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OPTOMETRY & VISUAL PERFORMANCE

A Deliberate Set of Examinations and the Application of Yoked Prisms in the Treatment of Visual Midline Shift Syndrome: A Case Report

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Optometry & Visual Performance (OVP) is an international, peer reviewed journal dedicated to the advancement of the role of optometry in enhancing and rehabilitating visual performance. The mission of *OVP* is to increase the awareness and availability of clinically relevant information in functional, developmental, behavioral, and vision therapy aspects of optometry through an internet based, open access format. *OVP* is a collaborative effort of the Australasian College of Behavioural Optometry and the Optometric Extension Program Foundation.

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Editorial ▸ Optometry: A Vital Member of the Rehabilitation Team

Eric T. Ikeda, OD, Bellflower, California

Four years after graduating from the Southern California College of Optometry, I eagerly began my career in vision therapy by joining the office of Drs. James L. Cox and Michael M. King (also SCCO graduates). Soon after joining the group, I had my first experience in working with a traumatic brain injured patient. This was approximately the same time that the Head Trauma Issue of the Journal of the American Optometric Association was published.[1] Dr. Allen H. Cohen wrote an inspiring editorial titled "Optometry: the invisible member of the rehabilitation team." What timing! I enjoyed reading each clinical article and have eagerly pursued my clinical practice of evaluating and managing patients with visual deficits resulting from traumatic brain injury.

Concussive injuries have become increasingly newsworthy, especially since many young athletes suffer from concussive sports injuries each year. Just recently, I viewed a report on the evening news of a young 17-year-old who actually died from a concussive brain injury. Is it because players are bigger and faster, creating greater force and injury? Regardless, what is being done to protect the athletes, and what changes are being made at all levels in contact sports? It is becoming evident that while the NFL knew about these issues many years ago, the public's awareness and concern began perhaps when former San Diego Chargers linebacker Junior Seau took his own life in 2012. Studies by the National Institute of Health concluded that Seau suffered from chronic traumatic encephalopathy (chronic brain damage) that resulted from repetitive concussive injury to his brain. Fortunately, there has been growing interest in finding effective and efficient screening strategies for those who suffer a head injury. Not surprisingly, vision is recognized as a significant factor when screening for concussive injury, which has resulted in the use of vision assessment tools as part of the screening battery. As a result, more athletes are being referred for vision consultation and treatment.

I learned about optometry's role in rehabilitation in the 1990s, when I attended the OEP-sponsored Optometry and Occupational Therapy Collaboration in Anaheim, California. Eager to learn more, I attended many educational conferences and meetings, including the Neuro Optometric Rehabilitation Association (where I served as President), the College of Optometrists in Vision Development, and the Optometric Extension Program Foundation Clinical Curriculum TBI course. Eventually, I was invited to provide hospital in-services to medical staffs and lectured to optometric colleagues, allied professionals, and optometry students on the topics of traumatic brain injuries and sports concussive injuries. This led to my appointment as the Neuro Optometry Program Director for Casa Colina Hospital and Centers for Healthcare (Pomona) and Rancho Los Amigos National Rehabilitation Center (Downey), as well as referrals to provide consultative vision care for the Providence Little Company of Mary (San Pedro), CareMeridian Rehabilitation Centers, and various local rehabilitation facilities. In just over ten years, I have seen optometry change from an "invisible member of the rehabilitation team" to an integral and visible member of the team. Optometry has made tremendous advances in clinical care, and vision rehabilitation is one area that continues to advance and grow. I am delighted to see many colleges of optometry recognize the role of optometrists in rehabilitative care. This is evidenced by the interest shown by many optometry students. Recently, the OEP student

clubs at the Southern California College of Optometry, Marshall B. Ketchum University, and Western University of Health Sciences College of Optometry attended my presentation on neuro optometric rehabilitation and toured the neuro optometry clinic and hospital and outpatient facilities at Casa Colina.

As I enjoyed reading each article in the special issue of the Journal in 1992, I hope that will you also enjoy reading this latest issue on brain injury rehabilitation. Many of our colleagues will share their cases that will help you expand your knowledge and grow your confidence in providing the necessary evaluation and treatment for those who have suffered a brain injury.

References

1. J Am Optom Assoc 1992;63(8).

Article ▸

A Deliberate Set of Examinations and the Application of Yoked Prisms in the Treatment of Visual Midline Shift Syndrome: A Case Report

Christopher H. Caldwell, OD, Joint Base Lewis-McChord, Washington

Evelyn Reyes-Cabrera, OD, MS, Fort Hood, Texas

ABSTRACT

Background: Visual midline shift syndrome (VMSS) is an uncommon sequela of mild traumatic brain injury (mTBI). Untreated, VMSS may contribute to vague complaints of visual discomfort and balance or postural dysfunction. Identifying and treating this condition may improve visual outcomes and quality of life for an mTBI patient.

Case Report: A 24-year-old female Soldier with a history of at least one concussive event and complaints of drifting when walking, photophobia, asthenopia, and persistent visual blur presented to the optometry clinic via a referral from her nurse case manager. Her initial visit failed to yield any reliable information regarding her symptoms due to fatigue, over-medication, and distraction, so we delayed any treatment until she could return in a better state of mind. Over her next four visits, we conducted a full ocular health examination and an extensive neuro-optometric evaluation, and we diagnosed her with convergence insufficiency and VMSS. We then issued corrective lenses and yoked prism in a stepped fashion. She responded progressively better to each intervention, and one month after her final visit, she was largely visually asymptomatic.

Conclusions: A deliberate approach to care and a thorough neuro-optometric examination is indicated for any patient manifesting visual sequelae of mTBI during their initial comprehensive ophthalmic examination. Proper treatment of subtle dysfunctions evident only upon careful evaluation and consideration of all of a patient's concerns can have a positive impact on the patient's quality of life.

Keywords: convergence insufficiency, egocenter, mild traumatic brain injury, neuro-optometry, post traumatic vision syndrome, visual midline shift syndrome, visuo-spatial, yoked prism

Introduction

Mild to severe traumatic brain injuries are well known to have immediate and persistent effects on vision and the visual system.[1] These effects are highly variable and in some cases may be quite subtle to the observer. Post-injury vision may be correctable to 20/20, and no significant binocular vision problems may be evident, yet the patient may still complain of vague visual discomfort and difficulty reading or

Table 1: Characteristics and Symptoms of Post-Trauma Vision Syndrome[3]

Common Characteristics	Common Symptoms
Exotropia	Diplopia
Exophoria	Blurred near vision
Convergence Insufficiency	Perceived movement of print or stationary objects
Accommodative Insufficiency	Asthenopia
Oculomotor Dysfunction	Headaches
Increased Myopia	Photophobia

Table 2: Common Symptoms of Visual Midline Shift Syndrome

Poor or altered posture
Poor or decreased balance
Altered gait or sense of drifting while walking; "tilted floor" phenomenon
Less commonly, vertigo
Less commonly, spatial disorientation in complex visual environments

concentrating. An acute observer may diagnose patients with slight vergence, accommodative, or oculomotor dysfunctions that either did not exist prior to the trauma or were appropriately compensated and therefore were not evident in previous examinations. This constellation of symptoms and their respective characteristics is frequently referred to as post-traumatic vision syndrome (PTVS; Table 1). Patients may also have complaints of imbalance or reduced coordination. In severe cases, a patient may develop inefficient compensatory postural habits, leading to neck, back, or leg pain after a head injury. Advanced testing may reveal that their sense of visual straight-ahead has changed and no longer aligns with the environment, causing slight physical contortions in an effort to rectify the disparity. This condition, called visual midline shift syndrome (VMSS), is a less common but well-documented phenomenon that may occur in up to 50% of patients after certain types of neurological insults.[2] Dr. William V. Padula of Guilford, CT has described VMSS as a mismatch of visual and spatial information processed by the brain. Common symptoms of this syndrome are listed in Table 2.

Padula has built greatly on the work of Trevarthen and Sperry to explain vision as a bimodal processing system composed of both focal and ambient vision.[2] He explains that the "focal" process is largely concerned with detail discrimination and is referenced primarily to function in the visual cortex. This focal visual system consists of around 80% of the visual fibers, either via the lateral geniculate body to the visual cortex or through the pretectal nucleus in the pupillary constrictive system. At the same time, and more germane to our discussion, the "ambient" process matches visual information coming from the environment with kinesthetic, proprioceptive, and vestibular information generated by the body to build a visuo-spatial environment. This system gets its visual input from the 20% of the visual fibers that travel to the superior colliculus (SC). The SC serves to merge these information feeds and to start a feed-forward system wherein the midbrain communicates with the visual cortex and other areas of the brain to establish a spatial construct and to provide an organizational framework for other cognitive perceptual processes. Accurate saccadic movement is an example of a visual task enabled by this framework. Saccades require non-occipital cognitive effort to anticipate movement, to release attention from the former object of regard, to release the visual image, and to recognize the intended new point of regard. A compromise in this process is not likely completely to prevent fixation but will very likely cause delays or inaccuracies in fixation. Subtle saccadic dysfunction is a potential source of visual inefficiency and general discomfort for an mTBI patient and is, in fact, measurable by a keen observer. Similarly, appreciation of an accurate midline is visual task-dependent on the ambient system and its associated non-occipital functions; deficiencies in appreciation of the midline are in many cases measureable in systems disrupted by brain injury.

Padula defines the concept of visual midline organization as "the pre-conscious ambient visual concept of the lateral and anterior-posterior axes related to the perceived body center for sense of position." This organization arises during childhood development as an infant gains the ability to focus attention on an object, then develops the ability to turn and flex his head and neck to follow an object in space. The infant eventually sits, crawls, stands, and walks only when it directly interacts with the physical world, appreciates its own orientation

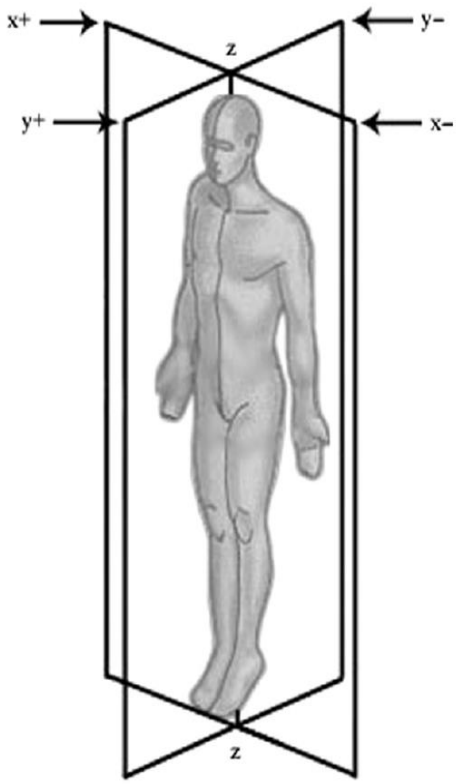

Figure 1. *A graphical representation of ambient visuo-spatial volume affecting egocenter*

against gravity, and allows the ambient system properly to merge its visual, proprioceptive, kinesthetic, and vestibular inputs. The child falls many times until these inputs align properly in a functional system; the result is appropriate posture and gait. A child with a successfully established ambient system tends to maintain an intact system (and therefore, proper posture and gait) throughout his life. Disruption or delay of visual or spatial signals into the system after an injury will force the system to adjust that midline organization, resulting in a compression or expansion of internal space to accommodate the new information.[3] This alteration in internal space is then transmitted via the feed-forward mechanism previously described to the occipital cortex to be projected into the subject's external space.[4] The subject will no longer be able accurately to assess his midline given this faulty projection; the disparity between the subject's physical axes and his ambient sense of midline (the "midline shift") is frequently detectable by careful measurement.

Clinically, a subject's perceptions of midline on his x- and z-axes (Figure 1)[2] are evaluated during a comprehensive neuro-optometric exam, where the examiner simply moves a pen or fixation wand in front of the patient and requests him to respond when the object is centered on his midline.[5] Alternately, a patient can face a large blank wall or whiteboard, and the examiner can present a stimulus either by moving a small high-contrast target or by using a laser pointer. Houston proposes a more precise method, identifying deficiencies in the "Eye-Head" and "Hand-Head" egocentric midlines while minimizing potentially confounding visual or proprioceptive cues. Regardless of technique, the examiner should present the stimulus multiple times to the patient to gain reliable information on right, left, anterior, or posterior shifts in midline.

Once identified, a common therapy to address the shift is the application of yoked prism effectively to recalibrate the focal and ambient systems by leveraging the optical properties of prisms. The term "yoked" refers to the practice of placing the prisms with the bases in the same direction (base left or base right). To understand the benefits of this technique for VMSS, a review of the optical properties of prisms is appropriate. Every prism shifts light towards its base, but a prism does not bend all light uniformly across its surface.[6] Relative to each other, light rays at the base are bent slightly less, and rays at the apex are bent slightly more. There occurs an overall shift in the image towards the apex, but the resulting image is not a perfect copy of the original image. The visual field closest to the base has been effectively compressed, and the visual field closest to the apex has been effectively expanded.

The focal system does not appreciate the shift or the expansive/compressive effects; the prisms shift the image of the world in one direction but continue to provide unadulterated details for the focal visual

process to continue to function. In fact, a patient can actually see a benefit from the lenses if the provider prescribes the appropriate ametropic correction. Concurrently, the slight alteration of space corrects the inappropriate expansion and compression occurring in the pre-conscious ambient system. The result is a newly restored visual processing system more closely aligned with the patient's proprioceptive, kinesthetic, and vestibular systems, thereby improving balance, gait, and, ultimately, posture.

In practice, the provider must not only carefully measure the patient's refractive error, but also properly address the direction and magnitude of the patient's midline shift. In cases of a lateral midline shift, the prism should be designed with the bases in the direction opposite to the patient's altered midline. For example, in a patient with a left midline shift, his ambient center is displaced to the left, and he will tend to stop the examiner's wand when it is to the left of his nose. Therefore, base right prisms will provide this patient the best benefit, thereby bringing the visual "center" into alignment with the patient's projected ambient "center." Determining the dioptric power of the prism required for a given patient is largely subjective; multiple combinations should be tried before settling on a final prescription. Generally, as the patient's midline shift is usually only a few degrees, the dioptric power will be relatively low.

