Vision Development & Rehabilitation

College of Optometrists in Vision Development
Prevention • Enhancement • Rehabilitation

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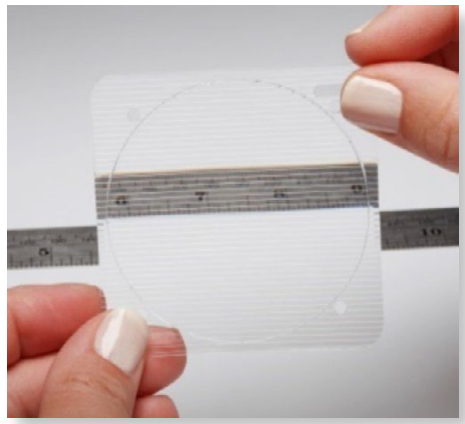
EDITORIAL

Press on Prism

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

Don't confuse the title of this editorial with the 3M Company's trademarked name for Fresnel prism, "Press-On". https://goo.gl/Z4FWOb



My purpose in this piece is to share a few thoughts about the use of prism, particularly as related to learning. As you may be aware, I formally sold my optometric practice in April, 2017, but will continue to see patients on a regular basis as well as being the site supervisor for our residency program administered through the Southern College of Optometry. Parenthetically our new practice owner is a former student extern, Dr. Laura Knapp, a testament to the synergistic fruits of labor in vision development and rehabilitation between private practices and optometric institutions. By coincidence our practice is situated geographically at the nexus of three booming Orthodox Jewish Communities in Monsey (New York), Brooklyn (New York), and Lakewood (New Jersey), and I mention this specifically because prism has become a buzz word in these circles.

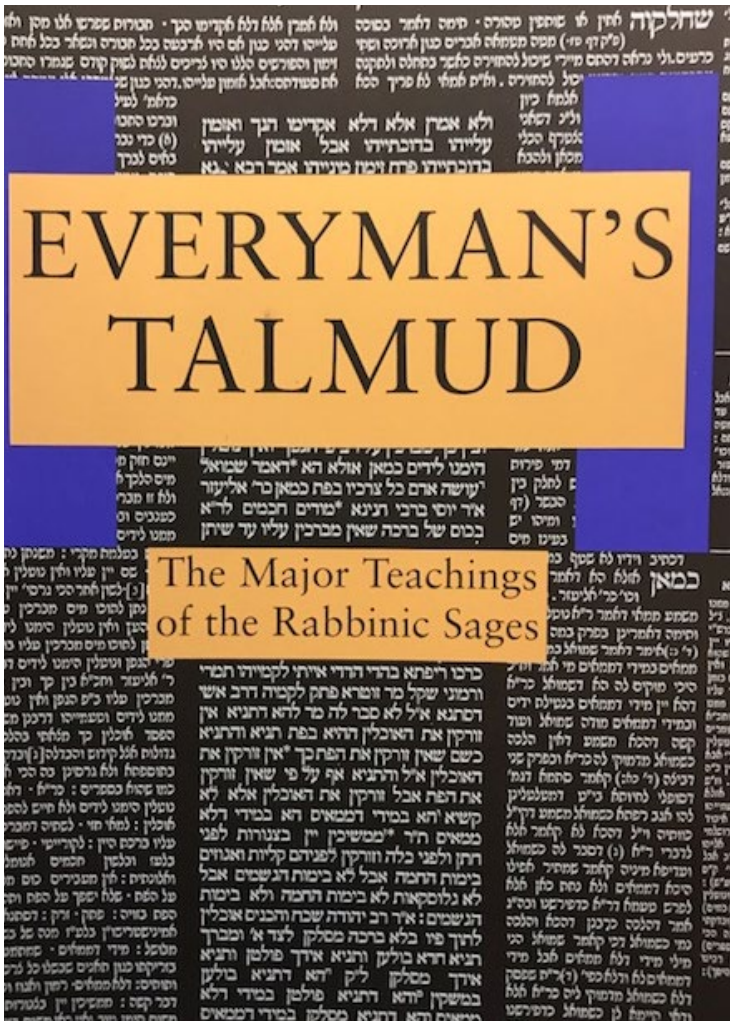
Prism is an attractive treatment option for patients because it is a form of wearable therapeutic optical technology. In lesser amounts, when ground into a lens or attained by decentration, no one but the patient knows that prism has been incorporated which addresses potential concerns about cosmesis. Rules of thumb and various criteria exist to determine whether prism can be beneficial, along with derivations for the amount and orientation of prism. Yet optometric heritage warns that prism should not be positioned as a cure-all, hence the aphorism about the judicious application of lenses and prisms.

It is tempting to think that if a little bit of prism is good, more is better, until one is reminded of the analogy to medication and its dosages. Hence the importance of assessment and measurement to see if prism is indicated. The use of prism has been advanced as an aid to reducing the impact of learning problems, postural skews. and visual field loss, ranging well beyond their original use to lessening eye strain and compensating for double vision. Here we should make a distinction between higher power prism used in the vision therapy room including yoked prism, typically between 5^ and 15^ and lower amounts of prism. Dr. Robert Fox has provided a nice overview in this regard.

Theories abound as to why low power prisms are effective, mirroring theories about why low plus power lenses are effective. A strong proponent for prescribing low power prism is Dr. Merrill Bowan, coining the phrase "miniprism" or "microprism".

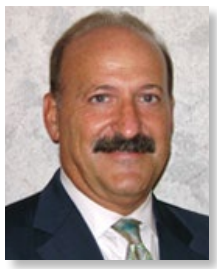
One of Dr. Bowan's theories is that a low amount of prism, typically 1^ BI OU, is effective in reducing the phenomenon of image aliasing. He provides a neurophysiological rationale for this, along with a grid to elicit the phenomenon and probe the effects of prism.

This brings us back to the Orthodox Jewish population mentioned earlier, and the Talmud - the principal compilation used for learning in this community. Expectations for mastery of the material in its printed form are high, particularly among boys. However the arrangement of the print, its variation in font size, and visual crowding combine to induce a form of textual aliasing, and in some instances even illusory motion to susceptible users.



To my knowledge there has never been a controlled study to investigate the effects of low power prism lenses on reducing textual aliasing, and that would be welcome. In the interim, anecdotal reports of improvement through prism have been compelling. How long should a patient anticipate needing prescriptive prism? How often should the doctor re-evaluate the prism to determine the need for a change in power, symmetry, or base orientation? These are determinations that are as much an art as a science, though there is no need to be apologetic about that. Most of our clinical advances flow through the confluence of art and science.

As our practices collectively wade into these waters, I am reminded of the classic Mosby volume edited by Dr. Susan Cotter in 1995, "Clinical Uses of Prism: A Spectrum of Applications". It would not be an exaggeration to state that now, twenty-two years later, the spectrum of applications has widened considerably.



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PERSPECTIVE

Neuro-Optometry: An Evolving Specialty Clinic … 40 Years Later: A Perspective

Kenneth J. Ciuffreda, OD, PhD, FCOVD-A, FAAO, FARVO

Diana P. Ludlam, BS, COVT

Barry Tannen, OD, FCOVD

The area of "neuro-optometry" has grown rapidly and broadly over the past several decades, and it will continue to evolve, as we learn from our past and make new inroads and advances into the future. But what exactly do we mean by "neuro-optometry" and perhaps now more accurately, "neuro-optometric rehabilitation (NOR)"? Some nicely articulated answers come from the COVD website: (www.covd.org). It " … represents a specialized area of optometry, which addresses the oculomotor, accommodative, visuomotor, binocular, vestibular, perceptual/visual information processing, and specific ocular/neurological sequelae of the acquired brain injury population. It includes "standard optometric modalities, such as corrective lenses, prisms, tints and coatings, and optometric vision therapy." This includes the diagnosis of the visual sequelae present and their remediation, that is, a dual-pronged neuro-optometric rehabilitative evaluation. These descriptions present and allow for a comprehensive approach and conceptualization of the area for the optometrist, as well as other professionals involved in their own related specialty (e.g., the physiatrist).

One might ask, "How did the area of neuro-optometry come about?" To such a broad and personalized question, there rarely is a single answer, as history typically reveals several independent avenues that develop over a similar timeframe, then overlap and converge, and 'suddenly' a new field emerges. The particular 'milestone' for the present authors was a paper from the Stark laboratory at Berkeley co-authored by the present first author (KJC): "Neuro-optometry: An Evolving Specialty Clinic" published in 1977,[1] in which the authors considered it to be "an optometric clinical specialty focusing on neurological dysfunctions of the visual system", a broad description at the time that included eye movements, accommodation, and the pupil using *objective,* laboratory-based protocols and instrumentation, in conjunction with the clinical findings. Larry Stark was a board-certified neurologist and widely considered to be the "father of bioengineering", a brilliant and futuristic thinker, and true friend of the profession of optometry. He did not care much about your professional degree, but rather your brain-power and creativity! Interestingly, both the second and third authors were also influenced by Larry: Barry Tannen spent an externship at his Berkeley laboratory while still an optometry student, and has subsequently spent much of his career in this field, and Diana Ludlam used Larry's theory

Correspondence regarding this article should be emailed to Kenneth J. Ciuffreda, OD, PhD, at kciuffreda@sunyopt.edu. *All statements are the authors' personal opinions and may not reflect the opinions of the College of Optometrists in Vision Development, Vision Development & Rehabilitation or any institution or organization to which the authors may be affiliated.* *Permission to use reprints of this article must be obtained from the editor. Copyright 2017 College of Optometrists in Vision Development.* *VDR is indexed in the Directory of Open Access Journals. Online access is available at www.covd.org.*

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Keywords: brain science, COVD, neuro-optometric rehabilitation, neuro-optometric rehabilitative evaluation, neuro-optometry, optometry, NORA, vision rehabilitation, vision

of oculomotor 'scanpaths'[2] (1971) to explain visual information processing problems to her patients over the past 45 years — Larry was clearly influential in optometry! While the original 1977 paper concentrated on eye movements, accommodation, and the pupil, it had broader implications, both for the care of these patients, as well as the scope of the care, and even more broadly, the optometric profession itself.

There were other significant early developments and milestones that were influential to the growth of neuro-optometry, some of which included the following. In 1990, the Neuro-Optometric Rehabilitation Association (NORA; www.nora.cc) was founded and remains an educational and political force in the area. NORA provides lectures, symposia, courses, and formal fellowships in the area in the United States, and internationally. Its intent is to integrate the best science and clinical aspects to improve the care of this broad population having neurological and developmental disorders, accompanied by problematic visual sequelae. Another key group is the College of Optometrists in Vision Development (COVD) founded in 1971, with a broad agenda of important visual dysfunctions to care for, including those individuals with visual developmental and neurological disorders, and others. It has an international examination certification board for both optometrists and vision therapists. COVD's mission is educational, both nationally and internationally. They too have a wide array of educational programs, most recently with emphasis on acquired brain injury. Lastly, from this historical perspective, some of those who pioneered the area, and involved in one or both of the aforementioned organizations, include: Irwin Suchoff, Lynn Hellerstein, Bill Ludlam, Vincent Vicci, Penelope Suter, Bill Padula, Danny Gottlieb, and Allen Cohen, and others, who have made a range of important contributions in both the clinical and research arenas.

And, in more recent times, advances in clinical research have elevated neuro-optometric rehabilitation to new heights. This too includes a range of *objective* approaches: documentation of the oculomotor system and reading in mild traumatic brain injury (mTBI) and stroke, both before and after vision therapy, as done in our SUNY Brain Injury Laboratory;[3,4] assessment of cortical responsivity and visual attention using the visual-evoked potential (VEP) following some vision intervention, such as vision therapy[5] or binasal occlusion,[6] with this also done in our laboratory; and on-going investigations using imaging techniques to record brain changes following vision therapy. Interestingly, all of the above "recent advances" were foreseen and predicted by the Stark et al paper[1] in 1977, namely objective approaches and instrumentation to assist in the diagnosis, prognosis, and therapy of these patients.

Thus, the future bodes well for optometry, and more specifically the area of neuro-optometric rehabilitation. There continues to be a rich, symbiotic relation between the clinical and laboratory domains, with resultant improvement in neuro-optometric rehabilitative care.

Acknowledgements

We thank Dr. Irwin Suchoff for his insightful discussions.

REFERENCES

1. Stark L Bahill AT, Ciuffreda KJ, Kenyon RV, Phillips S. Neuro-optometry: an evolving specialty clinic. Am J Optom Physiol Opt 1977; 54: 85-96.
2. Noton D, Stark L. Scanpaths in eye movements during visual perception. Science 1971; 171: 308-311.
3. Thiagarajan P, Ciuffreda KJ. Effect of oculomotor rehabilitation on vergence responsivity in mild traumatic brain injury. J Rehabil Res Dev 2013: 50: 1223-1240.
4. Thiagarajan P, Ciuffreda KJ, Capo-Aponte JE, Ludlam DP, Kapoor N. Oculomotor rehabilitation for reading in mild traumatic brain injury (mTBI): an integrative approach. NeuroRehabil 2014: 34: 129-146.
5. Yadav NK, Thiagarajan P, Ciuffreda KJ. Effect of oculomotor rehabilitation on the visual-evoked potential and visual attention in mild traumatic brain injury. Brain Inj 2014; 28: 922-929.

6. Yadav NK, Ciuffreda KJ. Effect of binasal occlusion (BNO) and base-in prisms on the visual-evoked potential (VEP) in mild traumatic brain injury (mTBI). Brain Inj 2014; 28 1568-1580.

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ARTICLE

Prevalence of Oculomotor Dysfunction in Healthy Athletes Preseason: Implications for Concussion in Sport

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Badovinac SD, Quaid P, Hutchison MG. Prevalence of oculomotor dysfunction in healthy athletes preseason: Implications for concussion in sport. Vision Dev & Rehab 2017;3(2):75-89.

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ABSTRACT

Background: Measures of oculomotor function are becoming more frequently employed as part of comprehensive concussion assessments. However, performances on many of these oculomotor measures have not been examined in a healthy athletic cohort. The purpose of this study was to characterize performance of university level athletes on a battery of oculomotor tests and identify any potential influence of gender and history of concussion.

Methods: 259 healthy university level athletes (males, n = 150; females, n = 109) completed an oculomotor screening battery prior to the start of their competitive season. The battery assessed stereopsis, visual acuity, monocular amplitude of accommodation, near point of convergence, monocular and binocular accommodative facility, vergence facility, positive and negative fusional vergence, and saccades. Athletes also completed the Convergence Insufficiency Symptom Survey (CISS).

Results: Three oculomotor tests (stereopsis, convergence, saccades) showed significant differences between male and female athletes at $P<0.05$, uncorrected. A high percentage of athletes were identified as having oculomotor deficiencies including abnormal acuity (34.2%), vergence infacility (28.6%), abnormal horizontal saccades (21.7%) and accommodative infacility (18.7%). Convergence insufficiency differed by method of assessment, ranging from 11.0-15.7%.

Conclusion: A significant proportion of healthy athletes displayed abnormal performance across a variety of oculomotor indices. A history of lifetime concussion (i.e., greater than 12 months prior to study) did not significantly influence oculomotor test performance. Clinicians should be aware of such differences and potential implications associated with postconcussion evaluations.

INTRODUCTION

Concussions are a form of mild traumatic brain injury (mTBI) estimated to account for approximately 75% of all traumatic brain injuries (TBI).[1] They are highly prevalent in sports, and the Centers for Disease Control (CDC) estimated that sports or recreation related traumatic brain injuries (TBI) accounted for 207,830 emergency department visits each year between the years 2001 and 2005.[2] In Canada, sports and recreational activities are the third leading cause of TBI hospital admissions.[3] It is estimated that the incidence of concussion is approximately 500 to 600 per 100,000 of the population with about 10-15% of affected patients experiencing symptoms even after 12 months.[4]

Many athletes report a constellation of vision-related symptoms following sport-related concussion.[5] This is not surprising, given that some neuroanatomical publications have indicated that between 40-50% of the primate brain has reciprocal connections to visual or visually-related areas, and therefore is likely to be involved to some extent in concussion-based injuries.[6,7] Many of the vision difficulties reported by concussed individuals are related to impairments in convergence and accommodation, and often include headaches, asthenopia, vision blurring in and out of focus, loss of one's place while reading and copying text from distance to near.[8] Despite the relationship between vision difficulties and symptoms postconcussion, oculomotor assessment is not currently a focus of concussion management protocols.[9] In a recent systematic review of oculomotor-based vision assessment following concussion,[10] the authors concluded that the evidence for the use of these tests following concussions is preliminary at this point, and not sufficient to warrant clinical recommendations for the use of oculomotor assessment following mTBI.

The presence of convergence-related difficulties following concussion has been one of the most commonly documented oculomotor complaints,[8,11-13] with impairments typically being present during the first week following sport-related concussion.[11,12] While normative data has been published for the near point of convergence (NPC) test in the general population,[14] the importance of measuring baseline NPC values for athletes has been emphasized, since previous work has shown the prevalence of convergence insufficiency to be higher among healthy athletes than among the general population.[12] Difficulties related to vergence function among individuals with mTBI have also been assessed by measuring fusional ranges,[8] and vergence facility.[15] Vergence facility describes the ability of the eyes to binocularly converge and diverge in a sequential manner with a fixed demand of accommodation and can be tested at distance and near. Although these tests are not generally included in standardized baseline protocols or postconcussion evaluations, abilities related to vergence have been found to be impaired postconcussion.[8,15]

Accommodation is the monocular process by which innervation to the ciliary body of the eye is changed resulting in contraction or relaxation of the ciliary body in order to adjust focus on objects at varying distances. Tests measuring accommodative ability often include amplitude of accommodation,[8,16] which relates to the minimum distance at which a target can be maintained in clear focus (converted to dioptres by taking the inverse of this distance in metres). Accommodative facility measures the ability to quickly change accommodative function to view a target at various distances (i.e. from distance to near and vice-versa). In essence, amplitude represents the power of the system whereas facility represents the flexibility of the system. In the acute phase of mTBI, accommodative insufficiency (i.e. the power) has been reported to occur among 65% of patients, and may persist for several years following injury.[16]

The 2012 Consensus Statement on Concussion in Sport did not identify measures of oculomotor function as a central focus

of concussion diagnosis or management.[9] However, they noted that future research should consider the efficacy of including vision tests such as the King-Devick,[9,12,17-19] which assesses saccadic eye movement. A basic saccadic eye movement is a rapid re-fixation from one point to another. In order to initiate a saccade, the visual system must not only release fixation from the point of regard, but also pre-plan where the next fixation will land, a process that ultimately requires peripheral awareness.[20] Many tests of saccadic function are designed for a clinical setting and require a computer and video-oculography. However, the King-Devick (K-D) test quickly evaluates saccadic eye movement and has shown promise in its ability to diagnose concussion in an athletic setting.[9,12,17-19]

In recent years, there has been a significant increase in research examining the utility of oculomotor tests post-concussion.[10,21-24,46,47] Although promising, the influence of pre-morbid oculomotor dysfunction on post-injury performance or the natural history of oculomotor function throughout clinical recovery is less well-understood. A recent systematic review found that few studies (20%) have assessed and reported oculomotor performance *prior* to injury.[10] Furthermore, it has been shown that reduced function on oculomotor testing (in the realms of vergence and accommodation specifically) is associated with prior concussions in athletes.[25] Since the reference values for normal vs. abnormal scores for vision-related deficits are often based on reference values of the general population, it would be potentially more appropriate to develop normative values specific to a student athlete population. This is particularly important since previous work has demonstrated that professional athletes exhibit superior ability in some oculomotor domains at baseline.[26,27] While it is possible that this finding is specific to athletes at the professional level, further research is required to examine this issue further and to determine whether unique norms are justified for the student athlete population. On the other hand, athletes would likely have greater exposure to repetitive head impacts and history of lifetime concussion compared to the general population. Therefore, the purpose of this study was to characterize performance on a battery of oculomotor measures in a cohort of healthy university level athletes. As part of this process, we examined the proportion of athletes classified as abnormal for each oculomotor test based on adopted cut-off values recommended in the literature. We also examined potential differences between male and female athletes, as well as influence of prior concussions.

METHODS

Participants

Between August and November 2014, 291 healthy inter-university student athletes completed the oculomotor testing battery prior to the start of their competitive season. These athletes were recruited from a single institution and participated in sports that presented an increased risk of concussion, which included basketball, field hockey, football, ice hockey, lacrosse, rugby, soccer, volleyball and wrestling. Eight participants were excluded because habitual eye correction was not worn at the time of testing. Given that our primary objective was to characterize performance among healthy athletes, we also excluded individuals who had sustained a concussion within the last 12-months or who were recovering from a concussion that had occurred greater than 12 months prior to the study (n = 24). A final dataset of 259 athletes was used for analyses (male = 150, female = 109). The study was approved by the Institutional Ethics Review Board (Protocol Reference # 30641).

Data Collection

All athletes completed the oculomotor test battery prior to the start of their competitive season. The battery consisted of eleven tests targeting a range of visual functions, and could be

administered in approximately fifteen minutes. All tests were administered in a randomized sequence. For all tests, habitual correction was used by participants when necessary (i.e. reading glasses or contacts). A complete exam was purposely not done, as *habitual status* was desired. This situation is also more reflective of real-life scenarios whereby not all athletes will have up-to-date eye care (i.e. optimal visual acuity for example). Tests were administered by individuals trained by an optometrist with advanced training in diagnosing and treating oculomotor dysfunction (PQ). Following the training period and prior to data collection, all examiners (6 total) had to demonstrate competence under supervision.

