patients include photosensitivity, decreased processing of peripheral stimuli, reduced visual-closure perceptual ability, reduced sensory integration, and reduced processing of faces and motion processing.[2] As optometrists, we have the ability to improve functional vision and enhance patients' quality of life with lenses, prisms, and/or vision therapy.

The clinician may face challenges when treating children with autism because they may have difficulty engaging in reciprocal conversation, have poor attention due to sensory overstimulation, be resistant to change, or be non-verbal. Through other multi-disciplinary therapies, this article seeks to compile effective behavior modifications, strategies, and supports used by other professionals, such as teachers, and physical, occupational, and behavioral therapists. These modifications may be incorporated into optometric vision therapy in patients with ASD to more successfully improve vision functions and visually-guided activities.

Table 1

Common visual deficits in children with autism
Reduced convergence[3]
Visuo-spatial processing deficits[2]
Photosensitivity[2]
Decreased processing of peripheral stimuli[2]
Reduced visual closure perceptual ability[2]
Reduced sensory integration[2]
Reduced processing of faces and motion processing[2]

Table 2

Signs of visual problems in children with autism[40]
Squints or closes an eye
Looks at objects sideways or with quick glances
Sensitivity to light
Becomes confused at changes in flooring or on stairways
Pushes or rubs eyes
Has difficulty making eye contact
Touches walls or tables while moving through space
Flaps hands, flicks objects in front of eyes
Stares at certain objects or patterns

Case Report: Patient AB

AB is a 9-year-old female of Hispanic descent, who presented for a comprehensive eye examination at University Eye Center. The chief complaints, reported by her mother, included bumping into objects, losing her place while reading, and needing to repeat sentences while reading. AB also easily became confused with stairways and flooring, and she often looked at objects sideways with quick glances. AB was born very prematurely at 30 weeks with Caesarian Section, and she was born weighing 1 lb. 15 oz. She was in the prenatal intensive care unit for three months after birth. Although she started walking on time, she was delayed in speech development. AB was diagnosed with mild, high functioning autism at two years of age. She received early intervention for occupational, speech, and physical therapy services since she was two years old. She currently attends a special education school for children with ASD, and her mother reported that she was doing well in school besides her difficulty with eye-tracking.

Upon entering the examination room, AB began to walk aimlessly in different directions, seemingly unaware of her surroundings. The refractive analysis revealed emmetropia with 20/20 visual acuity at distance and near OD, OS, and OU. Pupils were round and reactive to light without afferent pupillary defect. Anterior segment and posterior segment ocular health with dilated fundus exam were unremarkable

Table 3. Patient AB's post VT comparison

Examination Testing	Pre-Vision Therapy	Post-Vision Therapy
Symptoms	1. Loss of place while reading 2. Re-reading lines 3. Bumping into surroundings	1. No longer loses her place 2. No longer re-reads 3. Only occasionally bumps into surroundings
Distance and Near VA	20/20 OD/OS/OU	Stable
Distance Cover Test	Ortho	Ortho
Near Cover Test	8XP	Ortho
Near Point of Convergence	5"/recovery 7"	3"/recovery 4"
Pursuits	Several fixation losses, unable to follow accommodative target or fixation light.	No re-fixations, no head movements, smooth and accurate
Saccades	Profound inaccuracy, with severe head movements, 50-60% undershoots in all gazes	No more re-fixations, no head movements, about 10-20% undershoots in all gazes
Developmental Eye Movement Test (DEM) • Vertical • Horizontal • Error • Ratio	 1st%ile 1st%ile 15th%ile 15th%ile	 15th%ile 15th%ile 35th%ile 35th%ile
Vergence Ranges • Distance Base In • Distance BO • Near Base In • Near Base Out	 X/18/6 X/28/6 X/30/12 X/24/6	 X/8/4 X/12/10 X/6/2 X/35/25

for each eye. Binocular findings were adequate, and stereo-acuity at near was normal at 30 seconds of arc. The pertinent examination findings are summarized in Table 3.

Pursuit eye movement testing revealed profound fixation losses and an inability to follow one cycle of a moving target. Her saccades were also grossly inaccurate, and she was unable to complete a single cycle of fixating between two targets without many re-fixations and head movements. The Developmental Eye Movement (DEM) test[A] also demonstrated difficulties with horizontal eye movements. AB had little ability to control her eye movements and was diagnosed with severe deficits of saccades.

In-office vision therapy was recommended with an estimate of 20 sessions consisting of weekly 45-minute in-office sessions combined with daily home activities. The goal of vision therapy was to improve oculomotor control and improve visual spatial awareness. Though AB was light-hearted and eager to play, challenges in vision therapy included frustration when changing between activities, hypersensitivity to sensory stimulation, and difficulty understanding verbal directions. When focused on a particular activity, she experienced difficulty transitioning to a new activity. She was easily distracted with environmental noise and was easily frustrated when multiple sensory components were added, such as the balance board and metronome. Explaining activities required multiple forms of visual demonstrations. Though AB experienced significant challenges with sensory issues, transitioning, and comprehension, with the application of visual supports, accommodations, and behavior modifications to vision therapy as described in this article, AB was able to read more efficiently after 25 sessions of vision therapy, and her oculomotor skills improved to within age appropriate levels.

Interdisciplinary Behavior Modifications, Strategies, and Supports to Implement During In-office Vision Therapy

1. **Challenge:** Difficulty transitioning between activities.
   Strategy: Implement activity schedules and/or social stories, gradually change activities, and offer choices.

Psychologists have studied how people with ASD often show strong reaction to changes, which may be due to atypical development for change-detection among children with autism.[4] Because of this resistance to change, children with autism may struggle with transitions, which may lead to behaviors such as aggression, tantrums, and non-compliance. Their further difficulty with socialization may cause them to be unable to communicate their frustration with change.

Teachers have researched behavior modifications to improve this change, and provide support for every change within a schedule. These include choice-making, incorporating preferred activities, reinforcement, and visual support systems.[5] Teachers provide choices amongst different positive behaviors, which improves compliance.[6] In addition, special education teachers use activity schedules, which combines photographs, images, or drawings in a sequential format to provide structure for the child.[5] This allows children with autism to anticipate change throughout the day. Recent research studies since 2000 have shown how teachers use activity schedules to successfully help children progress easily between steps in an activity and improve compliance.[7,8,9,10,11,12] However, most of the research are either case reports or represent a small sample size. Further randomized controlled studies are needed to verify the effectiveness of activity schedules. Activity schedules may be created using a child's tablet. Choiceworks[B] and Brill Routines-Visual Timer[C] are examples of Apple and Android apps to implement in the therapy room and to incorporate into the children's given tablet from their special education schools.

Through a survey conducted by Vaz,[13] it has been shown how visual symbols can clarify procedures, re-assuring children with ASD that the procedures will not proceed indefinitely. Professionals in a hospital setting instructed 20 children with learning disabilities and ASD by using 150 visual symbols of the hospital setting, such as "doctor" and "hospital". The views of 50 professionals in a general hospital and a community child health service were analyzed through a brief questionnaire about the child's understanding and compliance. All professionals felt that the visual symbols helped increase understanding of the verbal explanation. They reported that the visual symbols reinforce what has been said for those with poor auditory memory.[13]

Social stories, used by parents, therapists, and other professionals, are narrative activity schedules that identify concerns and develop a story to support a desired outcome. These help children with challenges in verbal communication and social interactions and will help them predict what will happen in social situations.[14] Children with ASD can review the social story before the vision therapy sessions and thus, be more at ease by having a predictable outcome. Unlike an activity schedule, social stories require specific elements and structure, such as descriptive, perspective, affirmative, and directive text.[15] Descriptive statements tell the story specifically. Perspective sentences relate feelings and opinions. Directive sentences provide behavior choices, while affirmative statements highlight what is shared.[5] Further studies are needed to confirm the effectiveness of social stories.

Developmental optometrists can incorporate techniques researched by psychologists, teachers, and other professionals into optometric vision therapy. Giving children with autism a choice between activities on the schedule can help children feel more at ease with the activity. Additionally, having a consistent therapist is also important to minimize change to the patient. Finally, visual schedules of vision therapy techniques can be implemented by using technology already used in the classroom (see Table 4 for an example).

2. **Challenge:** Difficulty understanding verbal instructions.
   Strategy: Use visual stimuli when giving instructions.

Table 4. Here is an example of Patient AB's social story.

Photograph	Story Text
Patient AB walking into the eye center	There are a lot of students in the waiting room. It is okay to feel this way. Patient AB can choose to greet the other vision therapy students.
Patient AB reviewing the schedule of the day	There are going to be 3 activities. Patient AB gets to choose which one she wants to do first.
First, we will play with the Marsden Ball	Patient AB will try to catch the butterfly flying around. It is okay if she sometimes misses the butterfly.
Playing with the Vectogram	Patient AB will try to make the clown into one clown with her eyes so that she can paint the clown's face.
Playing with the Brock String	Patient AB will try to make each bead one and pretend like she's an elephant eating peanuts
Then, we will show your mom what you are going to play with at home!	It is okay for your eyes to feel tired afterwards. Patient AB will explain to her mom which one is her favorite.
It is time to leave	Say bye.

Children with ASD often struggle with comprehending verbal instructions, and have difficulty with receptive and expressive language.[3] Compared to auditory information, children with ASD respond more favorably to visual information. Thus, special education teachers minimize giving solely auditory instructions.[16]

A case-controlled study with 16 boys with ASD, conducted by Molloy et al. in 2003, showed the importance of visual cues in maintaining balance. When compared with neurologically normal controls, children with ASD had significantly ($p<0.05$) more difficulty maintaining an upright posture when visual cues were eliminated.[17] This demonstrates the need for treating patients with autism who have difficulty with binocular and visual information-processing deficits with in-office vision therapy. Improving visual efficiency in these patients can improve visual-vestibular interaction, and, in turn, reduce "clumsiness", which is sometimes described as a feature of ASD.[13] However, randomized controlled studies are still needed to confirm the significance of visual stimuli on postural control in children with ASD.

Similar to Social Stories and Activity Schedules, in a non-randomized controlled study with 33 children with ASD aged 13-16 years, visual stimuli were shown to enhance productivity in a physical education program and enable children with autism to achieve greater weight loss ($p<0.014$).[18] With the help of visual assistance, such as delineation of playing fields, posters, pictures, and demonstration of difficulty levels, children succeeded more and demonstrated greater improvements in physical activity, possibly due to greater understanding. In a small sample study, educators also showed how picture-activity schedules greater improved fine and gross motor skill performances, compared to traditional verbal descriptions and demonstration.[17] This provides further evidence on the importance of incorporating visual instructions with verbal instructions when explaining each vision therapy activity.

In the vision therapy room, as discussed previously, activity schedules, visual schedules, and social stories can be integrated. A compilation of activity schedules for commonly used activities can be implemented. Furthermore, to improve efficiency when giving instructions, short videos can be used, as well as taking actual pictures of each activity. Additionally, vision therapists can also demonstrate or model the activity to perform to provide visual support. Many offices have used white boards with Velcro attachments to improve flow of demonstrating visual schedules. Figure 1 is an examination of a Visual Schedule used with the Brill Routines – Visual Timer.[C]

3. **Challenge:** Difficulty with hyper- or hypo- sensitivity to sensory stimulation.

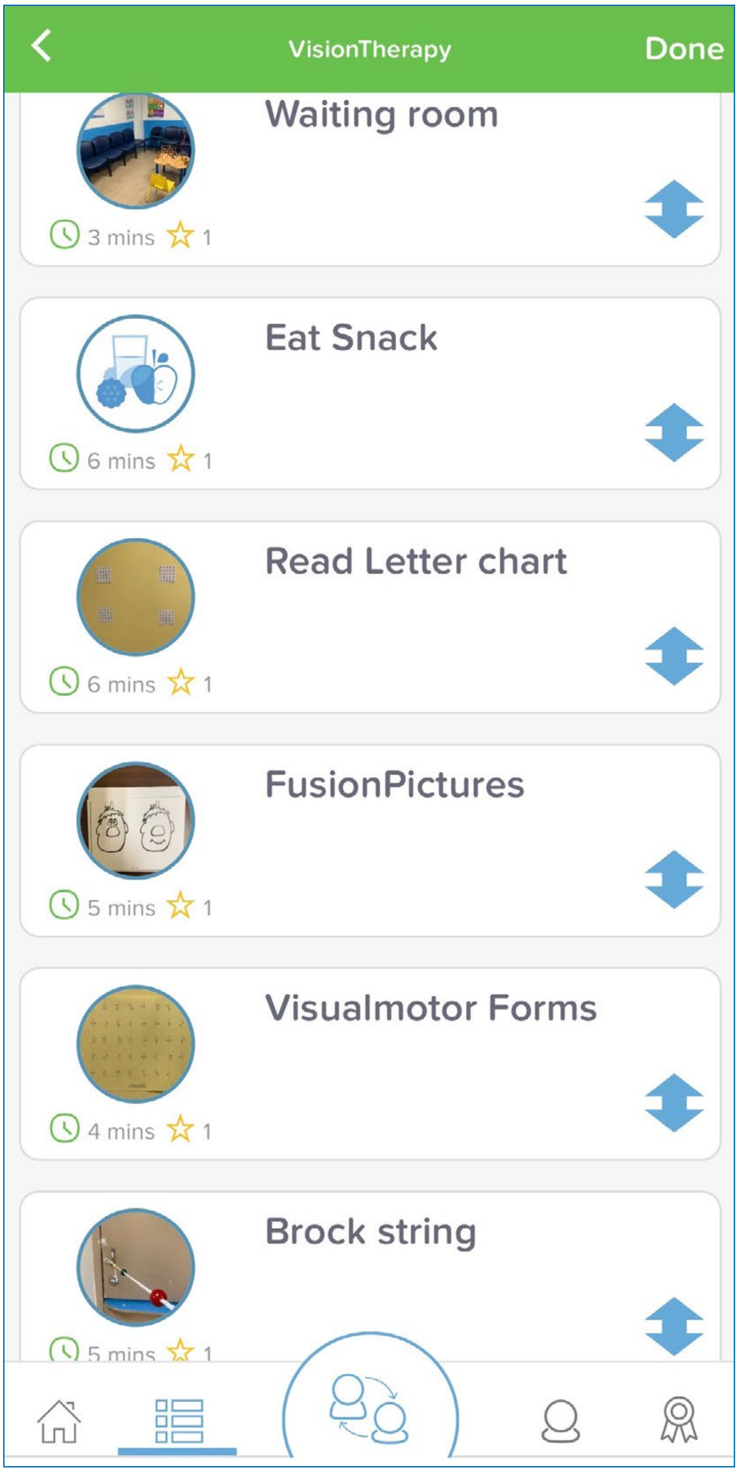


Figure 1: Example of Brill Routines-Visual Timer[C]

> **Strategy:** First, allow sensory supports, second, slowly emphasize sensory integration to improve compliance and motor planning.