A patient with an anterior shift presented with a vertically moving target will stop the examiner while the stimulus is still lower than eye level. This patient suffers from a compression of his ambient space at near and a corresponding expansion of ambient space in the distance. In a pre-conscious attempt to align the visual and proprioceptive systems, he may tend to employ flexion, or a constant bending forward. To compensate for this, a patient will commonly have "a tendency to extend [his] spine and thrust [his] weight backwards, leading to excessive lumbar lordosis, kyphosis of the thoracic spine, and hyperactivity of the lumbar paravertebrals."[7] This disparity is addressed with base down prisms. Base down prisms will optically expand the wearer's immediate visual space and compress the ambient space in the distance. Conversely, a posterior shift gives the patient inappropriate expansion of his immediate space and compression at distance, which causes a primary extension and a compensatory flexion. Constant flexion may "contribute to postural imbalances such as kyphosis, scoliosis, and sway back."[7] Base up prisms provide the appropriate counteraction to this deficiency in the ambient system. In both cases, the primary and compensatory spinal flexions and extensions are reduced, and the patient can ambulate through his environment more efficiently and, presumably, more comfortably. In at least one case study, an athlete with persistent sacroiliac dysfunction responded very well to this therapy even when traditional physical therapy methods failed.[8]

Case

A 24-year-old Hispanic female Soldier was referred by her nurse case manager to the optometry clinic at Fort Sam Houston, TX for a full optometric examination. At the time, the Soldier was undergoing a medical separation from the Army and was assigned to the Warrior Transition Battalion (WTB), a unit with the special mission to "provide competent and compassionate leadership through command and control, complex case management, comprehensive transition planning, and primary care in a safe environment that promotes the optimum healing for the wounded, ill, or injured Soldier that will return to the force or transition to civilian life as a productive Veteran."[9]

Medical History

In the summer of 2011, she was struck in the head by a vehicle turret during mobilization training for her military deployment. According

to the patient and subsequent medical records, she lost consciousness for around 2 minutes and was treated that day by the unit medic but did not seek medical care at a hospital or by a primary care provider. Over the next two years, the Soldier was a victim of at least two physical assaults. The first event occurred while she was deployed and caused a great deal of distress and anxiety. She was diagnosed with post-traumatic stress disorder (PTSD), and her command transferred her from theater to the WTB at the San Antonio Medical Military Center (SAMMC) to be evaluated for continued military service. While her case was under review, her anxiety and headaches increased, and her physical condition deteriorated to the point where she required a cane to ambulate. During a counseling session at the WTB, she mentioned to her case manager a variety of visual symptoms, including persistent visual blur that started after the head injury in 2011. She also complained of current excessive light sensitivity and headaches when reading or using the computer. These visual problems were not corrected adequately with the last pair of spectacles provided to her at Fort Bliss, TX. By the time we saw the Soldier, she had been diagnosed with mild traumatic brain injury (mTBI), PTSD, major depression, dyssomnia, chronic back pain, and headache syndromes.

Besides the problems described, she had no other significant medical conditions. She was a light smoker and denied alcohol or illegal drug use. Her only allergy was to latex, and she was taking the following medications: (for anxiety) clonazepam 1mg tab tid, hydroxyzine pamoate 50mg cap tid, venlafaxine 150mg qd; (for sleep) trazodone 1.5x100mg tab qhs, ramelteon 8mg qhs; (for pain) tizanidine 4mg tab tid; (for headaches) propranalol 80mg cap qd, rizatriptan 10mg tab at onset of migraine; (for birth control) drospirenone/ethinyl estradiol 20mcg/3mg qd; (for constipation) sennosides/docusate 50mg bid; (for allergies) loratadine 10mg tab qd; (for asthma) levalbuterol 45mcg as needed. Her last eye exam was 17 months prior (May 2012), and she noted headaches and progressively blurry vision since the concussive incident in 2011. The exam revealed a slightly myopic refractive error, for which she was issued full-time spectacles.

Vision Assessment

Our Soldier suffered her most recent assault about three weeks before we performed the first vision examination in our clinic. The incident exacerbated her existing back pain and, naturally, her general level of anxiety and emotional distress. This led to several medication adjustments; she was still being assessed for their effects on the day she presented to us. At her initial appointment with us in October 2013, she was late, mildly disheveled, and dysthymic. She did not bring her old glasses but reported that she rarely used them because they "no longer work[ed] for her." She reported that the visual blur and photophobia that started after the concussion in 2011 had persisted but were stable. When questioned more closely, she also reported several other visual changes since the injury: inability to read comfortably for more than 30 minutes, words blurring in and out of focus when reading, difficulty keeping place when reading, occasional closing or covering of one eye to see more comfortably, and a history of "drifting" or bumping into walls when walking. She also noted that the drifting feeling was worse while driving; she had recently been in several small automobile accidents when she inadvertently "side-swiped" other cars or structures. She was currently using a cane for chronic back pain; important to our case is the observation that her complaint of drifting while walking predated the back pain but did not exist before the head injury. She reported no ocular discomfort but suffered from chronic headaches.

Pupils were round, and we noted them to be excessively reactive during the swinging

Table 3: Medications and Known Side Effects

Medication	Known visual side effects (incidence)
Clonazepam	Vision changes (rare)
Hydroxyzine pamoate	Blurred vision
Venlafaxine	Changes in vision, such as blurred vision (more common)
Trazodone	Blurred vision (more common)
Ramelteon	None
Tizanidine	Blurred vision (less common)
Propranolol	Blurred or loss of vision, double vision, disturbed color perception, halos around lights, night blindness, overbright appearance of lights, red irritated eyes, tunnel vision, dry eyes (incidence not known)
Rizatriptan	Blurred vision, dry eyes, eye irritation (less common)
Drospirenone/ethinyl estradiol	Vision changes, yellow eyes (incidence not known)
Sennosides/docusate	None
Loratadine	None
Levalbuterol	Discharge from the eye, eye itch (less common or rare)

flashlight test. We noted that her extraocular movements were jerky in all directions with continuous, irregular, rapid fixation corrections, especially while in upgaze. Her confrontations were full to finger count, and the distance cover test revealed unsteady fixation with frequent, irregular, vertical corrections in gaze, equal between the eyes. At distance, the cover test revealed a highly variable exophoria that we estimated averaged around 10 prism diopters. Non-contact tonometry (NCT) was 13mmHg and 12mmHg. Her anterior segment findings were unremarkable, and an undilated posterior segment examination by 90D lens revealed fleeting views of a healthy fundus with a round healthy disc and a cup-to-disc ratio of approximately 0.25 OU.

That day's autorefraction (AR) revealed less myopia and more astigmatism than we expected from her previous manifest refraction (MR). Conversely, that day's MR revealed more myopia and astigmatism than her previous exam. Her responses during refraction by phoropter were very slow, incomplete, and variable. We halted her MR at OD: -1.25 -0.50x160 ($20/20^{-1}$) and OS: -1.00-0.25x020 (20/25) when it became clear that the patient could not concentrate on the task. At near, she subjectively benefitted from a +1.00 ADD to bring her to 20/20 OU. NRA/PRA through this ADD was +1.00/-0.75. Again, her responses were variable. During the refraction, the Soldier reported that she couldn't concentrate on the letters partly because they were "moving and shaking" left and right when her fellow eye was occluded. Examination of eye movements outside the phoropter did not reveal any monocular or binocular nystagmus.

At this point, we decided to halt the exam and bring the patient back at the next available opportunity, as it was clear that the patient would be unable to provide enough information to determine an accurate prescription or to identify any subtle binocular vision problems. We also wanted to provide the patient with a full dilated fundus exam (DFE) to rule out any organic etiologies for her visual complaints. The Soldier agreed, and we released her for the day. We contacted her case manager with our concerns and coordinated a date for her return. We strongly suggested that the Soldier return to our clinic early in the morning to mitigate the effects of fatigue, and we requested that she not have any other medical appointments set for that morning so that she wouldn't feel rushed or anxious during our exam. We evaluated her current medications for adverse effects on the visual system, especially accommodation; we found that eight of the Soldier's twelve medications have a known side effect of "visual changes" or "blurred vision" (Table 3).[10]

At the next exam (November 2013), the Soldier presented much more alert and responsive. Entering acuities were OD $20/20^{-1}$ and OS 20/20 without correction; NCT was 9mmHg OD and OS. We found her manifest refraction to be OD: -0.25-0.25x165 (20/20) and OS: -0.50 sph (20/20) with no ADD needed for 20/20 OU near vision. Her NRA/PRA

improved to +1.25/-1.00, which is balanced but still slightly lower than we expected for her age. Pupils, extraocular movements (EOMs), cover testing, confrontation fields, and anterior segment findings were all normal; she displayed no irregular eye movements at all. We trial framed the full MR, and the Soldier noted good subjective comfort in the chair and while walking around our hallway. We dilated her with 1 gtt cyclopentolate 1% and 1 gtt tropicamide 1% and waited 45 minutes for the full cycloplegic effect to occur. Her CRx was OD: plano-0.25x165 (20/20) and OS: -0.25 sph (20/20), which are normal values that reflect an appropriate release of tonic accommodation from an accurate dry MR. This "wet" refraction ruled out latent hyperopia, and given her myopic MR at the previous exam, provided an insight into the Soldier's original complaints of blurry vision. Her DFE was unremarkable. We ordered a two pairs of full-time distance vision (DV) spectacles for her (one with 15% grey tint and one clear) and instructed the patient to pick up the glasses from us in two weeks. At four weeks, our intent was to assess her comfort with the new spectacles and conduct an extended binocular vision assessment. Important for her ongoing medical fitness review, we determined that she met all requirements for ocular health and visual function per Army Regulation (AR) 40-501, Chapter 2-12 (Eyes) and Chapter 3-16 (Vision).

At one month, the Soldier reported to the clinic for her neuro-optometric exam but had not yet picked up her spectacles. When presented with the new DV spectacles, she reported good subjective comfort with both sets of lenses. All entrance and anterior segment findings were normal. We have summarized her findings from the examination in Table 4.

Table 4: Examination Information (all BV findings through manifest refraction)

Procedure	Findings	Normal Values	Interpretation
Manifest Refraction	OD: -0.25-0.25x165 20/20 OS: -0.50 sph 20/20	n/a	n/a
Cycloplegic Refraction	OD: pl-0.25x165 20/20 OS: -0.25 sph 20/20		
Stereoacuity	140" circles, 100" animals	20"	Low
Worth 4-Dot	Suppression OS at D; Fusion at I, N	Fusion at 6m, 1m, 40cm	Focal suppression OS
NPC (accommodative target)	14 cm/17 cm	Break 5 cm, recovery 7 cm	Low
NPC (non-accommodative target)	20 cm/24 cm	Break 7 cm, recovery 10 cm	Low
Distance Cover Test	1 XP	Ortho	Ok
Near Cover Test	4 XP'	4-6 XP'	Ok
Distance VG	Horiz: Ortho Vert: Ortho	Ortho H/V	Ok
Distance BI (step)	x/8/5	x/7/4	Ok
Distance BO (step)	x/12/6	x/11/7	Ok
Near VG	Horiz: 4 exo Vert: Ortho	4-6 exo	Ok
Near BI (step)	11/18/14	x/13/10	Ok
Near BO (step)	22/24/8	x/19/14	Ok
NRA/PRA	+1.25/-1.00	+2.00/-2.50	Both low, worse PRA
Accommodative Amps (ML)	6.5D OD/OS	18-(age/3)=10	Low
AC/A (+2.00 method)	9 exo VG => (9-4)/2 = 2.5:1	4:1	Low
Vergence Facility (12BO/3BI)	9 cpm, trouble with BO	15 cpm	Low
MAF (+2/-2)	OD 12 cpm, OS 13 cpm	11 cpm	Ok
BAF (+2/-2)	9 cpm, trouble with plus	8 cpm	Ok
Double Maddox Rod Test	No cyclotorsion	No torsion	Ok
Saccades	2 small under-shoot corrections in all gazes	Accurate in all gazes	Low accuracy
Pursuits	Smooth, full range of motion	Smooth, full	Ok
Midline Shift Test	2"-3" consistent left shift	No shift	Shift left

We performed the midline shift test in the following manner: The patient was comfortably seated in the exam chair in full illumination. We provided the patient with the following instructions: "I am going to move this pen slowly across your field of view. I would like you to tell me to stop when the pen is directly in front of your nose. Please don't look at the pen though, and don't look at me, just relax your eyes and try to focus on an imaginary point in the distance. You may use the chart on the wall if you'd prefer. Do you understand?" Once the Soldier acknowledged understanding, the examiner positioned himself directly in front of the patient at approximately 75 cm and started moving the pen inwards at about 4 inches per second from the patient's far left periphery. When the patient said "stop," the examiner assessed the location of the pen in relation to the patient's nose by quickly alternating between his eyes to reduce parallax. He repeated this patient-left to patient-center movement three times and estimated an average value based on patient response. He then conducted the same movement and analysis three times from the patient's far right. The examiner then averaged the results from the trials in each direction to determine the direction and magnitude of shift from the patient's center line, defined by the location of her nose. Important in these multiple measurements is the assessment of consistency in patient response. A consistent localization to the left or right is more diagnostically reliable. Equal lead or lag from each side may not reflect a true midline shift but may represent a cognitive or perceptual dysfunction outside the scope of this particular test. Our Soldier demonstrated a consistent shift of about 2" to 3" to the left of true midline on every trial.

Impression

The diagnoses were: history of concussion (V15.52.0), refractive error–myopia (367.1), astigmatism (367.20), gaze convergence palsy (378.83), visuospatial defect (799.53).

Treatment

Our initial strategy was to address the existing refractive error by prescribing her MR in FT DV spectacles in standard military issue frames, which the patient found adequately decreased her DV blur when trial framed. We also ordered pair of 15% tint spectacles with her MR to address the photophobia. At one of her follow-ups, she reported that she wore the DV spectacles most, but not all, of the time. She reported that wearing the tinted lenses was helpful, but she usually just wore civilian non-prescription sunglasses because they were darker and looked better. She elected not to order darker lenses in the standard fames at that time. Department of Defense policy authorizes us to provide photochromatic lenses to Soldiers diagnosed with mTBI, but this Soldier did not express a desire for that type of lens. Regarding her convergence insufficiency, our primary strategy was to provide a slight ADD (+1.00 sph OU) with a small amount of base in (BI) prism (3^BI OU). We trial framed the proposed NVO prescription in-office, and the Soldier reported good subjective comfort and vision. We also discussed a few of the more common home vision therapies, including Brock string and "pencil pushups," but the Soldier elected to wait for the NVO spectacles. When the spectacles arrived a few weeks later and the Soldier tried them on, she again reported good subjective comfort and vision, and we dispensed them to her, with the instructions to use them as much as possible when reading or using the computer for the next few weeks and return in six weeks for a follow-up.

The use of BI prism for CI is not an optimal solution; full correction of ametropia and vision therapy (VT) for sensory fusion are generally preferred as first options. If VT is unavailable or unsuccessful, small amounts of

BI prism can be used in cases of CI. A good fixation disparity analysis should be used to prescribe the appropriate BI prism.[11] Our facility is not equipped to measure fixation disparity accurately or to provide vision therapy services. Given these restrictions and the lack of patient motivation to start therapy, considering that the Soldier would very soon separate from the Army and keeping in mind the reduced accommodation identified during testing, we elected not to pursue VT but to provide near NV spectacles with a small ADD and low BI prism as a trial. We believed that this choice would likely provide the best benefit in the shortest amount of time in this particular case.

Regarding the midline shift, we trial framed the MR with a few combinations of base right prism, and the patient responded favorably to 3^ base right OU. We prescribed the right-yoked prisms with her MR, and the Soldier elected to get these spectacles with photochromatic lenses. At her dispensing visit a few weeks later, the Soldier remarked immediately that the vision and comfort of these yoked prism spectacles were superior to her current lenses and that she felt like "the world seemed straighter" to her right away. We walked the Soldier down the hallway and around the clinic with ease, and the Soldier reported no dizziness or distortion with the new lenses. Before completing the lap around the clinic, she requested permission to wear these new glasses "all the time" instead of her older ones. We encouraged her to allow for an adaptation period of a few days before she tried to drive with them, but full time wear was approved. The Soldier was very thankful and encouraged that her overall comfort would improve.

Outcome

Ten days later, after we dispensed the yoked prism spectacles, the Soldier medically retired from the Army; she let us know by a recent phone call that she still has all the glasses and always uses the yoked prisms to drive. She feels much more confident driving and has had no "side-swipes" or close calls while wearing these lenses. She uses the DV specs most of the day unless she is reading for more than a few minutes; in that case, she switches to her BI NVO spectacles. She is working full time and is very happy with the results of our therapy, especially her increased confidence in driving. As she is no longer eligible to be seen in our clinic, we are unable to follow her and measure quantitatively any changes in her visual comfort since our intervention. Given the opportunity, we would be very interested in objectively assessing any such changes. We are also curious to assess any changes in her back pain, as there is a remote possibility that at least some of her sacroiliac pain may have developed from poor posturing due to her visuo-spatial imbalance. We must note that while we are happy with the Soldier's response to our correction of her lateral shift, we failed to conduct testing for anterior or posterior shift, for which proper correction could potentially have an even greater effect on overall comfort.