Stereopsis

Global (random dot) and local (circles) depth perception were assessed using the Randot Butterfly Stereo Test. Participants were first asked to put on a pair of polarized glasses and identify a butterfly presented in a random dot display in order to establish a baseline depth perception capability (random dot target, global depth perception). Upon successful completion of the first task, participants were presented with a series of nine four-dot displays, and were told to indicate whether the top, bottom, left or right circle appeared slightly raised compared to the other three circles (local stereopsis). Each subsequent four-dot display represented stereopsis values ranging from 800 seconds of arc to 40 seconds of arc, with fewer seconds of arc indicating a smaller separation between two overlapping images. If the random dot target could not be seen, stereopsis testing was still undertaken with the local stereopsis targets and the local stereopsis limit recorded (two in a row incorrect required with latter level recorded as limit). The smaller the separation between the two images, the more difficult it was to perceive elevation of one of the circles. Participants' minimum seconds of arc and time to completion was recorded, with the latter representing "speed of stereopsis".

Visual Acuity

Monocular visual acuity was assessed using a standardized Snellen eye chart with more than half of the line required to give credit for the line of acuity. The limit of visual acuity on this chart was 20/10 (6/3) acuity.

Monocular Amplitude of Accommodation

Monocular amplitude of accommodation was calculated using the push-up method.[28] In this test, a "budgie-stick" with printed letters (equivalent to approximately 20/30 at 40cm) was held forty centimetres in front of the face. The budgie stick was gradually moved towards the face, and the minimum distance at which the participant was able to maintain a clear and focused view of the letters was recorded. This distance was measured three times with the last measure being taken for each eye. The corresponding amplitude of accommodation, expressed in diopters, was calculated by dividing this distance in cm into 100. As a reference, the expected minimum amplitude of accommodation for a given age was calculated according to Hofstetter's formula: 15 – 0.25 x age.[29]

Near Point of Convergence (NPC)

To measure the near point of convergence, the subject was instructed to use both eyes to focus on an accommodative target (i.e. a pen tip) as it was moved towards them. The distance at which the subject reported seeing double was recorded. In the event that the subject did not report seeing double, the point where one of the eyes was observed to turn outwards was recorded (i.e. if the patient suppressed one eye). The near point of convergence was calculated as the average of three trials, as has been recommended previously.[14]

Monocular / Binocular Accommodative Facility (MAF / BAF)

For the monocular accommodative facility test, a ± 2DS flipper lens were used to assess one eye at a time, while the other

eye was covered. A budgie stick with printed letters (equivalent to 20/30) was held in place approximately forty centimetres from the face. The examiner held the plus lens over the participant's eye, and the participant was instructed to indicate when the text on the card was clear. This was the examiner's cue to flip to the minus lens. The number of cycles completed in one minute (cpm) was recorded for each eye. In the binocular accommodative facility test, the lenses were placed over both eyes at once, and the same procedure followed. Normal values of 10 and 11 cpm (SD = 5.0) have been identified for monocular and binocular accommodative facility, respectively.[30] In testing MAF data, the fellow eye is observed (under the cover) to ensure that movement occurred (inward for minus lenses and outward for plus lenses) to ensure that the patient's own response was not the only response depended on. In addition, pupil sizes were monitored as when minus lenses are cleared (i.e., positive relative accommodation) pupil size decreases, and conversely, when plus lenses are cleared (i.e., negative relative accommodation) pupil size increases. In testing BAF data, the examiners monitored the participants' eyes to ensure convergence and divergence occurred with both positive and negative relative accommodation.

Vergence Facility

This was measured with a 12 base-out / 3 base-in prism flipper, which was positioned over one eye (right eye) with both eyes open during testing. The subject fixated on the tip of a pen at near (40cm), which would initially appear as two images due to the effect of the prism. The subject indicated when they had fused the image and the examiner accordingly changed the flipper position from 12 prism diopters base out to 3 prism diopters base in. The examiner continued to rotate between the two prisms as cued by subject with the eyes also being used as objective confirmation of movement (as suppression can occur and no diplopia be perceived for example). The number of cycles completed in one minute was recorded with general normative values being 16cpm (SD = 2.6) using 12BO/3BI.[31]

Convergence Amplitude (a.k.a. Positive Fusional Vergences or PFV function)

Convergence Amplitude (measured as base-out prism to break and base-out prism to recovery) is also known as positive fusional vergence (PFV). Convergent "step vergence" was determined at near (40 cm) using a prism bar in free space and a pen tip as a target. The "break point" was the prism value at which the subject saw double and could not re-fuse, and the "recovery" was the prism value at which the pen tip could be re-fused. If participants were able to fuse the full range of positive step vergences without break (including the maximum value of 45), a value of >45 was recorded as the break point and recovery point.

Divergence Amplitude (a.k.a. Negative Fusional Vergence or NFV)

Divergence amplitude, measured as base-in break and base-in recovery, is also known as negative fusional vergence (NFV). Divergent "step vergence" was determined at near using a prism bar in free space and a pen tip as a target. The "break point" was the prism value at which the subject saw double and could not re-fuse and the "recovery point" was the prism value at which the pen tip could be refused. If participants were able to fuse the full range of negative step vergences without break (including the maximum value of 45), a value of >45 was recorded as the break point and recovery point.

Developmental Eye Movement (DEM) Test

This is a standardized oculomotor skills test, which measures the speed at which subjects can recognize and verbally identify a series of numbers.[32] The first two subtests of the DEM involve reading an array of numbers vertically, while the third subtest

Table 1. Descriptions of criteria used to identify abnormal or clinically significant scores.

Condition	Criteria for abnormal score
Convergence Insufficiency	NPC > 6 cm[12] CISS score > 20[35]
Accommodative Insufficiency	Amplitude of Accommodation for at least one eye > 2D below age-related expected minimum value, based on Hofstetter's formula: [15-(0.25*age)][29]
Accommodative Infacility	MAF < 6cpm in at least one eye or BAF < 3 cpm (1 SD below mean reported in[30])
Vergence Infacility	< 13 CPM (1 SD below mean reported in[31]
Fusional Vergence Dysfunction (+ve) Fusional Vergence Dysfunction (-ve)	Break point < 10 or Recovery point < 7 (1 SD below mean reported in[36]) Break point < 7 or Recovery point < 5 (1 SD below mean reported in[36])
Abnormal Vertical Saccades	> 31.58 seconds (1 SD below mean obtained from preliminary young adult norms for DEM scores)[37]
Abnormal Horizontal Saccades	> 33.63 seconds (1 SD below mean obtained from preliminary young adult norms for DEM scores)[37]
Abnormal Acuity	Poorer than 20/20
Abnormal Stereopsis	> 40 seconds of arc[38]

involves reading horizontally, and is an analog of the King-Devick test. All sub-tests were timed to the nearest hundredth of a second, and the score of the horizontal sub-test was adjusted to account for subject errors (i.e. errors of addition, omission, substitution and transposition). The primary outcome variables included vertical saccade time in seconds and horizontal saccade time in seconds. While a ratio of vertical and horizontal saccades is often used as the primary outcome variable for the DEM, we opted to interpret vertical and horizontal saccades separately. From a clinical standpoint, we feel that discrepancies between vertical and horizontal saccades are more accurately captured by comparing the percentile ranks of the two, rather than using a ratio.

Table 2. Demographic characteristics.

	All	Male	Female
N		150	109
Age (years)	21.1 ± 2.2	21.4 ± 2.1	20.8 ± 2.5
History of concussion (%)	57.0	57.4	42.6

Convergence Insufficiency Symptom Survey (CISS)

This is a validated questionnaire that contains 15 questions addressing problems related to convergence impairments (e.g. "Do your eyes feel tired when reading or doing close work?"). Subjects rated agreement with each of the fifteen statements on a 5-point Likert scale (0 = never, 4 = always), for a maximum possible score of 60.[33]

Statistical Analysis

Descriptive statistics were calculated for all vision tests, with mean, standard deviation, 50th percentile, and cut-offs for the lowest 25th and 10th percentiles. Non-parametric Mann-Whitney U tests were conducted to identify differences in oculomotor scores associated with gender and history of concussion. For the current study, we report both the uncorrected p-values and the false-discovery rate (FDR) adjusted p-values, which correct for multiple comparisons as described in Yekuteli & Benjamini.[34]

We also calculated the proportion of athletes classified as abnormal for each oculomotor test based on adopted cut-off values recommended in the literature. The criteria used to identify abnormal scores are provided in table 1. Chi-square test of goodness-of-fit were employed to identify if the proportion of athletes with abnormal vision tests differed according to the presence or absence of a previous concussion; this test was performed separately on male and female athletes. Statistical significance in all analyses was indicated by a p-value of ≤0.05. All

Table 3. Summary of oculomotor test performance.

Oculomotor Test	All Athletes (n = 259) Mean ± SD (50th, 75th, 90th)	Males (n = 150) Mean ± SD (50th, 75th, 90th)	Females (n = 109) Mean ± SD (50th, 75th, 90th)	Unadjusted P value	Adjusted P value
NPC	3.7 ± 1.4 (3.5, 4.5, 5.7)	3.9 ± 1.5 (3.7, 4.7, 6)	3.5 ± 1.2 (3.3. 4.2, 5.2)	0.072	0.180
Stereo Arc[ac]	-- (40, 50, 100)	-- (40, 50,100)	-- (40, 50, 60)	0.012	0.120
Stereo Speed	15.1 ± 6.2 (14.0, 18.3, 24.5)	16.0 ± 6.0 (15, 21, 25)	14.2 ± 6.2 (12.7, 16.9, 22.5)	0.003	0.060
Acuity Right[ab]	20/20 ± 1.3 (20/20, 20/20, 20/25)	20/20 ± 1.4 (20/20, 20/20, 20/30)	20/20 ± 1.2 (20/20, 20/20, 20/25)	0.473	0.676
Acuity Left[ab]	20/20 ± 1.3 (20/20, 20/20, 20/25)	20/20 ± 1.3 (20/20, 20/20, 20/25)	20/20 ± 1.2 (20/20, 20/20, 20/25)	0.785	0.907
A of A Right	17.6 ± 4.6 (16.7, 20.0, 25.0)	17.7 ± 4.3 (16.7, 20, 23.8)	17.4 ± 5.0 (16.7, 20, 25)	0.519	0.692
A of A Left	17.6 ± 4.9 (16.7, 20.0, 25.0)	17.2 ± 4.8 (16.7, 20, 25)	18.1 ± 5.0 (16.7, 20, 25)	0.292	0.531
MAF Right[a]	14.7 ± 7.0 (15.0, 10.0, 5.0)	13.6 ± 7.4 (14, 9, 3.5)	15.7 ± 6.4 (17, 12, 6.5)	0.058	0.180
MAF Left[a]	15.3 ± 7.0 (16.0, 11.0, 6.0)	15.3 ± 7.3 (15.5, 11, 6)	15.2 ± 6.8 (16.3, 12, 6)	0.816	0.907
BAF CPM[a]	13.4 ± 5.5 (14.0, 10.0, 6.0)	13.4 ± 5.7 (13, 10, 5.5)	13.4 ± 5.2 (14, 11, 6)	0.943	0.979
Vergence Facility[a]	16.1 ± 6.8 (17.0, 12.0, 6.0)	15.6 ± 6.9 (16, 12, 6)	16.6 ± 6.5 (18, 12, 6)	0.192	0.384
Base Out Break[ac]	-- (40.0, 30.0, 25.0)	-- (35.0, 30.0, 25.0)	-- (40.0, 35.0, 25.0))	0.032	0.160
Base Out Recovery[ac]	-- (30.0, 25.0, 20.0)	-- (30.0, 25.0, 20.0)	-- (35.0, 25.0, 20.0)	0.051	0.180
Base In Break[ac]	-- (14.0, 12.0, 10.0)	-- (14.0, 12.0, 10.0)	-- (14.0, 12.0, 10.0)	0.735	0.907
Base In Recovery[ac]	-- (12.0, 8.0, 6.0)	-- (12.0, 8.0, 6.0)	-- (10.0, 8.0, 6.0)	0.373	0.622
CISS Total	12.5 ± 7.6 (11.0, 16.0, 23.0)	13.2 ± 8.1 (12, 17, 25)	11.6 ± 6.9 (11, 16, 22)	0.068	0.180
DEM Vertical	26.3 ± 3.7 (25.9, 28.9, 31.4)	26.7 ± 4.0 (26.2, 29.7, 32.7)	25.7 ± 3.2 (25.5, 27.8, 30.3)	0.100	0.222
DEM Horizontal	29.4 ± 5.3 (28.3, 32.5, 36.6)	29.6 ± 5.6 (28.3, 32.4, 38.2)	29.3 ± 5.0 (28.4, 32.7, 35.5)	0.979	0.979
DEM Ratio	1.1 ± 0.2 (1.1, 1.2, 1.3)	1.1 ± 0.2 (1.1, 1.2, 1.2)	1.1 ± 0.1 (1.1, 1.2, 1.3)	0.031	0.160

[a] Reverse scoring – meaning higher values = better performance; [b] Standard deviation value refer to lines on a Snellen chart; [c] No means reported as variables are categorical

statistics were completed using Stata Version 14.1 (StataCorp, TX, USA).

RESULTS

Of the 259 student athletes, the mean age was 21.1 years (SD = 2.3; range 18-33) with no significant difference in age between male and female athletes (difference, -0.47 [95% CI, −1.00 to 0.08]; P = 0.09). For athletes with a lifetime history (i.e. not within last 12 months with continuous play within last 12 months) of concussion, the distribution was equal between male and females (X^2 = 0.97, P = 0.32). See table 2 for a summary of demographic characteristics. Our sample consisted of athletes participating in basketball (n = 21), football (n = 55), field hockey (n = 14), ice hockey (n =40), lacrosse (n = 42), rugby (n =18), soccer (n = 36), volleyball (n = 29), and wrestling (n = 4).

Table 3 summarizes group averages of oculomotor performance for the various tests, as well as cut-values for the lowest 25th and 10th percentiles. Mann-Whitney U tests identified significant differences between male and female athletes on three oculomotor tests (stereopsis, vergence amplitude, and saccades), however, no tests attained significance after adjusting for multiple comparisons (table 3). Furthermore, after adjusting for multiple comparisons, no significant differences were observed in those with a lifetime history of concussion compared with athletes with no history of concussion. However, uncorrected p-values noted significantly higher CISS symptom scores in athletes with a lifetime history of concussion and poorer fusional vergence (by lower base out break scores) in male athletes with history of

Table 4. Proportion of athletes with abnormal or clinically significant oculomotor test scores.

Condition	% Abnormal (N=259)	% Males (N=150)	% Females (N=109)
Convergence Insufficiency NPC > 6 cm CISS score > 20	 11.0 15.7	 14.2 18.1	 10.4 14.6
Accommodative Insufficiency	1.4	1.0	1.9
Accommodative Infacility	18.7	20.9	15.6
Vergence Infacility	28.6	31.2	25.4
Fusional Vergence Dysfunction (+ve)	3.1	4.3	1.7
Fusional Vergence Dysfunction (-ve)	9.7	12.9	5.9
Abnormal Vertical Saccades	9.7	14.1	4.3
Abnormal Horizontal Saccades	21.7	22.5	20.7
Abnormal Acuity	34.2	38.1	29.0
Abnormal Stereopsis	9.1	12.6	6.7

* sample sizes may vary between tests.

concussion compared to male athletes with no history of concussion.

When basic monocular visual acuity levels of the 259 athletes were examined, 34.2% of athletes did not have 20/20 acuity in *at least* one eye. Table 4 provides the proportions of athletes with abnormal or clinically significant test scores based on established norms and cutoffs. 11% (using 6cm NPC cut point) were classified as having convergence insufficiency based on NPC results. When a CISS score of > 20 was applied as determining convergence insufficiency, 15.7% of the athletes were classified as abnormal. Apart from visual acuity, the most common oculomotor deficiencies were vergence infacility (28.6%), abnormal horizontal saccades (21.7%), and accommodative infacility (18.7%). Given the differences we observed between group averages of male and female athletes, we examined whether the proportion of abnormal test results were significantly different between genders. Vergence infacility was identified more frequently in male athletes compared to females (P = 0.02) and convergence insufficiency by NPC result trended toward significance in males (table 3); however, no significant difference was found when corrected for multiple comparisons. Finally, we investigated proportion differences for each gender and the relationship with lifetime history of concussion; no significant differences were observed.

It was decided at the onset of the study not to correct participants who did not wear habitual ophthalmic correction. However, a significant proportion of athletes (n = 85) did not have 20/20 visual acuity in at least one eye. We speculated that reduced visual acuity may have been due to simple uncorrected refractive error. Thus, we conducted a follow-up analysis to examine whether reduced acuity was playing a major role in the relatively high proportion of abnormal oculomotor findings. Within the group that did not have 20/20 in one eye, we identified significantly decreased stereopsis level (uncorrected P = 0.041, FDR = 0.369) and speed of stereopsis (uncorrected P < 0.001, FDR = 0.018). Also, when a more stringent threshold was applied (20/30 or worse in at least one eye), these athletes (n = 35) displayed reduction in stereopsis level (uncorrected P < 0.001, FDR = 0.009), base in break (NFV) (uncorrected P = 0.016, FDR = 0.096), base-in recovery (NFV) (uncorrected P = 0.009, FDR = 0.081), and lower values in amplitude of accommodation in at least one eye (uncorrected P = 0.022, FDR = 0.099).

DISCUSSION

The objective of this study was to characterize the performance of healthy athletes on a variety of oculomotor tests beyond simple visual acuity, with particular interest in identifying variation in performance related to gender and concussion history (more than 12 months out with the athlete still actively playing). Our results highlight that a significant proportion of athletes displayed abnormal oculomotor function across a variety of indices. In addition, a lifetime history of concussion did not significantly influence oculomotor test performance, although we observed trends

of gender-related differences, with females generally performing better than males.

We used near point of convergence as an objective measure to assess the prevalence of convergence insufficiency (CI), employing a conservative cut-off of 6cm. Using this criterion, 11% of the athlete cohort were considered to display CI, which is higher than the prevalence rates previously reported in university level students (3%)[39] and in the general population (5%).[14] Our present results are more comparable with a recent study of youth hockey players (11.5%), which employed the same cut-score of greater than 6cm.[12] Near point of convergence has also been utilized more frequently in post-concussion assessments and recently reported in approximately 42% of cases at the 1-month stage post-sports related concussion.[5,40] Furthermore, exposure to a competitive season in collision sports also appears to impact NPC scores as a recent study has shown that the repeated heading of a football alone can result in a convergence issue being induced.[41]

In addition to near point of convergence, the CISS symptom score was also used to detect convergence-related difficulties. This tool has been identified as a valid and reliable instrument for the measurement of convergence-related symptoms.[35] We employed the recommended cut-off score of >20, and found that over 15% of all athletes reported significantly symptomatology. While only 11% met the CI definition according to our criteria of 6cm, it has previously been shown that the CISS scores are associated with symptomatology in non-CI cases such as in accommodative dysfunction[42] or vergence facility dysfunction,[43] for example. Given that CI in particular appears to be a common finding post-concussion,[8,11-13] the relationship between symptom reports and objective NPC findings needs to be examined in more detail prior to concussion as it is possible that post-concussive symptomatology may be exacerbated in subjects with abnormal NPC findings and / or higher CISS symptom scores. It should be noted that while we defined convergence insufficiency using an NPC cut-point of 6cm, this is not the only method by which it can be defined. Previous work has employed a multi-criterion approach to the diagnosis of CI, and has relied on a combination of three criteria (i.e., exophoria at near > or = 4 delta than at far; insufficient fusional convergence; and receded NPC of > or = 7.5 cm break or > or = 10.5 cm recovery) to diagnose definite CI.[44]

Our group mean for vergence facility cycles completed per minute was 16.1 cycles, with a standard deviation of 6.8. This value is slightly lower than the normative value previously established for an athletic population.[45] However, the previous study used a near-far test of vergence facility (i.e., Haynes Distance Rock Test), which assesses concurrent changes in vergence and accommodation, rather than vergence facility, which includes variable vergence demand with fixed accommodative effort. We also found vergence infacility to be present in 28.6% of our study sample, which is important to note because vergence infacility has been found to be a very sensitive predictor of overall oculomotor dysfunction and reading performance,[38] with abnormal reading and tracking being common complaints post-concussion. However, little research on vergence facility at near has been done among young adults and athletes with little information on how it relates to concussion. Vergence facility testing measures the ability of the visual system to move the eyes from a converged (inward) position to a diverged (outward) position quickly and efficiently at a fixed plane (i.e. fixed accommodative effort), which can be done at distance or near. Vergence facility is essentially a reflection of the "flexibility" or "degrees of freedom" of the vergence system. Performance on this test has been found to be highly correlated with reading efficiency (using infrared eye tracking methods), therefore appearing to be a logical oculomotor test to utilize as an indicator of reading efficiency,[38] an issue which is often

reported post-concussion.[8,38,46] We observed the proportion of athletes with vergence infacility to be substantially higher than the percentage of athletes with convergence insufficiency. Given the natural link between near-distance visual system "flexibility" which is important in sports, vergence facility testing likely represents a more sensitive "litmus test" for rapidly identifying the presence of overall oculomotor dysfunction, and particularly when tested at distance.[47] Accordingly, patients with convergence insufficiency would be likely to fail vergence facility testing at near and vergence facility testing at distance.[48] Our findings provide justification for examining vergence infacility in this population in future studies; also, they prompt the evaluation of vergence facility testing at distance,[47] as this removes the confounder of accommodation at near, which can sometimes mask vergence dysfunction (as the accommodation system can "kick in" to compensate for the vergence deficit at near).