Children with ASD have difficulty integrating visual, tactile, and auditory stimuli.[19,20] Our senses provide important information about ourselves and our environments, such as the identities and locations of objects, and the speed of movement. We have a "comfort range" for each of these senses. For individuals with ASD, there is a reduced "comfort range"[20] leading to sensory disturbances, which include hyper- and hypo- sensitivities. These sensitivities may be due to differences in how these stimuli are processed in the brain. A more sensitive child may show withdrawal behaviors when being touched, avoid textures, and have poor coordination when there is too much activity in the environment. Hypo-sensitive children can be under-responsive to visual and auditory stimuli, as well as pain. This may mean that they place themselves in dangerous situations as they do not understand or interpret warning signs clearly. These characteristics can lead to poor organization and behavioral problems. Furthermore, in a study conducted by Hilton et al., those with atypical sensory responsiveness and social impairments seem to be strongly related.[21] Children with hyper-responsiveness to tactile stimuli were more likely to develop stereotypical behaviors, such as repetitive verbalizations, inflexible behaviors, and inattention.[21] Thus, identifying and treating sensory integration disorders can greatly impact a child with ASD's social characteristics.

Occupational therapists and psychologists have researched methods to improve sensory integration in children with ASD. In more than 80 studies, improving sensory integration has shown to be effective in some aspects, though some with limitations.[22] During occupational therapy, the therapist will slowly introduce vestibular, proprioceptive, and tactile stimuli while the patient is in motion.[23] Sensory-integration therapy (SIT) with occupational therapists can improve both organizational speed and smooth pursuit.[24]

In order to identify patients with sensory processing disorders, a questionnaire was developed by Robertson et al., which included questions like: *"Do you find yourself fascinated by small particles?", "Do you notice that you* *have hurt yourself but did not feel any pain?"*, and *"Do you react very strongly when you hear an unexpected noise?"*[20] This questionnaire can easily be implemented early in therapy to identify hyper- and hypo-sensitivities.[25]

Because children with autism may manifest sensory difficulties in many different facets, in vision therapy, developmental optometrists should consult with the patient's occupational therapist on how the patient modulates sensory information.[26] Sensory items provided by occupational therapists include chew toys, such as chewable jewelry, weighted sensory lap pads, slant boards, swings, and hammocks. Patients should be encouraged to use these sensory supports in the beginning of therapy. In order to improve sensory integration into vision therapy, activities should begin with gross motor skills, such as incorporating bilaterality while drawing on a chalkboard (Chalkboard circles) to provide a strong motor foundation.[19] Therapy should also first begin in a quiet environment free of clutter. In addition, regulating the patient's sensory system at the beginning of therapy prepares the patient to move to a more sensory challenging activity and environment.[21] This could mean incorporating rhythmic motor demands, such as bunting a ball repetitively before incorporating bilateral integration. Additionally, tactile defenses can be modulated by engaging in symbolic play, such as pretending to be a butterfly, pirate, or princess.[21]

As therapy advances, more sensory input can be added, such as a balance board, bilateral integration, and a metronome for auditory integration. Oftentimes, vertical yoked prisms are used to create an image shift, which requires a motor response to the sensory mismatch in space. Thus, yoked prism can improve visual spatial awareness in patients with ASD. Incorporating yoked prism in therapy can facilitate and improve sensory motor integration.[27]

Incorporating facets of sensory integration therapy from occupational therapists into vision therapy can improve attention and compliance, as well as improve motor planning and their social function. Allowing for sensory supports and motor breaks in therapy can also improve compliance for more sensory stimulating activities, such as walking in different patterns with a metronome, monocular accommodative rock with magazine saccades, and binocular activities on a balance board. Another method to improve sensory integration is providing a visual schedule, as discussed previously, which can allow unpleasant sensory stimuli to be more predictable, making them more tolerable.

4. **Challenge:** Poor compliance with therapy and homework, poor reciprocal communication.
   Strategy: Positive reinforcement and extinction (withdrawal of reinforcement), and Discrete Trial Therapy.

Reinforcement strategies have been shown by behavioral therapists to improve social skills, language, and academic tasks. In a case series with three children with autism, positive reinforcement has been shown to improve working memory, or the ability to "keep information online" while simultaneously processing it.[28] Behavioral therapists implement positive reinforcement through Applied Behavior Analysis (ABA).[29] ABA has shown to increase functional and cognitive skills in children with autism. Special education teachers have shown how implementing ABA in the classroom can decrease challenging behaviors and increase socially significant replacement behavior. Moreover, the frequency of reinforcement can be systematically decreased, in order to more closely mimic a natural environment.[30]

There are many dimensions to ABA training, which vary based on the 1) degree of structure in the curriculum, 2) depth of the curriculum, and 3) decisions regarding maintenance, modification, and control.[31] Discrete trial therapy (DTT) is one technique used within an ABA program, which includes a structured method of presenting stimuli in small steps in a controlled setting. ABA emphasizes repetition for rapid attainment of rote skills in the beginning, and then contextualizing what was learned into daily living skills once the initial skills are mastered.[30] Reinforcements can include food, toys, praise, sensory stimulation (vibration, music, strobe), and tokens, and allowing children to choose the form of reinforcement also enhances effectiveness. Studies have shown that multiple reinforcements can be effective, and Leaf et al. also demonstrated that verbal feedback alone might be a form of social reinforcement that might be effective.[29]

Though optometrists may not always be able to implement a highly structured ABA program during in-office vision therapy, incorporating certain aspects of ABA can allow vision therapy to be more efficacious for children with autism. Positive reinforcement seems to have a beneficial effect on learning new tasks for children with autism, such as working memory and social skills, which optometrists can translate into the therapy room. Positive reinforcement also allows for better compliance in therapy and a more structured setting, which helps children with autism cope with transitions. As developmental optometrists, a token economy developed from ABA can be implemented to decrease challenging behaviors. A token economy includes 1) a list of target behaviors, 2) tokens or points for achieving behaviors, 3) and a list of backup reinforcement items (see Figure 2).[25] An example of a token economy implemented in therapy is encouraging eye exercises for tokens, and achieving 10 tokens can lead to a sticker or a small snack. Special education teachers have shown how the inclusion of token economy improved the effectiveness of learning, partly because students are given a choice for their behaviors.[31] Optometrists can also implement DTT by breaking each vision therapy technique into small finite steps and reinforcing specific responses. Once compliance and therapy effectiveness have been achieved, optometrists can spread out the amount of tokens given (i.e. needing to achieve a greater quantity of favorable behaviors before receiving a token), and transition physical reinforcements into more verbal feedbacks. Through implementing positive reinforcements, choice making, and finite goals, developmental optometrists can effectively improve compliance and success in vision therapy.

5. **Challenge:** Reduced motor planning ability, increased restlessness, and clumsiness during therapy.
   Strategy: Incorporate rhythmic movement then incorporate bilateral integration activities.

Token Economy

1. Brock String	2. Letter Chart	3. Ball and Loop	4. Fusion Pictures	5. Trampoline
Token				
		GOAL REACHED: SNACK REWARD		

Figure 2: Example of a Vision Therapy Token Economy. Patient AB will receive 1 "token" for each procedure. When each box is complete, she gets a SNACK!

Children with autism may have motor deficiencies in addition to social and communication deficiencies such as deficiencies in walking, coordination, symmetry, and movement initiation.[18] Movement therapy can strengthen these deficits, improve sensory integration, and enhance social and cognitive learning. Movement therapy focuses on increasing vestibular and proprioceptive stimulation to increase body awareness, increase attention, and behavior regulation. Repetition and routine of sequences, such as yoga therapy, increases the child's motor planning ability, which improves balance and coordination.[32] Additionally, improving motor skills may allow children with autism to engage with other normally developing children with more ease, which can result in improved social interaction, self-confidence, and motivation.[18] In a study conducted by Askay et al., physical activity improved strength, walking, and running in children with ASD, which, in turn, decreased maladaptive behaviors. An observational study by See et al. involved 41 children and found that music and movement therapies are beneficial. Positive impact was evident in regards to behavioral problems, such as restlessness, temper tantrums, and inattention ($p<0.001$). Rhythmic songs alongside movement helped develop auditory-motor coordination and improve body awareness.[33]

Developmental optometrists can incorporate movement in vision therapy not only to improve motor planning, but also to improve social communication, cognition, and reduce maladaptive behaviors. Allowing for motor breaks, as discussed above, vision therapy should begin with rhythmic movements, such as stomping in place before incorporating bilateral integration and left-right awareness activities. Examples of incorporating movement with vision therapy include incorporating gross motor activities, such as tossing a bean bag, jumping in different directions corresponding to arrows, as well as incorporating movement alongside common therapy procedures, such as using a bead and string while walking in a straight line, or balance beam while reading a letter chart.

6. **Challenge:** Anxiety, poor attention, and inability to generalize learned ideas.
   Strategy: Incorporate problem solving techniques, repeated practice, and abstract reasoning influenced by cognitive behavioral therapy.

Children with ASD have difficulty relating to others as well as emotions to actions. Another theory used in behavioral therapy is cognitive behavioral therapy (CBT), which seeks to promote "retrievable memories of adaptive responses", which can override and suppress memories of maladaptive responses. Individuals in CBT learn skills to modify their thoughts and beliefs, as well as develop problem-solving strategies.[34] In order to promote deep processing of new concepts, behavioral therapists incorporate Socratic questions, or questions that incorporate hints of the correct answer. In this way, children will put the accurate answers in their own words, which could then convert the task into memory and, thus, be incorporated into their everyday lives. In a randomized controlled study with 70 children diagnosed with high functioning ASD, CBT significantly improved social communication skills and decreased anxiety ($p<0.04$). The program comprised of three main sections: part one emphasizes recognizing different emotions, part two focuses on management techniques, and part three focuses on problem-solving strategies, using the STAR (STOP, THINK, ACT, and REFLECT) strategy. CBT addresses the cognitive deficiencies that may cause communication, behavioral, and emotional difficulties.[35]

In vision therapy, optometrists can implement CBT during more difficult procedures that require visualization and cognitive thinking, such as visual perceptual training. Socratic questions can be asked during binocular training to promote awareness and control of convergence and divergence. During vectogram procedures, questions such as, *what do you think your eyes are doing? If the image seems to come closer to you, what could that mean about how you are controlling your eyes?* During difficult procedures, the STAR strategy can be implemented to lessen frustrations and improve communication with the therapist.

7. **Challenge:** Poor ability to relate to therapist and develop meaningful play. **Strategy:** Incorporate techniques of Symbolic Play (Development Individual-Differences-Relationship-Based Model "Floortime").

The Developmental Individual-Differences Relationship Based (DIR) model is developed by Greenspan and Wieder in the 1980s to provide a clinical model for therapeutic intervention in children with ASD.[36] The model is used by interdisciplinary professionals to emphasize the importance of emotional, affect-based experiences within a relationship, which children with ASD may lack.[37] The DIR model also addresses the differences in how children with ASD engage, relate, and communicate with their environment.[38] Children with ASD also often develop spontaneous behaviors, including wandering aimlessly without purpose, lining up toys, habitually placing toys at eye level, and demanding to play with the same toys over and over. Serena Wieder, a child psychologist, found good results in therapy with these children by matching the patient's developmental level and interacting with the child in ways that were meaningful and enjoyable.[36] In this way, meaningful play through the use of "Floortime" interactions can be encouraged. "Floortime" interactions occur when the therapist follows the patient's lead, and at the same time supporting their emotional and intellectual abilities.

First, by using the "developmental" aspect of the DIR model the therapist can understand the emotional developmental capacity of the child in how he or she engages and relates to others, in order to show intentional behavior, to communicate needs, and to stay calm and regulated. The therapist must also understand how the child processes sensory information, such as vision, sound, touch, balance, and motor movements, which is the Individual-Differences aspect of the model. Finally, Relationship-based refers to the importance of providing affect-based interactions with the child to facilitate learning. Incorporation of meaningful play, including incorporating emotions, will maximize the patient's understanding and also promote better communication with others.[31] In the earliest stages, occupational therapists, speech and language pathologists, teachers, and other professionals first try to establish a two-way communication. Later, "Floortime" activities are integrated into each specific therapeutic profession. Controlled research supporting "Floortime" is limited, but preliminary research shows a positive outcome. A pilot study by Solomon et al. in 2007, found that nearly half of the children (45.5%) made significant emotional developmental progress (using the Functional Emotional Assessment Scale created by Greenspan et al. in 2001) and reported a 90% approval rating from parents.[39]

In vision therapy, the DIR Model can be incorporated to better understand the patients' emotional developmental levels and to better connect with patients and facilitate progress in vision therapy. The therapist must first understand how a child responds to auditory or tactile stimuli, challenging motor tasks, and to language, gestures, and facial expressions. Then, the therapist can modify the task to meet the child at his or her developmental level, such as changing equipment use, instructions, and the pace of the procedure. In order to add affect and connect with patients, the therapist can add a simple story to procedures, for example, the therapist can pretend the Marsden ball represents a swimming fish, and the patient will try to catch the fish with the loop, which represents a net. Affect can be added by establishing the purpose of catching the fish, how it makes the child feel, and the difficulty of the task. Affect can also be added via nonverbal communication, such as facial expressions and gestures. See Table 5 for a summary of challenges and strategies to incorporate into optometric vision therapy.

Patient AB: Post-Vision Therapy with Behavior Modifications

At the conclusion of therapy, patient AB showed progress in many areas and her mother was thrilled with the dramatic transformation at the end of therapy. The clinical findings after completion of vision therapy are summarized in Table 3. There were marked improvements in ocular motility control. Pursuit eye movements also showed profound improvement. She was able to follow a target for 5 cycles, and initiated saccadic eye movements with only moderate undershoot. The horizontal component of the DEM test was significantly improved, and she performed within age norms. These findings correlated well with the symptomatic changes that her mother reports at school. Patient AB no longer has difficulty with keeping her place while reading, and she did not bump into objects as frequently.