Conclusion

Every effort must be taken to identify uncommon problems properly (in this case, VMSS) and to apply uncommon solutions (in this case, yoked prism spectacles) to special needs patients. A properly engaged, educated, and equipped optometrist will provide the best possible chance for positive outcome. Our second point is that the importance of a deliberate approach to optometric care when working with a special needs population cannot be overstated. Our mTBI patients frequently present with visual sequelae that are sometimes confounded by the effects of multiple medications, by physical restrictions that may prevent proper evaluation, and by emotional or cognitive challenges that may not be evident upon initial examination.

For example, mTBI patients frequently experience anxiety or fatigue during extended oculomotor testing and need more breaks than typical patients. In our case, the findings from our initial encounter with our Soldier were inaccurate and unreliable given her emotional and medical condition; subsequent examinations were much more productive. Work-ups like these may need to take place over several visits, and longer cycloplegic exams may require coordination with the patient's care manager to ensure that adequate time and attention are available for proper evaluation. With respect to treatment, we have also found that this population may benefit from frequent follow-ups, even for routine spectacle prescriptions. Very small adjustments that may seem negligible in the "normal" population provide outsized improvements in comfort in mTBI patients. Her small refractive error and slight convergence insufficiency may have been easily compensable by a patient without her medical history, but to her our intervention made a big difference.

References

1. Hudac CM, Kota S, Nedrow JL, Molfese DL. Neural mechanisms underlying neurooptometric rehabilitation following traumatic brain injury. Eye and Brain 2012;4:1-12. http://bit.ly/1m1YqwH
2. Padula W, Nelson C, Padula W, Benabib R, et al. Modifying postural adaptation following a CVA through prismatic shift of visuo-spatial egocenter. Brain Injury 2009;23(6):566-76. http://1.usa.gov/1jYFAoH
3. Padula WV. Post Trauma Vision Syndrome. In: Padula WV, Munitz R, Magrun WM, eds. Neuro-Visual Processing Rehabilitation: An Interdisciplinary Approach. Santa Ana, CA: Optometric Extension Publishers, 2012: 57-69. http://bit.ly/1Qp9Tn1
4. Sharieff K. From Braille to quilting: A neuro-optometric rehabilitation case report. Optom Vis Dev 2010;41(2):81-91. http://bit.ly/1Z8giV1
5. Houston KE. Measuring visual midline shift syndrome & disorders of spatial localization: A literature review and report of a new clinical protocol. J Behav Optom 2010;21:87-93. http://bit.ly/1g8sK4l
6. Padula W. Neuro-Optometric Rehabilitation: Serving the Neuro-Visual Consequences of a Neurological Event. Presented to the College of Optometrists for Visual Development (COVD), October 23-25, 2014. [COVD website; updated 7/1/2014; cited 2/5/2015]. Available from http://bit.ly/1XzfQ4R
7. Robey JH, Boyle K. The role of prism glass and postural restoration in managing a collegiate baseball player with bilateral sacroiliac joint dysfunction: A case report. Int J Sports Phys Ther 2013;8(5):716-28. http://1.usa.gov/1RNSrYC
8. Asher A. About.com – The Daily Spine – Spinal Flexion – Posture, Injury, and Back Pain [updated 4/26/2011; cited 4/17/2014]. Available from: http://abt.cm/1HGGce9
9. Warrior Transition Battalion, Brooke Army Medical Center, US Army Medical Department [homepage on the Internet; updated 8/30/2013; cited 4/17/2014]. Available from: http://1.usa.gov/1Ylt5gV
10. Drugs.com website for each medication, homepage for Drugs.com [last updated 4/15/2014; cited 4/21/2014]. Available from www.drugs.com
11. Scheiman M, Wick B. Clinical management of binocular vision: Heterophoric, accomodative, and eye movement disorders. Philadelphia, PA: Lippincott Williams & Wilkins, 2002: 234. http://bit.ly/1NlzYQa

Correspondence regarding this article should be emailed to Christopher H. Caldwell, OD at cnw247@hotmail.com.

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Caldwell CH, Reyes-Cabrera E. A deliberate set of examinations and the application of yoked prisms in the treatment of visual midline shift syndrome. Optom Vis Perf 2015;3(6):440-9.

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Article ▸ The Optometric Care of Vision Problems After Concussion: A Clinical Guide

Miki Lyn D'Angelo, OD, Riverhead, New York

Barry Tannen, OD, Hamilton, New Jersey

ABSTRACT

Over the past decade, there has been a surge in the diagnosis of sports-related concussions. The etiology of this increase may be the cultural emphasis placed on physical activity, the heightened awareness of the signs and symptoms of concussion leading to better, more accurate diagnosis, or a combination of these two factors. Regardless, there is increased recognition that a number of concussion patients retain persistent symptoms which do not resolve within three months, resulting in the diagnosis of Post-Concussion Syndrome. Often, many of the lingering symptoms are visual in nature, with patients having difficulty with visual attention, decreased reading speed and comprehension, headaches, asthenopia, photophobia, and difficulty processing in busy visual environments. Research over the past ten years provides evidence that post-concussion patients have accommodative, vergence, oculomotor, and visual-vestibular dysfunctions to correlate with these symptoms; research has further classified this type of traumatic brain injury as Post-Traumatic Vision Syndrome (PTVS). Awareness of this syndrome is in its infancy, and there is a paucity of clinical information to help optometrists provide care for these patients. The goal of this paper is to be a clinical guide to the identification, treatment, and management of patients with PTVS.

Keywords: concussion, Post-Concussion Syndrome (PCS), Post-Traumatic Vision Syndrome (PTVS), vision rehabilitation, vision therapy

Introduction

A concussion is classified as a mild traumatic brain injury (mTBI) caused by a bump, blow, or jolt to the head that can cause a disturbance of cerebral function.[1] It is estimated that 1.5-3.8 million concussions happen annually, with 200,000 of those being sports-related.[1] This number is only an estimate, as concussions are often under-diagnosed and under-reported.[2] All athletic activities pose some risk of a concussive injury, but studies show that football for males and soccer for females pose the highest risk.[1]

Concussion Effects

A concussion is caused by a blow to the head or body with a force strong enough for the brain to displace the cerebrospinal fluid, hitting one side of the skull then rebounding to the opposite side. This is known as the coup/contra-coup reaction. The primary injury during the coup/contra-coup force is the diffuse shearing, stretching, and twisting of the brain's axons, which slows signal transmission.[3] Secondary injury occurs days to weeks following the initial injury, resulting from neurotoxins that have been released and a subsequent biochemical cascade, which further slows and interrupts normal communication within the brain.

It should be noted that because most of the effects of concussion are biochemical in nature, there is often no sign of brain damage on a

Table 1: Common Symptoms of Post-Traumatic Vision Syndrome (PTVS)[11]

Blurred vision, Distance viewing	Face or head turn	Disorientation	Discomfort while reading	Easily distracted	Loss of balance	Dizziness
Blurred vision, Near viewing	Head tilt	Bothered by movement in spatial world	Unable to sustain near work	Decreased attention span	Poor eye-hand coordination	Poor coordination
Slow to shift focus, near to far to near	Covering, closing one eye	Bothered by noises in environment	General fatigue while reading	Reduced concentration ability	Poor handwriting	Clumsiness
Difficulty taking notes			Loss of place while reading	Difficulty recalling what had been read	Poor posture	
Pulling or tugging sensation around eyes			Eyes get tired while reading	Easily distracted		

standard MRI. However, Inglese et al. show that special imaging techniques, such as diffusion tensor imaging, may be used as early indicators and prognostic measures of subsequent brain damage.[4] With the continued advancement in technology, these tests will eventually allow for better, more accurate diagnosis of concussion.

Post-Concussion Syndrome and Post-Traumatic Vision Syndrome

Post-Concussion Syndrome (PCS) is a diagnosis that is given when symptoms of a concussion persist and become pervasive in a patient's life for a period of greater than three months.[5,6] These symptoms often include headaches, cognitive and emotional issues, nausea, dizziness, visual disturbances, and loss of balance.[6] Post-Traumatic Vision Syndrome (PTVS) is a sequela of any acquired brain injury that is diagnosed when the symptoms are largely visual in nature.[7,8] The high prevalence of convergence insufficiency (30-42.5%),[9-11] deficits in saccades (19.6-51.3%),[9-11] and deficits in accommodation (21.7-41%)[9-12] within this population are well documented. Seglaeter et al.[13] also reveal that 26% of mTBI patients manifest an abnormal horizontal distance phoria, while Hellerstein et al.[14] find that approximately 37% of mild traumatic brain injury patients have reduced negative fusional ranges in the distance. These clinical vision syndromes correlate with the most common PTVS symptoms described in Table 1.[8]

Clinical Management

Patients with PCS suffer with symptoms that can be grouped into the following categories: somatic, emotional/behavioral, and cognitive.[7] Success in the evaluation of these patients will be based on the consideration that this population will most likely be suffering from headaches, fatigue, sensitivity to light/noise, and emotional instability. They will often have slowed thinking, slowed response speed, and deficits in executive function.[7] Adapting the speed, environment, and elements of your exam will allow for the most effective patient evaluation and diagnosis.

It is important to recognize that the pervasive effects of concussions often lead to these patients having a team of professionals collaborating towards maximal patient care and rehabilitation. Neuro-optometric rehabilitation is a key element for the patient's care and requires constant communication and correspondence with other medical professionals to ensure comprehensive care of the concussed population.[15]

Methods/ Exam Considerations

To accurately assess the visual system in the concussed and post-concussion syndrome patient, careful consideration of the following should be considered: quality of life surveys, visual acuity, the vergence and accommodative systems, oculomotor function, and the vestibular system. Table 2 gives a broad overview of these,

Table 2: Summary of Examination Elements and Treatment Considerations

Exam Elements	Tests	Treatment Considerations
Quality of Life Survey		
Visual Acuity/ Refraction	• Snellen Chart (Projector or Computerized) • ETDRS Chart	• Spectacles
Contrast Sensitivity	• MARS Test • Pelli-Robson • Vistech	• Tints
Vergence System	Distance & Near: • Cover Test • Von Graefe Phorias • Negative/Positive Fusional Ranges Near Only: • Near Point of Convergence (NPC) • Vergence Facility (3BI/12BO)	• Prism • Vision Therapy
Accommodative System	• Minus Lens Accommodative Amplitudes • +/-2.00 Accommodative Facility	• Near Vision Spectacles • Vision Therapy
Oculomotor Skills	Fixation/Saccades/ Pursuits • Gross Observation • Visagraph	• Vision Therapy
Visual Vestibular	•OKN Drum	• Binasal occlusion • Tints • Vision Therapy
Photosensitivity		• Tints

with corresponding diagnostic methods and treatment considerations.

Quality of Life/Activities of Daily Living Surveys

According to Rutherford et al,[16] approximately 10-15% of those with mTBI have post-concussive symptoms persisting beyond one year post-injury. To get a full understanding of the patient's symptoms, a clinically studied quality of life (QOL) or activities of daily living (ADL) survey is beneficial. A commonly used survey is the College of Optometrists in Vision Development (COVD) quality of life questionnaire (COVD-QOL). It has been shown to be statistically accurate in identifying an at-risk population, with a cut-off score of 20. It can also be used as a pre- and post-vision therapy marker.[17]

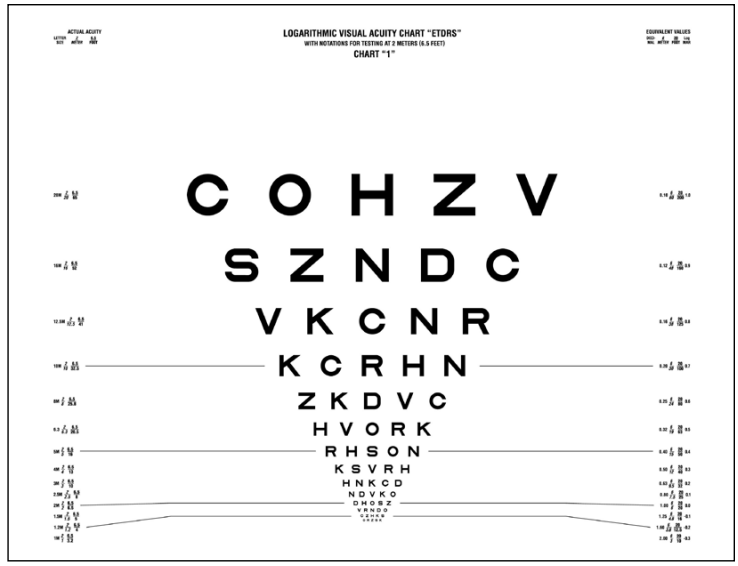

Figure 1. *Early Treatment Diabetic Retinopathy Study (ETDRS) Chart[19] (Precision Vision, La Salle, Illinois)*

Additionally, the authors recommend inquiring whether flickering or fluorescent lights are problematic for your patients. Many surveys do not include this question, but a study by Chang et al.[18] showed that the mean critical flicker fusion frequency (CFF) was found to be higher in mild TBI patients who suffered from light and motion sensitivity when compared to those not suffering from mTBI. The etiology of this correlation is perhaps neurological disinhibition as a result of the brain injury, but further studies are needed definitively to address this.[18]

Visual Acuity

Visual acuity should be taken with standard Snellen letters 20 feet from the patient. The authors use a method where visual acuity is assessed as single letter acuity to threshold and then by full-line assessment. This limits the amount of unnecessary distraction early in the exam. Computerized visual acuity systems allow for flexible testing in this manner.

If a computerized system is unavailable or there is concern of reduced contrast sensitivity, a clinician can opt to use the Early Treatment Diabetic Retinopathy Study (ETDRS) Chart (Figure 1). The ETDRS chart is commonly

Figure 2. *Pelli-Robson Chart*[21]

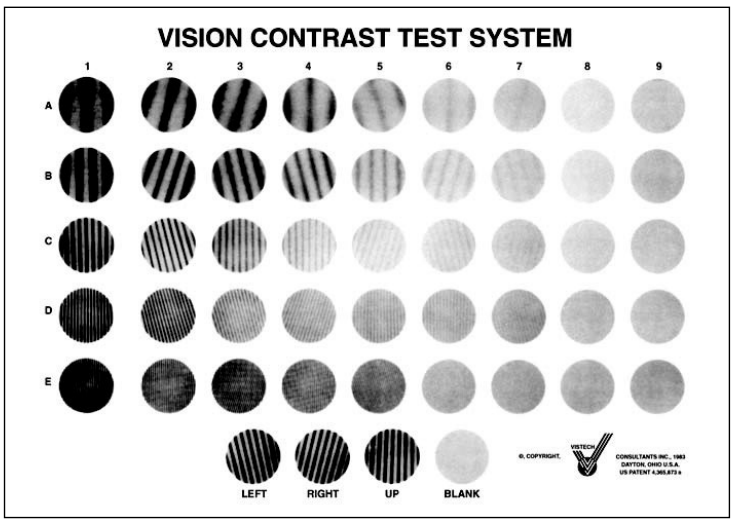

Figure 3. *Vistech Chart*[22]

Figure 4. *MARS*[23] *(Image courtesy of Mars Perceptix)*

used in large clinical studies because of result consistency. This chart is printed with high-contrast letters, is illuminated from behind, and has the same number of letters on each line (5) that decrease in size based on a mathematical formula. If necessary, this chart can be brought as close as 1 meter to evaluate visual acuity.[19]

Contrast Sensitivity

Many TBI patients present with 20/20 vision but have a series of complaints about the quality of their vision. If this is the case, testing a patient's contrast sensitivity is recommended. In simplest terms, contrast sensitivity is the ability to distinguish an object from its background. Lemke et al.[20] revealed that 21% of mTBI patients suffered from reduced contrast sensitivity, which correlated with poorer results on a quality of life survey.