We identified higher rates of negative fusional vergence dysfunction (9.7%) compared to positive fusional vergence dysfunction (3.1%). One possible explanation for observed differences in percentages between negative and positive fusional dysfunction is that our study sample consisted of individuals who do a lot of close work as full-time students (i.e., reading and studying) and being highly connected (i.e., handheld mobile devices, social media). Therefore, they are presumably good at near work and would be expected to have relatively low amounts of positive fusional deficits. It should be noted that we opted to record the break and recovery point and not include blur points (which is a limitation in the study) as we were interested in where the oculomotor system broke into diplopia rather than where it was "stressed". It is quite possible that the results may have shown a higher level of dysfunction had we looked at the blur point also. However, as this study was conducted in a sports medicine environment, the cut-off of diplopia was selected, since it can be both reported by the subject and reliably observed by the examiner (i.e., seeing the eyes deviate).

The prevalence of accommodative infacility in the general population is not well reported. Hennessey, Iosue and Rouse[49] assessed monocular and binocular facility in an asymptomatic sample of children and reported rates of 20% and 10% for binocular and monocular accommodative infacility, respectively. Although these previously reported rates were determined in a younger sample, the rates of accommodative infacility are in line with the 18.7% prevalence identified in our sample.

High rates of abnormal horizontal saccades were also identified within our sample (21.7%). Saccadic eye movement has been of particular interest in recent years in concussion assessment, with the increasing use of the King-Devick (K-D) test, which has been well validated as a sensitive sideline performance measure for concussion detection.[17,18] It is not surprising that saccades (a test of saccadic function which involves fixation and peripheral awareness, weighing aspects of the stimulus, the goal of the eye movement, motor planning and organization, and motivation) would be prone to malfunction from neurological trauma more readily than other eye movement types. It is however unclear as to the reasoning for the high percentage of abnormal saccades at baseline. One potential explanation could be the fact that DEM performance does not solely depend on saccades, and proficiency in other skills such as language speed, attention and visual processing speed are required. We did not observe proportional differences in those with a lifetime of history of concussion, however, we cannot rule out any remnant effects of repetitive head impacts associated with many sports or whether this incidence is truly this high in a non-professional level athletic cohort. Such association is aligned with studies showing that repetitive low-level impacts can be enough to even induce a convergence insufficiency,[41] therefore

potentially cumulative smaller hits may explain this result.

Collectively, results from the present study highlight the significant proportion of healthy, active athletes with what appear to be undiagnosed oculomotor-related abnormalities in addition to simple acuity issues (likely refractive in nature). This is particularly relevant in the context of post-concussion evaluations, as clinical vision assessments have become more frequent due to the constellation of visual problems secondary to sports-related concussions.[46] It is largely assumed that post-injury oculomotor abnormalities are most likely directly due to the concussive insult. Although this may be true to some extent, given the high proportion of oculomotor and basic visual acuity issues among athletes in this study, pre-morbid oculomotor dysfunction must be controlled for as a variable. Therefore, knowledge of an athlete's premorbid oculomotor status would likely be substantially beneficial in the context of a postconcussion evaluation. An optimal environment for concussion assessment and management has been recommended to include pre-season evaluations, although this is not always possible due to logistical challenges and resources. However, based on this paper, it is likely warranted to screen for individuals that may benefit from a comprehensive eye examination by a qualified eye care professional, in addition to a visual skills assessment looking in more detail at oculomotor function.

Including an oculomotor assessment as part of concussion baseline assessments would also allow for an opportunity to investigate the relationship between various oculomotor deficits and neuropsychological test (e.g., ImPACT) scores. Previous research has identified an association between oculomotor test scores and ImPACT scores post-concussion,[5,12,19] and thus it is logical to question how much the issue of oculomotor dysfunction at baseline affects performance on neuropsychological assessments.

Another possible explanation for the high proportion of athletes with oculomotor dysfunction at baseline may be that the previously available cut-points in the literature are not appropriate for a university level athletic population. Therefore, the development of normative data for athletic populations is warranted. Drawing from the field of neuropsychology, it has also been highlighted that comparing an individual's performance to some population average score is appropriate only when the score is uniformly present in all individuals and when performance is not related to age, gender, race, or education.[50] Significant differences were observed between male and female athletes on a number of measures; this result being unexpected, as gender differences have rarely been reported. Therefore, the development of group averages stratified by gender (table 3), as well as cut-values (lowest 25th and 10th percentiles) for a battery of oculomotor tests would be an appropriate reference for normative data of university level student athletes in the future.

There are some limitations resulting from our study design that may potentially impact interpretation of results. First, our findings suggest that a lifetime history of concussion – beyond 12 months from testing – does not significantly impact oculomotor test performance in this population. Despite no observed differences, we acknowledge the limitations of recall bias associated with self-reported history of concussion and the potential tendency of some athletes to under-report concussions.[51-53] We were also unable to account for potential inter- and intraexaminer variability. Data collection was part of a larger pre-season medical evaluation strategy; however, previous reports have identified high inter-examiner coefficients for a number of the oculomotor tests.[44] Also, prior research utilizing more sophisticated measures (using infra-red tracking) identified poor reading performance in conjunction with oculomotor dysfunction being associated with a 10.72x

increased likelihood of having previous suffered a concussion.[25] However, we omitted athletes with a history of concussion in the prior 12 months (or who had not been cleared following a concussion occurring > 12 months prior) so that our sample would be more likely to be a true representation of the healthy general athletic population, as residual effects of a recent concussion could not be ruled out. Second, although a relatively large proportion of the sample (34.2%) did not have 20/20 visual acuity in at least one eye, the nature of the underlying refractive error is important to discuss. If myopic or astigmatic in origin, then the effect on near point tasks is much less than if hyperopic in origin.[38,54] However, especially in a younger population, hyperopic refractive error rarely reduces distance visual acuity and therefore will typically reveal a "20/20" result in an eye examination (assuming no accommodative dysfunction present). Although not the aim of this paper, a refractive error assessment (including cycloplegic to relax accommodation) to determine accurately the underlying full refractive error may help to clarify how much this confounder is contributing to the relatively high amount of oculomotor dysfunction identified in this baseline athletic group. Our follow-up analyses of individuals with less than 20/20 suggests that reduced visual acuity impacts performance on a number of measures. Thus, proper correction should ideally be sought prior to formal baseline testing.

CONCLUSION

In conclusion, a significant proportion of healthy athletes displayed difficulties on a number of oculomotor measures during pre-season evaluations. Evaluation of oculomotor issues (i.e. not just simple visual acuity) in a comprehensive concussion model appears to be a useful tool in overall rehabilitation management and is in line with recently published multi-disciplinary guidelines (i.e. section 10.10).[55] Untreated persistent visual issues are likely important to recognize as a significant barrier to overall recovery post-concussion. Our findings highlight the need for an understanding of athletes' oculomotor status prior to concussion, particularly since pre-existing oculomotor dysfunction was not uncommon in our sample. Knowledge of preinjury oculomotor status of an athlete would: (i) screen for those that may benefit from a comprehensive eye examination (and where appropriate addressing significant oculomotor issues) by a qualified eyecare professional, and (ii) provide information about changes in oculomotor function in context to baseline data should a subsequent concussion occur.

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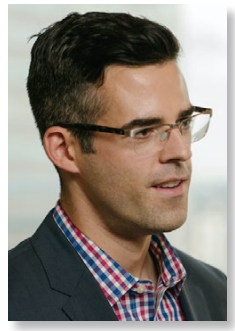
Conflict of Interests

None of the authors have a conflict of interest or financial disclosure to report.

REFERENCES

1. Centers for Disease Control and Prevention (CDC), National Center for Injury Prevention and Control. Report to Congress on mild traumatic brain injury in the United States: steps to prevent a serious public health problem. Atlanta (GA): Centers for Disease Control and Prevention; 2003.
2. Centers for Disease Control and Prevention. Nonfatal traumatic brain injuries related to sports and recreation activities among persons aged ≤19 years — United States, 2001–2009. MMWR 2011; 60(39):1337–1342.
3. Canadian Institute for Health Information. Head injuries in Canada: A decade of change (1994-1995 to 2003-2004). 2006.
4. Iverson GL. Outcome from mild traumatic brain injury. Curr Opinin Psychiatry 2005;18:301-317.
5. Master CL, Scheiman M, Gallaway M, Goodman A, Robinson RL, Master SR, Grady MF. Vision diagnoses are common after concussion in adolescents. Clin Pediatr 2016; 55:260-267.
6. Felleman DJ, Van Essen DC. Distributed hierarchical processing in the primate cerebral cortex. Cereb Cortex 1991;1:1–47.
7. Ventura RE, Balcer LJ, Galetta SL. The neuro-ophthalmology of head trauma. Lancet Neurol 2014;13:1006-1016.
8. Capó-Aponte JE, Urosevich TG, Temme LA, Tarbett AK, Sanghera NK. Visual dysfunctions and symptoms during the subacute stage of blast-induced mild traumatic brain injury. Mil Med 2012;177:804–813.
9. McCrory P, Meeuwisse WH, Aubry M, Cantu B, Dvorak J, Echemendia RJ, Engebretsen L, Johnston K, Kutcher JS, Raftery M, Sills A. Consensus statement on concussion in sport: the 4th International Conference on Concussion in Sport held in Zurich, November 2012. Br J Sports Med 2013;47:250-258.
10. Hunt AW, Mah K, Reed N, Engel L, Keightley M. Oculomotor-based vision assessment in mild traumatic brain injury: a systematic review. J Head Trauma Rehabil 2016;31:252-261.
11. Mucha A, Collins MW, Elbin RJ, Furman JM, Troutman-Enseki C, DeWolf RM, et al. A brief vestibular/ocular motor screening (VOMS) assessment to evaluate concussions: preliminary findings. Am J Sports Med 2014;42:2479–2486.
12. Vernau BT, Grady MF, Goodman A, Wiebe DJ, Basta L, Park Y, et al. Oculomotor and neurocognitive assessment of youth ice hockey players: Baseline associations and observations after concussion. Dev Neuropsychol 2015;40:7–11.
13. Ventura RE, Jancuska JM, Balcer LJ, Galetta SL. Diagnostic tests for concussion: Is vision part of the puzzle? J Neurooptha lmol 2015;35:73–81.
14. Scheiman M, Gallaway M, Frantz KA, Peters RJ, Hatch S, Cuff M, et al. Nearpoint of convergence: test procedure, target selection, and normative data. Optom Vis Sci 2003;80:214–25.
15. Szymanowicz D, Thiagarajan P, Ludlam DP, Green W, Kapoor N. Vergence in mild traumatic brain injury: a pilot study. J Rehabil Res Dev 2012;49:1083-1100.
16. Green W, Ciuffreda KJ, Thiagarajan P, Szymanowicz D, Ludlam DP, Kapoor N. Static and dynamic aspects of accommodation in mild traumatic brain injury: a review. Optometry 2010;81:129–36.
17. King D, Brughelli M, Hume P, Gissane C. Concussions in amateur rugby union identified with the use of a rapid visual screening tool. J Neurol Sci 2013;326:59–63.
18. Tjarks BJ, Dorman JC, Valentine VD, Munce TA. Comparison and utility of King-Devick and ImPACT® composite scores in adolescent concussion patients. J Neurol Sci 2013;334:148-153.
19. Galetta KM, Brandes LE, Maki K, Dziemianowicz MS, Laudano E, Allen M, et al. The King–Devick test and sports-related concussion: Study of a rapid visual screening tool in a collegiate cohort. J Neurol Sci 2011;309:34–39
20. Termsarasab P, Thammongkolchai T, Rucker JC, Frucht SJ. The diagnostic value of saccades in movement disorder patients: a practical guide and review. J Clin Mov Disord 2015;2:14.
21. Collins MW, Kontos AP, Reynolds E, Murawski CD, Fu FH. A comprehensive, targeted approach to the clinical care of athletes following sport-related concussion. Knee Surg Sports Traumatol Arthrosc 2014;22:235-246
22. Ellis MJ, Leddy JJ, Willer B. Physiological, vestibulo-ocular and cervicogenic postconcussion disorders: An evidence-based classification system with directions for treatment. Brain Inj 2015;29:238-248.
23. Ciuffreda KJ, Ludlam DP, Yadav NK, Thiagarajan P. Traumatic Brain Injury: Visual consequences, diagnosis, and treatment. Adv Ophthalmol Optom 2016;1:307-333.
24. Gallaway M, Scheiman M, & Mitchell GL. Vision therapy for post-concussion vision disorders. Optom Vis Sci 2017;94:68-73.
25. Poltavski DV, Biberdorf D. Screening for lifetime concussion in athletes: Importance of oculomotor measures. Brain Inj 2014;28:475–485.
26. Christenson GN, Winkelstein, AM. Visual skills of athletes versus nonathletes: Development of a sports vision testing battery. J Am Optom Assoc 1988;59:666-675.
27. Hughes PK, Blundell NL, Walters JM. Visual and psychomotor performance of elite, intermediate and novice table tennis competitors. Clin Exp Optom 1993; 76(2):51-60.
28. Koslowe K, Glassman T. Accommodative amplitude determination: Pull-away versus push-up method. Optom Vis Dev 2010;41:28.
29. Hofstetter HW. Useful age-amplitude formula. Optom World 1950;38:42-45.
30. Scheiman M, Wick B. Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders. Lippincott Williams & Wilkins, 2014.
31. Gall R, Wick B, Bedell H. Vergence facility: Establishing clinical utility. Optom Vis Sci 1998;75:731-742.

32. Orlansky G, Hopkins KB, Mitchell GL, Huang K, Frazier M, Heyman C, et al. Reliability of the Developmental Eye Movement Test. Optom Vis Sci 2011;88:1507-1519.
33. The Convergence Insufficiency Treatment Trial (CITT) Study Group. The Convergence Insufficiency Treatment Trial: Design, Methods, and Baseline Data. Ophthalmic Epidemiol 2009;15:24–36.
34. Yekutieli D, Benjamini Y. Resampling-based false discovery rate controlling multiple test procedures for correlated test statistics. J Stat Plan Inference 1999;82:171–96.
35. Rouse MW, Borsting EJ, Mitchell GL, Scheiman M, Cotter SA, Cooper J, et al. Validity and reliability of the revised convergence insufficiency symptom survey in adults. Ophthalmic Physiol Opt 2004;24:384–390.
36. Wesson MD. Normalization of prism bar vergences. Am J Optom Physiol Opt 1982;59:628–634.
37. Powell JM, Birk K, Cummings EH, Ciol MA. The need for adult norms on the Developmental Eye Movement test. J Behav Optom 2005;16:38–41.
38. Quaid P, Simpson T. Association between reading speed, cycloplegic refractive error, and oculomotor function in reading disabled children versus controls. Graefes Arch Clin Exp Ophthalmol 2013;251:169–87.
39. Porcar E, Martinez-Palomera A. Prevalence of general binocular dysfunctions in a population of university students. Optom Vis Sci 1997;74:111–113.
40. Pearce KL, Sufrinko A, Lau BC, Henry L, Collins MW, Kontos AP. Near point of convergence after a sport-related concussion: Measurement reliability and relationship to neurocognitive impairment and symptoms. Am J Sports Med 2015;43:3055–3061.
41. Kawata K, Rubin LH, Lee JH, Sim T, Takahagi M, Szwanki V, et al. Association of football subconcussive head impacts with ocular near point of convergence. JAMA Ophthalmol 2016;134:763-769.
42. Momeni-Moghaddam H, Goss DA, Sobhani M. Accommodative response under monocular and binocular conditions as a function of phoria in symptomatic and asymptomatic subjects. Clin Exp Optom 2014;97:36-42.
43. Kapoula Z, Morize A, Daniel F, Jonqua F, Orssaud C, Bremond-Gignac D. Objective evaluation of vergence disorders and a research-based novel method for vergence rehabilitation. Transl Vis Sci Technol 2016;5:8.
44. Rouse MW, Borsting E, Deland PN. Reliability of binocular vision measurements used in the classification of convergence insufficiency. Optom Vis Sci 2002;79:254-264.
45. Coffey B, Reichow AW. Optometric evaluation of the elite athlete. Problems in Optometry 1990;2(1):32-59.
46. Tannen B, Darner R, Ciuffreda KJ, Shelley-Tremblay J, Rogers J. Vision and reading deficits in post-concussion patients: A retrospective analysis. Vision Development and Rehabilitation 2015;3:206-213.
47. Tannen B, Ciuffreda KJ, Lyon E, Shelley-Tremblay J. Distance horizontal fusional facility (DFF): A proposed new diagnostic test for concussion patients. Vision Development and Rehabilitation 2016;3:170-175.
48. Trieu LH, Das S, Myung J, Hatch S, Scheiman M. The value of vergence facility testing for the diagnosis of convergence insufficiency. Poster presented at: Annual Meeting of the American Academy of Ophthalmology; 2016 Oct 15-18; Chicago, IL.
49. Hennessey D, Iosue RA, Rouse MW. Relation of symptoms to accommodative infacility of school-aged children. Am J Optom Physiol Opt 1984;61:177–183.
50. Lezak MD, Howieson DB, Bigler ED, Tranel D. Neuropsychological Assessment. Oxford University Press; 2012.
51. McCrea M, Hammeke T, Olsen G, Leo P, Guskiewicz K. Unreported concussion in high school football players: Implications for prevention. Clin J Sport Med 2004;14:13-17.
52. Kaut KP, DePompei R, Kerr J, Congeni J. Reports of head injury and symptom knowledge among college athletes: Implications for assessment and educational intervention. Clin J Sport Med 2003;13:213–221.
53. Kroshus E, Garnett B, Hawrilenko M, Baugh CM, Calzo JP. Concussion under-reporting and pressure from coaches, teammates, fans, and parents. Soc Sci Med 2015;134:66–75.
54. Rosner J. The relationship between moderate hyperopia and academic achievement: how much plus is enough? J Am Optom Assoc 1997;68:648–650.
55. Marshall S, Bayley M, McCullagh S, Velikonja D, Berrigan L, Ouchterlony D, et al. Updated clinical practice guidelines for concussion/mild traumatic brain injury and persistent symptoms. Brain Inj 2015;29:688–700.



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ARTICLE

Persistent Diplopia in Visually Mature Patients. Is it Intractable or something else? A Review and Case Series

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ABSTRACT

Diplopia is described as being intractable when there is inability to both fuse the two images and suppress the second image. Intractable diplopia persists despite achieving ocular alignment using either prisms, lenses,vision therapy,extraocular muscle surgery, or botulinum toxin injection. Treatment usually resorts to occluding or fogging the patient's nondominant eye. Often times, however, adults having other causative mechanisms for supposedly persistent diplopia are able to achieve comfortable single vision with treatment that either establishes fusion or reactivates a preexisting sensory adaptation. This case series reviews these other causes of diplopia.

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INTRODUCTION

Diplopia is the condition in which a single image is perceived simultaneously as being two rather than one.[1] When on examination there is inability to fuse the two images and also suppress the second image, the diplopia is intractable.[2,3] Intractable diplopia is constant and occurs in all positions of gaze despite achieving satisfactory ocular alignment with either prisms, lenses, vision therapy, extraocular muscle surgery, or botulinum toxin injection. Some patients may actively avoid fusion by performing involuntary ocular movements of their nonfixating eye, reporting one image jumping over the other when the strabismus is neutralized either with prisms or with haploscopic devices such as the synoptophore.[4-6]

Since fusion cannot be established, treatment for intractable diplopia is usually palliative. Occlusion or fogging of the nondominant eye with patches,[3] frosted spectacle lenses,[7] partially occlusive filters,[8,9] monovision glasses or monovision contact lenses,[10,11] occlusive contact lenses,[3,7,12] corneal tatooing,[13,14] opaque intraocular lenses,[15,16] and botulinum toxin induced ptosis[13] have been used to ameliorate intractable diplopia.

Although it has historically been reported to be a consequence of either amblyopia or strabismus treatment, the consensus is that intractable diplopia occurs infrequently.[17-21] Cumulative findings from three studies involving adults manifesting constant strabismus without diplopia prior to extraocular muscle surgery reported intractable diplopia developing in only 12 of 899 patients (1.3%) postoperatively.[22-24] Furthermore, strabismus that began in early childhood and either was untreated or treated and recurred is usually accompanied by sensory adaptations such as suppression

and/or anomalous retinal correspondence which prevent diplopia.[25,26] However, in some instances these sensory adaptations may not be permanent and can be changed or lost over the years as illustrated in Case 1.