CONCLUSION

With the help of a plethora of multi-disciplinary clinical practice models to improve the emotional, communicative, and motor development of children with ASD, optometrists can improve the effectiveness of vision therapy to promote better visual comfort in these patients' daily lives. Compared to the non-neurologically-impaired child, the child with ASD may present with many challenges, including lack of social and emotional reciprocity, sensory disorganization, as well as resistance to change. Eye care professionals have an opportunity to become an integral part to the growing need in improving ASD children's developmental level and, thus, maximize their daily function. Vision therapy not only can improve their academic function, but also, with the help of these multi-disciplinary modifications, strategies, and supports can improve their emotional and social functions. More controlled research is needed to outline the effectiveness of vision therapy in this population, as well as take into account low- versus high- functioning children with ASD. However, with an effective treatment plan that meets the child's developmental level, those with ASD can benefit significantly from vision therapy.

Table 5: Summary of challenges and strategies to incorporate into optometric vision therapy.

Challenges	Strategies
1. Difficulty transitioning between activities	• Implement activity schedules and/or social stories • Gradually change activities • Offer choices
2. Difficulty understanding verbal instructions	• Use visual stimuli when giving instructions Example: Short videos, pictures of each activity or vision therapists can demonstrate or model the activity to be performed.
3. Difficulty with hyper- or hypo-sensitivity to sensory stimulation.	• Allow sensory supports and slowly emphasize sensory integration to improve compliance and motor planning Example: As therapy advances add balance board, bilateral integration, and a metronome for auditory integration. Vertical yoked prisms to create an image shift, requiring a motor response to the sensory mismatch in space.
4. Poor compliance with therapy and homework, poor reciprocal communication	• Positive reinforcement, extinction and Discrete Trial Therapy Example: Token economy for positive reinforcement. Break each vision therapy technique into small finite steps and reinforce specific responses
5. Reduced motor planning ability, increased restlessness, and clumsiness during therapy	• Incorporate rhythmic movement • Incorporate bilateral integration activities Example: Incorporate gross motor activities, such as tossing a bean bag, jumping in different directions corresponding to arrows Incorporate movement alongside common therapy procedures, such as using a bead and string while walking in a straight line, or balance beam while reading a letter chart.
6. Anxiety, poor attention, and inability to generalize learned ideas	• Incorporate problem solving techniques, repeated practice, and abstract reasoning Example: Socratic questions during vectogram procedures. What do you think your eyes are doing? If the image seems to come closer to you, what could that mean about how you are controlling your eyes?
7. Poor ability to relate to therapist and develop meaningful play	• Incorporate techniques of Symbolic Play Example: Matching patient's developmental level and interacting in ways that are meaningful and enjoyable. Therapist follows the patient's lead, while supporting their emotional and intellectual abilities. Therapist must understand how a child responds to auditory or tactile stimuli, challenging motor tasks, and to language, gestures, and facial expressions. Then, therapist can modify the task to meet the child at his or her developmental level.

SOURCE LIST

A. Bernell VTP 4016 N Home Street Mishawaka, IN 46545 800-348-2225. https://www.bernell.com

B. Choiceworks. Computer Software. Apple App Store. 4.4.1. Bee Visual LLC. 2012-2014. https://apple.co/2RnM0CF

C. Brili Routines-Visual Timer. Computer Software. Apple App Store. 2.0.9. Brili GmbH, Inc. 2019. https://apple.co/2YRHJZM

REFERENCES

1. Blumberg SJ, Bramlett MD, Kogan MD, et al. Changes in prevalence of parent-reported autism spectrum disorder in school-aged U.S. children: 2007 to 2011-2012. National health statistics reports; no 65. Hyattsville, MD: National Center for Health Statistics, 2013.
2. Coulter RA. Understanding the visual symptoms of individuals with Autism Spectrum Disorder (ASD). Optom Vis Dev 2009;40(3):164-75.
3. Milne E, Griffiths E, et al. Vision in children and adolescents with Autistic Spectrum Disorder: Evidence for Reduced Convergence. J Autism Dev Disord 2009;39: 965-75. Web.
4. Simmons DR, Robertson AE, McKay LS, Toal E, McAleer P, Pollick Fe. Vision in Autism Spectrum Disorders. Vision Res 2009;49.22:2705-39.
5. Banda DR, Grimmett E, Hart, SL. Helping students with Autism Spectrum Disorders in general education classrooms manage transitions issues. Teach Escep Child 2009;41:16-21.
6. Coulter RA, Bade A, Tea, Y, Fecho G, Amster D, Jenewein E, Rodena J, Lyons K, Mitchell GL, Quint N, Dunbar S, Ricamato M, Trocchio J, Kabat B, Garcia C, Radik I. Eye examination testability in children with Autism and in Typical Peers. Optom Vis Dev 2014;92:31-43.
7. Dauphin M, Kinney EM, and Stromer R. Using video-enhanced activity schedules and matrix training to teach sociodramatic play to a child with autism. J Posit Behav Interv 2004;6:238-50.
8. Morrison RS, Sainato DM, Benchaban D, and Endo S. Increasing play skills of children with autism using activity schedules and correspondence training. J Early Interv 2002;25:58-72.
9. Bryan LC and Gast DL. Teaching on-task and on-schedule behaviors to high-functioning children with autism via picture activity schedules. J Autism Dev Disord 2000;30:553-67.
10. Dooley P, Wilczenski FL, and Torem C. Using an activity schedule to smooth school transitions. J Posit Behav Interv 2001;3:57-61.
11. Massey NG and Wheeler JJ. Acquisition and generalization of activity schedules and their effects on task management in a young child with autism in an inclusive pre-school classroom. Education and Training in Mental Retardation and Developmental Disabilities 2000;35:326-35.
12. Dettmer S, Simpson RL, Myles BS, and Ganz JB. The use of visual supports to facilitate transitions of students with autism. Focus Autism Other Dev Disabl 2000;15:163-9.
13. Vaz I. Visual symbols in healthcare settings for children with learning disabilities and Autism Spectrum Disorder. Br J Nurs 2013;22:156-9.
14. Briody J, McGarry K. Using social stories to ease children's transitions. Young Children 2005;60:38-42.
15. Gray C. The New Social Story Book: Illustrated Edition, 2nd ed. Arlington, TX: Future Horizons Inc.; 2000.
16. Liu T, Breslin CM. The effect of a picture activity schedule on performance of the MABC-2 for children with autism spectrum disorder. Res Q Exerc Sport. 2013;84:206-12.
17. Molloy CA, Dietrich KN, Bhattacharya A. Postural stability in children with Autism Spectrum Disorder. J Autism Dev Disord. 2003;33(6):643-52
18. Aksay E. Effects of physical activity programs with visual stimuli on physical development of children with Autism Spectrum Disorder. J Educ Psychol 2014;5(1)34-43.
19. Case-Smith J, Bryan T. The effects of occupational therapy with sensory integration emphasis on preschool-age children with Autism. Am J Occup Ther 1999;53(5):489-497.
20. Robertson AE, Simmons DR. The relationship between sensory sensitivity and autistic traits in the general population. J Autism Dev Disord 2013; 43:775-84.
21. Hilton CL, Harper JD, Kueker RH, Lang AR, Abbacchi AM, Todorow A, LaVesser PD. Sensory responsiveness as a predictor of social severity in children with high functioning Autism Spectrum Disorders. J Autism Dev Disord 2010;(40)-937-45.
22. Shaaf RC, Millder LJ. Occupational therapy using a sensory integrative approach for children with developmental disabilities. Ment Retard Dev Disabil Res Rev 2005;11:143-8.
23. Allison CL, Gabriel H, Schlange D, Frerickson S. An optometric approach to patients with sensory integration dysfunction. Optometry 2007;78:644-651.
24. Horowitz LJ, Oosterveld WJ, Adrichem R. Effectiveness of sensory integration therapy on smooth pursuits and organization time in children. Padiatrie und Grenzgebiete 1993;31:331-44.
25. Robertson AE, Simmons DR. The relationship between sensory sensitivity and autistic traits in the general population. J Autism Dev Disord 2013; 43:775-84.
26. Coulter RA, Tea Y, Weider S. Thinking goes back to school: providing better vision therapy to patients with autism spectrum disorder. Optom Vis Perf 2014;2(5):211-9.
27. Eubank TF, Ooi TL. Improving visually guided action and perception through use of prisms. Optometry 2001;72:217-27.
28. Baltruschat L, Hasselhorn M, Tarbox J, Dixon DR, Najdowski AC, Mullins RD, Gould ER. Further analysis of the effects of positive reinforcement on working memory in children with autism. Res Autism Spectr Disord 2011;5:855-63.

29. Leaf JB, Dale S, Kassardjian A, Tsuji KH, Taubman M, McEachin JJ, Leaf, RB, Oppenheim-Leaf ML. Comparing Different Classes of Reinforcement to Increase Expressive Language for Individuals with Autism. Educ Train Autism Dev Disabil 2014;49:533-46.
30. Hahs AD, Miller S. ABA in the special education classroom: promoting on-task behavior in an individual with autism. Education 2014;4:95-102.
31. Jensen VK, Sinclair LV. Treatment of autism in young children: behavioral intervention and Applied Behavior Analysis. Infants and Young Children 2002:42-9.
32. Kenny M. Integrated Movement Therapy: Yoga-Based Therapy as a viable and effective intervention for autism spectrum and related disorders. Int J Yoga Therap 2002;12:71-9.
33. See, CM. The use of music and movement therapy to modify behavior of children with autism. Partanika J Soc Sci & Hum 2012;20:1103-16.
34. Sung M, Ooi Y, Goh T. Effects of Cognitive-Behavioral Therapy on Anxiety in Children with Autism Spectrum Disorders: A Randomized Controlled Trial: Child Psychiatry Hum Dev 2011;42:634–49.
35. Wood J. Brief Report: effects of cognitive behavioral therapy on parent-reported Autism symptoms in school-age children with high-functioning Autism. J Autism Dev Disord 2009;39(11):1608-12.
36. Greenspan, S, Wieder S. Engaging autism: using the floortime approach to help children relate, communicate, and think. Cambridge, MA: Da Capo Press, Lifelong Books, 2006.
37. Coulter RA, Tea Y, Weider S. Thinking goes back to school: providing better vision therapy to patients with autism spectrum disorder. Optom Vis Perf 2014;2(5):211-9.
38. Azimi Green MD, Wachs H, Dee M. Successful optometric vision therapy with patients on the autistic spectrum: engaging patients with visual- cognitive therapy. Optom Vis Perf 2014;2(5):235-9.
39. Solomon R, Necheles J, Ferch C, and Bruckman D. Pilot Study of a parent training program for young children with autism: The PLAY project home consultation program. Autism 2007;11:205-24.
40. Lemer P, et al. Envisioning a bright future: interventions that work for children and adults with Autism Spectrum Disorders, 2008.

AUTHOR BIOGRAPHY: Jennifer Fisher, OD, FAAO

Berkeley, California

Dr. Jennifer Fisher completed her Doctorate of Optometry and Bachelor's in Integrative Biology at UC Berkeley. She graduated from UC Berkeley School of Optometry and completed a residency in Vision Therapy and Vision Rehabilitation at the State University of New York College of Optometry. She is a Fellow of the American Academy of Optometry and a member of the College of Optometrists in Vision Development. During her residency, she worked extensively with children on the autism spectrum and with special needs. Dr. Fisher currently works as an Assistant Clinical Professor at the Binocular Vision clinic at UC Berkeley.

AUTHOR BIOGRAPHY: Tyler Phan, OD, FAAO

Bronx, New York

Dr. Tyler Phan is the Director of Vision Therapy at Union Community Health Center where he founded the clinic's vision therapy and neuro-optometric rehabilitation program. He is also a Staff Optometrist in the Department of Ophthalmology at SBH Health System in Bronx, New York.

He is a Fellow of the American Academy of Optometry and a member of the College of Optometrists in Vision Development.

Dr. Phan graduated from the University of California Los Angeles (UCLA) with a Bachelor of Science in Psychobiology and received his Doctor of Optometry from Western University of Health Sciences College of Optometry. He completed a residency in Neuro-Optometric Rehabilitation and Vision Therapy at The State University of New York College of Optometry.

emergent
vision redefined

COLLEGE OF OPTOMETRISTS IN VISION DEVELOPMENT
ENDORSED

Online Vision Therapist Training Course

Our online vision therapist course is designed to provide a new vision therapist with foundational concepts and a basic knowledge of vision therapy. Our curriculum is **ideal for new Vision Therapists** or existing therapists preparing for their COVT boards.

The course has **over 50 videos with 45 comprehension checks**.

The course also includes:

- Access to a Certified Optometric Vision Therapist mentor
- A final written exam
- A final oral review with a Certified Optometric Vision Therapist
- A printed End of Course Report for the supervising doctor that includes course/exam scores, proof of credit hours, curriculum covered, strengths/weaknesses of the student, and personalized notes regarding the student's performance.
- A printed Emergent certificate (if the students receives a passing grade)

info@EmergentVT.com
emergent.teachable.com/p/therapist-training

ARTICLE

An Examination of the Oculomotor Metrics within a Suite of Digitized Eye Tracking Tests

Nicholas Murray, PhD
Karla Kubitz, PhD
Claire-Marie Roberts, PhD
Melissa Hunfalvay, PhD
Takumi Bolte, MSC
Ankur Tyagi, MSC

Statement of Conflict of Interest: *1st, 2nd, and 3rd authors are independent of the company (RightEye, LLC) with no conflicts of interest. All analysis and results are conducted by the 1st, 2nd, and 3rd authors. All data collection was conducted independently of RightEye by experienced vision specialists. The 4th, 5th and 6th authors work for RightEye as scientists, are blind to the analysis and follow all ethical considerations during the scientific and experimental process.*

ABSTRACT

Objective

The purpose of this study was to examine the reliability of oculomotor metrics in healthy individuals, to determine the normative values through cluster analysis, and to compare oculomotor metrics by age groups in a suite of digitized eye tracking tests.

Correspondence regarding this article should be emailed to Melissa Hunfalvay, PhD, at melissa@righteye.com.
 https://doi.org/10.31707/VDR2019.5.4.p269.

Hunfalvay M, Murray N, Kubitz K, Roberts CM, Bolte T, Tyagi A. An examination of the oculomotor metrics within a suite of digitized eye tracking tests. Vision Dev & Rehab 2019;5(4):269-84.

Keywords: cluster analysis, eye tracking, normative data, reliability, saccades, smooth pursuit

Design

Experimental cross sectional

Participants

A large sample of 2993 participants completed RightEye tests.