To test contrast sensitivity, there are three popular options: the Pelli-Robson wall-mounted chart, the Vistech chart, and the hand-held MARS letter tests (Figures 2, 3, and 4).

The Pelli-Robson chart is performed at 40 inches (1 meter), requiring the patient properly to identify the letters in decreasing contrast.[21] The Vistech chart has two test distances of near (16 inches/40cm) and far (10 feet/3.05 meters), but instead of letters, patients have to identify the orientation of the grating bars correctly (vertical, horizontal, or diagonally oriented to the left or right).[22] The MARS letter test has a very similar design to the Pelli-Robson test, using letters of decreasing sensitivity, but this test is portable and is completed at 20 inches (0.5 meters). It should be noted that any of these tests are useful, but the MARS letter contrast sensitivity chart is the most reliable, with a 28% increased precision compared to the Pelli-Robson.[23]

Vergence

Thiagarajan et al.[10] show that 56.3% of the mTBI population has some type of vergence dysfunction. Similarly, Ciuffreda et al.[11] show

that 42.5% suffers from signs and symptoms of convergence insufficiency. With the known prevalence of vergence issues in this population, it is essential to perform complete static vergence measurements at both distance and near. Performing at both test distances covers the association of common near findings with visual discomfort, as well as the abnormal distance phoria and reduced fusional capabilities found in mTBI.[13,14]

Testing includes unilateral and alternate cover tests, near point of convergence, von Graefe heterophoria measurements, positive and negative fusional ranges, and vergence facility. All should be performed using standardized clinical test procedures.[24] The near point of convergence should be performed three times, with both accommodative and non-accommodative targets, to allow for the observation of degradation with increased visual stress.

Accommodation

Accommodative dysfunctions in the population are frequent and include insufficiency, excess, and infacility. Accommodative insufficiency, defined as a reduction in amplitude of accommodation to maintain clear near vision, has an incidence between 10-40%.[12] Patients with accommodative excess often report distance vision blur, whereas difficulty with accommodative facility often results in the inability to make quick viewing changes and maintain clear vision.

The accommodative system should be evaluated with various tests, including negative and positive relative accommodation, monocular minus lens amplitudes, a measurement of the lag/lead of accommodation, and accommodative facility with +/-2.00 flippers. Each of these tests should be performed via standardized procedures with the patient wearing their full refractive correction, but adjustment can be made if neccessary.[24]

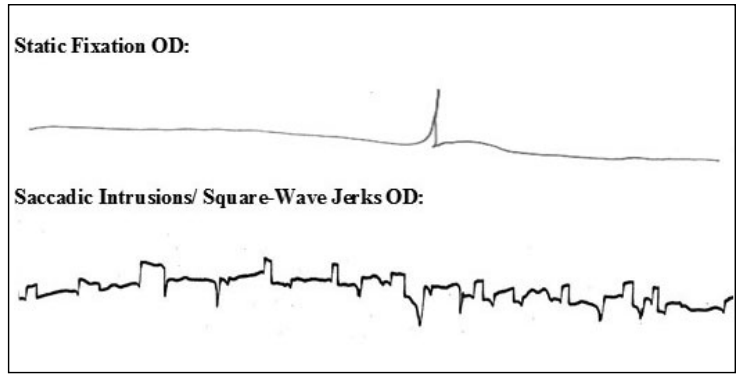

Figure 5. *Measurements taken by Visagraph (Dr. Barry Tannen)*

Eye Movement Evaluation

Fixation

Examination of the stability of the patient's fixation is essential in the assessment of our oculomotor system. The stability of fixation translates into the ability to gather visual information in a concise manner. If, however, there is an unintentional saccade, with a disruption in fixation, this is described as a saccadic intrusion or square-wave jerk.[25] The saccadic intrusions may cause symptoms such as floating/jumping of words, reduced reading speed, and an overall sense of instability in the patient's visual world.[26] Ciuffreda et al.[26] showed that fixation deficits were consistently found in individuals with ABI.

Fixation can be assessed grossly with the observation of both monocular and binocular fixation for ten seconds. There should be no loss of fixation from the target. Findings should be recorded as number of losses/ten seconds.[24]

A more precise measurement of fixation integrity would be to use the 'measurement only' function on the Visagraph/ReadAlyzer. Once the goggles are placed on the patient with their pupils aligned, the patient is encouraged to look back and forth between two objects about twelve inches apart on the observer's command, similar to the NSUCO method of assessing saccadic eye movements.[24] The scans should show steady fixation once the jump has been made. Figure 5 compares steady fixation with unsteady saccadic intrusions noted on Visagraph testing.

Saccades/Pursuits

Saccades are quick eye movements that allow a person to move their eyes from one object to another. Pursuits are the slow tracking eye movements that allow us to fixate and to follow an object. Both eye movements are essential for gathering visual information and maintaining a stable visual environment. Each should be assessed grossly by using standardized measures, such as the NSUCO methodology.[24]

Reading Eye Movements

Many mTBI patients report difficulty with tracking and visual attention and decreased reading comprehension. Ciuffreda et al.[11] report that there is a 90% occurrence of oculomotor dysfunction compared to 20% in the non-ABI symptomatic patients. They propose that if "an oculomotor dysfunction is not found after careful and comprehensive testing, it is unexpected and represents an exception to the rule."

Tools such as the Visagraph or ReadAlyzer are readily available for private practice use. The modified protocol for these instruments that Tannen and Ciuffreda adapted is recommended. This protocol calls for two recordings taken at the patient's independent reading level, with the second one being used for analysis, and one recording taken at least two grade levels below the patient's independent reading level.[27] A comprehension score of at least 70% is required to analyze data. If this level of comprehension is not achieved, the grade level of the text should be dropped until success is achieved. In theory, the grade level efficiency should remain relatively stable over the varying difficulty levels if an oculomotor dysfunction is present (+/-2 grade levels).[27]

Non-computer based methodologies such as King-Devick or Developmental Eye Movement (DEM) testing can also be used to evaluate oculomotor status.[28,29] The authors use the DEM/King-Devick alone for testing children who are unable to read fluently as these tests only require number recognition. These tests are also used as a basis for comparison to the Visagraph/ReadAlyzer to determine whether different types of visual stresses (letters/words vs. numbers) result in dissimilar findings, which contributes to understanding the patient's overall visual/cognitive function.

Vestibular System

The visual and vestibular systems are closely intertwined with the task of keeping a person's visual world stable. It is common for TBI patients to have a mismatch between these two systems, resulting in symptoms such as dizziness, blurred vision, difficulty with dynamic moving environments (i.e. supermarkets), and constant nausea.[30] To measure the mismatch between the visual and vestibular systems, a modification of the optokinetic drum proposed by Ciuffreda is used. It is recommended that testing should be done with the drum spinning slowly directly in front of the patient's fixation. Have the patient rate the level of discomfort on a scale of 1-10, with 10 eliciting severe vertigo-like symptoms. Repeat this procedure about 30 degrees to the left, to the right, above, and below eye level to assess the interaction fully and to ascertain whether there is one quadrant more bothersome.[31] The authors theorize that patients who suffer from visual-vestibular dysfunctions rely on visual information more than vestibular-proprioceptive cues, which results in an overwhelming vertigo-like response to visual environments. It is not fully understood why some quadrants are more bothersome to some patients than others, but it may be related to the type and area of the sustained injury. In non-TBI populations, the OKN drum should not elicit any symptoms.[31]

Treatment Options

Spectacles

We have found that TBI patients often have an increased sensitivity to small refractive errors. It is essential to do a careful refraction, keeping in mind that mild astigmatic, myopic, and

hyperopic corrections may result in subjective improvement. These prescriptions often have a two-fold benefit: 1) the correction of these small refractive errors gives the patient an extra bit of clarity and boldness that improves decreased contrast sensitivity, and 2) correcting for these small errors allows for the visual system to be balanced accommodatively, which removes this as a disruption to binocularity. However, one must be careful about pseudo-myopes who may subjectively take extra minus. Remember only to prescribe these small refractive errors when a patient's objective and subjective findings correlate. The consideration for near vision only spectacles should be made when a patient is suffering from a symptomatic accommodative insufficiency. When prescribing for near with the mTBI population, it is recommended for the prescriber to consider all objective and subjective information and to trial frame the proposed prescription to ensure that the patient will accept the added plus at near. If there is a mismatch between objective and subjective findings, two options present themselves: recheck the patient at a follow-up visit, or prescribe based on professional judgment.

Prism

Depending on the case, yoked or compensatory prism may be indicated to help alleviate symptoms and to encourage binocularity. We have found that patients with a larger-angle convergence insufficiency or a small vertical deviation respond well to low amounts of compensatory prism. The authors recommend an in-office trial of prism to evaluate patient response.

Tints

Photosensitivity is a prevalent problem in this population (57.8%).[32,33] People who suffer from PCS often retain sensitivity to light past the common healing time of 6 months.[34,35] Common brown and gray polarized tints may be added to lenses for outdoor use. For difficulty with fluorescent lights or motion sensitivity, a blue-ish purple tint from BPI (Omega) may alleviate symptoms.[35]

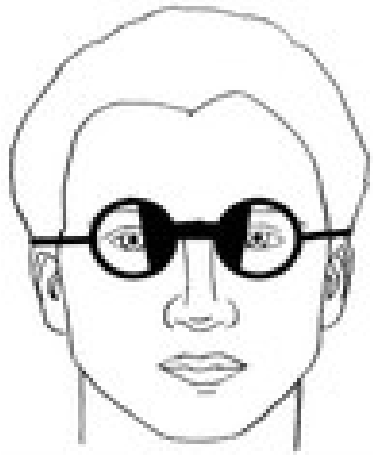

Figure 6. *Binasal occlusion (Image from VisionHelpBlog)*

Binasal Occlusion

Patients who suffer from severe motion sensitivity/vestibular dysfunction may benefit from binasal occlusion. Binasal occlusion can be achieved with black/clear tape or Bangerter foils oriented diagonally at the nasal limbus on the patient's glasses (Figure 6). Padula et al. showed that binasal occlusion helped those suffering from PTVS to organize their peripheral visual processes and aided in the improvement of binocularity and fusion.[36] A recent study by Yadav and Ciuffreda[37] also showed that binasal occlusion in the mTBI population improved VEP findings and also improved visual impressions and sensorimotor tasks in 90% of their subjects.

Vision Therapy

Research in neuroplasticity demonstrates that therapy enhances top-down (cognitive) processing, resulting in the recovery of function and reduction of symptoms.[38] In order to be productive when treating patients with TBI, the approach to vision therapy needs to take into account various processing mechanisms and neuroplasticity.[9] Cohen et al.[9] discuss five components to achieving effective therapy: motivation/goal-directed participation, repetition, feedback to recalibrate, refining encoded

responses, and creating a motor match to a sensory mismatch to encourage sensorimotor recalibration and multi-sensory (intermodal) integration. The authors have consolidated the vision therapy sequence into three phases of care.

Phase 1: Visual Stabilization

Initial therapy needs to work on building monocular oculomotor and accommodative skills, while simultaneously stabilizing the vergence system at both distance and near. Activities include basic convergence, Brock string, monocular accommodative rock, near-far Hart chart, Hart chart saccades, Michigan tracking, Marsden ball activities, and PTS II Visual Search and Scan.[39]

Phase 2: Binocular Vision Integration

Once monocular skills have improved, therapy focuses on binocular accommodative and oculomotor tasks, while challenging the vergence system to make quick changes in demand. This level should allow for the basic integration of vergence and accommodation and help to increase the speed and accuracy of all activities. Procedures in this phase include binocular accommodative rock, early BOP/BIM techniques, loaded Brock string with prism flippers, Hart chart saccades, and dynamic reader/visual search and scan with prism and accommodative flippers.[39]

Phase 3: Visual Automaticity

The last phase of therapy helps to ensure that the newly learned visual skills have become fully habituated. Therapy procedures are designed to challenge the patient continually to refine their visual skills, to increase speed of response, and to incorporate the vergence, accommodative, vestibular, and auditory systems. Therapy exercises in this phase are similar to the previous phases but are performed at a higher demand level by challenging further integration of systems. Incorporation of vestibular movements, such as head turns (VOR) and metronomes with higher-level accommodative/vergence procedures, builds and encourages the development of automaticity.[39]

Follow-Up Schedule

If a patient is in active therapy, a progress check is recommended after every 10-12 sessions of therapy to ensure that therapy is addressing all of the patient's symptoms and concerns. After therapy is complete and home maintenance is dispensed, it is our protocol to follow up with the patients six weeks after therapy, then again in three, six, and twelve months post-therapy.

Conclusion

With the growing number of patients who suffer from PCS and PTVS, it is essential as healthcare providers to identify, to diagnose, and to treat these conditions. Understanding the pervasive effects that concussions have and the application of this treatment model will serve as a starting point in providing comprehensive care to this in-need population.

References

1. Concussion and Traumatic Brain Injury. Centers for Disease Control and Prevention; [cited 29 May 2014]. Available from: http://www.cdc.gov/Concussion.
2. Shrey DW, Griesbach GS, Giza CC. The pathophysiology of concussion in youth. Phys Med Rehabil Clin N Am 2011:22577-602. http://1.usa.gov/1RkH9Ml
3. Blennow K, Hardy J, Zetterberg H. The neuropathology and neurobiology of traumatic brain injury. Neuron 2012;76(5):886-99. http://1.usa.gov/1lJlFvR
4. Inglese M, Makani S, Johnson G, Cohen B, et al. Diffuse axonal injury in mild traumatic brain injury: A diffusion tensor imaging study. J Neurosurg 2012;116:298-303. http://1.usa.gov/1QiMDY0
5. Ryan LM, Warden DL. Post concussion syndrome. Int Rev Psychiatry 2003;15:310-6. http://bit.ly/1RkHtdP
6. Kirkwood MW, Yeates KO, Wilson PE. Pediatric sport-related concussion: A review of the clinical management of an oft-neglected population. Pediatrics 2006;117:1359-71. http://bit.ly/1O0wsrA
7. Padula W, Argyris S. Post trauma vision syndrome & visual midline shift syndrome. Neurorehab 1996;6:165-71. http://bit.ly/1MdBX4Y