Case 1

A 17 year – old high school student was referred for constant and long- standing horizontal diplopia which he described as minimally bothersome. He desired to obtain a driver's license.

Ocular history was significant for bilateral optic neuritis and uveitis at 4 months of age. The patient's systemic history was unremarkable. Extraocular muscle surgery in the left eye for probable infantile esotropia occurred at 3 years of age. Bilateral cataract surgery was performed at 8 years of age and YAG capsulotomy in the right eye at 14 years of age. Prism glasses were prescribed when he was 12 years old but was not helpful. He denied having had any patching or orthoptics/ vision therapy.

On examination refractive correction and corrected visual acuities were right eye -1.25 – 2.50 x 14 (20/25) and left eye -1.50 -1.00 x 155 (20/25). The diplopia, described as being horizontal, was absent when either eye was covered. A constant left exotropia of 30 prism diopters (PD) was revealed with the cover test at distance and near. The exotropia was accompanied by bilateral dissociated vertical deviation. Latent nystagmus was present in the left eye. Versions showed bilateral superior oblique overaction with larger exotropia in downgaze.

The patient responded to sensory testing paradoxically as if he was esotropic. With the Worth 4-dot and red lens tests at both distance and near, the diplopia was uncrossed rather than crossed, suggesting anomalous retinal correspondence. The diplopia could neither be fused nor suppressed with any amount of prism. Synoptophore evaluation measured an objective angle of 30 PD base – in and a subjective angle of 22 PD - 24 PD base-out confirming anomalous retinal correspondence.

The diagnosis was consecutive exotropia with intractable diplopia. Bangerter filters and/ or +2 D (diopter) and +3 D Fresnel lenses placed over the left spectacle lens to eradicate the diplopia were uncomfortable for the patient. This was attributed to the blur possibly increasing the dissociated vertical deviation. A 0.2 density Bangerter filter providing sector occlusion and restricted to the central portion of the left spectacle lens used exclusively for driving was more acceptable.[27]

Strabismus beginning in adulthood causes diplopia that usually resolves and fusion reestablished when the eyes are accurately aligned prismatically or surgically. However, intractable diplopia has been reported following prolonged visual deprivation due to cataract or from uncorrected unilateral aphakia in adults with sensory strabismus who eventually received secondary intraocular lens implantation.[28-32] Intractable diplopia has also been reported following severe head injury, ocular trauma, central nervous system disorders, post viral syndromes, intracranial surgery, and cerebral vascular accident or stroke[3-5] as illustrated in Case 2.

Case 2

A 56 year-old man was referred with a complaint of constant diplopia which began following a stroke three years ago. He described the diplopia as being both vertical and horizontal and more bothersome when reading. The diplopia was absent with either eye covered. He denied having strabismus and any other eye disorders during childhood. The referring practitioner prescribed prism glasses that did not alleviate the diplopia. There had not been any strabismus surgery performed.

On examination, refractive correction and visual acuities were right eye Pl – 0.50 x 75 (20/20) and left eye Pl – 0.50 x 110 (20/20). He manifested with the cover test a left hypertropia of 10 PD in primary gaze at distance and near.

An 8 degree right excyclotorsion was measured with the double Maddox rod. Versions showed limited elevation in the right eye and limited depression in the left eye. With the doll's head (oculocephalic reflex) maneuver, versions in both eyes improved suggesting a supranuclear cause for the impaired ocular motility.

Sensory testing with the Worth 4-dot test showed vertical diplopia at both distance and near. Stereopsis with the contour stereotargets (circles) of the Randot stereotest (Stereo Optical Co, Inc, Chicago, ILL) was nil. With the synoptophore which compensated for the torsional as well as the vertical deviation, normal retinal correspondence and "fleeting sensory fusion" were demonstrated. Motor fusion was extremely poor. Moving the tubes of the synoptophore less than 1 PD in any direction caused diplopia.

It was noted that the present glasses had ground-in prism with 3 PD base-down in the right lens and 8 PD base-up in the left lens, which corrected for left hypotropia, not left hypertropia. Using glasses without ground-in prism, treatment included 8 PD base-up Fresnel prism over the upper portion of the right spectacle lens. Since diplopia was more bothersome when reading and achieving fusion at both distance near seemed improbable, a 0.6 density Bangerter filter over the lower portion of the right spectacle lens was included.

One month later diplopia persisted. In this clinician's opinion, the absence of motor fusion and impaired ocular motility in each eye made prism therapy and any other therapy likely ineffective. Treatment consisted of prescribing a 0.3 density Bangerter filter that eliminated the diplopia over the entire right spectacle lens.

Comment

Both patients, Case 1 with childhood onset strabismus and Case 2 with adult onset strabismus, met the criteria for intractable diplopia, showing absence of both fusion and suppression. Case 2 demonstrated "fleeting sensory fusion" with the synoptophore. Due to the lack of demonstrable motor fusion, this most likely indicated superimposition of the synoptophore targets rather than actual sensory fusion of the targets.

Since intractable diplopia can have a substantial impact on the patient's quality of life and occurs infrequently, it should be a diagnosis of exclusion and made only after other causes of diplopia have been ruled out.[3,22,33,34] Many patients I have examined over the years had other types of diplopia that could be treated without constantly occluding or fogging one eye. They were able to achieve comfortable single vision by either changing their optical prescription, altering their fixation pattern or head posture, using prisms, or undergoing vision therapy or extraocular muscle surgery.

The purpose of this report is to review other types of diplopia that should be considered in adults and visually mature patients with persistent diplopia. Representative case reports are included.

Monocular Diplopia

Monocular diplopia is when two images of the object of regard are seen by one eye alone.[34-39] It persists when one eye is covered. It may be constant or intermittent, unilateral or bilateral, vertical or horizontal, and occur with or without strabismus. The patient frequently describes perceiving a halo, ghost image, image overlapping or stacking rather than two distinct separate images. Approximately 6% to 12% of patients with diplopic symptoms have monocular diplopia.[36,40]

Causes of monocular diplopia are numerous being either extraocular or optical, or organic.[34] Extraocular or optical are the most common and can be associated with uncorrected or inadequately corrected refractive error (especially astigmatism), reflections from spectacle lens surfaces, incorrect placement of a bifocal segment, tear film abnormalities, lid abnormalities, corneal irregularity (i.e.,

scarring from refractive surgery or keratoconus which frequently can be corrected with rigid contact lenses), iris abnormalities, cataract, or a dislocated intraocular lens.[34,35,36,40] Incipient cataract with zones of increased optical density in the anterior and posterior subcapsular or nuclear layers of the lens is frequently causative.[34,35] Monocular diplopia attributed to optical causes usually resolves when placing a pinhole before the affected eye.[34]

Organic causes include neurogenic disease, migraine, and retinal abnomalities such as epiretinal membrane, choroidal neovascular membrane, and macular edema. Monocular diplopia that is organic does not resolve with a pinhole.[34]

When occurring with strabismus where diplopia is expected to be binocular, monocular diplopia may not be considered as the source of the patient's symptoms.

Case 3

A 62 year-old woman was referred for long-standing diplopia. She described the diplopia as being vertical in direction. She reported her eyes being misaligned since childhood. Treatment included glasses and vision therapy for many years, the latter given both as a child and as an adult. She denied having had prism glasses, patching, or surgical treatment.

On examination, refractive correction and visual acuities for the right eye were +5.75 - 0.50 x 70 (20/30) and the left eye +6.50 - 3.50 x 107 (20/50). Cover test revealed orthophoria at distance and intermittent left exotropia of 10 PD at near. With the vertical Maddox rod, a left hyper deviation of 0.5 PD was measured at both distance and near. Versions were full in all positions of gaze. Stereopsis with the contour stereotargets of the Randot stereotest was 200 arc seconds. Fusional vergence amplitudes at near were 16/10 and 10/8 for divergence and convergence, respectively.

Further testing indicated that when the right eye was covered, diplopia persisted and when the left eye was covered, diplopia was absent, confirming monocular diplopia. When placing a pinhole aperture before her left eye, diplopia resolved. Biomicroscopy and dilated ophthalmosopy revealed no apparent causes for the monocular diplopia. It was noted that her spectacle lenses were scratched, a large diagonal scratch occurring on the left lens. A manifest refraction was right eye + 6.25 - 0.50 x 73 and left eye + 7.00 - 3.00 x 100 and gave 20/30 visual acuity in each eye. Placing the refraction in a trial frame eliminated the monocular diplopia in her left eye.

The diagnosis was intermittent exotropia with optically-induced monocular diplopia. She was prescribed the updated refraction with a +2.50 add and no longer had diplopic symptoms.

Comment

Case 3 illustrates the importance of ruling out monocular diplopia with patients manifesting strabismus having diplopic symptoms. The patient had intermittent exotropia and had undergone vision therapy both as a child and as an adult. Checking the patient's refraction and quality of the spectacle lenses should not be overlooked or discounted in patients having monocular diplopia.[37,38]

Spontaneous and Rapid Alternate Fixation

Adults having strabismus since childhood may notice a shift or jump in apparent position of the fixation target if they rapidly change fixation from one eye to the other eye.[25,41] They frequently describe this phenomenom as diplopia. Rapid alternate fixation is more common with patients unable to fuse having small-angle deviations and is particularly bothersome when driving, playing sports, and doing close work.

Observing the patient's fixation pattern and relating it to findings from sensory tests is essential for the correct diagnosis. With the Worth 4-dot test, for example, patients may report diplopia, seeing five lights. The clinician should inquire whether the five lights

are present simultaneously or whether they alternate between two and three lights very quickly. When maintaining fixation with each eye in turn, "diplopia" is not present. Treatment of rapid alternate fixation encourages the patient to develop and fixate exclusively with his/her dominant eye.

Case 4[8]

A 44 year-old woman was referred for diplopia which reportedly began in childhood. She described the diplopia as being both horizontal and vertical. Ocular history included amblyopia treatment as a child that involved full-time alternate day patching, patching the right eye one day and patching the left eye the next day. This was done for 3 years. There had been no other treatment.

On examination visual acuity refractive error and visual acuties were +0.50 -0.25 x 115 and 20/20 in each eye. The patient manifested a constant, alternating esotropia of 2 PD at distance and 14 PD at near. No vertical deviation was detected. Versions were full in all positions of gaze.

Sensory testing with the Worth 4-dot test revealed uncrossed diplopia at both distance and near. Stereopsis was nil with the contour stereotargets of the Randot stereotest. The synoptophore indicated normal retinal correspondence without evidence of fusion.

Observation of the patient's fixation pattern indicated that she rapidly alternated fixation between the eyes. Repeating the Worth 4-dot test showed that she did not perceive five lights simultaneously. Maintaining fixation with either eye resulted in suppression of the contralateral eye, seeing either two or three lights depending on which eye was fixating at the time.

The diagnosis was esotropia with rapid alternating fixation. In an attempt to disrupt her habitual fixation pattern, a 0.3 density Bangerter filter was placed over the right spectacle lens. This was the least dense filter that eliminated the rapid alternate fixation. On follow up, the patient reported less "diplopia" but found wearing the filter annoying. Switching the filter to the left spectacle lens was more acceptable.

The patient returned to her referring practitioner with the treament plan of gradually tapering the density of the filter and possibly its discontinuation while maintaining fixation exclusively with her right eye.

Case 5

A 45 year-old police officer was referred for diplopia since childhood. The referring practitioner diagnosed intractable diplopia and treated him with an occlusive contact lens. Ocular history included four strabismus surgeries, the first at 4 years of age and the last when he was 25 years old. He had undergone vision therapy from 4 years old to 12 years old. The latter was given by an orthoptist and reportedly included fusion and antisuppression procedures. The patient denied any patching or prism therapy.

Present examination revealed refractive correction and corrected visual acuities for right eye Pl – 1.00 x 73 (20/25) and for left eye -0.75 – 0.75 x 75 (20/20). The patient manifested a constant right exotropia of 12 PD at distance and 18 PD at near. Despite the slight difference in visual acuities, he could readily maintain fixation with his right eye. No vertical or torsional deviation was detected. Versions indicated full ocular motility in all positions of gaze.

When covering either eye, diplopia was absent. With the red lens test, uncrossed diplopia was perceived at distance and crossed diplopia at near. The Worth 4-dot test showed uncrossed diplopia at distance and near. Fusion could not be demonstrated when neutralizing the strabismus with prisms. The Randot stereotest, however, revealed 70 arc seconds and 250 are seconds with contour stereotargets and random dot stereotargets, respectively. Synoptophore testing indicated normal retinal correspondence, sensory fusion with stereopsis, and a limited range of motor fusion.

Due to the presence of reasonably good stereopsis, It was concluded that the patient did not have constant exotropia with intractable diplopia but rather intermittent exotropia with fusion capability, albeit tenuous. Home- based vision therapy procedures using stereoscopic vectograms 20 minutes per day were prescribed to enhance fusional vergence amplitudes and vergence facility.

On follow-up, he reported compliance with the home-based exercises but no relief of diplopia. Sensorimotor testing showed findings consistent with the previous visit. Careful questioning with the Worth 4-dot test, however, indicated that he perceived not five lights simultaneously but rather two and then three lights seen in rapid succession. Spontaneous alternate fixation between eyes was apparent. Having the patient consciously maintain fixation with either eye in turn resulted in suppression of the contralateral eye. These findings were confirmed with the red lens and Bagolini striated lens tests.

The diagnosis was recurrent intermittent exotropia with rapid spontaneous alternate fixation. Treatment was altered to encourage fixation exclusively with his dominant left eye. Placing a 0.4 density Bangerter filter on the spectacle lens over his right eye was the minimal density filter that inhibited the rapid alternate fixation and resolved the "diplopic" symptoms. The goal was to eventually taper and discontinue the filter with the possibility of improvement in fixation.

Comment

The extensive treatment during childhood for both patients may have caused the problematic fixation pattern in adulthood. Case 4 had full time alternating patching for 3 years and Case 5 had vision therapy for 8 years and also four extraocular muscle surgeries. Additional vision therapy and/or surgery to enhance the fusion capability for Case 5 were declined by the patient.

Both patients were given Bangerter filters (Reyser Optik AG, St. Gallen, Switzerland) which are used to treat amblyopia in children,[42] and in adults having intractable diplopia[8,9] (Case 1 and Case 2), and other types of diplopia that may not be immediately amenable to prisms, vision therapy, or extraocular muscle surgery. Bangerter filters or foils are thin plastic and translucent partially occlusive filters that degrade central vision and eliminate diplopia while allowing a full visual field and favorable cosmesis. Similar to a Fresnel prism, they are usually placed on the inner surface of the spectacle lens usually before the patient's nondominant eye. The filters create a functional central scotoma in the eye with the filter under binocular viewing conditions.[9] They vary in strength from the most dense (<0.1), which produces marked reduction in visual acuity (20/300), to the barely occlusive (0.8), in which nearly normal visual acuity (20/25) is possible. Since degradation in visual acuity is patient dependent and not always equal to manufacturer estimations,[43] the weakest density filter that resolves the diplopic symptoms is prescribed.[7-9] With less dense filters, peripheral fusion and stereopsis can be maintained in some cases.[7,9]

The long term treatment effect of Bangerter filters for diplopia is unknown. The preferred method is to initially prescribe the weakest density filter that eliminates the diplopia and over time taper the filter's density either partially or entirely if possible.[8,9]

Fixation Switch Diplopia

As illustrated in Cases 4 and 5, when alternating strabismus occurs in patients with a history of childhood onset strabismus, suppression usually changes from one eye to the other eye with a switch in fixation.[25] Patients having nonalternating strabismus and strong fixation preference may not be able to transfer suppression to their preferred or fixating eye. They instead experience diplopia when fixating with their nonpreferred or deviating eye.[25,29] For example, a patient having left esotoropia with

suppression may experience diplopia when forced to fixate with the left eye. Referred to as fixation switch diplopia, the diplopia can be either intermittent or constant.[44-50] Its diagnosis is confirmed when diplopia is present with only the right eye or only the left eye fixating under binocular viewing conditions.

Although it can be idiopathic, fixation switch diplopia is more frequently associated with a reduction in visual acuity in the preferred or fixating eye.[49] This can occur naturally when a myopic refractive shift occurs either exclusively or more so in the preferred eye relative to the nonpreferred or deviating eye. If the distance visual acuity is reduced to a level below that of the nonpreferred eye, fixation may be switched to the nonpreferred eye, causing diplopia. Prescribing the optimal refractive correction usually restores the previous fixation preference and suppression. Fixation switch diplopia can also occur when a cataract or retinal disorders such as macular edema, age related macular degeneration, and retinal detachment reduces visual acuity in the preferred eye causing fixation to be switched to the nonpreferred eye having better visual acuity.[4,49]

Fixation switch diplopia can be iatrogenically induced. Refractive corrections determined subjectively and without cycloplegia for adults having strabismic amblyopia can be causative.[45] The possibly better visual acuity obtained in the amblyopic eye can lead to a fixation switch to that eye, resulting in diplopia. Adults with mild strabismic amblyopia without fusion capability should not have the visual acuity in their deviating eye improved to a level equal or better than that of their fixating eye to avoid fixation switch diplopia.

The use of monovision to treat presbyopia using contact lenses, corneal refractive surgery, or cataract surgery with intraocular lenses has become more common. Since monovision forces fixation with one eye at distance and the other eye at near, it may induce fixation switch diplopia in visually mature patients with a history of nonalternating childhood onset strabismus.[45-47,49] Restoration of the original fixation pattern by reversing the monovision and prescribing the optimal refractive correction for each eye with glasses, contact lenses, or further refractive surgery usually resolves the diplopia.

Fixation switch diplopia is not uncommon. In a report on 152 adults with history of childhood strabismus having diplopic symptoms, 20 (13%) cases were attributed to fixation switch diplopia.[40]

Case 6

A 34-year-old physician's assistant was referred for long-standing and occassional diplopia. She had undergone two surgeries for childhood onset esotropia, the first at 5 years of age and the second two months before the current examination. The recent surgery did not alleviate the horizontal diplopia which was reported to be variable and more problematic when doing suturing. The patient also reported poor depth perception and blurred vision. She denied having had any occlusion, prisms, or vision therapy. She wore contact lenses for myopic anisometropia (right eye -7.00 D and left eye -5.25 D).

On examination corrected visual acuities were 20/20 in each eye. The patient manifested an esotropia that was barely noticeable at casual glance. Cover testing revealed an incomitant deviation. When fixating with her right eye at both distance and near, esotropia of 25 PD was measured whereas when fixating with her left eye, esotropia of 12 PD was measured. Version testing revealed a mild adduction deficiency in the right eye causing exotropia in extreme left gaze.

Sensory testing indicated that the diplopia was dependent upon which eye was fixating. With right eye fixation, diplopia occurred whereas with left eye fixation, there was suppression. This was confirmed with the Worth 4-dot test which showed with right eye fixation, five lights (uncrossed diplopia) and with left eye fixation, two or three lights (suppression) at distance and near. Sensory testing also

revealed that the strabismus was intermittent as indicated with the Randot stereo test (100 arc seconds for contour stereotargets and 250 arc seconds for random dot stereotargets). Cover test during stereotesting revealed absence of strabismus.

The diagnosis was incomitant strabismus with fixation switch diplopia. Since the visual problems were more troublesome for near and the patient had fusion capability, the minimum prism (8 PD base-out) and minimum added plus lenses (+1.50 D) that provided continuous fusion when suturing were prescribed over her contact lenses.

Comment

Treatment for fixation switch diplopia involves restoring the patient's original fixation pattern which allowed suppression to occur. Case 6 was atypical for fixation switch diplopia since there was absence of visual acuity reduction and presence of fusion. The latter was revealed with stereotesting and reinforced with treatment. Additional extraocular muscle surgery was declined by the patient.

Changes in Ocular Alignment

Suppression associated with childhood onset strabismus tends to be regional as well as facultative.[25,41] Hemiretinal suppression zones are specific to the direction of the strabismus developing mostly in the nasal hemiretina with esotropia and mostly in the temporal hemiretina with exotropia. Since suppression is not developed immediately or at all in visually mature patients, diplopia can result when there is a sudden change in the angle of strabismus. This can occur either spontaneously or following strabismus surgery, trauma, illness, paresis or mechanical restriction of an extraocular muscle(s), or changes in the patient's refractive management or refractive needs.[21-23,51]

Diplopia is likely when strabismus changes direction.[51,52] If the eyes were esotropic since early childhood and become exotropic, diplopia is probable since the extra-foveal image is no longer on the nasal hemiretina but on the non-suppressed temporal hemiretina. Similarly if the eyes were exotropic since early childhood and become esotropic, diplopia is probable since the extra-foveal image is no longer on the temporal hemiretina but on the non-suppressed nasal hemiretina. Diplopia may also occur when the angle of strabismus increases or decreases yet remains in the same direction. Reverting back to the previous angle of deviation either with prisms or extraocular muscle surgery usually eliminates the diplopia.[23,25,29]

When there is anomalous retinal correspondence and the magnitude of the strabismus changes, diplopia can be paradoxical.[25] For example, in a patient with moderately- sized esotropia and anomalous retinal correspondence preoperatively and a small angle esotropia postoperatively, any diplopia will be perceived as being crossed rather than uncrossed.