Results

These tests demonstrated acceptable or higher reliability on 85% of the eye movement metrics and the clustering analysis distinguished 5 distinct age groups. Furthermore, group differences were found between age clusters.

Conclusions

Overall, the findings represent the reliability of a computerized oculomotor measure and the importance to consider individual and group characteristics for clinical applications as well as applied settings.

Vision in humans is the dominant sensory system with specific characteristics and capabilities. The purpose of eye movements within the oculomotor system is to move salient information into the fovea to see it clearly. Oculomotor function (OMF) is broadly composed of smooth pursuits, saccades, and fixations.[1] Smooth pursuit (SP) occurs when the eyes track a slow-moving stimulus, usually toward its intended target or location, while keeping the object on the fovea.[2,3,4] Saccades are voluntary or reflexive movements of the fovea from one fixation point to the next.[5] Finally, fixations represent a pause or stop of the eye on a target of interest.[6] Given the importance of eye movements, there is a need to incorporate reliable and accurate measures of OMF into clinical practice and in research. As such, the purpose of this project is to test the reliability (i.e., degree to which these test consistently measure OBF metrics) of OMF metrics from the RightEye tests in a large sample of healthy individuals and to determine the normative values of OMF metrics for healthy individuals. Furthermore, to compare OMF metrics by age.

Therefore, the review of literature is meant to serve as a survey of recent work to this end.

Deficits in the oculomotor system can result in lower visual acuity, changes in visual perception, and reduced visual stability.[7,8] Furthermore, the oculomotor system can be an indicator of the neurological status of an individual. For example, it is well-known that individuals with traumatic brain injury (TBI) and mild traumatic brain injury (mTBI) suffer from some vision and visual processing dysfunctions, including visual field defect, visual motion sensitivity, and oculomotor deficits (e.g.,[9]). Further, oculomotor control differs from many psychiatric and neurological pathologies.[7] For example, saccades and smooth pursuit eye movements have been identified as endophenotypes in several neurological and psychiatric conditions including reading efficiency,[8] schizophrenia (e.g.,[10,11]), Parkinson's disease (e.g.,[12]), essential tremor (e.g.,[13]), attention deficit hyperactivity disorder (ADHD[14,15]), fetal alcohol spectrum disorder,[16] bipolar disorder,[17] borderline personality disorder (e.g.,[18]), and anxiety and depression.[19] With the proper measurement of eye movements, scientists and clinicians could utilize OMF to indicate certain neurological diseases. Also, eye movement measurement may indicate current disease state and efficacy of therapy even when other measures (such as magnetic resonance imaging (MRI)) fail to indicate a deficit.[20]

OMF is controlled by specific brain structures, starting with ganglion cells in the retina, spanning across each lobe and involving both excitatory and inhibitory neurons through direct and indirect pathways. Depending on the type of eye movement, this function can involve components of the following key brain areas: superior colliculus, thalamus (lateral geniculate nucleus), parietal cortex (parietal eye field), frontal cortex (dorsolateral prefrontal cortex), prefrontal cortex, frontal eye field, supplementary eye field), basal ganglia (striatum, globus pallidus, subthalamic nucleus), cerebral cortex, brain stem reticular formation, and cerebellum.[21,22] Also, saccades directed towards a remembered target involve working memory and will, therefore, require activation of neurocircuitry including the prefrontal cortex, frontal eye field, or supplementary eye field regions.[23,24,25,26]

Beyond a potential indicator of neurological disease, OMF has shown to be affected by age. Smooth pursuit eye movements typically decline with age[7] and some saccades, such as the disjunctive component of eye movements, are also influenced by age. Children demonstrate an increase in vertical conjugate post-saccadic drift after upward saccades with less horizontal vergence.[26] Vernet et al.,[27] using the gap and overlap paradigms, demonstrated switching latencies are age-dependent in saccadic direction and by paradigm. They also found that frontal eye field layers are optimal in early adulthood and decline with age. Further, these authors demonstrated differences in horizontal and vertical saccades with longer latencies for middle-aged adults. Also, Contreras et al. found that younger participants could synchronize their eye movements better than older participants and that age is a critical factor when comparing impaired groups as well as normative data.

Assessment of OMF is also examined, both directly and indirectly for elite levels of performance, including military, police, and athletic.[28,29] A superior ability to anticipate the flight of a ball (Croft, Button, & Dicks, 2010[29]) or target an area[30] can, at least in part, be associated with effective smooth pursuit, saccades, and fixations.[31] The OMF form a foundation in which other higher order processing occurs such as superior reaction time,[32] memory,[33] information processing speed,[34] and decision making[35] even in children as young as eight years old.[36] Elite level OMF allows for the athlete, officer, or warfighter to be efficient and effective in performance-related environments and in situations that are stressful or time constrained.

Given the factors that influence OMF and the current standards of assessment, there is a need for objective and reliable measures of OMF. Leigh & Zee,[7] in their classic textbook, describe the clinical examinations of saccades, smooth pursuit, gaze behavior, and eye-head movements among others. Typically, these clinical evaluations involve a "bedside" approach and instruction which include 'follow the tip of my finger' and require the physician to detect the salient characteristics of OMF by the naked eye.[37] The recommendations for saccadic eye examinations included naked eye evaluations are aimed at answering such questions as: "Are saccades promptly initiated? Are they of normal velocity? Are they accurate? Do the eyes move together? Do they go straight to the target?" ([7], p 243). Examinations for smooth pursuit eye movements are similar. In addition to the standard bedside evaluations, the most commonly used protocol by practitioners, recommended by the Neuro-Optometric Rehabilitation Association, is the Vestibular Ocular-Motor Screening Assessment (VOMS;[38]). This protocol is used in many settings by various professionals from general medical practitioners, to physical therapists, to neurologists, and even athletic coaches. However, several problems exist with this approach. First, the reliability of VOMS is low irrespective of the administrator's level of experience.[39,40] Second, it is only possible to detect gross eye movement through the naked eye. Third, measuring a change in eye movements over time is difficult when employing the "bedside" or VOMS assessments. It should be noted that these assessments of oculomotor function are only one part of a more holistic assessment which often includes short-term memory and balance tests. In addition to these tests, there are several other vision specific tests that have been shown to be reliable including the King-Devick (K-D) test which assesses horizontal saccadic eye movement and attention, the Developmental Eye Movement (DEM) test that identifies oculomotor dysfunction in children, and the prism bar measurement which reliability measures vergence ranges. However, there is limited work on the usefulness of these test outside their specific purpose.[36] Therefore, with the knowledge that eye movements are an essential factor in overall health,[37] it is imperative that they are examined with the highest level of accuracy needed to provide reliable data and interpretation.

Eye tracking has recently been used to examine OMF for overall visual and neurological health and wellness.[41,42,43,44] Eye tracking provides a highly specific, objective measurement especially when the sample rate is high (120-1000 Hz;[6]). Research to date shows promise in the employment of eye tracking devices to measure characteristics indicative of healthy eyes, in turn, provide evidence for a healthy brain.[37]

A current limitation of eye movement research is a lack of data examining the reliability of oculomotor metrics.[45] Furthermore, many of the psychometrically focused studies have included relatively low sample sizes (e.g., Bedell & Stevenson,[37] n = 39; Contreras et al.,[8] n = 45; Vernet et al.,[27] n =10), however, there are some notable exceptions (e.g., Bargary et al.,[46] n = 1058; Evdokimidis et al.,[47] n = 2,006; Lenzenweger and O'Driscoll,[48] n = 300, Murray, Hunfalvay, and Bolte[29] n = 416). Therefore, this study has three main purposes. The first purpose was to examine the reliability of OMF metrics from the RightEye tests in a large sample of healthy individuals. The second was to determine the normative values of OMF metrics for healthy individuals, and the third was to cluster these normative values by age.

METHODS

Participants

For the normative data analysis, 2993 participants completed of the RightEye tests. Participants were between the ages of 5-62 years (M = 20.87, SD = 12.45); 2030 were males

(67.85%), 962 were females (32.15%). Of the 2993 participants, 61.63% were white, 6.85% black, 8.32% Hispanic, 0.20% Native American and 8.96% opted not to report ethnicity.

To establish test-retest reliability, a subset (n = 201) completed RightEye tests twice (i.e., Trial1 and Trial2) on two separate days. These participants were between the ages of 5-62 years (M = 25, SD =17.47); 108 were males (53.73%), 93 were females (46.27%). Of the 201 participants, 66.67% were white, 3% black, 1.5% Hispanic, and 28.83% opted not to report ethnicity.

Testers

The Testers who conducted the RightEye tests were American Board-Certified optometry doctors. Furthermore, they had undergone a training course on how to administer the RightEye tests, including set-up and test taking procedures. This standardized the testing process and viability of the results. One example of the RightEye training ensured patients were at the correct distance from the screen.

Apparatus

Stimuli were presented using the RightEye tests on NVIDIA 24-inch 3D Vision monitor fitted with an SMI 12" 120 Hz remote eye tracker connected to an Alienware gaming system, and a Logitech (model Y-R0017) wireless keyboard and mouse. The participants were seated in a stationary (non-wheeled) chair that could not be adjusted in height. They sat in from of a desk in a quiet, private testing room. Participants' heads were unconstrained.

The accuracy of the SMI eye tracker was 0.4 degrees within the desired headbox of 32cm x 21cm at 60cm from the screen. For standardization of testing, participants were asked to sit in front of the eye tracking system at an exact measured distance of 60cm (ideal positioning within the headbox range of the eye tracker). A nine-point calibration was conducted with points spanning the computer screen.

Oculomotor Tasks

Five RightEye oculomotor tests are described below. From these 5 tests, 54 different metrics of digitized oculomotor function was assessed (for full description see Appendix I).

Circular smooth pursuit test (CSP). In the CSP test, participants were instructed to track a target stimulus, a black dot of 0.2 degrees' diameter at a 10-degree radius at a rate of 0.4Hz, in a clockwise direction, for 15 seconds. The 0.4 Hz = 1 revolution/0.4 revolutions per sec = 2.5 sec. To find linear velocity, we multiply the angular velocity. The CSP test provides measures of fixation percentages, saccade percentages, latent smooth pursuit, and smooth pursuit target accuracy.

Horizontal smooth pursuit test (HSP). In the HSP test, participants were asked to focus on a dot (same size and speed as the CSP test) on the screen and follow the dot horizontally across the screen for 25 seconds, moving to the far right, then to the far left, and back to the center. The stimuli moved in a sinusoidal way from the left to right and right to left in a straight line. For a participant to be considered "on target," they were required to follow the stimuli within an error of 2.4 degrees. A participant could also be ahead or behind a stimulus and can still be labeled as 'following' if they are within an error of 4.8 degrees. The HSP test also provides measures of fixation percentages, saccade percentages, latent smooth pursuit, and smooth pursuit target accuracy.

Vertical smooth pursuit test (VSP). The protocol for the VSP test was the same as the protocol for the HSP test. However, the VSP test was in a vertical plane.

Horizontal saccades test (HS). In the HS test, participants were asked to look at a countdown of 3, 2, 1 in the center of the screen before moving their eyes back and forth between 2 dots. Their goal was to "target each dot" on the left and right of the screen as quickly and accurately as possible. The dots on the screen turned green when the participants' eyes hit the targets. The test lasted 10 seconds. The HS test provides measures of fixation percentages, saccade percentages, and target accuracy.

Vertical saccades test (VS). The protocol for the VS test was the same as that for the HS test. However, the VS test was in a vertical plane.

Procedure

Participants were recruited through advertisements placed on the internet, social media, bulletin boards, and word of mouth. The study was conducted in accordance with the tenets of the declaration of Helsinki. The study protocols were approved by the Institutional Review Board of East Carolina University. The nature of the study was explained to the participants, and all participants were provided a written University Approved informed consent to participate. Following informed consent, participants were asked to complete a pre-screening questionnaire, to identify four shapes at 4mm in diameter and a basic optometry clinical exam. If any of the pre-screening questions, clinical exam or calibration were failed, then the participant was excluded from the study.

Prescreening Questionnaire: The prescreening questionnaire required participants to self-report any of the following conditions: neurological disorders (such as concussion, traumatic brain injury, Parkinson's Disease, Huntington's Disease, cerebral palsy), consumption of drugs or alcohol within 24 hours of testing. Five participants were excluded from the self-reported conditions.

Basic Optometemy Clinical Exam: During a basic clinical exam with the Optometry doctor, participants were also excluded if they were found to have: vision-related issues (such as extreme tropias (e.g.,[49,50]), phorias (e.g.,[49,50]), static visual acuity of greater than 20/400,[52] nystagmus (e.g.,[52,53]), cataracts, eyelash impediments.[53] Twelve participants were excluded due to the basic clinical exam.

Calibration: Participants were also excluded if they were unable to pass a 9-point calibration sequence. Six participants were excluded as they were unable to pass calibration. Qualified participants who successfully passed the 9-point calibration sequence completed the RightEye tests. The calibration sequence required participants to fixate one at a time on 9 points displayed on the screen. The participants had to successfully fixate on at least 8 out 9 points on the screen to pass the calibration sequence.

For each test, the participant was asked to follow the stimuli as "accurately as possible with their eyes." Written instructions on screen and animations were provided before each test to demonstrate appropriate behavior required in each of the tests.

Data Analysis

Given the three aims of this study, we conducted several statistical analyses. First, the reliability of RightEye Test was evaluated using Cronbach's Alpha (CA). The CA indicates the relative reliability and is interpreted using the following criteria CA > .9 specifies excellent reliability above .7 indicates acceptable, and less than .6 represents poor reliability.[54] The alpha level was set at $p<.05$ for all statistical test.

Second, to describe the normative features of the data, we performed exploratory data analysis and conducted model-based clustering using expectation–maximization (EM) algorithm analysis. We chose this approach because it has several advantages over k-means or hierarchical clustering approaches. First, both k-means and hierarchical approaches are mainly heuristics thus not model-based and not well suited for inference.[55] Second, a model-based approach uses a density function with an associated weight that will 'suggest' the optimal number of clusters. Lastly, the model approach is based on the Bayesian Information Criterion (BIC) values which help to determine the most appropriate clusters. Third, we examined group differences including age clusters and gender with a series of five multivariate ANOVAs, one for each test (CSP, HSP, VSP, HS, and VS).