8. Chang A, Cohen AH, Kapoor N. Top-down visual framework for optometric vision therapy for those with traumatic brain injury. Optom Vis Perform 2013;1:48-53. http://bit.ly/1YfXL6X
9. Goodrich GL, Kirby J, Cokerha G, Ingalla SP, et al. Visual function in patients of a polytrauma rehabilitation center: a descriptive study. J Rehabil Res Dev 2007:44(7):929-36. http://1.usa.gov/1I57kUz
10. Thiagarajan P, Ciuffreda KJ, Ludlam DP. Vergence dysfunction in mild traumatic brain injury (mTBI): A review. Ophthalmic Physiol Opt 2011;31:456-68. http://bit.ly/1cvAo81
11. Ciuffreda KJ, Kapoor N, Rutner D, Suchoff IB, Han ME. Occurrence of oculomotor dysfunctions in acquired brain injury: A retrospective analysis. Optometry 2007;78:155-61. http://bit.ly/1cvAo81
12. Green W, Ciuffreda KJ, Thiagarajan P, Szymanowicz D. Accommodation in mild traumatic brain injury. J Rehabil Res Dev 2010;47:183-200. http://1.usa.gov/1P35If6
13. Schlageter K, Gray B, Hall K, Shaw R, Sammet R. Incidence and treatment of visual dysfunction in traumatic brain injury. Brain Inj 1993:7:439-48. http://1.usa.gov/1TLM7Q6
14. Hellerstein LF, Freed S, Maples WC. Vision profile of patients with mild brain injury. J Am Optom Assoc 1995:66:634-9. http://1.usa.gov/222DlTO
15. Suter PS, Harvery L. Vision Rehabilitation: Multidisciplinary Care of the Patient Following Brain Injury. Boca Raton, FL: CRC Press, Taylor & Francis Group, 2011. http://amzn.to/1ItitfV
16. Rutherford WH, Merrett JD, Mcdonald JR. Symptoms at one year following concussion from minor head injuries. Injury 1979;10:225-30. http://1.usa.gov/1J7B5yT
17. Bleything WB, Landis SL. Effectiveness of the College of Optometrists in Vision Development – QOL questionnaire in a socially at risk population of youth. Optom Vis Dev 2008:39:82-90. http://bit.ly/1I7p6oc
18. Chang TT, Cuiffreda KJ, Kapoor N. Critical flicker frequency and related symptoms in mild traumatic brain injury. Brain Inj 2007;21:1055-62. http://1.usa.gov/1I58dwm
19. Kaiser PK. Prospective evaluation of visual acuity assessment: A comparison of Snellen versus ETDRS charts in clinical practice. Trans Am Ophthalmol Soc 2009;107:311-24. http://1.usa.gov/1J7Bh12
20. Lemke S, Cockerham GC, Glynn-Milley C, Cockerham KP. Visual quality of life in veterans with blast-induced traumatic brain injury. JAMA Ophthalmol 2013;131:1602-9. http://bit.ly/1RO4khf
21. Pelli DG, Robson JG, Wilkins AJ. The design of a new letter chart for measuring contrast sensitivity. Clin Vis Sci 1988;2:187-99. http://bit.ly/1NtpVWV
22. Pesudovs K, Hazel CA, Doran RML, Elliot DB. The usefulness of Vistech and FACT contrast sensitivity charts for cataract and refractive surgery outcomes research. Br J Ophthalmol 2004;88:11-6. http://1.usa.gov/1QiPCj8
23. Arditi A. The MARS letter contrast sensitivity test. [Cited 12 Dec 2014] Available from: http://bit.ly/1MWxaIT
24. Scheiman M, Wick B. Clinical Management of Binocular Vision: Heterophoric, Accommodative, and Eye Movement Disorders (3rd ed). Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2008. http://bit.ly/1NlzYQa
25. Weissberg E, Lyons SA, Richman JE. Fixation dysfunction with intermittent saccadic intrusions managed by yoked prisms: A case report. Optometry 2000; 71:183-8. http://1.usa.gov/1Ntqa4l
26. Ciuffreda KJ, Han Y, Kapoor N. Oculomotor fixation and its rehabilitation in acquired brain injury. Invest Ophthalmol Vis Sci 2005:45 E-Abstract 2325. http://bit.ly/1Qplr9K
27. Tannen B, Ciuffreda KJ. A proposed addition to the standard protocol for the Visagraph II eye movement recording system. J Behav Optom 2007;18:143-7. http://bit.ly/1RO4w00
28. Ciuffreda KJ, Rutner D, Kapoor N, Suchoff I, et al. Vision therapy for oculomotor dysfunction in acquired brain injury. Optom 2008;79:18-22. http://1.usa.gov/1mknGP0
29. Dhawan P, Starling A, Tapsell L, Adler J, et al. King-Devick test identified symptomatic concussion in real-time and asymptomatic concussion over time. Neurol 2014;82:S11.003. http://bit.ly/1RklYZx
30. Craig S, Kapoor N, Ciuffreda KJ, Suchoff IB, et al. Profile of selected aspects of visually-symptomatic individuals with acquired brain injury: A retrospective study. J Behav Optom 2008;19:7-10. http://bit.ly/1P36OHU
31. Ciuffreda KJ. Visual vertigo syndrome: Clinical demonstration and diagnostic tool. Clin Eye Vis Care 1999;11:41-4. http://bit.ly/1MdD0lx
32. Digre KB, Brennan K. Shedding light on photophobia. J Neuro-Ophthalmol 2012;32:68-81. http://1.usa.gov/1I8U0dG
33. Vos PE, Battistin L, Birbamer G, Gerstenbrand F, et al. EFNS guideline on mild traumatic brain injury: Report of an EFNS task force. Eur J Neurol 2002;9:207-19. http://1.usa.gov/1IJorkS
34. Bohnen N, Twijnstra A, Wijnen G, Jolles J. Tolerance for light and sound of patients with persistent post-concussional symptoms 6 months after mild head injury. J Neurol 1991;238:443-6. http://bit.ly/1IQSTDA
35. Ciuffreda KJ, Ludlam D. Conceptual model of optometric vision care in mild traumatic brain injury. J Behav Optom 2011;22:10-3. http://bit.ly/opstudents
36. Padula W, Argyris S, Ray J. Visual Evoked Vision Syndrome (PTVS) in patients with traumatic brain injury (TBI). Brain Injury 1994;8(2):125-33. http://1.usa.gov/1RkJoiP
37. Ciuffreda KJ, Yadav NK, Ludlam DP. Effect of binasal occlusion (BNO) on the visual-evoked potential (VEP) in mild traumatic brain injury (mTBI). Brain Inj 2013; 7:41-7. http://bit.ly/1P37gWy
38. Tani J. Learning to generate articulated behavior through the bottom-up and top-down interaction processes. Neural Networks 2003;16(1):11-23. http://bit.ly/1P37gWy
39. Tannen B, Kapoor N, Ritter S. General vision therapy: Diagnostic and therapy procedures laboratory manual, 6th edition. SUNY State College of Optometry 2009:1-227.

Correspondence regarding this article should be emailed to Miki Lyn D'Angelo, OD at drdangelo@soundvisioncare.com. *All statements are the authors' personal opinions and may not reflect the opinions of the representative organizations, ACBO or OEPF, Optometry & Visual Performance, or any institution or organization with which the authors may be affiliated. Permission to use reprints of this article must be obtained from the editor. Copyright 2015 Optometric Extension Program Foundation. Online access is available at* www.acbo.org.au, www.oepf.org, and www.ovpjournal.org.

D'Angelo M, Tannen B. The optometric care of vision problems after concussion: A clinical guide. Optom Vis Perf 2015;3(6):451-61.

Appendix 1

JP- Age 16

A concussion specialist referred JP, a sixteen-year-old male, for a neuro-optometric work-up after he was still suffering symptoms months after his concussion. JP reported that he sustained a concussion from swimming full-speed into a concrete wall during a swim meet. He lost consciousness on the ride to the hospital and experienced amnesia directly following the incident. JP noted that he had a few other minor head injuries, the most notable a fall due to loss of balance two months after the initial concussion. Prior to our exam, JP underwent vestibular, exertional, physical, occupational, and cognitive therapies. Despite all of these therapies, JP still experienced severe eyestrain, headaches, fatigue with near work, nausea with busy visual environments, loss of place while reading, and decreased reading comprehension.

A full optometric exam, performed similarly to the one outline above, revealed that JP suffered from severe accommodative instability. He had reduced amplitude of 4.75/4.25D (target: 12.67D). (Table 3).

His vergence testing revealed a receded near point of convergence of eight inches with reduced compensatory vergence ranges, indicating a large convergence insufficiency. Distance vergence ranges showed fusional instability, with JP's distance positive fusional ranges breaking at 4Δ (19Δ is expected) and recovering at 0Δ (10Δ is expected). His negative fusional ranges were also reduced, indicating a lack of fusional stability.

Oculomotor assessment revealed a small saccadic intrusion on fixation, resulting in two losses of fixation/ten seconds. Visagraph testing also revealed a lower than grade level efficiency of 7.8, when JP was a sophomore (Grade 10) in high school.

JP was diagnosed with convergence insufficiency, fusional instability, oculomotor dysfunction, and accommodative insufficiency, for which neuro-optometric rehabilitative therapy was recommended. After 24 sessions, JP's objective findings improved, but more importantly, his symptoms subsided. Table 4 shows the post-vision therapy findings. The pre and post COVD-QOL questionnaires were 72 and 13, respectively. After 6 months of home vision therapy, JP reported that he felt 'visually normal' again and was able to return to all of his regular activities with no issue.

Table 3. JP Initial Findings-Vision Efficiency and Processing Evaluation

Test	Exam Results	Normal Range	Interpretation
Best Corrected Visual Acuity	RE: 20/20 LE: 20/20	20/20	Normal
Refractive Status	RE: -2.75 DS LE: -2.75 DS	n/a	Mild Myopia
Distance Phoria	2 exophoria	0-2 exophoria	Normal
Near Phoria	6 exophoria	0-6 exophoria	Normal
Nearpoint of Convergence	8 inches	3 inches	Convergence Insufficiency
Near Convergence Range	16 pd	18-24 pd	Convergence Insufficiency
Near Convergence Recovery	8 pd	7-15 pd	Normal
Near Divergence Range	16 pd	18-24 pd	Fusional Instability
Near Divergence Recovery	6	10-16 pd	Normal
Distance Convergence Range	4 pd	14-24 pd	Fusional Instability
Distance Convergence Recovery	0 pd	6-12 pd	Fusional Instability
Distance Divergence Range	4 pd	5-10 pd	Fusional Instability
Distance Divergence Recovery	2 pd	2-6 pd	Normal
Vergence Facility	17 cpm	>15 cpm	Normal
Accommodative Amplitude	RE: 4.75 D LE: 4.25 D	11.00 D	Accommodative Insufficiency
Test of Silent Word Reading Fluency	2.7 %	38th %	Reading Fluency Deficit
Visagraph Reading Eye Movement Test 7th Grade text	7.9 grade level	9.0 or greater grade level	Oculomotor Dysfunction

Table 4. Pre & Post Vision Therapy Evaluation

Test	Initial Evaluation	Re-Evaluation	Interpretation
Best Corrected Visual Acuity	RE: 20/20 LE: 20/20	20/20	Normal
Refractive Status	RE: -2.75 DS LE: -2.75 DS	n/a	Mild Myopia
Distance Phoria	2 exophoria	2 exophoria	Normal
Near Phoria	6 exophoria	6 exophoria	Normal
Nearpoint of Convergence	8 inches	1 inch	Normal
Near Convergence Range	16 pd	36 pd	Normalized
Near Convergence Recovery	8 pd	24 pd	Normalized
Near Divergence Range	16 pd	28 pd	Normalized
Near Divergence Recovery	6 pd	20 pd	Normalized
Distance Convergence Range	4 pd	10 pd	Normalized
Distance Convergence Recovery	0 pd	6 pd	Normalized
Distance Divergence Range	4 pd	22 pd	Normalized
Distance Divergence Recovery	2 pd	16 pd	Normalized
Vergence Facility	17 cpm	19 cpm	Normalized
Accommodative Amplitude	RE: 4.75 D LE: 4.25 D	OD: 11.00 D OS: 12.50 D	Normalized
Test of Silent Word Reading Fluency	2.7 %	79 %	Normalized
Visagraph Reading Eye Movement Test 7th Grade text	7.9 grade level	13.4 or greater grade level	Normalized

Article ▸ Visual Field Defects Secondary to a Cerebrovascular Accident

Briana Larson, OD, Austin, Texas

ABSTRACT

Background: Visual field defects occur frequently with acquired brain injury (ABI). Complex visual-motor and sensory-motor processes are affected in visual field defects, often resulting in significant impact to overall function, demeanor, and quality of life. Neuro-optometric rehabilitation, which may include lenses, visual rehabilitation therapy, and referrals to other professionals, can result in improvement in overall function, demeanor, and quality of life for individuals suffering from visual field defects secondary to ABI.

Case Summary: A 74-year-old Caucasian female presented 1 month following a cerebrovascular accident of the right side with concern about left visual field loss. Symptomatology included that her "eyes do not seem to focus together," blurry vision especially in left field, re-reading lines or words while reading, poor depth perception, loss of balance, disorientation, and difficulty with peripheral vision. The evaluation revealed the diagnosis of a superior left field quadrantanopia OD, left hemianopia OS, intermittent alternating exotropia of convergence insufficiency type, and oculomotor deficits of saccades and pursuits. Neuro-optometric rehabilitation, consisting of an active vision rehabilitative therapy program of in-office sessions and prescribed home activities, was prescribed. The vision rehabilitative therapy program encompassed compensatory oculomotor scanning, sensory-motor integration, and development of deficient binocular and oculomotor skills. In addition, the program also included the development of improved visual processing skills in the areas of visual spatial relations, visual memory, and processing speed (including span and tachistoscopic recognition). The outcome of this case yielded successful overall achievement of the patient's and doctor's goals, as well as significantly improved symptomatology and enhanced quality of life.

Conclusion: Appropriately prescribed neuro-optometric rehabilitation yields successful outcomes on quality of life post ABI with visual field defects. Diagnostic scrutiny of visual field defect-related conditions, such as visual midline shift, unilateral spatial inattention (USI), and focal-ambient integrative processing dysfunction, is important to optimize treatment plans. In addition, consoled phasic induction of applicable prismatic lenses maximizes prism acceptance and improves the outcome of neuro-optometric rehabilitation.

Keywords: acquired brain injury, cerebrovascular accident, focal and ambient processing, hemianopia, neuro-optometric rehabilitation, unilateral spatial inattention, visual field defect, visual midline shift

Background

Visual field defects of various types and degrees of impairment are common among individuals affected by an acquired brain injury (ABI) and have significant impact on recovery and overall function. Visual field defects affect the visual processes involved in sensory-motor relations to organize and gain meaning of visual space and to administer related executive functions. For instance, small scotomas near

central fixation can disrupt tasks requiring fine visual discrimination, such as reading.[1] In individuals with an ABI, the disruption in the previously intact system leads to a series of visual processing disruptions and resulting dysfunction.

The visual field defect homonymous hemianopia, which is loss of vision to the same side of half of each eye's visual field, occurs in approximately 10% of stroke patients.[2] Homonymous visual field defects are among the most frequent disorders that occur in the elderly after vascular brain damage and can have a major impact on quality of life.[3] Homonymous hemianopias are commonly associated with an egocentric visual midline shift (perceived straight-ahead is usually in the direction opposite to the affected visual field), resulting in veering to one side and running into or tripping over objects in the affected field during ambulation.[1]

Visual field defects following an ABI can be associated with unilateral spatial inattention (USI), which is a lack of awareness and attentional control on the contralesional side of space in which individuals are unresponsive to events, objects, and people in the related side of external space.[4] USI patients may even fail to acknowledge contralesional parts of their own body or of mental representations.[5] The acute onset of these visual disruptions of external space caused by the visual field defect can be debilitating to both overall function and demeanor. Past experiences developed from intact sensory-motor systems have minimal value to the visual system with new visual field defects.[6] Frustration and difficulty dealing with inadequacies and loss of independence typically result, in addition to apprehensions about safety. USI has a significant impact on the overall outcome following brain damage.[7]

Neuro-optometric rehabilitation, which includes lenses, visual rehabilitation therapy, and referrals to other professionals, can result in improvement in overall function, demeanor, and quality of life for individuals suffering from visual field defects secondary to ABI. Studies have shown that individuals with visual field deficits who are active in vision rehabilitation show the best prognosis for achieving their personal goals of safety and independence.[1] For instance, a study showing that stroke patients with persisting visual field defects possessed significantly reduced mental health compared to that of stroke patients without visual field defects.[2] This study concluded that individuals with persistent visual field defects should be offered additional neuropsychological rehabilitation that may improve visual functioning or supportive psychotherapeutic interventions.[2] Furthermore, a high incidence of patients with USI following stroke are misdiagnosed as having dementia.[8] Many patients with a cerebrovascular attack (CVA) have undiagnosed USI, which puts them at risk for injury.[9]

The present case of a visual field defect secondary to CVA emphasizes the importance of diagnostic scrutiny of visual field defects and their effect on sensory-motor function following an ABI. The case also highlights the significance of consoled phasic induction of applicable prismatic lenses to maximize prism acceptance and to improve the neuro-optometric rehabilitative outcomes.