Rather than actual hemiretinal suppression zones, a "trigger mechanism" operating on a hermiretinal basis has been proposed. This "trigger mechanism" determines whether suppression or diplopia occurs and is activated by the image of the fixation target crossing the midline of the retina from the nasal side to the temporal side or vice versa.[41,53]

Changes in ocular alignment are a common cause of diplopia, occurring in 95 of 152 adults (63%) having strabismus dating back to childhood.[40]

Case 7

A 39 year-old woman was referred for recent onset diplopia. The diplopia, which was horizontal in direction, developed three months earlier. She denied having recent trauma, illness, ocular surgery, or taking any medication. The patient's ocular history included childhood onset left esotropia which was treated with glasses and surgery, the latter occurring at 7 years of age. Occlusion therapy was given for amblyopia. Records from the

referring practitioner indicated esotropia of 30 PD at distance and near prior to the onset of diplopia. All current ocular, systemic, and neurological health evaluations were normal.

On examination, corrected visual acuities with small hyperopic refractive correction were 20/25 and 20/50 for the right and left eyes, respectively. A constant left esotropia of 50 PD was measured at distance and near. Version testing revealed full ocular motility in all positions of gaze. Sensory testing with the Worth 4-dot and Bagolini striated lens tests at distance and near showed uncrossed diplopia which did not resolve with neutralizing prisms. Synoptophore evaluation showed an objective angle of 38 PD base-out and a subjective angle of 18 PD base-out, suggesting unharmonious anomalous retinal correspondence. With 15 PD base - out before her left eye, the diplopia was abated.

Treatment included prescribing the minimum prism power that provided continuous suppression (Fresnel prism 15 PD base-out over the left spectacle lens) and subsequent strabismus surgery.

Comment

When prior records indicate a change in the angle of strabismus for patients with recent onset diplopia, prisms can frequently provide single vision. For Case 7, 15 PD base-out prism optically simulated her prior angle of strabismus and placed the extrafoveal image within a preexisting suppression zone on her nasal hemiretina. Extraocular muscle surgery or botulinum toxin injection would be expected to produce similar results.

Regarding prisms, they can be used diagnostically to help identify patients at risk for diplopia by simulating changes in ocular alignment to be achieved surgically.[22,40] This is more of a concern when strabismus surgery is to be performed on adults having neither diplopia nor fusion capability.

While wearing his/her refractive correction, the patient fixates an isolated Snellen optotype at distance. Loose prisms, a prism bar, or rotary prisms of increasing power are placed before the deviating eye. The patient is instructed to report if and when diplopia occurs and disappears as prism power is changed. When diplopia is reported, prism is decreased to determine at what point it can be eliminated and if findings are reproducible. During testing, the patient maintains fixation with his/her preferred eye so that rapid alternate fixation, which can mimic diplopia, is not induced (Case 4 and Case 5). If diplopia is elicited at or near the target angle to be achieved with surgery, Fresnel prisms can be prescribed to determine whether the diplopia subsides or persists over time.

Regarding usefulness of this procedure, reports indicate that although it is very sensitive, it has a relatively low positive-predictive value.[22] Perceiving diplopia with prisms revealed only a small risk for temporary diplopia and minimal risk for developing intractable diplopia postoperatively.[22] On the other hand, absence of diplopia with prisms was excellent assurance that diplopia would not occur postoperatively, indicating a high negative-predictive value.

The low positive-predictive value has been attributed to the different mechanisms involved when comparing the effect of prisms and extraocular muscle surgery. The use of prisms to simulate postoperative alignment does not account for changes in proprioceptive afferent input that occur following extraocular muscle surgery.[22] Botulinum toxin injection reportedly allows better simulation of surgical alignment and has been shown to reduce the proportion of false-positive diplopia tests.[54]

Diplopia occurring with horizontal strabismus that is vertically incomitant can appear to be intractable. When the magnitude of esotropia or exotropia changes by 10 PD-15 PD or more in up versus downgaze, it is referred to as an alphabet pattern strabismus (i.e., A, V, X, or Y pattern). For patients with alphabet patterns,

sensorimotor testing should not be restricted to primary gaze. Diplopia that appears to be intractable in one position of gaze does not rule out either suppression or fusion in other positions of gaze.

Case 8

A 42 year-old man was referred because of long-standing diplopia which became more troublesome with his new glasses. The diplopia was described as being horizontal and worse when reading and less when when depressing his chin. The patient recalled having strabismus as a child. Prism glasses and contact lenses had been prescribed but did not relieve the diplopia. There had not been any strabismus surgery. He denied head trauma or any health concerns. The referring practitioner recently prescribed progressive addition lenses for incipient presbyopia.

On examination, refractive correction and visual acuities were right eye +1.25 D (20/20) and left eye +1.00 D (20/20) with a +1.50 D add. The patient manifested a constant left esotropia of 18 PD and 20 PD at distance and near, respectively. The amount of esotropia was similar in right and left gaze. In upgaze, there was absence of strabismus whereas in downgaze, esotropia measured 25 PD. Version testing showed full ocular motility in all positions of gaze.

Sensory findings concurred with the patient's ocular alignment status. With the Worth 4-dot test, uncrossed diplopia occurred in primary and downgaze whereas in upgaze, the patient reported 4 lights (fusion). Stereopsis with the contour stereotargets of the Randot stereotest was nil in primary and downgaze whereas 100 arc seconds was measured in upgaze. Synoptophore testing showed normal retinal correspondence, sensory fusion with stereopsis, and fusional vergence amplitudes.

The diagnosis was diplopia due to long-standing V-pattern esotropia. Because the esotropia and diplopia increased in downgaze and there was demonstrable fusion, progressive addition lenses were discontinued. Diplopia resolved and fusion was maintained using multifocal contact lenses combined with prism glasses (9 PD base-out), the prism amount being the minimum that permitted fusion.

Comment

Progressive addition lenses may be contraindicated in presbyopes with alphabet pattern strabismus and fusion potential.[55] *Since having a wide transition zone between the distance and near segment, progressive addition lenses force patients to view further into downgaze when reading than with conventional flat top "line" bifocal lenses. Switching to high- set flat top bifocals or multifocal contact lenses may be helpful. This justifies the patient in Case 8 with V-pattern esotropia reporting the diplopia being more troublesome with his new glasses.*

Torsion

Torsion or cyclotorsion is a wheel-like movement of the eye around its antero-posterior or fixation axis. Extorsion or excyclotorsion is wheel-like movement of the eye outward away from the nose whereas intorsion or incyclotorsion is wheel-like movement of the eye inward toward the nose.

Torsion occurs mostly with cyclovertical strabismus and can affect one or both eyes.[3,56] When misalignment between the eyes involves the cyclovertical extraocular muscles, torsional diplopia may occur. Since patients with cyclovertical strabismus usually have coexisting vertical and horizontal deviations, the diplopic images will be described as being not only tilted, oblique, or slanted, but also vertical and horizontal, the amount being incomitant.

The most common cause of torsional diplopia is superior oblique palsy, accounting for approximately 67% of all cases.[56-58] Other causes include palsies or restrictions involving the other cyclovertical extraocular muscles, thyroid myopathy, and skew deviation.[56-58]

Optical conditions such as uncorrected astigmatism with an oblique axis or improper placement of the axis of an astigmatic correction can also cause torsion.[59] Torsional diplopia can occur secondary to certain retinal disorders and macular translocation surgery.[60]

Assessment of torsion is advisable for any patient whose diplopia cannnot be resolved with offset of the deviation with prisms, particularly when the deviation is vertical. Torsion is measured in degrees subjectively using the double Maddox rod test which is considered the gold standard, the Bagolini striated lenses, the synoptohore, or the Hess-Lancaster test.[25,61] It can also be measured objectively using either binocular indirect ophthalmoscopy or fundus photography to determine whether the fundus is anatomically rotated from its normal position.[25,56] The fovea is normally 0.3 disc diameters below and temporal to the center of the optic disc. While viewing the retina with indirect ophthalmoscopy when there is no torsion, an imagined horizontal line through the fovea will pass through the upper half of the optic disc. When there is torsion, the imagined line will appear either above (excyclotorsion) or below (incyclotorsion) the upper half of the optic disc in the involved eye. This technique requires much skill and experience to observe.

An amount of 6 degrees or more can degrade fusional vergence amplitudes and reduce stereopsis.[62] Excyclotorsion exceeding 10-15 degrees frequently accompanies bilateral superior oblique palsy and can disrupt fusion entirely.[56] Since prisms only have horizontal and vertical effect, torsional diplopia cannot be neutralized and thus can mimic intractable diplopia. It can be offset with the synoptophore along with any coexisting vertical/ horizontal deviations, allowing assessment for fusion. When fusion is demonstrated with the torsion neutralized, the diplopia is not intractable. Similar results can likely be achieved with accurate extraocular muscle surgery that specifically addresses the torsion along with other aspects of the strabismus.[63]

Case 9

A 33 year- old man reported long-standing diplopia which he described as being torsional, vertical, and horizontal. Ocular history included three surgeries for strabimus caused by a car accident when he was 21 years-old. The last surgery was six years ago. Prism therapy had not been successful and it was suspected by the referring practitioner that he had intractable diplopia. The patient denied having strabismus or any other binocular vision disorder as a child.

On examination, corrected refractive error and visual acuities were for the right eye -0.75 – 0.50 x 22 (20/25) and for the left eye -0.25 D sphere (20/20). A compensatory head posture was not present. The patient manifested at distance a constant right exotropia of 14 PD with 2 PD right hypotropia and at near right exotropia of 20 PD. In upgaze, there was 10 PD right exotropia and in downgaze, 10 PD right hypotropia. Versions showed moderate superior oblique underaction and inferior oblique overaction in the left eye. With the double Maddox rod, 26 degrees excyclotorsion (18 degrees right eye and 8 degrees left eye) was measured in primary gaze. The bilateral excyclotorsion was confirmed with fundus photography and binocular indirect ophthalmoscopy. Compensating for the strabismus with prisms did not eliminate the diplopia.

Sensory testing with the Worth 4-dot test revealed diplopia at distance and near. Stereopsis was nil with the contour stereotargets of the Randot stereotest. With the synoptophore which also offset the torsional deviation, normal retinal correspondence, sensory fusion with stereopsis, and fusional vergence amplitudes were demonstrated.

The diagnosis was residual cyclovertical strabismus with large excyclotorsion. Since fusion capability was present, additional extraocular muscle surgery was recommended. However, the patient decided against surgery at this time and used a 0.2 density Bangerter filter that resolved the diplopia over his right spectacle lens.

Comment

Diplopia accompanied by large amounts of torsion can mimic intractable diplopia. That fusion could be demonstrated with the synoptophore implies a favorable prognosis once torsion is reduced or eliminated and ocular alignment is achieved with extraocular muscle surgery.[63]

Accommodative Disorders

Patients with strabismus may have poor control of accommodation and not accommodate as well or as steadily as patients without strabismus. Accommodative insufficiency and accommodative spasm have been reported with both exotropia and esotropia.[25,64-70]

Exotropia occurring with accommodative insufficiency can be either primary and the accommodative insufficiency secondary or vice versa.[64] When exotropia is primary, the increased vergence demand stimulates the accommodative system to produce additional accommodative convergence to compensate for the exotropia and maintain fusion. The accommodative function can become chronically fatigued, leading to accommodative insufficiency. When accommodative insufficiency is primary, the poor focus of the target and a low accommodative vergence level can cause the vergence function to become fatigued, leading to exotropia.

A series of adolescent and young adults having exotropia and severely reduced accommodation was reported by Rutstein and Daum.[64] Symptoms included long-standing diplopia and blurred vision that had not responded to either to vision therapy, prisms, or extraocular muscle surgery. Orthophoria or small exophoria was present at distance and intermittent exotropia ranging from 5 PD to 20 PD was present at near. Clinical testing indicated that the patients had markedly reduced amplitudes of accommodation and difficulty sustaining accommodation. Accommodative response determined with dynamic retinoscopy (monocular estimate method) showed a large and varying accommodative lag relative to the accommodative demand indicating inability to sustain accommodation for any period of time. This involuntary fluctuation of accommodation as determined with dynamic retinoscopy appeared to be related to the presence or absence of the exotropia. With accommodation sustained, there was ocular alignment as revealed by cover test and single, clear vision whereas with accommodation released, there was exotropia, diplopia, and blurred vision. Rutstein and Daum concluded that the defective accommodation was a contributing cause for the exotropia and persistent diplopia. Treatment with base-in prism and bifocal glasses resolved the chronic visual symptoms for most patients.

Some investigators have attributed the poor accommodation as being secondary to the exotropia, it being due to under accommodation resulting from loss of convergence.[70-72] Horwood and Riddell, for example, reported that during decompensation of distance intermittent exotropia, accommodation normally driven by disparity cues becomes reduced as the drive from convergence is extinguished.[70-72] Regardless of the exact mechanism, the resulting diplopia can be persistent if the accommodative disorder is not addressed.

Case 10

A 27 year-old man was referred for long-standing horizontal diplopia and blurred vision which were more bothersome at near. He wore glasses for a small refractive error. He denied a history of strabismus, head trauma, systemic illness, or taking any medications. Prism glasses did not alleviate the diplopia.

Examination revealed refractive error and visual acuities for the right eye +0.75 – 0.75 x 110 (20/24) and the left eye +0.50 – 0.50 x 91 (20/25) Cover testing showed poorly controlled intermittent and alternating exotropia of 6 PD at distance and 14 PD at near. Versions and pupillary testing were normal. Near point of convergence was 50 cm. With the Worth 4-dot

test, fusion was present at distance and crossed diplopia at near. Stereopsis with the contour stereotargets of the Randot stereotest was 140 arc seconds.

The possible source of the exotropia, diplopia, and blurred vision became evident when evaluating accommodation. His amplitude of accommodation was less than 1 D in each eye. Dynamic retinoscopy showed a variable and large underaccommodative response (accommodative lag ~ 2.25 D) in each eye.

The diagnosis was intermittent exotropia with severe accommodative insufficiency. The minimum prism (6 PD base – in) and plus lens add (+1.75 D) that provided single and clear vision combined with the refractive correction was prescribed.

Two months later, he reported absence of diplopia and clear vision. Exotropia could not be elicited with the new glasses. Home based vision therapy utilizing vergence and accommodation procedures were given. The goal was to gradually taper and possibly eliminate the prism and bifocals.

Seven months following his initial visit, the patient reported absence of diplopia and blurred vision even without his glasses. Examination without optical correction indicated visual acuities of 20/20 in each eye. Cover test revealed 8 PD exophoria at distance and near. Near point of convergence was 3 cm. The patient fused the Worth 4-dot at distance and near and had 70 arc seconds with the contour stereotargets of the Randot stereotest. Accommodative amplitudes were 6 D and dynamic retinoscopy showed a stable accommodative response (accommodative lag ~ 0.50 D) in each eye.

The prism and bifocals were discontinued.

Comment

Clinical notes from the referring practitioner made no mention of any accommodative disorder. It cannot be assumed that patients with intermittent exotropia or any type of strabismus accommodate as well or as steadily as non-strabismic patients.[70]

Spasm of the near reflex or convergence spasm is a rare syndrome that can either mimic or occur simultaneously with esotropia.[73-76] It usually consists of intermittent and variable episodes of sustained maximal convergence, accommodative spasm, and pupillary miosis.[77,78] Ocular symptoms include diplopia, blurred vision, ocular pain, photophobia, or eye strain. Nonspecific symptoms such as headache, nausea, and dizziness may also occur. Dynamic retinoscopy shows a large and unstable over accommodative response or lead of accommodation.[75,77] Limited abduction, unilateral or bilateral, can accompany the episodes.[79] Mostly functional in etiology and self-limiting, organic causes have also been reported.[80]

Spasm of the near reflex is more frequent in children, adolescents, and young adults and is a differential diagnosis for other acquired esotropias, both comitant and incomitant.[74,76,78,79] Treatment usually includes inhibiting the accommodative and convergence spasm using cycloplegic agents combined with bifocal glasses. For persistent cases, botulinum toxin has had some success.[81,82]

When occurring in older patients, convergence spasm and pupillary miosis can occur without accommodative spasm. The accompanying diplopia can be persistent and diagnosed as being intractable.

Case 11[83]

A 65 year-old man was referred for long-standing horizontal diplopia. When he was 19 years old, he suffered an injury to his right cornea that required pressure patching. He reported that the patch was applied for 3 months. Following its removal, his right eye turned inward and he had experienced diplopia ever since. He had been examined annually the past 10 years in the primary care clinic at

our institution and esotropia with diplopia had always been documented. Since prism glasses had not helped, he was diagnosed with intractable diplopia and placed on medical disability. Ocular history also included myopic astigmatism, bilateral vitreous detachment, and bilateral cataracts. There had not been any orthoptics/vision therapy or extraocular muscle surgery.

Examination revealed refractive error and visual acuities for the right eye -8.00 – 1.75 x 75 (20/40) and left eye -6.00 – 1.50 x 95 (20/40). The patient manifested a fluctuating and difficult to measure right esotropia of approximately 25 PD at both distance and near. The variability of the strabismus appeared to be due to a superimposed convergence spasm. With versions, a marked abduction deficit occurred bilaterally accompanied by pupillary miosis. With duction eye movements, abduction in each eye became full and the pupils were not miotic.

Despite reporting diplopia, the patient suppressed the right eye with the Worth 4-dot test at distance and near. Stereopsis with the contour stereotargets of the Randot stereotest was nil. With the synoptophore, normal retinal correspondence, sensory fusion with stereopsis, and fusional vergence amplitudes were demonstrated.

One month later, the patient also reported experiencing occassional superimposition of two dissimlar images, suggesting possible visual confusion.[25] Corrected visual acuities were 20/30 in each eye. A variable right esotropia of 25 PD with an apparent superimposed convergence spasm continued to be measured. Sensory testing was consistent with the previous visit.

Because of the variable esotropia, pupillary miosis, and limited abduction which improved with duction eye movements, the diagnosis was diplopia due to esotropia and convergence spasm. The patient was referred for strabismus surgery which included a 7.5 mm recession of the right medial rectus. Postoperative visits revealed visual acuity of 20/30 in each eye, absence of diplopia, orthophoria at distance and near, normal pupillary size, full ocular motility, and 100 arc seconds with the contour stereotargets of the Randot stereotest.

Comment

It was speculated that the convergence spasm increased the magnitude of the esotropia and thus the separation between the diplopic images, allowing the patient to occassionally ignore rather than suppress the second image as revealed with the Worth 4-dot test.[25,82] Eliminating the esotropia eliminated the need for the convergence spasm.

Retinal Disorders/Surgery

Diplopia can occur secondary to retinal disorders such as epiretinal membrane, retinal wrinkling, vitreomacular traction, macular hole, macular edema, subretinal neovascularization, macula-off retinal detachment, and surgery that involves the macula.[8,84-86] Its cause is attributed to mechanical distortion and displacement of the macula from its normal centration relative to the peripheral retina leading to rivalry between central and peripheral fusional mechanisms.[84-86] The ocular alignment necessary to superimpose corresponding points in both foveae is different from the alignment that superimposes corresponding points in the peripheral retina of each eye. The patient's peripheral fusion tries to help align the anatomically misaligned central macular points preventing central fusion, resulting in diplopia. The diplopia is frequently intermittent and tends to improve in dim illumination.[84-86] Cover testing usually reveals a small-angle comitant vertical strabismus.[8,60,84-86] Cyclotorsion may also occur with absence of any cyclovertical muscle dysfunction.[60] Often visual acuity is not greatly affected.[60]

Aniseikonia and metamorphopsia frequently contribute to the diplopia and make it more difficult to treat.[60,87-93] The perceived image seen by the affected eye is distorted and of a different shape and size from that seen by the

other eye. The image size disparity, referred to as anatomic or retinally induced aniseikonia, results from forces causing either stretching or compression of the retinal photoreceptors, the former producing micropsia (perceiving a smaller image) and the latter macropsia (perceiving a larger image).[60] As this disruption of the photoreceptors is typically not uniform across the retina, the amount of aniseikonia is heterogenous and field dependent.[89,91] It can be larger or smaller in one meridian than the other. Methods used to treat optically induced aniseikonia due to anisometropia such as iseikonic lenses, contact lenses, or contact lens/spectacle lens combinations (Galilean telescopes) may be less helpful for aniseikonia that is retinally induced.[88,89,91] Anectodotal case reports have reported some success.[91,94]

Treatment with prisms alone for the small-comitant vertical strabismus should be attempted initially.[85] Although rarely successful, if compensating for the deviation provides stable fusion, prisms can be prescribed.

Case 12[89]

A 66 year-old woman was referred for diplopia. Its onset was 3 months ago and coincident with retinal detachment surgery in her right eye. The diplopia was vertical in direction, more troublesome at distance, and absent when either eye was occluded. She also reported the image in her right eye being approximately 20% smaller than the image in her left eye. She presently used "over the counter" reading glasses. The patient denied having strabismus or any other binocular vision disorder as a child.

Habitual visual acuities were 20/40 in each eye. With refraction right eye -0.75 - 0.25 x 35 and left eye +1.50 - 0.75 x 100, visual acuities improved to 20/30 and 20/20 for the right and left eyes, respectively. Amsler grid testing revealed slight metamorphopsia along the horizontal meridian in the right eye.