RESULTS

Test-Retest Reliability Analysis

All fifty-four eye tracking variables from trials 1 and 2 were analyzed using R (statistical package) reliability procedure. Tables 1-5 presents the means and standard deviations for trials 1 and 2, the Cronbach's Alpha correlations between the Trial 1 and Trial 2, and associated the test-retest reliability decisions. Eighty-five percent of eye tracking variables demonstrated *Acceptable* (.7) to Excellent (.9) test-retest reliability. Eight synchronization eye tracking variables were demonstrated *poor reliability* (<.6).

Cluster Analysis

The model-based clustering using EM algorithm analysis created five distinct age group: 5-8, 9-16, 17-28, 29-52, and 53-62. Further, we conducted stability testing to establish that the data sample used for cluster analysis that is representative of the entire population. The stability testing involved sub-sampling 10 individuals from the experimental

Table 1. Test-retest Reliability of Circular Smooth Pursuit Digitized Eye Tracking Variables

Variable	Trial 1 Mean	Trial SD	Trial 2 Mean	Trial 2 SD	CA	Decision
E/T VR (°) (Left)	14.92	3.13	14.89	2.84	0.9	Acceptable
E/T VR (°) (Right)	14.71	2.49	14.75	2.46	0.9	Acceptable
Fixation (%) (Left)	5.12	6.3	5.6	6.84	0.8	Acceptable
Fixation (%) (Right)	5.3	6.23	5.54	6.86	0.7	Acceptable
Sync X (0-1) (Left)	0.88	0.08	0.87	0.08	0.6	Poor
Sync X (0-1) (Right)	0.88	0.08	0.88	0.08	0.6	Poor
On-Target SP (Left)	62.05	22.56	62.72	24.13	0.7	Acceptable
On-Target SP (Right)	61.01	22.25	61.52	21.48	0.7	Acceptable
Saccade (%) (Left)	5.94	5.29	5.47	5.01	0.8	Acceptable
Saccade (%) (Right)	5.74	5.16	5.44	5.18	0.8	Acceptable
Latent SP (%) (Left)	13.85	14.15	13.77	14.56	0.9	Acceptable
Latent SP (%) (Right)	13.99	13.72	13.93	13.12	0.9	Acceptable
SP (Left) (%)	87.46	12.88	88.26	11.33	0.7	Acceptable
SP (Right) (%)	87.83	11.67	88.38	10.77	0.7	Acceptable
Predictive SP (%) (Left)	5.23	8.45	5.09	8.25	0.9	Acceptable
Predictive SP (%) (Right)	6.77	9.6	6.36	9.52	0.9	Acceptable
Sync Y (0-1) (Left)	0.85	0.09	0.86	0.08	0.5	Unacceptable
Sync Y (0-1) (Right)	0.85	0.08	0.85	0.07	0.4	Unacceptable

E/T VR (°) = eye/target velocity error, SP = Smooth pursuit

Table 2. Test-retest Reliability of Horizontal Smooth Pursuit Digitized Eye Tracking Variables

Variable	Trial 1 Mean	Trial 1 SD	Trial 2 Mean	Trial 2 SD	CA	Decision
E/T VR (°) (Left)	18.91	5.27	18.57	5.14	0.7	Acceptable
E/T VR (°) (Right)	18.84	5.03	18.59	4.87	0.7	Acceptable
Fixation (%) (Left)	8	6.63	7.84	6.71	0.8	Acceptable
Fixation (%) (Right)	7.64	6.27	8.26	6.09	0.7	Acceptable
Sync X (0-1) (Left)	0.95	0.07	0.96	0.06	0.3	Unacceptable
Sync X (0-1) (Right)	0.95	0.07	0.96	0.05	0.3	Unacceptable
Saccade (%) (Left)	4.95	5.23	4.63	5.16	0.8	Acceptable
Saccade (%) (Right)	4.92	5.2	4.74	5.36	0.9	Acceptable
SP (Left) (%)	86.54	10.79	86.38	11.34	0.9	Acceptable
SP (Right) (%)	87.05	9.57	86.6	9.74	0.8	Acceptable

Table 3. Test-retest Reliability of Vertical Smooth Pursuit Digitized Eye Tracking Variables

Variable	Trial 1 Mean	Trial SD	Trial 2 Mean	Trial 2 SD	CA	Decision
E/T VR (°) (Left)	23.17	9.2	22.4	9.82	0.9	Acceptable
E/T VR (°) (Right)	23.11	8.96	22.45	9.79	0.8	Acceptable
Fixation (%) (Left)	23.37	11.38	22.03	11.68	0.7	Acceptable
Fixation (%) (Right)	23.38	11.65	22.61	11.87	0.7	Acceptable
Saccade (%) (Left)	24.6	8.54	25.09	9.27	0.7	Acceptable
Saccade (%) (Right)	25	9.24	25.38	10.13	0.7	Acceptable
SP (Left) (%)	50.21	12.95	51.55	12.99	0.7	Acceptable
SP (Right) (%)	50.06	13.3	51.1	12.81	0.7	Acceptable
Sync Y (0-1) (Left)	0.73	0.08	0.73	0.07	0.4	Unacceptable
Sync Y (0-1) (Right)	0.73	0.08	0.73	0.07	0.4	Unacceptable

Table 4. Test-retest Reliability of Horizontal Saccades Digitized Eye Tracking Variables

Variable	Trial 1 Mean	Trial SD	Trial 2 Mean	Trial 2 SD	CA	Decision
Fixation (#) (Left)	17.75	9.76	20.22	8.58	0.7	Acceptable
Fixation (#) (Right)	17.45	9.39	20.1	8.49	0.7	Acceptable
On-Target (#) (Left)	2.57	2.84	2.88	2.86	0.9	Acceptable
On-Target (#) (Right)	2.15	2.65	2.28	2.61	0.9	Acceptable
Saccade (#) (Left)	18.29	9.53	21.04	8.08	0.7	Acceptable
Saccade (#) (Right)	18.38	9.18	21.15	8.18	0.7	Acceptable
All Bandwidths (#) (Left)	9.42	7.07	10.91	6.55	0.7	Acceptable
All Bandwidths (#) (Right)	8.91	6.31	10.63	6.42	0.7	Acceptable

Table 5. Test-retest Reliability of Vertical Saccades Digitized Eye Tracking Variables

Variable	Trial 1 Mean	Trial SD	Trial 2 Mean	Trial 2 SD	CA	Decision
Fixation (#) (Left)	16.01	6.56	17.76	6.66	0.8	Acceptable
Fixation (#) (Right)	15.21	6.9	16.45	6.76	0.8	Acceptable
On-Target (#) (Left)	3.73	3.87	3.92	4.09	0.7	Acceptable
On-Target (#) (Right)	3.87	4.04	3.84	4.06	0.8	Acceptable
Saccade (#) (Left)	16.49	6.72	17.92	6.91	0.7	Acceptable
Saccade (#) (Right)	16.51	6.8	18.06	7.54	0.7	Acceptable
All Bandwidths (#) (Left)	7.25	5.26	8.18	5.26	0.7	Acceptable
All Bandwidths (#) (Right)	7.31	4.86	7.97	5.36	0.7	Acceptable

population for each age group. These sub-samples were then compared against the entire population norm to assess cluster solution (See Figure 1). The comparison of the sample norms and the population norms showed the cluster solution was appropriate in numbers and quality (Calinski-Harabasz Index = 16.61 with average inter-cluster distance = 56.73). The descriptive statistics for all variables derived from the five RightEye tests for the 5 clusters are shown in Tables 6-10.

Group Differences

To provide a descriptive indication of the strength of our cluster solution, we conducted a MANOVA on the multivariate effect of the cluster membership (Age) for each test (CSP, HSP, VSP, HS, and VS). All five MANOVAs revealed a significant multivariate effect on cluster membership thus indicating reasonable support for our cluster solution.

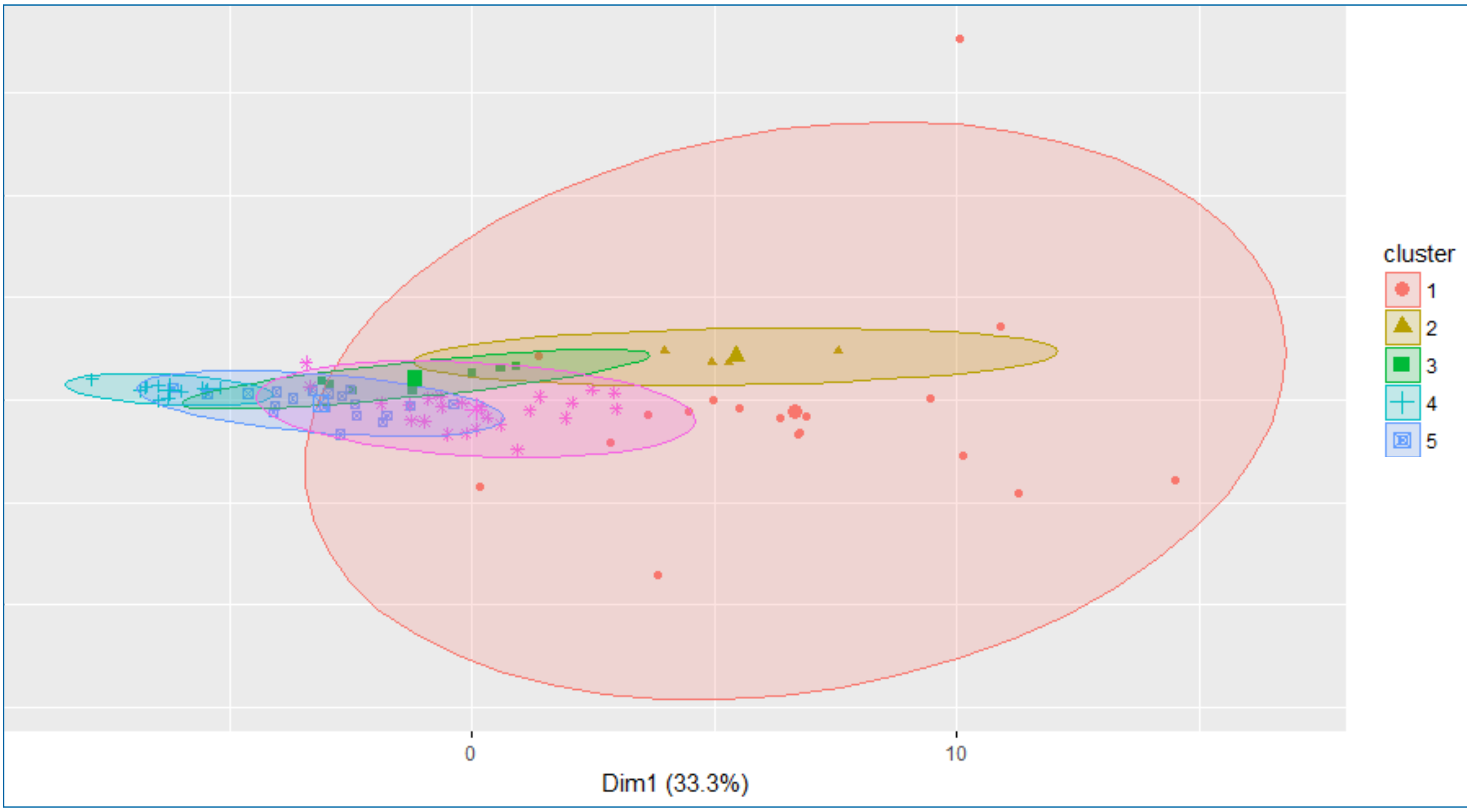


Figure 1: Figure 1: Five Cluster Solution

Table 6. Descriptive Statistics Circular Smooth Pursuit Clustered by Age

	5-8				9-16				17-28				29-52				53-62			
	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper
E/T VR (*) (Left)	17.45	5.18	16.81	18.09	15.62	3.7	15.32	15.91	14.26	1.84	14.1	14.42	14.38	2.91	14.06	14.69	15.11	1.9	14.74	15.48
E/T VR (*) (Right)	17.56	5.13	16.93	18.18	15.84	4.08	15.51	16.16	14.39	2	14.22	14.56	14.36	1.85	14.16	14.56	15.1	1.74	14.76	15.44
Fixation (%) (Left)	8.65	8.98	7.54	9.75	6.26	8.05	5.62	6.91	4.23	5.95	3.72	4.73	3.93	4.05	3.5	4.37	5.39	5.75	4.26	6.52
Fixation (%) (Right)	9.01	9.5	7.85	10.18	6.55	8.02	5.91	7.19	4.35	6.01	3.83	4.86	4.13	3.98	3.71	4.56	5.4	5.39	4.35	6.46
Sync X (0-1) (Left)	0.86	0.08	0.85	0.87	0.88	0.08	0.87	0.89	0.9	0.05	0.89	0.9	0.89	0.05	0.89	0.9	0.89	0.07	0.87	0.9
Sync X (0-1) (Right)	0.85	0.09	0.84	0.86	0.88	0.08	0.88	0.89	0.9	0.05	0.9	0.9	0.9	0.06	0.89	0.9	0.9	0.05	0.89	0.91
On-Target SP (Left)	56.75	21.23	54.15	59.36	63.64	21.8	61.9	65.38	67.35	20.09	65.63	69.06	64.31	20.98	62.07	66.55	62.53	21.81	58.25	66.8
On-Target SP (Right)	54.06	20.45	51.55	56.57	61.24	21.07	59.56	62.93	65.54	19.61	63.86	67.21	63.37	19.92	61.24	65.5	59.16	18.9	55.45	62.86
Saccade (%) (Left)	8.94	6.76	8.11	9.77	6.4	5.48	5.96	6.84	4.61	4.46	4.22	4.99	5.47	5.8	4.85	6.09	6.46	5.07	5.46	7.45
Saccade (%) (Right)	8.74	6.57	7.93	9.54	6.46	6.11	5.97	6.95	4.48	4.91	4.06	4.9	5.12	5.04	4.59	5.66	6.49	5.49	5.42	7.57
Latent SP (%) (Left)	13.54	13.31	11.9	15.17	14.36	14.98	13.17	15.56	16.89	15.43	15.58	18.21	20.47	18.17	18.53	22.41	17.06	18.51	13.44	20.69
Latent SP (%) (Right)	14.44	13.88	12.73	16.14	14.76	14	13.64	15.88	17.14	15.84	15.79	18.49	20.47	17.26	18.63	22.32	17.32	14.19	14.54	20.1
SP (Left) (%)	82.41	12.44	80.88	83.94	87.34	11.11	86.45	88.23	91.17	8.24	90.47	91.87	90.58	8	89.73	91.44	88.15	8.55	86.48	89.83
SP (Right) (%)	82.25	12.84	80.67	83.83	86.99	11.46	86.07	87.9	91.18	8.53	90.45	91.91	90.74	7.61	89.93	91.56	88.1	8.33	86.47	89.73
Predictive SP (%) (Left)	11.54	12.33	10.03	13.05	8.93	12.02	7.97	9.89	6.88	10.47	5.99	7.77	5.7	10.1	4.62	6.78	8.47	11.78	6.16	10.78
Predictive SP (%) (Right)	13.02	11.88	11.57	14.48	10.63	13.55	9.55	11.71	8.42	12.03	7.4	9.45	6.85	10.57	5.72	7.98	11.42	14.29	8.62	14.22
Sync Y (0-1) (Left)	0.84	0.07	0.83	0.85	0.86	0.07	0.86	0.87	0.87	0.07	0.87	0.88	0.86	0.08	0.85	0.87	0.85	0.08	0.84	0.87
Sync Y (0-1) (Right)	0.83	0.07	0.82	0.84	0.85	0.08	0.84	0.85	0.86	0.07	0.85	0.86	0.86	0.06	0.85	0.86	0.85	0.07	0.83	0.86