Course of this Disorder

CVA is a leading cause of death in the United States, killing nearly 130,000 individuals each year.[10] CVA is more common in the elderly, although it can occur at any age.[11] CVA, otherwise known as stroke, occurs when there is an interruption in blood flow that disrupts oxygen to the brain cells.[12] Eighty-five percent of strokes are ischemic, in which an artery in the brain becomes blocked.[13] The other two common types of stroke are hemorrhagic and transient ischemic attack.[13] Eighty percent of all strokes are preventable, which begins with management of risk factors, including high blood pressure, cigarette smoking, atrial fibrillation,

and physical inactivity.[14] The most important risk factor to control is hypertension, which is the cause of more than half of all strokes.[14]

A CVA can be debilitating to one's overall function and can negatively affect cognitive functions such as memory, attention, perception, and planning.[15] Visual impairments contribute to these deficits and are common after a CVA. A recent study showed that of 915 patients following a CVA, 54% were diagnosed with ocular motility abnormalities.[16] These were isolated impairments in 50% of cases and were associated with other ocular abnormalities in the other 50%, including impaired convergence, nystagmus, and lid or pupil abnormalities.[16] Furthermore, 35% of the patients experienced symptoms of diplopia and blurred vision.[16] Visual field loss has been reported in up to a quarter of stroke survivors.[17] Visual field loss is a loss of part of the field of vision and typically involves the peripheral field of vision with a CVA.[18] The most common type of visual field loss associated with CVA is homonymous hemianopia, which occurs in approximately two-thirds of those with visual field loss.[18] Other common types of visual field loss following a CVA is the loss of a quarter of the visual field (homonymous quadrantanopia) and an island-like area of blindness (scotoma).[17] Visual field loss following stroke has been attributed to cortical strokes, in which the visual pathway is damaged.[18] Vision care of an individual who has suffered a stroke is crucial given the high prevalence of visual deficiencies after stroke.

Case Report

History

CC, a 74-year-old white female, was referred by a hospital-based physical therapist for a consultation regarding treatment options for visual field loss. CC was retired, and prior to her insult, she enjoyed reading, sewing, line-dancing, and cooking. She lived with her husband in a private home with moderate independence, with the exception of her husband performing all of the driving. CC's main reason for the consultation was to determine the probability of gaining function in her left visual field and partaking in her pastime activities with greater ease and enjoyment.

Table 1: Presenting Signs and Symptoms

Eyes do not seem to focus together
Dry/irritated eyes, gritty/sandy sensation
Halos around lights
Itchy eyes
Blurry vision, especially in left field
Shadowy vision
Tired/sore/uncomfortable eyes
Re-reads lines or words
Loss of place while reading
Reduced memory
Uses finger as a marker to keep place while reading
Tilts/turns head when reading and writing
Poor depth perception
Loss of balance
Disorientation
Poor sense of direction
Decreased memory/forgetfulness
Poor posture
Sensitivity to light and glare
Difficulty with peripheral vision

CC experienced a stroke on her right side. She was hospitalized for 3 days and was released with instruction to enter rehabilitation. She was also advised to lower her dietary fat and salt intake and was referred to her cardiologist and general physician for routine care. CC was prescribed Tylenol and Plavix-potassium. CC recalled no double vision immediately following the insult. She reported complete loss of her left field with some recovery and consistent blur of her left field of vision.

Table 1 shows CC's presenting visually related signs and symptoms. Her major concern was the effect of her reduced left visual field on her activities of daily living. She reported that she no longer enjoyed reading, could not drive, and was constantly frustrated because she did not see things that her husband informed her were right in front of her. Furthermore, she reported that she was often startled as her husband would appear immediately before her and she could not see him approaching. She noted that since the insult, her activities

of daily living mostly entailed sleeping, eating, and visiting with neighbors. She was on the computer 1 hour per day and read about 1-2 hours per day. CC also expressed a strong desire to line dance again, especially to turn without losing her balance and feeling disoriented. She also desired to improve her memory.

CC's medical history was positive for fatigue, headaches, heart disease, an allergy to penicillin, hypertension, elevated cholesterol, and arthritis. Her surgical history was positive for a hysterectomy, gall bladder removal, and hernia repair. Her daily medications included the following: Atacand, Coreg, Hydrochlorothiazide, Plavix, Tums Ultra, Lipitor, Zocor, Vitamin D3, multi-vitamins with minerals, Lotrisone topical cream, and Elocon 0.1% topical cream. Her family history was positive for heart disease, cancer, and macular degeneration. CC's eye health history was positive for cataract removal with Phaco-Restor-Aspheric lens implant (+22.50) of both eyes and selective laser trabeculoplasty in both eyes. Her ocular daily medications included Optive in both eyes as needed. She wore over-the-counter reading glasses.

CC received a few hours of occupational and physical rehabilitation immediately following her hospital release post-insult. She walked without aid with mildly decreased gait, and her dexterity and coordination appeared intact. Her posture appeared well-balanced and aligned both sitting and walking. Her cognitive function appeared intact, with adequate recall, conversation skills, and illustration of wit. Her receptive and expressive language and speech pronunciation also appeared to be within normal ranges. Energy level, responsiveness, mood, and social interactions all appeared within normal levels. CC independently performed and maintained proper hygiene and physical appearance. She cooked and performed household chores independently, and she refrained from driving.

Existing Condition Background

Vessel occlusion causes approximately 80% of strokes; the rest are hemorrhagic.[9] The specifics of CC's cerebrovascular accident (CVA) are unknown; however, based on her medical history review, she was at risk secondary to her hypertension and high cholesterol. She was currently monitored routinely by her cardiologist and general practitioner, and she reported compliance with her medications to regulate her blood pressure and cholesterol for prevention of reoccurrence.

Diagnosis

Table 2 illustrates the diagnostic data from CC's initial assessment. The following are her diagnoses:

1. Mild compound hyperopic astigmatism OU and presbyopia
2. Concomitant alternate intermittent exotropia of convergence type, greater OS, with suppression OS
3. Oculomotor dysfunction of pursuits and saccades
4. Superior left field quadrantanopia defect OD and left hemianopia OS

Coordination with other disciplines:

CC was released from physical therapy prior to her first assessment. I recommended that she continue to be followed by her neurologist, cardiologist, primary care physician, and ophthalmologist. Initial testing results and progress results throughout the neuro-rehabilitation treatment process were sent to all professionals involved in CC's care.

A comprehensive consultation took place after CC's initial assessment to educate CC and her husband about her conditions, the recommended treatment plan, and home/lifestyle recommendations. Specifically, CC's husband was educated about how he could best support CC's visual field defect in her personal, peri-personal, and extra-personal spaces. Specifically, with regard to CC's personal

Table 2: Diagnostic Data from Initial Assessment

REFRACTIVE STATUS
Unaided Distance Visual Acuity:
OD 20/50 full Snellen chart, OS 20/25 full Snellen chart (slow and laborious)
Corrected Near Acuity (cc habitual readers +2.50 OU):
OD reduced Snellen (RS) 80 full Snellen chart, OS RS 30 full Snellen chart (slow and laborious)
Autorefraction: OD +1.00-1.75x055, OS +0.25-0.50x132
Retinoscopy: OD +1.25-1.75x055, OS +0.25-0.50x130
Subjective Refraction:
OD +1.25-1.75x060 Distance Visual Acuity: 20/20 Near Visual Acuity: RS 20 Add: +2.50
OS +0.50-0.50x135 Distance Visual Acuity: $20/30^{+2}$ pinhole no improvement Near Visual Acuity: RS 30 Add: +2.50
Keratometry: OD 45.50 x 44.50 @ 051, OS 44.75 x 42.00 @ 007

BINOCULAR VISION
Cover Testing: Distance: no movement/ortho Near: intermittent alternating exotropia, 90%, >OS, 12-14 prism diopters/6 exophoria
Near Point of Convergence: 7/8 inches OS out, no diplopia reported
Comitancy Testing, Near Cover Testing 9 fields: Unilateral: intermittent alternating exotropia, 90%, >OS, 12-14 prism diopters; Alternate: 6 exophoria
Motor Fusion:
Von Graefe Phoria Distance and Near: constant OS suppression
Positive and Negative Vergence Ranges Distance and Near: constant OS suppression
Vergence Facility: suspect possible constant OS suppression
Sensory Fusion:
Worth 4 Dot: flat fusion at distance, intermediate, and near (pulled head away at 5 inches-repeatable) ranges with no movement on unilateral cover test
Stereo Acuity Testing: appreciation of 1 out of 4 on 500 seconds of arc Randot stereo, 40 seconds of arc on Wirt circles

OCULOMOTOR
Gross Pursuits Monocular and Binocular: full and smooth with 1+ head movement
Gross Saccades Monocular and Binocular: 90% gross undershoots, 1+ head movement
Visagraph Testing: Increased number of fixations, regressions, and duration of fixation; decreased span of recognition, words per minute, and comprehension OD, OS
Developmental Eye Movement (DEM) Test: Horizontal 28th percentile, Error >60th percentile (based on ceiling age of 13.11 years)

LENS/PRISM TRIALS
Trialed subjective refraction for distance and near, CC preferred her habitual readers at this time.
Trialed base-in prism in 2-6 prism diopter ranges, CC felt discomfort with prism in place.
Trialed yoked prisms base left in 2-15 prism diopter ranges, CC felt discomfort with prism in place.

OCULAR HEALTH
Pupils: Equal, round, reactive to light with no afferent pupillary defect
Confrontation Fields (single presentation):
OD: reduced accuracy and appreciation in inferior left and right quadrants
OS: reduced accuracy and appreciation in left superior and inferior quadrants
Anterior and Posterior Segment: Unremarkable other than posterior chamber intraocular (PCIOL) lenses clear OD, OS and 1+ vitreous degeneration OD, OS dilated fundus examination included OD, OS
Noncontact Tonometry: 15 mm Hg OD, 14 mm Hg OS
24-2 supra-threshold visual field test (1 gtt Proparacaine 0.5%, 1 gtt Tropicamide 1% OU): Absolute superior left field quadrantanopia defect OD and left superior and inferior temporal hemianopia OS (showing absolute defect in superior quadrant and dense defect inferior)
Humphry visual field 30-2 test (from exam with ophthalmologist): Absolute superior left field quadrantanopia defect OD and left superior and inferior temporal hemianopia OS (showing absolute defect in superior quadrant and dense defect inferior; Figure 1)
USI Testing:
Clock Test: 100% completion, filled in clock counter-clockwise
Line Bisection Cross Out Test: CC bisected all lines in the middle section. Note: CC completed upper-right quadrant first, lower-right quadrant second, lower-left quadrant third, and upper-left quadrant last.

space, CC's husband was cautioned about her reduced balance and depth perception and was informed specifically to refrain from turns in their line-dancing class and to stay beside her while walking on stairs, uneven surfaces, and curbs. Likewise, in relation to peri-personal space, he was educated to assist in dissipating any inattention of CC's visual field by engaging her in joint attention to her left field, reinforcing scanning into the left field, and also being

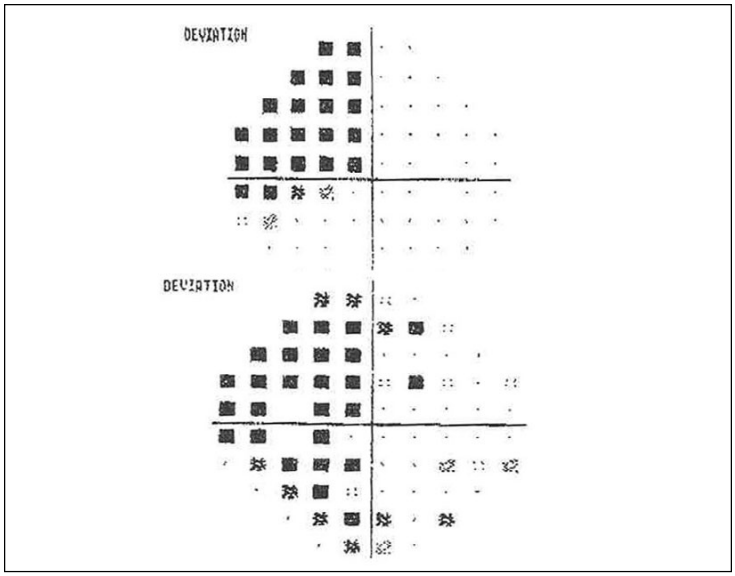

Figure 1. *Pre-therapy visual field (top OD, bottom OS)*

aware of her left field of vision (e.g., reminding her to finish food on the left side of her plate, to apply make-up to the left side of her face, etc.). Lastly, in relation to extra-personal space, he was instructed to use verbal cues when entering a room or field of view from her left side. He was cautioned about safety regarding this concern (e.g., cooking together in the kitchen) and CC's anxiety about being startled by unexpected appearances in her left field.

Treatment Plan

New visual space

Goals as reported by CC included: to enjoy reading again, to increase awareness of her left field of vision, and to improve balance (specifically for the capability to perform turns in line-dancing again).

Doctor's goals included: achieving the patient's goals by eliminating/minimizing presenting signs and symptoms, increasing her left visual field awareness and function, heightening vestibular function, enhancing focal-ambient processing, improving best-corrected visual acuity at near and distance of the left eye, eliminating exotropia, enhancing stereopsis, developing accurate and efficient saccadic and pursuit abilities, and enhancing visual processing with specific focus on visual memory, visual spatial, and processing speed.

The prognosis through a neuro-optometric rehabilitation program for the above-mentioned patient and doctor goals was overall fair for the reasons outlined below.

Visual Field Influencing Factor:

Improved functionality and awareness of CC's left visual field showed adequate prognosis as CC showed a strong cognitive ability and appeared to respond favorably to scanning techniques and compensating strategies. She also showed minimal signs of USI at the initial assessment. Proposed prognosis of adaptation to spectacle prism training (i.e., Fresnel sector prism on left lens base out) was likewise deemed favorable. CC and her husband were clearly educated about the set goals and expectations with regard to the visual field defect. Prior to initiation of the treatment plan, it was concurred that the neuro-optometric rehabilitation plan would aim to improve functional vision post-insult to obtain stabilization, organization, and awareness of her peripheral vision. CC and her husband were educated that the rehabilitation program would aim to improve performance in activities of her daily living and give improvements in her presented signs/symptoms rather than actual visual field recovery. CC and her husband were informed that actual visual field recovery is modest (averaging 5-7 degrees in hemianopia),[1,8] and they were shown specifically that if any modest recovery occurred, it would take place at the gradient inferior border of the inferior left field defect.

Strabismic Influencing Factors:

It was suspected (however unknown) that the exotropia was acute-onset secondary to the neurological insult of the CVA. Exotropia and high exophoria have been found to be comorbid with ABI and associated with imbalances in focal and ambient processes.[6] Flat fusion was obtained, illustrating possible acute standing, as suppression typically ocurs

over time. The exotropia was also intermittent, and suppression was only noted during phorometry (phoria and fusional measures), which may have posed a higher convergence demand and further imbalance of the focal-ambient processing. Furthermore, the favorable prognostic characteristics of her strabismus included concomitancy, small magnitude, exo direction, intermittent frequency, and normal correspondence.