The patient manifested a constant right hypertropia of 8 PD at distance and 4 PD at near. The hypertropia was similar in amount for right and left gaze and with forced right head tilt and forced left head tilt. Versions revealed full ocular motility in all positions of gaze. With the double Maddox rod test, 5 degrees right excyclotorsion was measured.

The Worth 4-dot test indicated vertical diplopia at distance and fusion at near. Stereopsis was nil with the contour stereotargets of the Randot stereotest.

Aniseikonia measured with the New Aniseikonia Test (Handaya Co.,Tokyo, Japan) showed 10% image disparity between the eyes, the right eye requiring the magnification. Despite the aniseikonia, metamorphopsia, and torsion, prism 7 PD base-down right eye combined with the refractive correction produced stable fusion and was prescribed.

More frequently, diplopia induced by retinal disorders or surgery can be intractable and requires occlusion or fogging of the nondominant eye. Bangerter filters alone or combined with Fresnel prisms have been used with intractable diplopia due to maculopathy.[86]

Case 13[8,89]

A 78 year-old diabetic man complained of long-standing vertical diplopia and image size disparity. The image seen by his right eye was described as being much smaller than that of his left eye. Ocular history included bilateral pseudophakia, bilateral pars plana vitrectomy, bilateral macular edema worse in the left eye, and bilateral epiretinal membrane peel. The visual symptoms commenced with the retinal disorders. Prism glasses had been unsuccessful. The patient was presently using "over the counter" reading glasses.

On examination, unaided visual acuities were 20/30 and 20/25 for the right and left eyes, respectively. With the Amsler grid, metamorphopsia without scotoma was present in both eyes, the right eye more than the left eye. Cover test showed right exotropia of 6 PD with right hypotropia of 2 PD at distance and 8 PD right exotropia with 2 PD right hypotropia

at near. The vertical deviation was confirmed subjectively with the Maddox rod. Versions were full in all positions of gaze. With the double Maddox rod, 5 degrees right excyclotorsion was measured. Sensory testing with the Worth 4-dot test showed diplopia at distance and near. Stereopsis with the contour stereotargets of the Randot stereotest was nil.

With the New Aniseikonia Test, 3.5% aniseikonia was present, the right eye requiring the magnification. Placing a 3.5% size lens before his right eye provided more visual comfort.

The diagnosis was retinally induced aniseikonia and small-angle strabismus. Since the patient used "over the counter" reading glasses and was nearly emmetropic, prescribing iseikonic lenses to minimize aniseikonia was not feasible. Applying 2 PD base-up Fresnel prism before the right eye did not eliminate the diplopia. Adding a low density Bangerter filter (0.6) with the prism provided single vision.[86] The patient was bothered by the blur with the filter and after two months used only the prism which did not provide single vision.

The patient had been examined 4 years earlier, prior to any cataract and retinal surgery. His refractive error and best visual acuities at that time were right eye -1.00 - 0.75 x 107 (20/40) and left eye -2.25 - 0.75 x 105 (20/50). He reported vertical diplopia and the two images being unequal in size. Orthophoria and 7 PD exophoria were present at distance and near, respectively. A 4 degree right excyclotorsion was measured with the double Maddox rod. Findings with the Worth 4- dot test and stereotesting were identical to the later visit. Interestingly, the aniseikonia measured 11% in the vertical meridian and 9% in the horizontal meridian, the left eye rather than the right eye requiring the magnification.

Comment

Both patients had diplopia and aniseikonia secondary to retinal disease or surgery. Case 12 surprisingly did well with vertical prism despite the large aniseikonia whereas Case 13 with less aniseikonia could not achieve fusion with either size lenses or prisms and was diagnosed with intractable diplopia.

CONCLUSION

Although intractable diplopia can develop in visually mature patients having either childhood or adult onset strabismus, it occurs rarely and therefore should be considered a diagnosis of exclusion. Being diagnosed with intractable diplopia can cause significant stress and has substantial quality of life implications. In this case series, one patient (Case 11) had been placed on medical disability for many years, another patient (Case 5) used an occlusive contact lens while serving as a police officer, and another patient (Case 6) could not perform suturing procedures as a physician's assistant. All three patients had long-standing diplopia that had been resistant to earlier treatment and demonstrated fusion capability on examination.

Gruzensky and Palmer's paper in 1988 categorized complicated cases of diplopia into two types, absolute and resistant.[3] Absolute diplopia is synonymous with intractable diplopia as presently defined and implies constant diplopia in all positions of gaze and fixation distances that cannot be eliminated with prisms or other types of treatment. Occlusion or fogging of the nondominant eye is usually required. Resistant diplopia includes patients who sometimes may seem to have intractable diplopia but are found on examination to be able to obtain single vision with treatment that either establishes fusion or reactivates a preexisting suppression and/or anomalous retinal correspondence. The other types of diplopia reviewed in the present manuscript fit the resistant diplopia category described by Gruzensky and Palmer .

Summarizing, the diverse causes of diplopia in this case series emphasize the fact that when examining an adult with persistent diplopia that is possibly intractable, these mechanisms for

diplopia should be considered first. Determining the exact cause of diplopia requires a careful case history, thorough examination, and appropriate use of specialized testing. Many of these patients can be spared of either occluding or fogging their nondominant eye and should have access to other forms of treatment.

REFERENCES

1. Mashige KP, Munsamy AJ. Diplopia. S Afr Fam Pract 2015;18:1-6.
2. Khan AO. Intractable diplopia following monocular patching. Middle East Afr J Ophthalmol 2007;14;79-80.
3. Gruzensky WD, Palmer EA. Intractable diplopia: a clinical perspective. Graefe's Arch Clin Exp Ophthalmol 1988;226:187-192.
4. Rutstein RP, Bessant B. Horror fusionis: a report of five patients. J Am Optom Assoc 1995;67:733-739.
5. Pratt-Johnson JA, Tillson G. The loss of fusion in adults with intractable diplopia (central fusion disruption). Aust N Z J Ophthalmol 1988;16:81-85
6. London R, Scott SH. Sensory fusion disruption syndrome. J Am Optom Assoc 1987;58:544-546.
7. Fraine L. Nonsurgical mangement of diplopia. Am Orthopt J 2012;62:13-18.
8. Rutstein RP. Use of Bangerter filters with adults having intractable diplopia. Optometry 2010;81:387-393.
9. Silverberg M, Schuler E, Veronneau-Troutman S, et al. Nonsurgical management of binocular diplopia induced by macular pathology. Arch Ophthalmol 1999;117:900-903.
10. London R. Monovision correction for diplopia. J Am Optom Assoc 1987;58:568-570.
11. Bujak MC, Leung AK, Kisilevsky M, et al. Monovsion correction for small-angle diplopia. Am J Ophthalmol 2012;154:586-592.
12. Chia SN, Hegarty B, Weir CR. Intractable diplopia - do patients like occlusive contact lenses? Invest Ophthalmol Vis Sci 2014;55:2567 (meeting abstract).
13. Stone NM, Somner JE, Jay JL. Intractable diplopia: a new indication for cornea tattooing. Br J Ophthalmol 2008;92:1445, 1561-1562.
14. Dawson E, Maino A, Lee J. A unique use for a corneal tattoo. Strabismus 2009;17:98-100.
15. Kwok T, Watts P. Opaque intraocular lens for intractable diplopia - UK survey. Strabismus 2009;17:167-170.
16. Hadid OH, Wride NK, Griffiths PG, et al. Opaque intraocular lens for intractable diplopia: experience and patients' expectations and satisfaction. Br J Ophthalmol 2008;92:912-915.
17. Newsham D, O'Connor AR. Intractable diplopia resulting from the treatment of amblyopia and use of the Sbisa bar in the UK to assess the risk. Invest Ophthalmol 2010;51:4347 (meeting abstract).
18. Scheiman MM, Hertle RW, Beck RW, et al. Randomized trial of treatment of amblyopia in children aged 7 to 17 years. Arch Ophthalmol 2005;123:437-447.
19. Newsham D, O'Connor AR. Assessment of the density of suppression to identify risk of intractable diplopia in the United Kingdom. Strabismus 2016;24:45-50.
20. Mills MD, Coats DK, Donahue S, et al. Strabismus surgery for adults: A report by the American Academy of Ophthalmology. Ophthalmology 2004;111:1255-1262.
21. Holgado S. Diplopia after strabismus surgery. Am Orthopt J 2012;62:5-8.
22. Kushner BJ. Intractable diplopia after strabismus surgery in adults. Arch Ophthalmol 2002;120:1498-1504.
23. Scott WE, Kutschke PJ, Lee WR. Diplopia in adult strabismus. Am Orthopt J 1994;44:66-69.
24. Gill MK, Drummond GT. Indications and outcomes of strabismus repair in visually mature patients. Can J Ophthalmol 1997;32:436-440
25. Rutstein RP, Daum KM. Anomalies of binocular vision: diagnosis and management. St.Louis: Mosby, 1998. p 111-146, p 285-287, p 227.
26. Kushner BJ. The benefits, risks, and efficacy of strabismus surgery in adults. Optom Vis Sci 2014;91:e102-109.
27. Kirschen D, Flom MC. Monocular central - field occlusion for intractable diplopia. Am J Optom Physiol Opt 1977;54:326-331.
28. Pratt-Johnson JA. Fusion ability lost and regained in visual adults. Graefe's Arch Clin Exp Ophthalmol 1988;226:111-112.
29. Pratt-Johnson JA, Tillson G. Intractable diplopia after vision restoration unilateral cataract. Am J Ophthalmol 1989;107:23-28.
30. Ahn YJ, Park SH, Shin SY. Risk factors of persistent diplopia following secondary intraocular lens implanatation in patients with sensory strabismus from uncorrected monocular aphakia. Graefes Arch Clin Exp Ophthalmol 2016;254:129-133.
31. Sharkey JA, Sellar PW. Acquired central fusion disruption following cataract extraction. J Pediatr Ophthalmol Strabismus 1994;31:391-393.
32. Sloper JJ, Kousoulides L, Garnham C. Delayed visual evoked potentials in patients with intractable diplopia following removal of a longstanding catract or with prolonged uncorrected aphakia. Invest Ophthalmol Vis Sci 1996;37:S440.
33. Clark JT. Obstacles to fusion: strabismus and diplopia. Am Orthopt J 2008;58:14-17.
34. Rutstein RP. Diagnosis and management of the patient with diplopia. Contemporary Optometry 2007;5(9):1-6.

35. Amos JF. Diagnosis and management of monocular diplopia. J Am Optom Assoc 1982;53:101-115.

36. Comer RM, Dawson E, Plant G, et al. Causes and outcomes for patients presenting with diplopia to an eye casualty department. Eye 2007;21:413-418.

37. Coffeen P, Guyton DL. Monocular diplopia accompanying ordinary refractive errors. Am J Ophthalmol 1988;105:451-459.

38. Archer SM. Monocular diplopia due to spherocylindrical refractive errors (An American Ophthalmological Society Thesis). Trans Am Ophthalmol Soc 2007 ;105:252-271.

39. Kushner BJ. Diplopia associated with refractive surgery. Am Orthopt J 2012;62:34-37.

40. Kushner BJ. Recently acquired diplopia in adults with long-standing strabismus. Arch Ophthalmol 2001;119:1795-1801.

41. Pratt-Johnson JA, Tillson G. Management of strabismus and amblyopia: a practical guide, 2nd ed. New York: Thieme, 2001. p.251 - 256.

42. Pediatric Eye Disease Investigator Group. Rutstein RP, Quinn GE, Lazar EL, et al. A randomized trial comparing Bangerter filters and patching for the treatment of moderate amblyopia in children. Ophthalmology 2010;117:998-1004.

43. Rutstein RP, Foster NC, Cotter SA, et al. Visual acuity through Bangerter filters in nonamblyopic eyes. J AAPOS 2011;15:131-134.

44. Boyd TA, Karas Y, Budd GE, et al. Fixation switch diplopia. Can J Ophthalmol 1974;9:310-315.

45. Kushner BJ. Fixation switch diplopia. Arch Ophthalmol 1995;113:896-899.

46. Kushner BJ, Kowal L. Diplopia after refractive surgery. Occurrence and prevention. Arch Ophthalmol 2003;121:315-`321.

47. Pollard ZF, Greenberg MF, Bordenca M, et al. Strabismus precipitated by monovision. Am J Ophthalmol 2011;152:479-482.

48. Rutstein RP. Fixation switch diplopia: an unusual cause for adolescent and adult- onset diplopia. J Am Optom Assoc 1985;56:862-865.

49. Pineles SL. Fixation switch diplopia. J Neuro-Ophthalmol 2016;36:118-119.

50. Arnoldi K. Case Corner: Complicated fixation switch diplopia. Am Orthopt J 2009;59:111-115.

51. Eskridge JB. Persistent diplopia associated with strabismus surgery. Optom Vis Sci 1993;70:849-853.

52. Simon JW, Paskowski JR. Intractable diplopia after overcorrection of vertical strabismus. Am J Ophthalmol 1994;117:675-676.

53. Pratt-Johnson JA, Tillson G. Suppression in strabismus - an update. Br J Ophthalmol 1984;68:174-178.

54. Khan J, Kumar I, Marsh IB. Botulinum toxin injection for postoperative diplopia testing in adult strabismus. J AAPOS 2008;12:46-48.

55. Kushner BJ. Management of diplopia limited to down gaze. Arch Ophthalmol 1995;113:

56. Miller AM. Torsional diplopia. Am Orthopt J 2015;65:21-25.

57. von Noorden GK, Murray E, Wang SY. Superior oblique paralysis: A review of 270 cases. Arch Ophthalmol 1986;104:1771-1778.

58. Woo SJ, Seo JU, Hwang JM. Clinical characteristics of cyclodeviations. Eye 2005;19:873-878.

59. Guyton DL. Prescribing cylinders: the problem of distortion. Surv Ophthalmol 1977;22:177-188.

60. Benegas NM, Egbert J, Engel K, et al. Diplopia secondary to aniseikonia associated with macular disease. Arch Ophthalmol 1999;117:896-899.

61. Flodin S, Karlsson P, Gronlund MA. Cyclotorsion measured in a patient population using three different methods: A comparision study. Strabismus 2016;24:28-26

62. Georgievski Z, Sleep M, Koklanis K. Simulated torsional disparity disrupts horizontal fusion and stereopsis. J AAPOS 2007;11:120-124.

63. Arlaster GE, Burke JP. Surgery to treat residual acquired excyclotropia without change in the primary position vertical deviation. A case report. Strabismus 2016;24:93-96.

64. Rutstein RP, Daum KM. Exotropia associated with defective accommodation. J Am Optom Assoc 1987;58;548-554.

65. Somer D, Demirci S, Cinar FG, et al. Accommodative ability in exotorpia: predictive values of surgical success. J AAPOS 2007;11:460-464.

66. Stark L, Ciuffreda KJ, Grisham DJ, et al. Acommodative disfacility presenting as intermittent exotropia. Ophthalmic Physiol Opt 1984;4:233-244.

67. Rutstein RP, Daum KM, Amos JF. Accommodative spasm: a study of 17 cases. J Am Optom Assoc 1988;59:527-538.

68. Shanker V, Ganesh S, Sethi S. Accommodative spasm with bilateral vision loss due to untreated intermittent exotropia in an adult. Napal J Ophthalmol 2012;4:319-322.

69. Ha SG, Jang SM, Cho YA, et al. Clinical exhibition of increased accommodation loads for binocular fusion with basic intermittent exotropia. BMC Ophthalmol 2016;16:77-83.

70. Horwood AM, Riddell PM. Decreased accommodation during decompensation of distance exotropia. Br J Ophthalmol 2012;96:508-513.

71. Horwood AM, Riddell PM. Evidence that convergence rather than accommodation controls intermittent distance exotropia. Acta Ophthalmol; 2012;90:e109-e117.

72. Horwood AM, Riddell PM. Disparity-driven vs blur-driven models of accommodative and convergence in binocular vision and intermittent strabismus. J AAPOS 2014;18:576-583.
73. Lyons C. Where the wild things are: When esotropia misbehaves. Am Orthopt J 2012;62:61-69.
74. Rutstein RP. Spasm of the near reflex mimicking deteriorating accommodative esotropia. Optom Vis Sci 2000;77:344-346.
75. Hussaindeen JR, Mani R, Agarkar S, et al. Acute adult-onset comitant esotropia associated with accommodative spasm. Optom Vis Sci 2014;91:S46-S51.
76. Shanker V, Nigam V. Unusual presentation of spasm of near reflex mimicking large-angle acute acquired comitant esotropia. Neuro-Ophthalmology 2015;39:187-190.
77. Goldstein JH, Schneekloth BB. Spasm of the near reflex: a spectrum of anomalies. Surv Ophthalmol 1996;40:269-278.
78. Fresina M, Giannaccare G, Versura P, et al. Accommodative spasm might influence surgical planning and outcome in acute acquired distance esotropia in myopia. Med Hypotheses 2016;94:66-67.
79. Griffin JF, Wray SH, Anderson DP. Misdiagnosis of spasm of the near reflex. Neurology 1976;26:1018-1020.
80. Dagi LR, Chrousos GA, Cogan DC. Spasm of the near reflex associated with organic disease. Am J Ophthalmol 1987;103:582-585.
81. Laria C, Merino-Suarez ML, Pinero DP, et al. Spasm of the near reflex. Treatment with botulinum toxin. Semin Ophthalmol 2015;30:393-396.
82. Rutstein RP, Than TP, Cogen MS. Botulinum toxin injection for a patient with chronic spasm of the near reflex. Poster presented at the 2002 meeting of the American Academy of Optometry in San Diego, CA.
83. Rutstein RP. Presumed intractable diplopia with chronic esotropia and convergence spasm. Poster presented at the 2012 meeting of the American Academy of Optometry in Pheonix, AZ.
84. Shippman S, Cohen KR, Heiser L. Macular diplopia. Am Orthopt J 2015;65:26-30.
85. Hodgetts DJ. Nonsurgical management of diplopia after retinal surgery. Am Orthopt J 2012;62:38-4.
86. Iacobucci IL, Furr BA, Archer SM. Management of binocular diplopia due to maculopathy with combined Bangerter filter and Fresnel prism. Am Orthopt J 2009;59:93-97.
87. Ugarte M, Williamson TH. Aniseikonia associated with epiretinal membranes. Br J Ophthalmol 2005;89:1576-1580.
88. de Wit GC. Retinally-induced aniseikonia. Binocul Vis Strabismus Q 2007;22:96-101.
89. Rutstein RP. Retinally induced aniseikonia: A case series. Optom Vis Sci 2012;89:e50-e55.
90. Rutstein RP. Update on aniseikonia. Contemp Optom 2010;8:9:1-8.
91. de Wit GC, Muraki CS. Field-dependent aniseikonia associated with an epiretinal membrane: a case study. Ophthalmology 2006;113:58-62.
92. Lee HN, Lin KH, Tsai HY, et al. Anisekionia following pneumatic retinopexy for rhegmatogenous retinal detachment. Am J Ophthalmol 2014;158:1056-1061.
93. Han J, Han SH, Kim JH, et al. Restoration of retinally induced aniseikonia in patients with epiretinal membrane after early vitrectomy. Retina 2016;36:311-320.
94. Currie D. Partial correction of irregular aniseikonia secondary to retinal traction. Optom Vis Sci 2012;89:1081-1086.

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Dr. Rutstein is retired and holds rank of Professor Emeritus.

ARTICLE

Neuro-Optometric Rehabilitation of Visual and Visual-Vestibular Symptoms Following Acquired Brain Injury

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ABSTRACT

Primary Objectives: To review the neurology of the vestibular and balance processing systems, the visual-vestibular symptoms experienced as a result of deficits in integrating visual and vestibular sensorimotor function, and to describe a neuro-optometric rehabilitation protocol for the management of those with visual and visual-vestibular symptoms.

Background: Patients with acquired brain injury (ABI) often experience sensorimotor visual deficits and symptoms of disequilibrium affecting their activities of daily living and quality of life. Dizziness, vertigo and gait disturbance, and their effect on balance, are among the most common complaints in this population. The visual system is a major component of balance and although often overlooked, it contributes heavily when vestibular dysfunction is present. When patients are unable to compensate for deficits in vestibular function with vestibular rehabilitation alone, one of the more common reasons is impairment in one or both of the other sensory inputs necessary for balance (visual and proprioceptive).

Conclusion: The visual system plays a crucial role in the overall sense of balance. Even minor oculomotor deficits and binocular vision dysfunction can have a negative effect on the vestibulo-ocular reflex (VOR) by creating mismatches between the visual and vestibular systems. This mismatch may exacerbate the sensation of disequilibrium, especially when patients are in multi-visually stimulating environments with motion, such as shopping in a grocery store, or without motion, such as watching TV or working on a computer monitor. As awareness of visual and visual-vestibular dysfunctions following ABI increases, the demand for optometrists who offer neuro-optometric rehabilitation will increase as well. Understanding the visual sequalae following ABI and coupling it with the current research on neuro-plasticity, optometrists who specialize in neuro-optometric rehabilitation can better aid in the rehabilitation of these individuals.