Table 7. Descriptive Statistics Horizontal Smooth Pursuit Clustered by Age

	5-8				9-16				17-28				29-52				53-62			
	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper
E/T VR (*) (Left)	24.29	8.44	23.26	25.33	20.14	6.63	19.61	20.67	16.97	2.92	16.72	17.22	17.06	3.56	16.68	17.44	17.74	2.91	17.17	18.31
E/T VR (*) (Right)	24.31	8.13	23.32	25.31	20.14	6.37	19.63	20.64	17.15	3.7	16.84	17.47	17.2	3.75	16.8	17.6	17.56	2.53	17.06	18.06
Fixation (%) (Left)	10.07	9.77	8.87	11.27	8.87	7.85	8.24	9.49	6.91	5.55	6.44	7.39	7.26	4.81	6.75	7.78	8.08	6.14	6.88	9.29
Fixation (%) (Right)	10.34	8.68	9.27	11.41	8.94	7.91	8.3	9.57	7.13	5.93	6.62	7.63	7.21	5.11	6.66	7.75	8.41	6.44	7.15	9.67
Sync X (0-1) (Left)	0.94	0.06	0.93	0.95	0.96	0.06	0.95	0.96	0.97	0.02	0.97	0.97	0.97	0.02	0.97	0.97	0.97	0.02	0.96	0.97
Sync X (0-1) (Right)	0.94	0.06	0.93	0.95	0.96	0.06	0.95	0.96	0.97	0.02	0.97	0.97	0.97	0.03	0.97	0.97	0.97	0.02	0.97	0.97
Saccade (%) (Left)	10.7	10.56	9.4	11.99	6.27	7.53	5.67	6.87	3.64	5.62	3.16	4.12	3.93	4.08	3.49	4.36	6.13	10.52	4.07	8.19
Saccade (%) (Right)	10.6	10.77	9.28	11.92	6.32	8.08	5.68	6.97	3.63	5.83	3.13	4.12	3.97	4.28	3.51	4.43	5.4	7.67	3.89	6.9
SP (Left) (%)	79.23	15.53	77.32	81.14	84.87	12.37	83.88	85.85	89.45	8.9	88.69	90.21	88.81	7.49	88.01	89.61	85.78	12.33	83.37	88.2
SP (Right) (%)	79.06	14.85	77.24	80.88	84.74	12.44	83.75	85.73	89.25	9.32	88.45	90.04	88.83	7.79	87.99	89.66	86.2	10.86	84.07	88.32

Table 8. Descriptive Statistics Vertical Smooth Pursuit Clustered by Age

	5-8				9-16				17-28				29-52				53-62			
	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper
E/T VR (*) (Left)	35.33	14.5	33.35	37.11	26.76	13.59	25.68	27.85	19.42	8.05	18.73	20.1	20.32	7.57	19.51	21.13	22.7	8.94	20.95	24.46
E/T VR (*) (Right)	35.27	14.09	33.55	37	27.17	14.69	26	28.34	19.76	12.72	18.67	20.84	20.26	7.6	19.45	21.08	22.23	8.56	20.55	23.9
Fixation (%) (Left)	28.24	12.51	26.7	29.77	26	11.88	25.05	26.95	20.16	10.35	19.28	21.04	20.06	8.76	19.3	21	19.49	9.4	17.64	21.33
Fixation (%) (Right)	28.28	13.33	26.65	29.92	25.92	12.02	24.96	26.88	20.76	10.24	19.89	21.63	20.22	9.07	19.25	21.19	20.49	9.05	18.71	22.26
Saccade (%) (Left)	26.72	11.35	25.33	28.11	24.2	8.68	23.51	24.9	24.4	9.52	23.59	25.21	26.46	9.31	25.46	27.45	28.32	11.16	26.13	30.51
Saccade (%) (Right)	26.6	11.35	25.2	27.99	24.42	9.58	23.66	25.18	23.96	9.85	23.12	24.8	26.24	9.61	25.21	27.27	29.69	12.79	24.18	29.19
SP (Left) (%)	45.11	12.35	43.59	46.62	49.79	12.26	48.81	50.77	55.44	12.04	54.41	56.47	53.52	10.83	52.37	54.68	52.09	10.97	49.94	52.24
SP (Right) (%)	45.14	12.85	43.57	46.72	49.65	12.46	48.65	50.64	55.28	12.15	54.25	56.32	53.56	11.28	52.36	54.77	52.74	11.21	50.54	54.94
Sync Y (0-1) (Left)	0.69	0.1	0.68	0.7	0.71	0.08	0.7	0.71	0.74	0.06	0.73	0.74	0.74	0.06	0.73	0.74	0.73	0.06	0.72	0.74
Sync Y (0-1) (Right)	0.69	0.1	0.68	0.7	0.7	0.08	0.7	0.71	0.74	0.06	0.73	0.74	0.74	0.06	0.73	0.74	0.73	0.06	0.71	0.74

Table 9. Descriptive Statistics Horizontal Saccades Clustered by Age

	5-8				9-16				17-28				29-52				53-62			
	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper
Fixation (%) (Left)	12.77	10.14	11.53	14.02	16.31	7.87	15.68	16.94	20.88	9.28	20.09	21.67	16.18	7.6	15.37	16.99	15.58	7.86	14.04	17.12
Fixation (%) (Right)	12.42	7.74	11.47	13.37	16.15	7.4	15.56	16.74	20.73	9.14	19.95	21.5	1625	7.82	15.41	17.09	15.58	7.9	14.03	17.13
On-Target (#) (Left)	2.14	2.2	1.87	2.41	3.06	2.98	2.83	3.3	3.7	3.47	3.41	4	2.9	3.17	2.57	3.24	2.95	3	2.36	3.54
On-Target (#) (Right)	2.04	2.37	1.75	2.33	2.78	2.68	2.56	2.99	3.43	3.27	3.15	3.71	2.89	2.95	2.57	3.2	2.69	3.11	2.08	3.3
Saccade (#) (Left)	13.85	9.7	12.66	15.05	17.2	7.62	16.6	17.81	21.48	9.02	20.72	22.25	17	7.39	16.21	17.79	16.35	7.46	14.89	17.81
Saccade (#) (Right)	13.63	7.22	12.74	14.51	17.21	7.3	16.62	17.79	21.49	9.06	20.72	22.26	16.91	7.26	16.13	17.68	16.35	7.2	14.94	17.76
All Bandwidths (#) (Left)	5.78	3.9	5.3	6.26	8.23	5.22	7.82	8.65	11.2	6.51	10.65	11.76	8.83	5.63	8.23	9.43	8.07	5.3	7.03	9.11
All Bandwidths (#) (Right)	5.69	3.63	5.25	6.14	8.22	4.96	7.82	8.61	10.83	6.35	10.29	11.37	9.02	5.75	8.4	9.63	7.81	5.82	6.67	8.95

Table 10. Descriptive Statistics Verticall Saccades Clustered by Age

	5-8				9-16				17-28				29-52				53-62			
	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper	Mean	SD	CL Lower	CL Upper
Fixation (%) (Left)	11.44	4.74	10.85	12.02	15.05	6.1	14.56	15.53	19.88	7.23	19.27	20.5	16.74	6.41	16.05	17.42	15.98	7.09	14.59	17.37
Fixation (%) (Right)	11.53	4.74	10.95	12.02	15.03	6.28	14.53	15.53	19.84	7.02	19.24	20.44	16.63	6.26	15.96	17.3	16.12	8.05	14.54	17.7
On-Target (#) (Left)	2.13	2.28	1.85	2.41	3.13	3.04	2.88	3.37	4.59	4.24	4.23	4.95	4.12	3.8	3.71	4.53	3.77	4.18	2.95	4.59
On-Target (#) (Right)	2.12	2.37	1.83	2.41	3.11	3.04	2.87	3.36	4.48	4.26	4.12	4.85	4.34	4.11	3.9	4.78	4.17	4.44	3.3	5.04
Saccade (#) (Left)	12.97	4.82	12.38	13.56	16.22	5.88	15.75	16.69	20.91	6.99	20.31	21.51	17.72	5.98	17.08	18.36	16.81	6.98	15.44	18.18
Saccade (#) (Right)	13.05	4.63	12.48	13.61	16.15	6.16	15.66	16.65	20.95	6.9	20.36	21.53	17.65	6.03	17.01	18.3	16.87	7.41	15.42	18.32
All Bandwidths (#) (Left)	4.9	3.3	4.49	5.3	7.17	4.51	6.81	7.53	10.01	5.59	9.53	10.48	8.05	4.86	7.53	8.57	7.44	4.3	6.6	8.28
All Bandwidths (#) (Right)	4.92	3.27	4.52	5.32	6.92	4.47	6.56	7.27	10.02	5.5	9.56	10.49	8.01	4.72	7.5	8.51	7.12	4.74	6.19	8.05

CSP Test

The MANOVA for the CSP Test revealed a significant multivariate effect on cluster membership, *Wilks' Lambda = .829, F*(64, 11,374) = 8.69, *p* < .0001. Descriptive CSP statistics for the five clusters were evaluated by separate one-way analysis of variance. The follow-up ANOVAs revealed significant Age Cluster differences for all circular smooth pursuit variables (p < .001). Tukey post hoc analysis for CSP variables indicated there were no significant differences between Age Clusters 17-28 and 29-52 however, these clusters were significantly different from Age Clusters 5-8, 9-16, and 53-62 for E/T VR Error, Fixation (%), On-Target SP, Saccade (%), Latent SP, and Predictive SP. Age Cluster 5-8 significantly differed from each Age Cluster (i.e., 9-16; 17-28; 29-52; and 53-62) for all CSP variables.

HSP Test

Similarly, the MANOVA for the HSP Test demonstrated a significant multivariate effect on cluster membership, *Wilks' Lambda = .729, F*(32, 7889.837) = 15.845, *p* < .0001. The follow-up ANOVAs for HSP further supported our cluster solution as significant Cluster differences were found for all HSP variables (p < .001). Age Clusters 17-28, 29-52, and 53-62 did not differ for E/T VR, Saccade %, and SP %, however, were significantly different for the remaining Age Clusters (i.e.,5-8, 9-16). Age Cluster 5-8 differed on all clusters for all HSP variables except Fixation %. In this case, Age Cluster 5-8 was not significantly different from Clusters 5-8, 9-16, and 53-62.

VSP Test

Likewise, the MANOVA for the VSP Test also showed a significant multivariate effect on cluster membership, *Wilks' Lambda = .739, F*(32, 7528.43) = 20.11, *p* < .0001. The follow-up ANOVAs for VSP also supported our cluster solution as significant Age Cluster differences were found for all VSP variables (p < .001) and Tukey's Post Hoc test demonstrated the same findings as the HSP Test.

HS Test

For the Horizontal Saccade Test, the MANOVA revealed a significant multivariate effect on cluster membership, *Wilks' Lambda = .851, F*(32, 10,486.01) = 14.684, *p* < .0001. Our Cluster solution was support by significant follow-up ANOVA for all HS variables (p < .001). Post Hoc test revealed Cluster 5-8 and Cluster 17-28 were significantly different from Clusters 9-16, 29-52, and 53-62 on Fixation %, On-target %, Saccade %, and All Bandwidths.

VS Test

Lastly, the Vertical Saccade Test revealed a significant multivariate effect on cluster membership, *Wilks' Lambda = .817, F*(32, 7972.35) = 12.956, *p* < .0001. Similar to the other analyses, follow-up ANOVAs for each

VS test demonstrated support for our Cluster solution as all VSP variables were significantly different ($p < .0001$). Post Hoc test revealed the Age Cluster 5-8 was significantly different on all variables. Age Cluster 17-28 differed from the all Age Clusters on All Bandwidths, Saccade, and Fixation %.

DISCUSSION

The purposes of this study were to use an empirical, data-driven approach to examine the reliability of RightEye Neuro Vision and to determine the normative values of OMF metrics for healthy individuals, and to cluster these variables by age through cluster analysis.

Reliability of RightEye Tests

Eighty-five percent of variables resulted in acceptable or higher reliability. Synchronization was the only unreliable metrics within smooth circular pursuit and vertical pursuit. Synchronization analysis, in this study, is modeled by separating the horizontal (x-axis) and vertical (y-axis) components of the eye position in relation to the same components of the target's position, as proposed by Contreras, et al.[8] However, there are no known tests of reliability for synchronization in previous literature, and thus questions group differences usually found using synchronization metrics via this method. Future experiments should analyze all eye movement metrics for reliability and explore other methods of quantifying synchronization such as that outlined by Samadini and colleagues.[56] The remaining tests, including circular smooth pursuit, horizontal smooth pursuit, vertical smooth pursuit, vertical saccade, and horizontal saccade, demonstrated excellent reliability and potentially represents an acceptable alternative to standard bedside clinical assessment. The circular smooth pursuit (CSP) test is not typically found in clinical practice primarily because it involves recruiting many areas of brain circuitry,[57] the clinical relevance is not entirely clear, and there has been a lack of reliable circular smooth pursuit (CSP) test. With reliable CSP, horizontal smooth pursuit, and vertical smooth pursuit tests, it may be possible to examine how competing signals are affected by brain injury, patient state, or disease state. For example, recent research has demonstrated functional differences in circular smooth pursuit for TBI/concussion (e.g.,[58,59]) and others have indicated the influence of drug intervention on smooth pursuit.[60] Additionally, deficits in smooth pursuit eye movement may be driven by impairments in low-level motion processing x and/or higher-level predictive mechanisms.[63,64] Lastly, for all smooth pursuit test the sampling rate was 120 Hz and recent research has demonstrated this to be a sufficient sampling rate to detect and reliably analyze smooth pursuit (e.g.,[65,66]).