Age-related Influencing Factors:

CC's age did not pose a significant prognosis risk, as she possessed high cognitive function. She lived in a private residence with her supportive husband who adequately provided transportation. It was expected that CC would have a good prognosis with vision rehabilitative therapy technique, application, and compliance.

Neuro-Optometric Rehabilitation Plan

Inter-disciplinary Approach

CC was compliant with my recommendations to continue to be followed routinely by her neurologist, primary care physician, and ophthalmologist. I also recommended that CC continue with physical and occupational therapy; however, CC insisted that she was dismissed with goals obtained in acute care.

Lenses

CC was opposed to using glasses even during vision rehabilitative therapy sessions other than her over-the-counter readers. I recommended that CC receive two single vision prescription lenses (versus progressives, which might exacerbate her disorientation), one for near and one for distance. CC was also educated that single vision lenses increase accuracy and stability for the proposed use of Fresnel prism trials. It is typical for individuals affected by an ABI to show elevated responses to subtle refractive error corrections to further support stability of the focal-ambient process change. CC also had a reasonable amount of refractive error in her right eye and showed significant visual acuity improvements in the right eye in the distance and near with the corrected prescription in place as compared to her current habitual system of unaided acuities for the distance and readers at near. CC deferred the recommended change to her habitual system. Furthermore, it was recommended that CC try a sector Fresnel prism base left on her left lens at minimum during vision rehabilitative therapy sessions in which prism lens adaptive training would be administered. During initial prism trials of small amounts of base-in prism (2-6 pd) to assist with her exophoric posture and focal-ambient processing, and during trials of full base left yoked prisms (2-15 pd) and sectoral base left prism (2-15pd) on the left eye, CC felt that the prisms were discomforting and deferred their application.

Vision Rehabilitative Therapy

CC was prescribed 24 in-office weekly sessions with prescribed home activities encompassing compensatory oculomotor scanning training, sensory-motor integration therapy, and development of deficient binocular and oculomotor skills, in addition to improving the visual processing skills of visual spatial relations, visual memory, and processing speed.

Phase 1

This phase emphasized the development of monocular fixation and transfer to accurate gross pursuit movements. Activities included star fixation, straw and pointer, visual-motor forms, Marsden ball tracking, rotator board, Wayne saccadic fixator board, computer perceptual therapy (CPT) pursuits, Sanet vision integrator (SVI), and Neuro Vision Rehabilitator (NVR). Sweeping gross eye movements into the left field and inferior left field were emphasized in pursuit training with controlled head and body movement with and without prediction. In addition, compensatory oculomotor scanning

training with strategizing and target populated left field technique (on SVI, NVR, Wayne saccadic fixator board) was implemented. Stimulation of convergence abilities with suppression checks began with instrumentation such as bead and string and 3-dot card. Peripheral awareness training at near with activities including ET touch and MacDonald cards was prescribed. Visual spatial processing development was prescribed to aid in organizational processing of her altered visual spatial schema status-post her visual field defect. These activities included bilateral integration (Randolph shuffle, chalkboard circles, etc.) right-left hand identification, and sequencing in eye-hand coordination tasks (e.g., bean bag toss, SVI, Wayne saccadic fixator board). Rehabilitation of body image, directionality, visual spatial midline, and visual spatial relations are necessary to reestablish matches between the perceived space and physical reality in conditions of visual field defects or USI.[1]

Progress Evaluation 1 (concluding 8 in-office sessions):

Per CC, she had improved in the following signs and symptoms: rereading lines, skipping words, use of finger to keep place for reading, balance, and posture (Table 3). She noted that although improved, she still experienced some dizziness and disorientation and had no noticeable improvement in peripheral vision. Pertinent objective measures are shown in Table 4, which illustrate her improved distance and near visual acuity of the right eye, DEM performance, and near point of convergence. She also eliminated her intermittent alternating exotropia, head movements during gross pursuits, and gross undershooting during gross saccades. CC was educated about trialing of the sector prism at minimum during vision rehabilitative therapy sessions and again deferred. CC was also educated about the test results and the treatment modifications in her vision rehabilitative therapy program.

Table 3: Symptomatology and Activities of Daily Living Progression

Initial Assessment
No longer enjoys reading
Frustrated because she does not see things that her husband informs her are right in front of her
Often startled as her husband would appear immediately before her and she could not see him approaching
Strong desire to line-dance again, especially to turn without losing her balance and feeling disoriented

First Progress Evaluation (concluding 8 sessions)
Improvements in reading (rereading lines, skipping words, and using finger to keep place less)
Balance and posture improving, still experiencing some dizziness and disorientation
No noticeable improvement in peripheral vision

Progress Evaluation 2 (concluding 16 sessions)
Improvment in reading skills
Improvment in balance abilities
More stable in line-dancing but still gets dizzy when performing a turn

Progress Evaluation 3 (concluding 24 sessions)
Improved side vision, proudly demonstrated her awareness of both of her thumbs simultaneously with her arms stretched out to her sides
Enjoying reading again
All presenting signs and symptoms improved, except difficulties with finding objects that she reported were directly in front of her
Memory skills still reduced

2-month Follow-up Evaluation (completed 24 in-office sessions, moderate compliance with prescribed home maintenance program)
Improved balance, could now perform some turns when line-dancing
Visual field remained intact, no longer surprised by her husband "popping up" in her left visual field
Reading and cooking easily
Still struggled to find things (e.g., her watch sitting on her dresser or calendar which had been propped up on wall, rather than lying flat)

Phase 2

Binocular fixation and binocular fine pursuit development ensued with integration of vestibular work on the NVR balance board and standard balance board. Gross saccadic activities were implemented (mastered monocularly and then binocularly). Activities included Hart chart saccades and Track and Read saccades. Voluntary convergence and positive fusional ranges were developed (e.g., tranyglyphs, vectograms, eccentric circles, and Vision Therapy System 3 (VTS3)). Peripheral awareness training extended to intermediate and distance ranges with emphasized work on SVI and NVR programs and included transfer to movement through space (e.g., bean bag toss to peripherally placed targets while walking on walking rail). Visual spatial processing development ensued with focus on directionality activities (e.g., floor map, CPT directionality, stick-tap, etc.); visual memory and visual sequencing processing development was incorporated per CC's complaint of affected memory skills post stroke and included memory activities on Visual Information Processing Skills (VIPS) and multi-matrix.

Progress Evaluation 2
(concluding 16 in-office sessions):

Symptomatology review (Table 3 outlines progression) illustrated that CC was still improving in her reading and balance abilities. She was more stable in her line-dancing but still got dizzy when performing a turn. Table 4 illustrates pertinent objective measures and shows improved unaided distance visual acuity of the right eye, near point of convergence, depth perception, and confrontation field performance.

Phase 3

Emphasis was on fine saccadic activities with processing speed, metronome integration, and cognitive loading (e.g., symbol tracking sequences, spell tracking, percon saccades, etc.). Advanced scanning activities ensued with concentration on left field and integration of processing speed. It has been proposed that for best outcome, processing speed and span of recognition should be maximized along with scanning training.[1] Tachistoscopic and span of recognition development were included through related activities on SVI, CPT, and Track and Read programs. Peripheral awareness with dynamic free space movement and vestibular components were integrated (e.g., windmill finger tracking, wall-spotting turns, step-turn sequences). Positive fusional step vergence, jump ductions, and divergence abilities were developed with movement and peripheral awareness as applicable (e.g., vectograms walking, distance eccentric circle chart walk-aways, etc.).

Progress Evaluation 3
(concluding 24 in-office sessions):

CC ecstatically reported improved side vision and proudly demonstrated her awareness of both of her thumbs simultaneously with her arms stretched out to her sides. She noted that she was enjoying reading again. All presenting signs and symptoms had improved except that CC was still concerned with her difficulties with finding objects that she reported being directly in front of her. She also noted that her memory skills were still reduced. Table 3 shows symptomatology progression. CC noted excessive fatigue during testing due to lack of sleep the night prior, which may support resurfacing of exotropia, although it presented with significantly reduced frequency and magnitude and a shift to unilateral as compared to initial testing. She showed improvement in depth perception and fusional facility. CC deferred the visual field testing recommended. Sectoral and bi-nasal prism trials were administered and again deferred by CC. Maintenance home activities were prescribed, including red and green far-near chart tracking, MacDonald card, and physiological diplopia

Table 4: Key Findings Pre- and Post-Treatment

	Baseline (Pre-Treatment)	Progress Evaluation 1 (Concluding 8 sessions)	Progress Evaluation 2 (Concluding 16 sessions)	Progress Evaluation 3 (Concluding 24 sessions)	Follow-up assessment (2 months post-treatment)
Visual Acuity Unaided (distance)	OD: 20/50 OS: 20/25	OD: 20/40 OS: $20/30^{-2}$	OD: $20/30^{-1}$ OS: $20/30^{-2}$	OD: $20/50^{+2}$ OS: $20/40^{+2}$	OD: $20/40^{+2}$ OS: $20/25^{-}$
Visual Acuity Corrected (near)	OD: RS 80 OS: RS 30	OD: RS 30 OS: RS 30	OD: RS 30 OS: RS 30	OD: RS 25^{-2} OS: RS 25^{-1}	OD: RS 20^{-2} OS: RS 20^{-2}
Cover Testing (distance)	No movement/ortho	No movement/ortho	No movement/ortho	No movement/ortho	No movement/ortho
Cover Testing (near)	Intermitttent alternate exotropia, 90%, >OS 12-14 pd/6 XP (equal in 9 fields of gaze)	No movement/6-8 XP (equal in 9 fields of gaze)	No movement/6 XP (equal in 9 fields of gaze)	Intermittent left exotropia, <5%, 4-6 pd/4xp (equal in 9 fields of gaze)	No movement/4xp (equal in 9 fields of gaze)
Near Point of Convergence	7/8" OS out, (-) diplopia	3/4" OS out, (-) diplopia	2/3" (+) blur	2/3" OS out, no diplopia reported	4/6" OS out, no diplopia reported
Worth 4 Dot	Flat fusion at distance, intermediate, and near (pulled head away at 5 inches-repeatable) ranges with no movement on unilateral cover test	Flat fusion at distance, intermediate, and near with no movement on unilateral cover test	Flat fusion at distance, intermediate, and near with no movement on unilateral cover test	Flat fusion at distance, intermediate, and near with no movement on unilateral cover test	Flat fusion at distance, intermediate, and near with no movement on unilateral cover test
Gross Pursuits (monoc and binoc)	Full and smooth, 1+ head movement	Full and smooth, no head movement	Full and smooth, no head movement	Full and smooth, 1+ head movement	Full and smooth, no head movement
Gross Saccades (monoc and binoc)	90% gross undershoots, 1+ head movement	Accurate, no head movement	Accurate, no head movement, reports "dizzy"	20% mild undershoots to CC's left field, no head movement	Accurate, no head movement
DEM Test (based on ceiling age of 13.11 years)	Horiz: 28th percentile Error: >60th percentile	Horiz: 60th percentile Error: 60th percentile	Horiz: 60th percentile Error: 60th percentile	Not tested	Not tested
Stereo Acuity Testing	1/4 500", 1/4 250" Randot Stereo, 40" Wirt Circles	Not tested	4/4 500", 1/4 250" Randot Stereo, 20" Wirt Circles	4/4 500", 4/4 250" Randot Stereo, 20" Wirt Circles, 100" Animals	3/4 500", 3/4 250" Randot Stereo, 20" Wirt Circles, 100" Animals
Confrontation Fields	Reduced accuracy and appreciation of inferior left and right quadrants, reduced accuracy and appreciation on patient's left superior and inferior quadrants	Single presentation: full to finger count OD, OS	Single presentation: full to finger count OD, OS	Single presentation: full to finger count OD, OS	Single and simultaneous presentation: full to finger count OD, OS
Fusional Facility	Suspect possible constant OS suppression	Not tested	Not tested	8 cpm	Not tested
Visual Midline (spatial localization)	Not tested	Not tested	Not tested	Not tested	Mild consistent visual midline shift to her right with no significant response to 2-10 pd trials yoked prism base left
Humphrey Visual Field 30-2	Absolute superior left field quandrantanopia defect OD and left superior and temporal hemianopia OS	Absolute superior left field quadrantanopia defect OD and left superior and resolving temporal hemianopia OS (absolute defect in superior quadrant and possible rise of edge border in inferior defect)	Not tested	Not tested	Not tested
Easygraph 24-2 (Sup Thr2) Visual Field	Absolute superior left field quadrantanopia defect OD and left superior and inferior temporal hemianopia OS (showing absolute defect in superior quadrant and dense defect inferior)	Superior left quadrant-anopia OD and decreased size of left hemianopia inferiorly OS (Figure 2)			

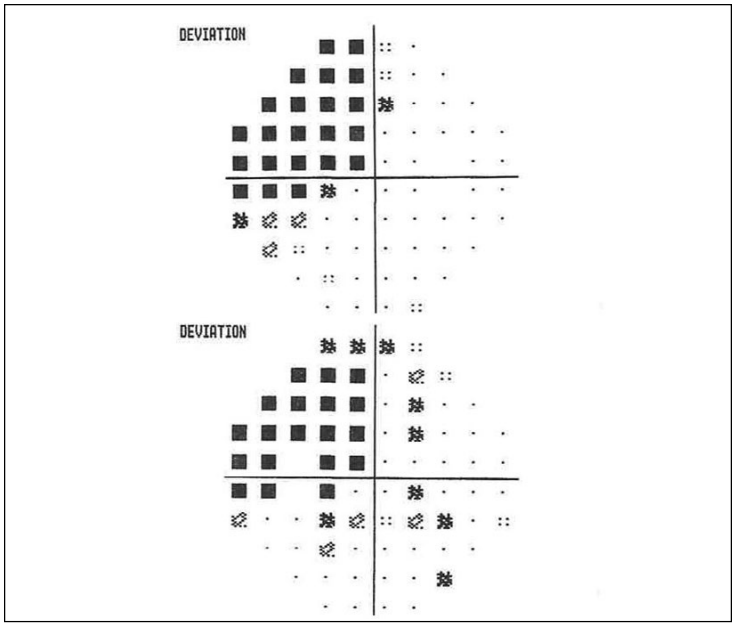

Figure 2. *Post-therapy visual field (top OD, bottom OS)*

stick facility. CC was instructed to return in 2 months for a follow-up assessment. She was educated on the importance of compliance with the home maintenance program to prevent regression of developed skills. CC was instructed on the importance of continuing to be monitored by her neurologist, cardiologist, primary care provider, and ophthalmologist.

2-month Follow-up Assessment (completed 24 in-office sessions, moderate compliance with prescribed home maintenance program):

Symptomatology review (Table 3 shows progression) revealed that CC's balance had improved and she could now perform some turns when line-dancing. She reported that her visual field remained intact, and she was no longer surprised by her husband "popping up" in her visual field. CC reported that she was now reading and cooking easily; however, she felt like she still struggled to find things (e.g., her watch sitting on her dresser or a calendar which had been propped up on wall, rather than lying flat).

Table 4 shows pertinent objective data which reveals improved near acuity for both eyes and slightly regressed near point of convergence with no exotropia found. Threshold visual field testing was deferred by CC. Visual midline (spatial localization) testing showed mild consistent visual midline shift to her right with no significant response to yoked prism base left. CC was educated to continue the maintenance home program and to return for a follow-up assessment in 4-6 months (which will include threshold visual field testing). CC was also educated on the importance of repeating visual fields in order to monitor the defect and, if needed, to make any modifications to her treatment plan. She was educated to continue to be monitored by her neurologist, cardiologists, primary care physician, and ophthalmologist. Lastly, she was instructed to continue to utilize scanning strategies and to take safety precautions related to her visual field defects.