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INTRODUCTION

Acquired Brain Injury

Acquired brain injury (ABI) may have a negative impact on an individual's quality of life as it affects physical, cognitive and psychosocial functions. ABI is defined as a sudden-onset, non-congenital and non-degenerative conditions that alter neurological function. ABI

may be a result of external insults leading to traumatic brain injury (TBI) such as motor vehicle accidents, gunshot wounds, sports-related or combat-related injuries. ABI may also occur from internal damage due to cerebral vascular accident (CVA) (stroke), tumors, aneurysms, vestibular dysfunction, and anoxia or hypoxia due to post-surgical complications.[1] Injury to the brain can be focal or global in nature. A focal injury is more localized to the location of impact and may be evident in stroke, which is an infarct in the brain and may be hemorrhagic or ischemic in nature, aneurysm, or brain tumor. On the other hand, a global insult suggests more diffuse neurological compromise and is often a sequalae of motor vehicle and sports-related accidents which often lead to decelerated/accelerated coup-contrecoup injury resulting in diffuse axonal injury (DAI).[1]

Epidemiology

The reported incidence of civilians with TBI in the United States is approximately 1.7 million each year of which 1.3 million cases are classified as mild TBI, while approximately 52,000 cases result in death.[2] About 5.3 million Americans live with long-term disability as a result of TBI.[3] TBI also contributes to 30.5 percent of all injury-related deaths in the United States.[3] In young-adult recreational sports, it has been found that 170,000 TBIs occur annually.[4] Additionally, approximately 750,000 people in the United States suffer from stroke annually.[5] Even with preventative efforts, CVA is still one of the leading causes of death in the America and is the leading cause of disability amongst adults.[5]

Furthermore, soldiers returning from Iraq and Afghanistan are also prone to TBI from the battlefield. It is estimated between January 2000 and December 2014 that more than 313,816 service members were identified as having suffered a TBI.[6] Many military service members and veterans with moderate or severe TBI also have physical injuries and are seen in polytrauma treatment facilities in the Department of Veterans Affairs (VA) system of care. Patients with polytrauma usually have access to advanced eye care as part of the treatment plans. However, there may be over 180,000 Active Duty service members and veterans with mild TBI from recent conflicts who do not have polytrauma. Therefore, they may not have access to eye care services, including vision rehabilitation services, and thus, their visual complaints may be overlooked.[7]

Common Symptoms Associated with ABI

Because of the recent advances in technology, the survival rate of patients with ABI has increased. However, this also means that more individuals may be suffering from sensorimotor visual deficits as a result of the ABI affecting their activities of daily living (ADLs) and quality of life (QOL).[8] These visual deficits vary from mild to severe, depending on the etiology, location and severity of the ABI. The disturbance can affect the patient's ability to process visually related tasks which require higher level cortical processing, such as visual closure and figure ground, or visually guided information such as encoding visual spatial relations with the motor output system in directing themselves in a crowded environment or grasping an object.[9] The most common problems associated with ABI have been categorized as Post Trauma Vision Syndrome[10] which includes: binocular dysfunctions (specifically, convergence insufficiency), acquired strabismus, diplopia, blurred vision, ocular motility deficits, visual field loss, photosensitivity, vestibular dysfunction, and visual perceptual-motor dysfunctions.[11,12,13]

The prevalence of binocular vision problems as a result of ABI is well documented in both the civilian and military populations. Below are just a few of the key literature studies demonstrating the reality of these deficits and the effectiveness of optometric vision therapy in the rehabilitation of these individuals.

According to a study by Ciuffreda et al in 2007, the most common ocular motor dysfunctions in a visually symptomatic TBI

sample included convergence insufficiency (56.3%), deficits of saccades (51.3%), accommodation (41%), strabismus (25.6%), and cranial nerve palsy (6.9%).[14] In terms of the effectiveness of rehabilitation, one study with nine TBI and five stroke patients suggested that both subjective symptoms and objective eye movement recordings significantly improved post vision therapy.[15] A larger scale study by Ciuffreda et al in 2008 also demonstrated that 90 percent of patients had improvements in signs and reduction in symptoms after the completion of in office vision therapy targeting oculomotor dysfunctions.[16]

Additionally, according to a study by Cockerham et al in 2009, in a non-selected TBI sample of veterans, approximately 20 percent presented with signs and symptoms of oculomotor and accommodative dysfunction, which is higher than the percentage of binocular vision disorders in the general population.[17] The percent may even be higher because not all VA facilities have polytrauma department and, therefore, may not have formal eye care services to diagnose these problems. The primary care provider may refer some of these patients to optometry or ophthalmology if there is a visual complaint. However, those who do not complain may be overlooked.[18] Another study by Goodrich et al in 2007 demonstrated that in a sample of veterans with TBI, 74 percent self-reported visual complaints of which 38 percent were diagnosed with a visual impairment.[19]

As discussed, vision and visual processing deficits are highly prevalent consequences of ABI, specifically TBI. Additionally, vestibular and balance issues are also often experienced. It has been reported that up to 98 percent of patients with TBI initially present with symptoms of dizziness, while 70 percent of individuals initially present with hearing loss and/or tinnitus.[20] Due to the close anatomical and functional relationships between the visual and vestibular systems, dysfunction in either can affect the other.

Why Is This Important for Optometrists?

Dizziness, vertigo and gait disturbance, and their effect on balance, are among the most common complaints by patients seeking medical attention.[13] This is often the case in patients with ABI and TBI. The visual system is a major component of the sense of balance. Although often overlooked, the visual system contributes heavily when vestibular dysfunction is present. When patients are unable to compensate for deficits in vestibular function with vestibular rehabilitation alone, one of the more common reasons is impairment in one or both of the other sensory inputs necessary for balance (visual and proprioceptive).[21] In the presence of vestibular dysfunction and associated symptoms of disequilibrium, the visual system may compensate as the primary sensory input for posture and balance, even if the visual system itself is compromised.[22] Errors in the visual input may cause these patients to lose balance or even fall.[23] Thus, it is important that visual function be assessed and addressed as part of the rehabilitation process, creating an important role for the primary care optometrist.

The purpose of this paper is to review the neurology of disequilibrium and to present a neuro-optometric rehabilitation model for the management of visual and visual–vestibular symptoms secondary to ABI.

Key Definitions

Symptoms of dizziness can result from any disturbance to the balance control systems. This can include the peripheral vestibular system, the cardiovascular system, the central nervous system, or the visual pathways. Damage along any pathway in the vestibular system can also lead to the sensation of dizziness, disequilibrium and/or vertigo. Patients, and even some clinicians, often use these terms interchangeably. However, because of the differences in the underlying etiology, it is important to distinguish between them.

Dizziness can refer to any subjective feeling of nausea, light-headedness or disorientation

and is a commonly experienced sensation following TBI. A feeling of dizziness often, but does not necessarily, involve vestibular dysfunction.[24]

Vertigo, on the other hand, is the illusion of movement or spinning of surroundings when there is none. Unlike dizziness, by definition, true vertigo implies vestibular involvement.[26]

Lastly, disequilibrium is the sensation of walking on soft/uneven ground or the loss of balance without any illusion of movement.[25] It is often accompanied by spatial disorientation and dizziness, although it can also occur independently. Patients with this sensation often describe that their brain is overloaded as if in a vice, or that there is tightness around the head. Disequilibrium is a generalized term where etiology may be multi-factorial. It does not necessarily imply vestibular dysfunction. A proposed theory is that these symptoms are a result of the inability of the brain's processing system to adjust to a mismatch of information between the auditory, visual and proprioceptive processing systems.[13]

Another important definition to be familiar with is diffuse axonal injury (DAI).[26] DAI is characterized by axonal stretching, tearing and bulbous formation (swellings at the ends of axons) which results in deficits in multiple areas of the brain and subsequently, a global insult to the brain.[27] It is often a result of acceleration-deceleration injuries found in motor vehicle and some sports-related injuries. The primary stretching of the axons occurring at the time of impact and secondary biochemical changes are thought to be the cause of symptoms related to altered sensorimotor, cognitive and mood following TBI.[28,29]

NEUROLOGY OF DISEQUILIBRIUM

Brief Overview of the Vestibular System

The human balance system has three afferent systems: vestibular, visual, and somatosensory, while the efferent system is composed of multiple neurological pathways that partially overlap and are redundant.[13] Maintaining a sense of balance during changing situations requires all of these pathways to be in sync in order to coordinate motor responses of the limbs, trunk and eyes to the incoming afferent information.

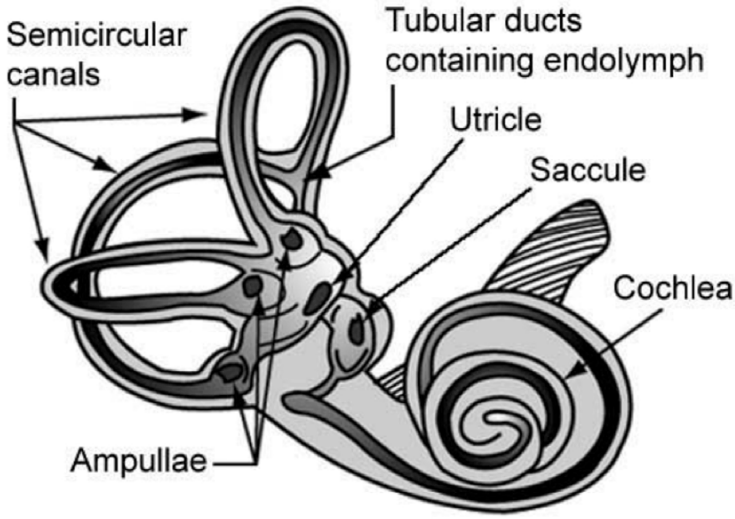

Figure 5: Vestibular apparatus- semi-circular canals otoliths organs. Courtesy of Wikipedia Commons.

The vestibular system includes the vestibular apparatus **(see Figure 1)** located bilaterally within the inner ear: the semi-circular canals and otoliths. The semi-circular canals are ring shaped structures that are sensitive to rotational acceleration in all directions of space. There are three canals in each ear: horizontal, anterior and posterior. This bilaterality is responsible for the yaw, pitch, and roll of head movement as well as sensing when the head turns right or left. They communicate via the vestibular nuclei with motor neurons of the extraocular muscles (EOMs).[26]

The second subdivision of the vestibular apparatus is the otoliths composed of the utricle and saccule, which are sensitive to gravitational and linear acceleration forces in the vertical, lateral and fore-aft directions. The bending of vestibular receptor hair cells with bundles of cilia, located within the maculae of the utricle and saccule and within the crista ampullaris of the semi-circular canals, in response to accelerating motion is responsible for the enhancement or suppression of aberrant neural activity (information derived from the bending of the receptor hair cells) that is ultimately

transmitted to the association cortex regions for decoding.[13]

Vestibulo-Ocular Reflex (VOR) and Its Association with Cerebellum & Sensory Mismatch

Central processing of vestibular information takes place in two main locations: the vestibular nuclear complex and the cerebellum.[30] Vestibular afferent information from receptor hair cells travels to the vestibular nuclei and brainstem (central component) via cranial nerve VIII where it is combined with visual, somatosensory, and cerebellar information for maintenance of balance and equilibrium.[13] From the vestibular nuclei, axons then split and some fibers extend and communicate with the cerebellum. The cerebellum serves as an adaptive role and modifies the VOR as needed and recalibrates the neural input from the vestibular system within the inner ears to the motor output of the EOMs.[32] Additionally, in 1992 Scudder and Fuchs[31] described eye velocity-head velocity neurons as being part of the pathways allowing vision to override or enhance vestibular information. This study sets the groundwork for neuro-optometric rehabilitation in the management of patients with vestibular dysfunction.

Each semi-circular canal has major neural connections to one ipsilateral and one contralateral EOM. The orientation of the three semi-circular canals within each ear parallels the action of each of the EOMs. This relationship is the basis of the VOR, which is responsible for maintaining stable retinal images during head movement. Vestibular signals that are produced as a consequence of VOR are suppressed when appropriate, allowing for appropriate saccade or pursuit eye movements.[13] Therefore, the mismatch of visual information with other sensory motor feedback systems may lead to the perception that an image is jumping and moving with shifting of our eyes.[13] **(See Figure 2)**.

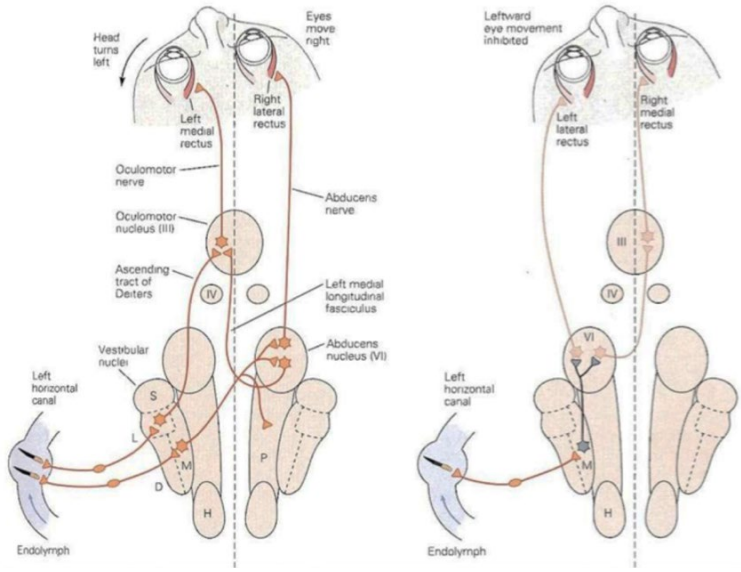

Figure 2: Vestibulo-ocular reflex. S: superior vestibular nucleus, M: medial vestibular nucleus, L: lateral vestibular nucleus, D: inferior vestibular nucleus, P: pons, H: medulla. Courtesy of Wikipedia Commons.

In addition to the central component, vestibular information is also transmitted to various association cortexes for further decoding. In humans, unlike other sensory modalities such as vision and somatosensation, there is no primary vestibular cortex. Once decoded, vestibular data is integrated with visual and somatosensory inputs, allowing for the final step in the process to occur: the vestibular motor output. This is accomplished via different motor neurons that produce both eye and postural movements resulting in maintenance of visual stability and overall body balance and coordination.[32]

Patients with ABI often have difficulty facilitating the filtering of multi-sensory inputs because the interaction between the vestibular and visual pathways is often compromised. Normally, the activation of vestibular pathways results in inhibition of visual information and vice-versa. However, neurologically-compromised patients are often unable to reconcile conflicting information between visual, vestibular, and somatosensory input (a complex system of nerve tactile feedback from touch, pressure, vibration/discriminative movement) resulting in balance instability, symptoms of disequilibrium, and dizziness.[13] In conclusion,

the visual system is a major component of the afferent-efferent model of balance because of its association with the VOR and vestibular nucleus.

Dorsal Stream Processing Deficit

The dorsal stream pathway begins sub-cortically and receives input through the magno-retinogeniculate pathway.[28] The magnocellular pathway begins in the retina and projects to the lateral geniculate nucleus (LGN) and ultimately to the primary visual cortex (V1). The majority of magnocellular information is processed in the parieto-occipital cortex and the extended dorsal stream pathways.[28] The cortical pathway begins at V1, then projects to the middle temporal area (MT), the middle superior-temporal area (MST), the posterior parietal cortex, the prefrontal cortex and to the inferior parietal lobules.[33]

Another important anatomical structure that is related to the dorsal stream is the pulvinar nucleus located at the thalamus. Recent research has considered this structure to be important for visual attention, saccadic eye movement, and visual motion detection via MT area of the dorsal stream, which impacts visual-vestibular performance.[34]

The dorsal stream pathway's main functions include spatial localization and motion perception.[35] Recent studies have shown that the pathway may not be used solely for the perception of space, but for visual calculation of specific actions, i.e. locating a cup of coffee in space and reaching for it.[36] Thus, it is now being thought that this pathway is an action-driven "how" pathway and not purely a spatially perceptive "where" pathway.

Dysfunction in the dorsal stream processing may lead to problems with spatial working memory, executive control of visuo-spatial processing, and difficulty adjusting to visually stimulating environment. Additionally, dorsal stream deficits are often seen in patients with TBI due to the anatomy and location of the posterior parietal lobe, making it more susceptible to injuries. Because of its vast connections throughout the brain, damage to this pathway in ABI often leads to more sensorimotor visual deficits such as: accommodation, versional ocular motility, vergence ocular motility and visual-vestibular dysfunction. Of all these common visual dysfunctions, the most common diagnoses include convergence insufficiency and deficits of saccades.[14]

NEURO-OPTOMETRIC REHABILITATION MODEL

Neuro-Optometric Rehabilitative Therapy (NORT) expands classic optometric management modalities, such as corrective lenses, prisms, tints and coatings, selective occlusion and optometric visual therapy, by incorporating principles of neuroscience involving top-down processing visual-motor and perceptual learning reflective of the underlying visual/neural system plasticity.[37] The goals of NORT are to eliminate or reduce any ocular motor, accommodative, binocular vision problems and to enhance the speed and facility of visual processing and intermodal processing. The detailed research of neuro-plasticity is beyond the scope of this paper. However, an overview of top-down processing and its application and relevance in NORT will be discussed.

How to Avoid Sensation of Disequilibrium

Dizziness and symptoms of disequilibrium are common complaints brought forth to the primary care physician, especially by those with ABI. Patients often describe a sensation of disequilibrium as a generalized imbalance when standing and walking or the feeling that their head is overloaded during near tasks. As previously mentioned, the vestibular system is considered to be the center of balance. There is no localized primary vestibular cortex; instead, the neurological control is integrated into many regions of the cerebral cortex, namely: temporal, parietal and frontal lobes. Damage from DAI can consequently affect one or all of these regions. Additionally, the most

dominant connection between the visual and vestibular system is the VOR, with its main purpose is to maintain a steady image on the retina during head movement. While there are many etiologies to the various symptoms of dizziness and disequilibrium, it is important to rule out the contribution of visual deficits in those with vestibular dysfunction, given the close relationship between the two systems.

Oculomotor deficits and binocular dysfunction can create mismatches of information between visual and vestibular systems and, thus, negatively affect the performance of the VOR creating discomfort and symptoms of disequilibrium. It is critical that the VOR has stable, bifoveal retinal input. Uncompensated binocular deviations such as fixation disparity, heterophorias, convergence insufficiency, and accommodative dysfunctions can exacerbate an existing vestibular disorder. In neurologically non-compromised patients, even with mild to moderate binocular vision problems, they may have been well compensated for most of their lives because the vestibular and somatosensory systems can mask some of the deficits of the visual system. However, an illness or stress can result in breakdown of fusional control and decompensation, creating further mismatch of visual input affecting the VOR and balance.

Optometrists who specialize in neuro-optometric rehabilitation can effectively reduce or eliminate symptoms of disequilibrium by enhancing the facility and accuracy of the binocular and ocular motor systems if the visual deficit is the primary contributing factor to the sensation of imbalance or enhance vestibular therapy by stabilizing visual-vestibular integration. Eliminating the visual triggers often increases progress in vestibular rehabilitation, while at other times treating the vestibular dysfunction often facilitates NORT progress.

TREATMENT AND MANAGEMENT

Cohen developed his model for effective NORT by utilizing the research of Eric Kandel and suggestions of Kleim.[38] He refers to five

Table 1: 5 Components for Effective NORT with Examples

Component	Description	Example
Motivation and Active Participation	-Motivation empowers the patient to be an active participant. -Goals allow therapists and patients to measure success and keep patients engaged. -Therapy procedures are presented using various problem-solving tasks allowing for active participation.	-Set realistic goals at the beginning of therapy (both doctor and patient) -Design therapy procedures that may be incorporated into every day life
Feedback	-Using multi-sensory alerts to recalibrate and refine encoded responses	-Anti-suppression procedures -Physiological diplopia -Polarized vectograms -Buzzers and beep -Verbal feedbacks
Repetition	-Repetition helps to encourage synaptic strength	-Home therapy techniques performed 3 times a week for 20 minutes each duration
Motor Match to Sensory Mismatch	-Patient visually guides motor response to specific visual input enhancing sensorimotor recalibration	-Localization with a pointer such as with vectograms -Prism lens shift -Stereoscopic cards in a stereoscope
Intermodal Integration	-Procedures should incorporate multi-sensory tasks for patients to react to. -Procedures may entail gradual multi-sensory input and output requiring visual, touch, auditory, proprioceptive and balance. -Requires patients to filter extraneous sensory information while attending to stimuli important for solving the visual task.	-Metronome -Balance board -Auditory beep -Yoked prism -Distraction

components that should be incorporated into NORT for maximum rehabilitation success: motivation, feedback, repetition, sensory-motor mismatch, and intermodal integration. **Refer to Table 1 for a summary of the components and examples.** All of these components involve top-down processing.