Vertical saccade and horizontal saccade RightEye are similar to clinical "bedside" evaluation and produced reliable data which is not always seen in clinical practice. A reliable test could be the first line of evaluation rather than a follow-up to suspected saccadic abnormality. As noted before, the "bedside" evaluation involves asking the patient to alternatively fixate on two targets.[7] This represents not only a shift of attention from one target to another but also a measure of oculomotor performance. As is found here, saccades were measured regarding their accuracy and could indicate lesions in frontal eye fields, motor neurons and oculomotor nerves, Basal Ganglia deficits, etc.[7]

Cluster Analysis

The cluster analysis represents a robust method to demonstrate distinct groups by age. We observed 5 distinct clusters which indicate the need to consider age ranges in an oculomotor test. The MANOVAs for circular, vertical, and horizontal smooth pursuit, horizontal saccades, and vertical saccades revealed a significant multivariate effect on cluster membership for Age, thus indicating reasonable support for our cluster solution.

Follow-up analysis indicated a majority of the eye tracking variables represent distinct differences for Age. Most measurements demonstrate a curvilinear relationship with peaks occurring for the 17-28 age groups and 29-58 age groups (See Figures 2, 3, 4 and 5 as examples). The results are in-line research indicating saccadic control increases from ages 3-14 and saccade latencies decrease until age 15.[67] In addition, other investigators have noted age-related declines in smooth pursuit and saccades (e.g.,[66]) and the underlying visual central-peripheral integration mechanisms such as those of the DLPFC.[68]

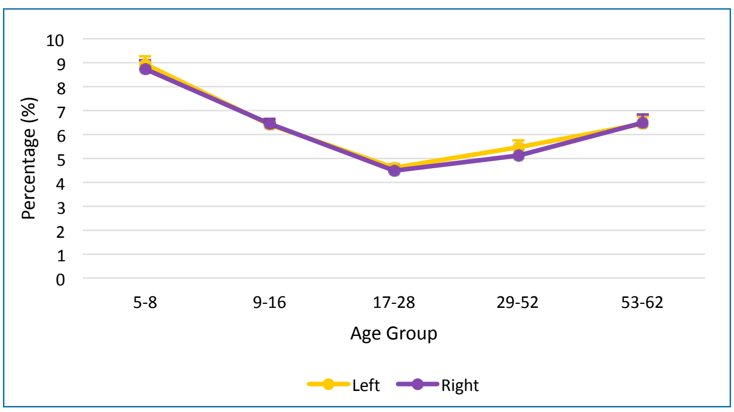


Figure 2: Circular Smooth Pursuit: Saccades (%)

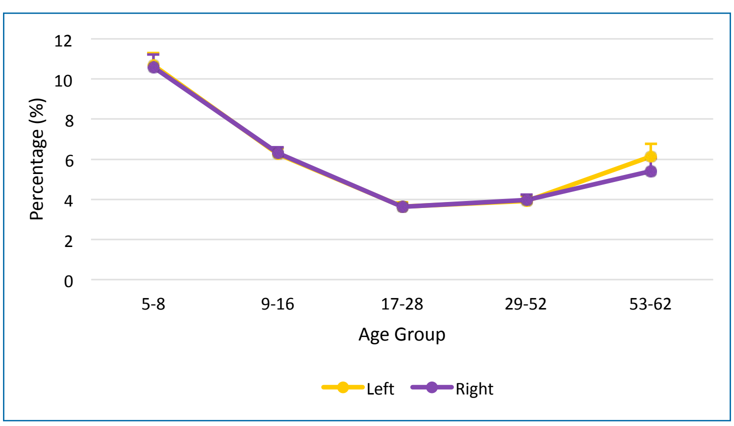


Figure 3: Horizontal Smooth Pursuit: Saccades (%)

The oculomotor function develops from childhood and into adulthood. Complex aspect of the visual systems tend to stabilize in later adolescent and remain stable until late adulthood.[69] Evaluation of oculomotor function is a relatively simple and potentially cost-effective approach to assess neurophysiological and neurodegenerative disorders and injury,

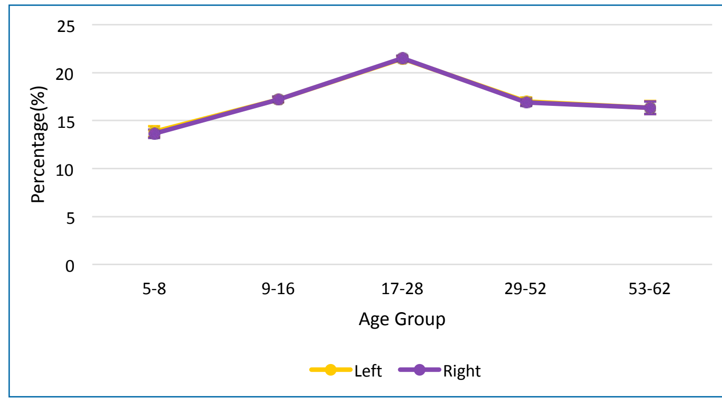


Figure 4: Horizontal Saccade: Saccades (%)

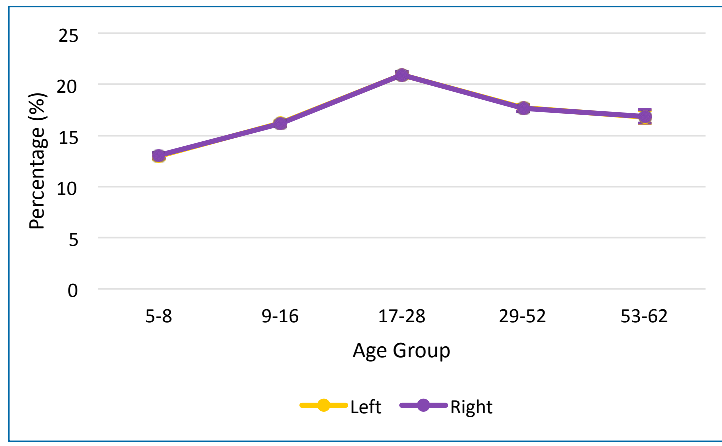


Figure 5: Vertical Saccades: Saccades (%)

however, future research should examine age as factor given a neurological disorder or injury. Furthermore, future research should also consider sustained static fixations as both a dedicated pre-screening method and for evaluation across age-span to identify normative data. More rigorous pre-screening methods, such as those outlined in Quaid & Simpson,[8] may also help to reduce the variability within groups and possibly identify further clusters across the lifespan.

CONCLUSION

Overall, the results demonstrated the RightEye reliable, and the clustering method presented here represents a reasonable method to demonstrate distinct differences in eye tracking variables by Age. Findings represent the sensitivity of OMF measures and the importance to consider individual and group characteristics for clinical applications as well as applied settings. Future studies should also consider normative values for OMF variables to enhance interpretation of findings. Furthermore, group analysis indicates the need to consider individual characteristics in eye tracking research.

REFERENCES

1. Michael, L., & Benjamin, T. (2009). Looking and acting: Vision and eye movements in natural behaviour. Oxford University Press: Oxford.
2. Barnes, G. (2008). Cognitive processes involved in smooth pursuit eye movements. Brain and Cognition, 68, 309-326.
3. Duchowski, A. T. (2007). Eye tracking methodology: Theory and practice. New York Springer
4. Poole, A., & Ball, L. J. (2005). Eye tracking in human-computer interaction and usability research: Current status and future prospects. In C. Ghaoui (Ed.) Encyclopedia of Human-Computer Interaction. Pennsylvania Idea Group: Hershey, PA.
5. Møllenbach, E., Hansen, J. P., & Lillholm, M. (2013). Eye movements in gaze interaction. Journal of Eye Movement Research 6, 1-15.
6. Komogortsev, O. V., & Karpov, A. (2013). Automated classification and scoring of smooth pursuit eye movements in the presence of fixations and saccades. Behavior Research Methods, 45, 203-215.
7. Leigh, R.J. & Zee, D.S. (2015). The Neurology of Eye Movements. 4th Ed. Oxford University Press. New York, New York.
8. Quaid P, & Simpson T. (2013). Association between reading speed, cycloplegic refractive error, and oculomotor function in reading disabled children versus controls. Graefe's Archive for Clinical and Experimental Ophthalmology; 251: 169-87.
9. McDowell, J. E., Clementz, B. A. (1997). The effect of fixation condition manipulations on antisaccade performance in schizophrenia: studies of diagnostic specificity. Experimental Brain Research, 115, 333-344.
10. Sereno, A. B., & Holzman, P. S. (1995). Antisaccades and smooth pursuit eye movements in schizophrenia. Biological Psychiatry, 37, 394-401.
11. Gitchel, G.T., Wetzel., P.A., & Baron, M.S. (2012). Pervasive ocular tremor in patients with Parkinson disease. Archives of Neurology, 70, E1-7. doi:10.1001/archneurol.2012.70.
12. Gitchel, G.T., Wetzel., P.A., & Baron, M.S. (2013). Slowed saccades and increased square wave jerks in essential tremor. Tremor and Other Hyperkinetic Movements, 3, 1-7.
13. Munoz, D. P., Armstrong, I. T., Hampton, K. A., Moore, K. D. (2003). Altered control of visual fixation and saccadic eye movements in attention-deficit hyperactivity disorder. Journal of Neurophysiology, 90, 503-514.
14. Overton, P. G. (2008). Collicular dysfunction in attention deficit hyperactivity disorder. Medical Hypotheses, 70, 1121-1127.
15. Paolozza, A., Titman, R., Brien, D., Munoz, D. P., & Reynolds, J. N. (2013). Altered accuracy of saccadic eye movements in children with fetal alcohol spectrum disorder. Alcoholism, clinical and experimental. Alcoholism, Clinical and Experimental Research, 37, 1491-1498.
16. Mueller, S. C., Ng, P., Temple, V., et al. (2010). Perturbed reward processing in pediatric bipolar disorder: an antisaccade study. Journal of Psychopharmacology, 24, 1779-1784.
17. Kaufman, D. M. (2007). Clinical Neurology for Psychiatrists. Elsevier: Philadelphia, PA
18. Jazbec, S., McClure, E., Hardin, M., Pine, D. S., & Ernst, M. (2005). Cognitive control under contingencies in anxious and depressed adolescents: an antisaccade task. Biological Psychiatry, 58, 632-639.
19. Bigler, Erin D. 2013. Neuroimaging biomarkers in mild Traumatic Brain Injury (mTBI). Neuropsychology Review 23, 169-209.
20. Munoz, D. P., & Coe, B. C. (2011). Saccade, search and orient - the neural control of saccadic eye movements. European Journal of Neuroscience, 33, 1945-1947.
21. Munoz, D. P., & Everling, S. (2004). Look away: The anti-saccade task and the voluntary control of eye movement. Nature Reviews Neuroscience, 5, 218-228.
22. Alvarez, T. L., Alkan, Y., Gohel, S., Ward, B. D., & Biswal, B. B. (2010). Functional anatomy of predictive vergence and saccade eye movements in humans: A functional MRI investigation. Vision Research, 50, 2163-2175.
23. Burke, M. R., & Barnes, G. R. (2006). Quantitative differences in smooth pursuit and saccadic eye movements. Experimental Brain Research, 175, 596-608.
24. Ito, N., Barnes, G. R., Fukushima, J., Fukushima, K., & Warabi, T. (2013). Cue-dependent memory-based smooth-pursuit in normal human subjects: Importance of extra-retinal mechanisms for initial pursuit. Experimental Brain Research, 229, 23-35.
25. Gaertner C. & Kapoula Z. (2015). Up/down anisotropies of vertical saccades in healthy children from 6 to 9 years of age. Investigative Ophthalmology and Visual Science, 56, 1901-1908.
26. Vernet, M., Yang, Q,, Gruselle, M., Trams, M., & Kapoula, Z. (2009) Switching between gap and overlap pro-saccades: cost or benefit? Experimental Brain Research, 197,49- 58.
27. Laby, D. M., Davidson, J. L., Rosenbaum, L. J., Strasser, C., Mellman, M. F., Rosenbaum, A. L., Kirschen, D. G. (1996). The Visual Function of Professional Baseball Players. American Journal of Ophthalmology, 122, 476-485.

28. Murray, N. P., Hunfalvay, M., & Bolte, T. (2017). The Reliability, Validity, and Normative Data of Interpupillary Distance and Pupil Diameter Using Eye-Tracking Technology. Translational Vision Science & Technology, 6 (4), 1-12.

29. Croft, J.L., Button, C., & Dicks, M. (2010). Visual strategies of sub-elite cricket batsmen in response to different ball trajectories. Human Movement Science, 29, 751-763. doi:10.1016/j.humov.2009.10.004.

30. Hunfalvay, M., Orr, R., Murray, N., & Roberts, C.M. (2017). Evaluation of Stereo Acuity in Professional Baseball and LPGA Athletes Compared to Non-Athletes. Vision Development and Rehabilitation, 3 (10) 33-41.

31. Oudejans, R.R.D., Michaels, C.F., & Bakker, F.C. (2011). The effects of baseball experience on movement initiation in catching fly balls. Journal of Sport Sciences, 15, 587-595. doi.org/10.1080/026404197367029.

32. Beilock, S. L., Wierenga, S. A., & Carr, T. H. (2002). Expertise, attention and memory in sensorimotor skill execution: Impact of novel task constraints on dual-task performance and episodic memory. The Quarterly Journal of Experimental Psychology A: Human Experimental Psychology, 55A, 1211-1240.

33. Williams, M.A. & Ward, P. (2003) Perceptual expertise: Development in sport. In J. L., Starkes, J & K. A., Ericsson (Eds.). Expert performance in sports: Advances in research on Ssort expertise by Human Kinetics, Champaign, IL.

34. Tenenbaum, G. (2003). Expert athletes: An integrated approach to decision making. In J. L., Starkes, J & K. A., Ericsson (Eds.). Expert performance in sports: Advances in research on Ssort expertise by Human Kinetics, Champaign, IL.

35. Verburgh, L., Scherder, E.J.A., Lange, A.M., & Oosterlaan, J. (2016). Do elite and amateur soccer players outperform non-athletes on neurocognitive functioning? A study among 8-12 year old children. PLoS One,11,1-12. doi:10.1371/journal.pone.0165741

36. Bedell, H. E., & Stevenson, S. B. (2013). Eye movement testing in clinical examination. Vision Research, 90, 32-37. doi: 10.1016/j.visres.2013.02.001

37. Mucha, A., Collins, M.W., Elbin, R.J., Furman, J.M., Troutman-Enseki, C., DeWolf, R.M., Marchetti, G., & Kontos, A.P. (2014). A brief vestibular/ocular motor screening (VOMS) assessment to evaluate concussions: Preliminary findings. American Journal of Sports Medicine, 42, 2479-2486.

38. Singh, H., Giardina, T.D., Meyer, A.N.D., Forjuoh, S.N., Reis, M.D., Thomas, E.J. (2013). Types and origins of diagnostic errors in primary care settings. JAMA Internal Medicine, 173, 418-425. doi:10.1001/jamainternmed.2013.2777.

39. Winters, B., Custer, J., Galvagno Jr, S.M., Colantuoni, E., Kapoor, S.G., Lee, H.W., et al., (2012). Diagnostic errors in the intensive care unit: A systematic review of autopsy studies. BMJ Quality and Safety, 21, 894-902. doi:10.1136/bmjqs-2012-000803.

40. Ciuffreda, K. J., Kapoor, N., Rutner, D., Suchoff, I. B., Han, M., & Craig, S. (2007). Occurrence of oculomotor dysfunctions in acquired brain injury: A retrospective analysis. Optometry - Journal of the American Optometric Association, 78, 155-161.

41. Contreras, R., Kolster, R., Voss, H. U., Ghajar, J., Suh, M., & Bahar, S. (2008). Eye-Target Synchronization in Mild Traumatic Brain-injured Patients. Journal of Biological Physics, 34, 381-392.

42. Samadani, U., Ritlop, R., Reyes, M., Nehrbass, E., Li, M., Lamm, E., Huang, P. (2015). Eye tracking detects disconjugate eye movements associated with structural traumatic brain injury and concussion. Journal of Neurotrauma, 32, 548-556.

43. Suh, M., Basu, S., Kolster, R., Sarkar, R., Mccandliss, B., & Ghajar, J. (2006). Increased oculomotor deficits during target blanking as an indicator of mild traumatic brain injury. Neuroscience Letters, 410, 203-207.

44. Pel, J. J. M., Manders, J. C. W., & van der Steen, J. (2010). Assessment of visual orienting behavior in young children using remote eye tracking: Methodology and reliability. Journal of Neuroscience Methods, 189, 252-256. doi:10.1016/j.jneumeth.2010.04.005.

45. Bargary, G., Bosten, J. M., Goodbourn, P. T., Lawrance-Owen, A.J., & Hogg, R.E. (2017). Individual differences in human eye movements: An oculomotor signature? Vision Research. 141, 157-169.

46. Evdokimidis, I., Smyrnis, N., Constantinidis, T., Stefanis, N., Avramopoulos, D., Paximadis, C., Theleritis, C., Efstratiadis, C., Kastrinakis, G., & Stefanis, G. (2002). The antisaccade task in a sample of 2,006 young men. Experimental Brain Research, 147,45-52.

47. Lenzenweger, M. F., & O'Driscoll, G. A. (2006). Smooth pursuit eye movement and schizotypy in the community. Journal of Abnormal Psychology, 115, 779-786. doi:10.1037/0021-843X.115.4.779.

48. Niehorster, D.C., Cornelissen, T.H.W., Holmqvist, K., Hooge, I.T.C., & Hessels, R.S. (2017). What to expect from your remote eye-tracker when participants are unrestrained. Behavior Research, 1-15. doi:10.3758/s13428-017-0863-0

49. Renard, D., Ferraro, A., Lorenzini, M-C., Jean, L., Portal, M-C., Llinares, E., Labauge, P., & Castelnovo, G. (2015). Orthoptic and video oculographic analyses in oculopharyngeal muscular dystrophy. Muscle Nerve, 52, 554-558.

50. Han, S.J., Guo, Y., Granger-Donetti, B., Vicci, V.R., & Alvarez, T.L. (2010). Quantification of heterophoria and phoria adaptation using automated objective system compared to clinical methods. Opthalmic and Physiological Optics. 30, 95-107.

51. Kooiker, M.J.G., Pel, J.J.M., Verbunt, H.J.M., de Wit, G.C., van Genderen, M.M., & van der Steen, J. (2016). Quantification of visual function assessment using remote eye tracking in children: Validity and applicability. Acta Ophthalmologica, 94, 599-608. doi:10.1111/aos.13038.

52. Holmqvist K, & Nystrom M. (2011). Eye Tracking: A Comprehensive Guide to Methods and Measures. Oxford University Press.

53. Fleiss, J.L. (1986) Design and analysis of clinical experiments. Willey, New York.

54. Hill, S. M. and Mukherjee, S. (2013). Network-based clustering with mixtures of L1-penalized Gaussian graphical models: An empirical investigation. The Annals of Applied Statistics, 7(4), 2157-2179.

55. Samadani, U., Li, M., Qian, M., Laska, E., Ritlop, R., Kolecki, R., Marmar, C. (2015). Sensitivity and specificity of an eye movement tracking-based biomarker for concussion. Concussion, 1-14.

56. Ilg U. J., & Their, P. (2008). The neural basis of smooth pursuit eye movements in the rhesus monkey brain. Brain and Cognition, 68, 229-240. doi:10.1016/j.bandc.2008.08.014.

57. Hunfalvay, M. (2017). Lingering binocular vision issues after a suspected concussion: A case study. Translational Biomedicine, 8, 115.

58. Zhang, K. (2014). Multimodal functional neuroimaging of cculomotor deficits in athletes with concussion (Doctoral dissertation). Retrieved from: http://bit.ly/33ZzcoC.

59. Naicker, P., Anoopkumar-Dukie, S., Grant, G. D., & Kavanagh, J. J. (2017). Medications influencing central cholinergic neurotransmission affect saccadic and smooth pursuit eye movements in healthy young adults. Psychopharmacology, 234, 63-71. doi:10.1007/s00213-016-4436-1.

60. Chen, Y. (2011) Abnormal visual motion processing in schizophrenia: a review of research progress. Schizophrenia Bulletin, 37, 709-715.

61. Nagel, M., Sprenger, A., Steinlechner, S., Binkofski, F., & Lencer, R. (2012). Altered velocity processing in schizophrenia during pursuit eye tracking. PLoSOne 7, e38494.

62. Hong, L. E., Turano, K. A., O'Neill, H., Hao, L., Wonodi, I., McMahon, R. P., Elliott, A., & Thaker, G. K. (2008) Refining the predictive pursuit endophenotype in schizophrenia. Biological Psychiatry, 63, 458-464.

63. Keedy, S. K., Ebens, C. L., Keshavan, M. S., & Sweeney, J. A. (2006). Functional magnetic resonance imaging studies of eye movements in first episode schizophrenia: smooth pursuit, visually guided saccades and the occularmotor delayed response task. Psychiatry Research, 146, 199 -211.

64. Gibaldi, A., Vanegas, M., Bex, P. J., & Maiello, G. (2017). Evaluation of the Tobii EyeX Eye tracking controller and Matlab toolkit for research. Behavior Research Methods, 49, 923-946. doi:10.3758/s13428-016-0762-9.

65. Seferlis, F., Chimona, T. S., Papadakis, C. E., Bizakis, J., Triaridis, S., & Skoulakis, C. (2015). Age related changes in ocular motor testing in healthy subjects. Journal of Vestibular Research: Equilibrium & Orientation, 25, 57-66.

66. Irving, E.L., Steinbach, M.J., Lillakas, L., Babu, R.J., Hutchings, N. (2006) Horizontal saccade dynamics across the human life span. Investigative Ophthalmology & Visual Science, 47(6), 2478-2484.

67. Sharma S., Ray, B., Bhardwaj, D., Dwivedi, A. K., & Roy, T.S. (2009). Age changes in the human oculomotor nerve— a stereological study. Annals of Anatomy 191,260- 266.

68. Luna, B., Velanova, K., Geier C.F. (2008). Development of eye-movement control. Brain and Cognition, 68(3), 293-308.

69. Lange, B., Hunfalvay, M., Murray, N., Roberts, C.-M. & Bolte, T. (2018). Reliability of computerized eye-tracking reaction time tests in non-athletes, athletes, and individuals with traumatic brain injury. Optometry & Visual Performance, 6 (3), 165-180.

AUTHOR BIOGRAPHY:
Melissa Hunfalvay, PhD
Bethesda, Maryland, USA

A vision scientist and practitioner using eye tracking technology, Dr. Hunfalvay has written, assessed and trained on the science of eye tracking for 20 years. She has worked with professional athletes, Military personnel and others for clinical and performance improvement of the visual system.

Appendix I

SMOOTH PURSUIT
Smooth Pursuit (%): are eyes movements that follow the target within a velocity range of the target and are reported as a percentage of the test time.
Saccade (%): are fast eye movements, that move the eyes from one point of interest to the next. They are calculated outside (above or below) the velocity range of the target and reported as a percentage of test time.
Fixation (%): is a stopping point of the eye that allows the user to see in detail. Fixations are reported as a percentage of the test time.
Eye/Target Velocity Error (°/s): refers to how far the user's eyes were away from the target (non-directional). This metric is calculated by subtracting the location of the stimuli and the user's eyes at same sample time, and reported as degrees per second.
Horizontal synchronization SPEM (0-1): refers to how far off on the X plane (co-ordinate) the user's eyes were during the test. Perfect synchronization is a score of 1.0.
Vertical Synchronization SPEM (0-1): refers to how far off on the Y plane (co-ordinate) the user's eyes were during the test. Perfect synchronization is a score of 1.0.
On Target Smooth Pursuit (%): refers to the user's eyes within a velocity range of the target and positioned on the stimuli within and 2cm and reported as a percentage.
Predictive Smooth Pursuit (%): refers to the user's eyes within a velocity range of the target and positioned ahead or in-front-of the stimuli between 2 and 5cm and reported as a percentage.
Latent Smooth Pursuit (%): refers to the user's eyes within a velocity range of the target and positioned behind the stimuli between 2 and 5cm and reported as a percentage.

SACCADES
Smooth Pursuit (%): are eyes movements that follow the target within a velocity range of the target and are reported as a percentage of the test time.
Saccade (%): are fast eye movements, that move the eyes from one point of interest to the next. They are calculated outside (above or below) the velocity range of the target and reported as a percentage of test time.
Fixation (%): is a stopping point of the eye that allows the user to see in detail. Fixations are reported as a percentage of the test time.
Eye/Target Velocity Error (°/s): refers to how far the user's eyes were away from the target (non-directional). This metric is calculated by subtracting the location of the stimuli and the user's eyes at same sample time, and reported as degrees per second.
Horizontal synchronization SPEM (0-1): refers to how far off on the X plane (co-ordinate) the user's eyes were during the test. Perfect synchronization is a score of 1.0.
Vertical Synchronization SPEM (0-1): refers to how far off on the Y plane (co-ordinate) the user's eyes were during the test. Perfect synchronization is a score of 1.0.
On Target Smooth Pursuit (%): refers to the user's eyes within a velocity range of the target and positioned on the stimuli within and 2cm and reported as a percentage.
Predictive Smooth Pursuit (%): refers to the user's eyes within a velocity range of the target and positioned ahead or in-front-of the stimuli between 2 and 5cm and reported as a percentage.
Latent Smooth Pursuit (%): refers to the user's eyes within a velocity range of the target and positioned behind the stimuli between 2 and 5cm and reported as a percentage.

CALENDAR OF EVENTS

ONLINE TRAINING
COVD Endorsed

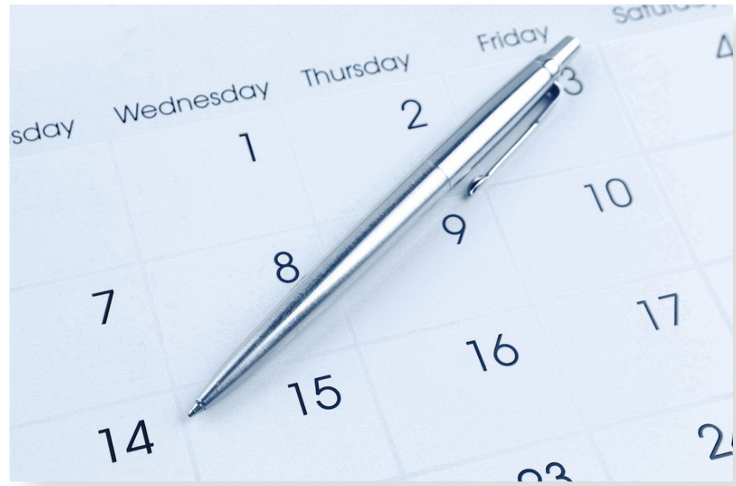
JANUARY 2020
January 23-25, 2019
Nassau, Bahamas
AOE Winter Meeting
Dry Eye Summit | January 23
Myopia Control | January 24
Neuro Optometry & Vision Therapy | January 25

FEBRUARY 2020
February 6-8
San Diego, California
International Sports Vision Association Annual Conference

MARCH 2020
March 20-22
Scarborough, Ontario, Canada
Comprehensive Foundational Course in Vision Therapy
COVD Endorsed
Multiple dates throughout Spring/Summer 2020

Optometrist's Program
100-hour course taking place over five weekends during Spring/Summer 2020. Register for the whole program or individual weekends.

Vision Therapist's Program
Specialized 30-hour course taking place over three weekends.



March 20-23
Cedarville, Ohio
Optometric Vision Therapy Retreat Seminars by Dr. Brenda Montecalvo

March 27 (TBD)
Vision Expo East
Confirmed speakers include Dr. Christine Allison, FCOVD and Dr. Ida Chung, FCOVD
Details coming soon!

APRIL 2020

COVD 20/20 50th ANNUAL MEETING

April 21-25
Toronto, Ontario, Canada
COVD 2020 50th Annual Meeting

College of Optometrists in Vision Development
215 W. Garfield Road • Suite 200 • Aurora, OH 44202
330.995.0718 (voice) • 330.995.0719 (fax)
info@covd.org • www.covd.org

COVD 20/20

50th ANNUAL MEETING

TORONTO

APRIL 21-25, 2020

College of Optometrists in Vision Development

www.covd2020.org

info@covd.org