1. Motivation and active participation: It is crucial to set realistic goals at the start of NORT. These goals allow therapists to measure success post therapy and allow the patient to stay engaged and focused on the tasks to be completed. Therapy procedures are presented in various problem-solving tasks allowing for active participation. The level of difficulty is also gradually increased.
2. Feedback: Feedback is achieved via anti-suppression procedures, physiological diplopia, stereopsis, buzzers and other alerting systems. Verbal feedback is also important in enabling the patient to know when they have done well or improve in areas where they are not.
3. Repetition: It is important to use different therapy procedures in order to keep patients engaged. Additionally, home visual therapy should be performed approximately three times a week for about 20 minutes each time. It is more beneficial to break up home therapy in smaller duration of time than to complete all tasks in one sitting.
4. Motor Match to a Sensory Mismatch: Functioning in a multi-visually stimulating environment requires integration of various streams in the cortex. However, in a traumatized brain, the speed of processing is often reduced and visually-guided motor skills are often compromised.[39] Therapy procedures that incorporate various lens types, such as prism, for example, can manipulate how visual information is perceived (sensory mismatch). If the therapy procedure requires a motor response (motor match) to this mismatch, at the same time providing a neural feedback (a beep or buzz) as to whether the response is correct or incorrect will further facilitate the recalibration and enhancement of this sensorimotor response. An example of this concept can be illustrated with a common therapy procedure utilizing polaroid filters and vectograms, in which localization of image is the motor match and the "Small In, Larger Out" (SILO) phenomenon is the sensory mismatch. Other procedures utilizing this concept can include the use of yoked prism lenses, polarized lenses and red/green glasses.
5. Intermodal integration: NORT procedures should incorporate multi-sensory tasks. Procedures may entail gradual multi-sensory input and output requiring visual, touch, auditory, proprioceptive and balance. ABI patients often have filtering difficulty and are easily overwhelmed with information. Thus, this top-down processing component requires patients to filter extraneous sensory information while attending to stimuli important for solving the visual task.

Lenses, Prisms and Tints

The obvious first step of treatment is to maximize the clarity of vision. The goal is to eliminate even minor visual distortions, which may add to over stimulating an already fragile neurological processing system. This is usually achieved with separate distance and near single vision lenses. Bifocals, progressive addition lenses (PALs), and multifocal contact lenses are contraindicated for those with ABI because of gait and/or vestibular issues. PALs have peripheral distortion when patients look away from the center and thus, may exacerbate the patients' symptoms. Bifocals may be prescribed for stationary activities such as reading and watching television. However, thorough patient education is required and the proper way to utilize the lenses must be taught.

Presently there is not a significant body of research regarding how tinted lenses reduce symptoms of disequilibrium and photosensitivity. It is our clinical experience that a fifteen percent blue Omega tint often reduces symptoms of disequilibrium and increased sensitivity to fluorescent lighting. For patients with general photosensitivity, tints may also be incorporated into the spectacles. Indoor lenses will often be prescribed with 30-40 percent tint, while 75-80 percent will be indicated for outdoor use.[28] Fusional prism may also be prescribed in conjunction with NORT in those with diplopia. Most practitioners will first utilize Fresnel prism for a trial period. If patients respond favorably to the Fresnel prism, then ground-in prism can then be incorporated into the patient's habitual prescription.

Phase 1 of NORT: Enhance the Stability of the Visual Input System

In phase 1 the goal is to enhance basic visual input to the highest level of accuracy as possible to provide fewer visual information processing conflicts. This phase lays the foundation for all future therapy. Visual therapy procedures need to adequately normalize ocular motor control, accommodation, quality of fixation, binocular stability, and VOR stability.

Phase 2 of NORT: Enhance Binocular Control Alignment and Sustenance

There are two main goals in this phase. The first is to develop adequate binocular control and second is to enhance speed of fusion recovery. The former is achieved as a result of neuro-muscular and visuo-motor control of sustaining ocular alignment with clear and comfortable single vision. Various procedures are used to increase the facility of the accommodative-convergence system. Procedures may include vectograms with emphasis on localization, stereopsis and perception of SILO; Brock string with emphasis on physiological diplopia; computerized equipment with emphasis on integration of vision with other sensory inputs.

Enhancement of fusional recovery utilizes both static and dynamic therapy procedures such as headshake and walking with Brock string. Procedures often incorporate prisms and lenses to provide feedback and to quickly re-establish single vision with changes in gaze. An excellent procedure is performed with the Brock string in which the patient closes his or her eyes and visualizes where the bead is located. As soon as they open their eyes, they must refuse the bead back to one as quickly as possible.

Phase 3 of NORT: Develop Speed of Visual Information Processing and Stability of Output

In Phase 3, both vestibular and multi-sensory stimulations are incorporated in the majority of the therapy procedures. This phase presents many types of sensory inputs such as auditory, balance, and visual distractions, in which the brain must selectively attend to specific targets and use the information to re-construct the spatial orientation of objects. The efficiency and accuracy of this task is achieved through top-down processing via the dorsal and ventral streams. The ventral stream is responsible for recognition of objects and their details. After identification of the objects, the dorsal stream is then activated to perform the planned motor act.

Refer to Figure 3 for schematic of how each phase of NORT addresses visual-vestibular symptoms.

CONCLUSION

The visual system plays a crucial role in the overall sense of balance. Even minor oculomotor deficits and binocular vision dysfunction can have a negative effect on the VOR by creating mismatches between the visual and vestibular systems. This mismatch may exacerbate the sensation of disequilibrium, especially when patients are in multi-visually stimulating environments with or without motion. As awareness of visual and visual-vestibular

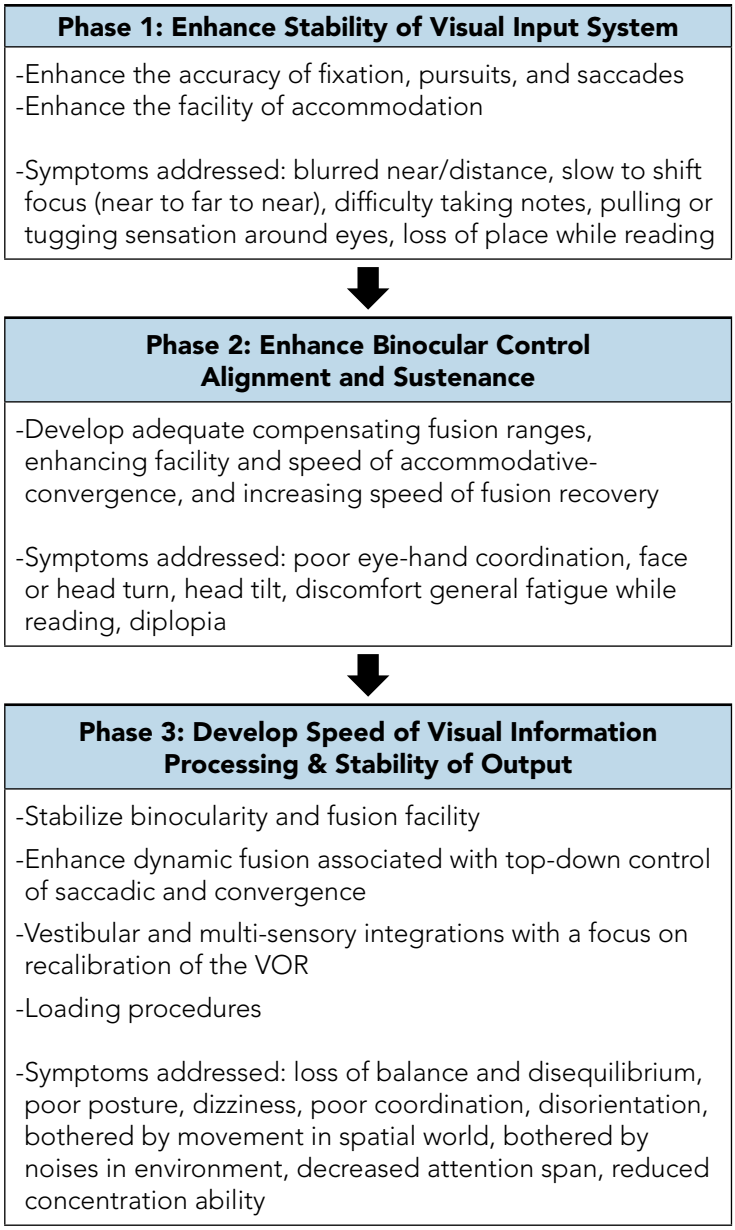

Figure 3: Flow chart highlighting how NORT addresses visual-vestibular symptoms

dysfunctions following ABI and concussion increases, the demand for optometrists who offer neuro-optometric rehabilitation will increase as well. Understanding the visual sequalae following ABI and coupling it with the current research on neuro-plasticity, optometrists who specialize in neuro-optometric rehabilitation can better aid in the rehabilitation of these individuals.

Acknowledgment

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REFERENCES

1. Suchoff IB, Ciuffreda KJ, Kapoor N, eds. Visual and vestibular consequences of acquired brain injury. Santa Ana: Optometric Extension Program Foundation, Inc, 2001.
2. Faul M, Xu L, Wald NM, Coronado VG. Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations, and Deaths. Atlanta, GA. Centers for Disease Control and Prevention, National Center for Injury Prevention and Control, 2010.
3. Biausa.org. Virginia: Brain Injury Association of America. Brain injury statistics. [updated 2015; cited 2017 Jan]. Available from: www.biausa.org.
4. Gilchrist J. Nonfatal traumatic brain injuries related to sports and recreation activities among persons aged less than 19 years- United States, 2001-2009. MMWR 2011;60(39):1337-1342.
5. Stroke.org. Colorado: National Stroke Association. Stroke 101. [updated 2017; cited 2017 Jan] Available from: www.stroke.org.
6. Dvbic.org. Maryland: Defense and Veterans Brain Injury Center. DoD worldwide numbers for TBI. [updated 2017 Jan 6; cited 2017 Jan 6]. Available from: https://goo.gl/720fpq.
7. Goodrich GL, Martinsen GL, Flyg HM, Kirby J, Asch SM, Brahm KD, et al. Development of a mild traumatic brain injury-specific vision screening protocol: A Delphi study. J Rehabil Res Dev. 2013;50(6):757–68.
8. Kapoor N, Ciuffreda KJ. Vision deficits following acquired brain injury. In Cristian A (ed) Medical Management of Adults with Neurological Disabilities. New York, NY: Demos Medical Publishing, 2009;407-23.
9. Cohen A. Acquired visual information-processing disorders: closed head trauma. In: Press L. Applied Concepts in Vision Therapy. Santa Ana, CA: Optometric Extension Program, 2008: 154-67.
10. Padula WV, Shapiro JB, Jasin P. Head injury causing post trauma vision syndrome. N Engl J Optom 1988; Dec/Winter:16-22.
11. Cohen AH, Soden R. An optometric approach to the rehabilitation of the stroke patient. J Am Optom Assoc 1984;52:795-800.
12. Fraco RF, Fells P. Ocular motility problems following road traffic accidents. Br Orthoptic J 1989;46:40-8.
13. Cohen AH. Vision rehabilitation for visual-vestibular dysfunction: The role of neuro-optometrist. NeuroRehab 2013;32:483-92.
14. Ciuffreda KJ, Kapoor N, Rutner D, Suchoff IB, Han ME, Craig S. Occurrence of oculomotor dysfunctions in acquired brain injury: a retrospective analysis. Optometry 2007;78(4):155-61.
15. Ciuffreda KJ, Han Y, Kapoor N, Ficarra AP. Oculomotor rehabilitation for reading in acquired brain injury. NeuroRehab 2006;21(1):9-21.
16. Ciuffreda KJ, Rutner D, Kapoor N, Suchoff IB, Craig S, Han ME. Vision therapy for oculomotor dysfunctions in acquired brain injury: a retrospective analysis. Optometry. 2008;79:18-22.
17. Cockerham GC, Goodrich GL, Weichel ED, Orcutt JC, Rizzo JF, Bower Ks, et al. Eye and visual function in traumatic brain injury. J Rehabil Res Dev.2009;46(6):811-818.

18. Goodrich GL, Martinsen GL, Flyg HM, Kirby J, Asch SM, Brahm KD, et al. Development of a mild traumatic brain injury-specific vision screening protocol: A Delphi study. J Rehabil Res Dev. 2013;50(6):757–68.
19. Goodrich GL, Kirby J, Cockerham G, Ingalla SP, Lew HL. Visual function in patients of a polytrauma rehabilitation center: A descriptive study. J Rehabil Res Dev.2007;44(7):929-936.
20. Maskell F, Chiarelli P, Isles R. Dizziness after traumatic brain injury: overview and measurement in the clinical setting. Brain Inj. 2006;20(3):293-305.
21. Luxon LM. Evaluation and management of the dizzy patient. J Neurol Neurosurg Psychiatry 2004;75:45-52.
22. Hellerstain L, Winkler P. Vestibular dysfunction associated with traumatic brain injury: collaborative optometry and physical therapy treatment. In: Suchoff I, Ciuffreda K, Kapoor N. Visual and Vestibular Consequences of Acquired Brain Injury. Santa Ana, CA: Optometric Extension Program, 2001, 220-35.
23. Manchester D, Woolacott M, Zederbauer-Hylton N, Marin O. Visual, vestibular and somatosensory contributions to balance control in the older adult. J Gerontol 1989;44:M1 18-27.
24. Bryan V. The vestibular system: anatomy, function, dysfunction, assessment, and rehabilitation. In: Suter PH, Harvey L. Vision Rehabilitation Multidisciplinary Care of the Patient Following Brain Injury. Boca Raton, FL: CRC Press, 2011:301-45.
25. Anderson DC, Yolton RL, Reinke AR, Kohl P, Lundy-Ekman, L. The dizzy patient: A review of etiology, differential diagnosis, and management. J Am Optom Assoc 1995;66:545-58.
26. Iskander D, Cohen AH, Kapoor N. Optometric management of a patient with parietal lobe injury. J Behav Optom 2010;21;6:143-49.
27. Vik A, Kvistad KA, Skandsen T, Ingebrigtsen T. "Diffuse axonal injury in traumatic brain injury" (original article in Norewegian). Tidsskrift for den Norske Laegeforening 2006;126:2940-4.
28. Wolf JA, Stys PK, Lusardi T, Meaney D. Traumatic axonal injury induces calcium influx modulated by tetrodotoxin-sensitive sodium channels. J Neurosci 2001;21:1923-30.
29. Iwata A, Stys PK, Wolf JA, Chen XH. Traumatic axonal injury induces proteolytic cleavage of the voltage-gated sodium channels modulated by tetrodoxin and protease inhibitors. J Neurosci 2004;24:4605-13.
30. Solan HA, Shelley-Tremblay J, Larson S. Vestibular function, sensory integration, and balance anomalies: A brief literature review. Optom Vis Dev 2007;38:13-7.
31. Scudder CA, Fuchs AF. Physiological and behavioral identification of vestibular nucleus neurons mediating the horizontal vestibulo0ocular reflex in trained rhesus monkeys. J Neurophysiol;68:244-64.
32. Hain T, Helminski J. Anatomy and physiology of the normal vestibular system. In: Herdman S. Vestibular Rehabilitation 3rd edition. Philadelphia, PA: F.A. Davis Company, 2007:2-218.
33. Goodale MA, Milner AD. Separate visual pathways for perception and action. Trends Neurosci 1992;Jan 15:20-5.
34. Arend I, Machado L, Ward R, McGrath M, Ro T, Rafal R. The role of the human pulvinar in visual attention and action: evidence from temporal-order judgment, saccade decision, and antisaccade tasks. Prog in Brain Res 2008. Volume 171, 475-483.
35. Wang J, Zhou T, Qiu M, Du A, Cai K, Wang Z. Relationship between ventral stream for object vision and dorsal stream for spatial vision: an fMRI + ERP study. Hum Brain Mapp 1999;Jan:8(4):170-81.
36. Milner AD, Goodale MA. Two visual systems re-viewed. Neuropsychologia 2006 Jan:44(5):828-33.
37. Kandel ER. In search of memory: The emergence of a new science of mind. New York: W.W. Norton & Company;2006
38. Chang A, Cohen A, Kapoor N. Top-down visual framework for optometric vision therapy for those with traumatic brain injury. J Optom Visual Performance 2013;1(2)82-93.

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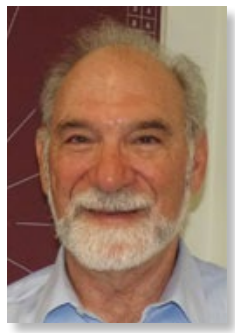

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Dr. Allen Cohen was the Chief of Optometry Service at the Northport VA Medical Center from 1973 to 2007. He was a partner in a multi-doctor private since 1965 and he specialized in visual therapy and neuro-optometric rehabilitation. He retired from private practice in 2007. Dr. Cohen has been a Professor of Clinical Optometry and the supervisor of the Neuro-Optometric Rehabilitation residency at SUNY College of Optometry since 2010. Dr. Cohen has lectured extensively on the diagnosis, treatment and visual consequences of patients with acquired brain injuries.

BOOK REVIEW

Aging: Oxidative Stress and Dietary Antioxidants

Preedy, V (Ed). Aging: Oxidative Stress and Dietary Antioxidants. Academic Press (Elsevier), Waltham, MA. 2014. 301 pages including index. ($94.00-$130 [on Amazon]) Kindle version available for $102.00

Reviewed by:
Dominick M. Maino, OD, MEd, FAAO, FCOVD-A,

This oversized text has 29 chapters, 82 contributing authors, multiple figures and diagrams in each chapter and an index. These chapters include but are not limited to the effects of oxidative stress upon frailty, skin aging, cardiovascular disease, diabetes, and elderly women. Other chapters review the health impact of various diets (vegetarian, etc) and a discussion of spices/herbs, vitamins, tryptophan, melatonin, soy and other supplements. Oxidative stress and depression, Alzheimer's disease, brain injury, heart disease, hypertension and arthritis are reviewed as well.

Since I have lectured on evidenced based clinical practice and often speak about the science behind the use of antioxidants, diet, and supplements; this particular text was a timely addition to the resources I have available in this area. And ever since I have become officially chronologically enhanced (AKA "old guy" who is eligible for Medicare), a particular interest in aging and the topics discussed in this book have become not only personally important to me, but also for many of the patients I serve.

The first chapter (Oxidative Stress and Frailty: A Closer Look at the Origin of a Human Aging Phenotype) immediately impacts any of us who are aging (this means all of us, of course). This chapter defines oxidative stress as a disturbance in the prooxidant-antioxidant balance leading to oxidative damage and the resultant outcomes of aging including frailty. Up to 25% of those 65 years of age and 45% of those 85 years of age and older are considered frail. Frailty (a state of increased vulnerability with a decreased ability to maintain homeostasis) has been repeatedly linked to an increase in detrimental outcomes above and beyond those expected from aging and disease state alone.

The frailty phenotype (which is nicely explained by figure 1.1) includes insulin resistance, inflammation, sarcopenia (age related loss of skeletal muscle), adiposity (increase in and redistribution of fat), age-related hormone decline, and nervous systems dysfunction. The latter results in fatigue, slowed walking speed, a decline in physical activity, weight change/loss and muscle weakness. This breakdown of homeostasis leads to an increase in falls/injuries, acute illness, hospitalization, disability, dependency, institutionalization and finally death. Obviously, oxidation is a topic that should be important to all of us who wish to have an active, happy, disability-free life as we experience our senior years.

About a third of the way into the book, I wished that I had additional degrees in biochemistry, genetics, cardiology and many other sciences from geriatrics to enteral nutrition (i.e. tube feeding). The good news is that our background and training does prepare you fairly well for the information within this book.

Since I am an Italian who does a fair amount of cooking using many different spices, chapter 10 really sparked my interest. This chapter discussed the use of herbs and spices as related to aging. Did you know that clove has an insulin enhancing ability; turmeric an anti-inflammatory, anticancer and anti- angiogenic affects and that cinnamon, black pepper, and cumin all appear to have a positive impact on the unwanted effects of aging? Ginger, oregano, gingko biloba, garlic, red pepper, seaweed, and pennywort may all

be useful as an anti-aging agent as well. The research that supports the possible anti- aging effects of these herbs and spices included both laboratory and clinical based studies. (Although not noted in this chapter, basil may also have anti-aging effects.)

Other chapters within this text discuss the anti-oxidative effects of fruits and vegetables (cherries, bananas, strawberries, apples and pears as well as rice white asparagus, lentils). Various vitamins and supplements may decrease bone loss (vitamin C), increase muscle performance (magnesium), decrease depression (gingo biloba, resveratrol [found in red wine]) and melatonin (scavenges free radicals). Vitamins and various supplements have been shown in clinical trials to reduce cardiovascular disease as well.

Aging in humans is usually accompanied by a decrease in brain volume and impairment in cognitive function rather than a loss of neurons within the brain. This process (along with any traumatic brain injury the individual may have experienced) can then promote the onset of Alzheimer's disease (chapter 19). Since 44 million individuals worldwide now live with Alzheimer's disease and up to 135 million individuals will have Alzheimer's disease and dementia by 2050, research in this area and the role of anti-oxidants is critical for all.

This book is made understandable for those of us who do not have an extensive background in biochemistry, genetics and other, perhaps unfamiliar areas, because of the many informative figures, diagrams and photographs. When the text I read resulted in some head scratching on my part, the accompanying figures or diagrams usually brought meaning to the fore.

If you want a better understanding of aging, oxidative stress and overall senior health, I can highly recommend this book to any who are experiencing chronological enhancement and have an interest in this area.

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