Summary

Outcome

The neuro-optometric treatment plan met the goals of both the doctor and the patient, in which CC's functional vision improved. CC was especially elated, expressing extreme excitement that she could appreciate her left visual field and could enjoy reading and line-dancing again.

Diagnostic Reflection

Overall, this case followed expectations with regard to prognostic factors assessed. The visual field gradient border of the left inferior temporal field of the left eye resolved slightly as anticipated, and CC's awareness of her left field significantly improved. Although the initial presenting signs and symptoms and USI testing performed did not yield a manifestation of USI, extinction confrontation fields were not performed; therefore, CC's marked improved awareness of her left field is difficult to gauge. In future related cases, more detailed assessments of USI will be performed at the initial testing, with specific emphasis on the individual's behaviors and conventional and extinction visual field tests, which are key factors in the diagnosis.[2] For

further assessment of suspect USI, future history intakes may include the following question: Does it ever seem like one side of your world is missing? CC answered that she did not bump into things when walking and that she did not just eat half of the food on her plate, while her husband said otherwise (varied responses between individuals and family members can be consistent with USI).[4]

Amsler grid testing would have been helpful to measure the extent of a suspected visual defect in relation to central vision, which would have given more functional insight on near tasks such as reading for CC.[4] In addition to providing insight into interventions for reading and writing, Amsler grid testing could have assisted in the performance expectations for search tasks by detecting visual distortions within the macular area and to serve as a measure of the proximity of the hemianopia edge to fixation.[1]

CC noted a significant improvement in her balance abilities, and while the oculomotor assessments of fixation, pursuits, and saccades provided insight into the integrity of the vestibular system, it would have been beneficial to monitor more specific measures of vestibular function. Future related testing may include dynamic acuity, Romberg testing, and routine gait/posture observation. Also, it would have been beneficial to refer CC to an orientation and mobility expert to provide further care.

In retrospect, a baseline of entering visual processing skills would have helped objective measures of progress in this area and would have aided in allowing specific direction of the development of processing skills in the treatment plan. Specific to this case, measures of tachistoscopic skills and span of recognition, processing speed, visual spatial relations, directionality, visual memory, and sequential memory would have assisted the treatment plan direction and progress monitoring. With regard to CC's complaint of difficulty locating objects amongst other objects/backgrounds and in different positions, measures of figure ground, spatial relations, and form constancy would have best assisted her care.

Treatment Reflection

In future related cases of visual field loss, more extensive patient and family education will be performed to highlight the importance of various prism trials for overall success of the neuro-optometric rehabilitation program. Recovery from an ABI can be aided by various uses of prisms.[19] Moreover, future case management will include prism trials outside of the exam room instead of solely in the vision rehabilitative therapy setting, with an emphasis on active closed-loop prism adaptation work with yoked prisms and movement.[19] Furthermore, with regard to prescribed prism for outside-clinic use, more education will transpire to ensure that the patient understands the basis of the prism application and safety while wearing the prism, including a review of the induced blind spot, diplopia, and image parameters while viewing through the prism. Also, specific prism scanning training will be outlined with future cases beginning with static modality mastery to advancement of dynamic modality.

With regard to CC's concerns about balance, it would have been beneficial to prescribe bi-nasal occlusion to determine whether increased focal-ambient processing may have further aided the progress of her balance abilities. Furthermore, the integration of trials of small-magnitude base left yoked prism into the vision therapy rehabilitation sessions may have also improved balance by shifting the visual midline. In addition, co-management of the balance concerns with occupational and physical therapy treatment plans may have also enhanced gains, especially with appropriate therapeutic lenses in place during those therapies.

Conclusion

A neuro-optometric rehabilitation plan yielded a successful outcome of improving functional vision following a CVA causing visual field defect. Diagnostic scrutiny of visual field defect-related conditions, such as visual midline shift, USI, and focal-ambient integrative processing dysfunction, are crucial for the development of the treatment plan and for monitoring objective progress throughout treatment. Integration of prism into the vision rehabilitative therapy sessions and extensive patient education of applicable prismatic lens options is imperative to achieve maximized benefits from the neuro-optometric rehabilitative treatment plan.

References

1. Sutter P, Margolis N. Managing visual field defects following acquired brain injury. Brain Inj Professional 2005;2(3):8-10.
2. Gall C, Franke GH, Sabel BA. Vision-related quality of life in first stroke patients with homonymous visual field defects. Health Quality of Life Outcomes [PMC2859371]. v.8; 2010 PMC2859371 http://bit.ly/1NlOOpQ
3. Papageorgiou E, Hardiess G, Schaeffel F, Wiethoelter H, et al. Assessment of vision-related quality of life in patients with homonymous visual field defects. Graefes Arch Clin Exp Ophthalmol 2007;245(12):1749-58. http://1.usa.gov/1SXUR5B
4. Suchoff I. The diagnosis of visual unilateral spatial inattention. Brain Inj Professional 2005;2(3):21-4.
5. Sarri M, Greenwood M, Kalra L, Papps B, et al. Prism adaptation aftereffects in stroke patients with spatial neglect: pathological effects on subjective straight ahead but not visual open-loop pointing. Neuropsychologia 2008;46:1069-80. http://1.usa.gov/1TLT6IE
6. Padula WV. Neuro-optometric rehabilitation. In: Padula WV, ed. Post-trauma Vision Syndrome Caused by Head Injury. Santa Ana, CA: Optometric Extension Program Foundation, Inc., 2000:179-93.
7. Chechlacz M, Rotshtein P, Humphreys G. Neuroanatomical dissections of unilateral visual neglect symptoms: ALE meta-analysis of lesion-symptom mapping. Front Hum Neurosci 2012 Aug 10;6:230. doi: 10.3389/fnhum.2012.00230. eCollection 2012. http://1.usa.gov/1jYURG4
8. Margolis N. Evaluation and Treatment of Visual Field Loss and Visual-Spatial Neglect. In: Suter P, Harvey L, eds. Vision Rehabilitation Multidisciplinary Care of the Patient Following Brain Injury. Boca Raton, FL: CRC Press, 2011:153-87. http://amzn.to/1ltitfV
9. Suter P, Hellerstein L, Harvey L, Gutcher K. What is Vision Rehabilitation Following Brain Injury. In: Suter P, Harvey L, eds. Vision Rehabilitation Multidisciplinary Care of the Patient Following Brain Injury. Boca Raton, FL: CRC Press, 2011:1-27. http://amzn.to/1ltitfV
10. Kochanek K, Xu J, Murphy SL, Miniño A, et al. Deaths: final data for 2009. Nat Vital Stat Rep 2011;60(3). http://1.usa.gov/1OsxL35
11. Scheiman M, Wick B. Binocular and Accommodative Problems Associated with Aquired Brain Injury. In: Scheiman M, Wick B, eds. Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders. Philadelphia, PA: Lipponcott Williams & Wilkins, 2008:578-99. http://bit.ly/1NlzYQa
12. Cdc.gov. Georgia: Center for Disease Control and Prevention [updated 2012 November 14]. Available from: http://www.cdc.gov/stroke/about.htm.
13. Cdc.gov. Georgia: Center for Disease Control and Prevention [updated 2012 November 14]. Available from: http://1.usa.gov/1NqSnfM.
14. Strokeassociation.org. 2014 Texas: Stroke Association. Available from: http://bit.ly/1TvUEa6.
15. Pearn J, O'Connor RJ. Community stroke rehabilitation helps patients return to work. Practitioner 2013;23(7):2-3. http://bit.ly/1O5EIN6
16. Rowe F, Wright D, Brand D, Jackson C, et al. Profile of gaze dysfunction following cerebrovascular accident. ISRN Ophthalmology 2013, Article ID 264604, 8 pages. http://bit.ly/1NHT1Y1
17. Stroke.org. 2012 National Stroke Association [updated 2012 August]. Available from: http://bit.ly/1XHCrHf.
18. Rowe F, Wright D, Brand D, Jackson C, et al. A prospective profile of visual field loss following stroke: prevalence, type, rehabilitation, and outcome. [PMC3782154]. Biomed Res Int. 2013; 2013: 719096. Published online 2013 Sep 9. doi: 10.1155/2013/719096. http://1.usa.gov/1O5ENk8
19. Harris P. The Use of Lenses to Improve Quality of Life Following Brain Injury. In: Suter P, Harvey L, eds. Vision rehabilitation multidisciplinary care of the patient following brain injury. Boca Raton, FL: CRC Press, 2011:213-81. http://amzn.to/1ltitfV

Correspondence regarding this article should be emailed to Briana Larson, OD at dr.larson@ocvt.info. *All statements are the author's personal opinions and may not reflect the opinions of the representative organizations, ACBO or OEPF, Optometry & Visual Performance, or any institution or organization with which the author may be affiliated. Permission to use reprints of this article must be obtained from the editor. Copyright 2015 Optometric Extension Program Foundation. Online access is available at* www.acbo.org.au, www.oepf.org, and www.ovpjournal.org.

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Article ▸ Multi-Sensory Factors When Examining Visual Fields in Unilateral Spatial Inattention and Its Effects on Treatment

Tonya Marie Smith Umbel, OD, Culpeper, Virginia

Curtis R. Baxstrom, OD, Federal Way, Washington

ABSTRACT

Unilateral spatial inattention (USI) is sometimes challenging to differentiate from true hemianopsia. Visual field testing can appear to be variable and inconsistent. The results of field testing may be conflicting depending on the method used. Factors to consider are the presentation method of targets, dual presentation, differences in target size, and other variables demanding increased attention. It is also important to examine the multi-sensory influence on the performance of the patient based on the neural pathways being utilized in each task. The following is a discussion of evaluation techniques of visual fields in patients who have suffered an acquired brain injury. Further, there will be a case example of an individual diagnosed with a right middle cerebral artery stroke and secondary visual field defect who presented with unique visual field results. Knowledge gained from comparison of these procedures may affect treatment. For example, a patient may show no signs of USI while sitting but exhibit USI when asked to stand. This individual may demonstrate visual field difficulties when attending to anything involving movement and balance. This unique aspect of processing of vision from a multi-sensory model can make treatment for such individuals more efficient, well-defined, and effective. This information will be essential for multi-disciplinary interactions to create better treatment strategies among rehabilitation disciplines. The procedures to be evaluated will be confrontation fields, static automated perimetry, and tangent screen fields under varied circumstances and test conditions to optimize treatment and to maximize performance of daily living activities.

Keywords: brain injury, evaluation techniques, rehabilitation, unilateral spatial inattention, visual field

Background

Visual information begins at the gathering of stimuli at the retina. This information comes down the optic nerve, and the information is sent to four areas of the brain: the lateral geniculate nucleus, the superior colliculus of the midbrain, the pretectum of the midbrain, and the suprachiasmatic nucleus of the hypothalamus. Information received by the superior colliculus is used in the control of eye movements. Information sent to the pretectum is used in pupillary reflex mechanisms. Information to the suprachiasmatic nucleus of the hypothalamus is used for diurnal rhythms and hormonal responses. Last, the majority of neural information is taken to the lateral geniculate nucleus (LGN) for visual perception.[1]

The LGN interprets visual information in segregated monocular form. It separates the information into three tracts of interpretation: magnocellular, parvocellular, and koniocellular. The magnocellular tract has large center/surround receptive fields, which detect movement. The parvocellular tract has small

center/surround receptive fields, is sensitive to color, and discriminates shape, form, and contrast. The koniocellular tract has very small concentric receptive fields, which are very strong in detailed color discrimination. All of these LGN fibers travel along the optic radiations and terminate in the striate cortex.[1]

The striate cortex, also known as the primary visual cortex (V1), analyzes the data coming from the magnocellular, parvocellular, and koniocellular pathways, on/off receptive fields, directional orientation, ocular dominance, and directional dominance.[2] The koniocellular pathway is interpreted by V1 blob cells. These cells process information in a monocular process, determining color. They maintain the concentric receptive fields as in the LGN and are found in clusters. These cells also obtain information from parvo cells for color perception and discrimination and are responsible for the memory of object colors. A second type of cells in V1 are the interblob cells. These interpret binocular information via elongated rectangular receptive fields, which detect orientation and ocular dominance. These cells are clustered around the blob cells. Parvocellular information is interpreted here to understand object perception and discrimination, spatial orientation, form, and shape. Magnocellular information is interpreted with subsets of interblob cells, which detect motion, direction of motion, and velocity of object to guide in eye movements. If V1 is damaged, it causes complete blindness.[1]

After this visual information is interpreted in the striate cortex, it divides into areas of the extra-striate cortex for more detailed interpretation. Damage to any of these areas causes problems with visual information processing, such as issues with identifying color, shape, location, or motion.[1] These areas send information to the visual association cortex, located in the parietal and temporal lobes. Here, visual information is separated into the dorsal ("where") and ventral ("what") streams of information.

The dorsal stream is found in the parietal association cortex and the superior/middle temporal association cortex. Information is interpreted in binocular receptive fields. Object location and movement help to determine spatial orientation, depth perception, and velocity of objects. Damage to this area causes deficits in spatial orientation, motion detection, and guidance in tracking eye movements.[1]

The ventral stream is located in the inferior temporal association cortex. This area is used in the recognition of objects and the ability to recognize and to interpret text (reading). Damage to this area causes issues in complex visual perception tasks that require attention and memory.[1] Although damage to V1 causes functional blindness, a secondary visual pathway composed of visual fibers to the pretectal nucleus of the superior colliculus creates a bypass of some 'visual' information. These fibers leave the pretectal nucleus and feed into the Edinger-Westphal nucleus and send connections to the frontal eye fields.[3] The frontal eye fields are responsible for saccadic function and react to motion. The lateral intraparietal area has also shown that it independently controls visual saccadic response and visual attention.[4] This motion processing can be observed in complete hemianopia and is hypothesized as the possible connection explaining the Riddoch phenomenon. This phenomenon occurs when motion is detected in an otherwise blind field.[5] Studies using fMRI comparing a patient with known V1 damage to controls have shown that this is from direct activation of V5, V3, and the parietal cortex, although the exact mechanism still is not well understood.[6] Research has demonstrated that if V1 is removed in monkey models, the animals continue to respond to orientation, shape, brightness, size, and motion of stimuli, but this disappears if the superior colliculus is destroyed. Functional imaging has shown that remaining activity is present in all of

the extra-striate cortex and parts of the parietal lobe without the involvement of V1.[7] There have also been reports that there is direct subcortical input to V5, bypassing V1.[8] This supports the idea that the superior colliculus could be the link for "blind sight."

In the case of unilateral spatial inattention, it is not caused by problems with neurology of V1. Instead, it is a problem with attention processes. Attention is "the entire family of processes that mediate the choice of suitable mental or external events for consciousness and action."[9] Attention lies along a continuum of consciousness, which can be described as follows: arousal→ attention→ memory→ language→ praxis→ recognition→ visual-spatial recognition→ complex cognition. Bottom-up impairment includes issues with arousal and attention, which have a negative effect on the rest of the series.[9] Therefore, any damage to attention will also affect visual-spatial recognition, defined as "abilities involving visual processing skills, spatial awareness, self-object spatial relationships, visual spatial memory, and navigation of extra-personal space."[9] A disturbance in this spectrum causes unilateral spatial inattention. Characteristics of USI are deficits in "perceiving, attending, representing, and/or performing actions within" the left visual field.[10]

Defining which areas of the brain control attention is unclear. It is well accepted that USI is primarily a dysfunction of the parietal lobe, with issues also found from lesions in the cingulate gyrus, frontal eye fields, and brain stem.[11] Interestingly, USI only seems to manifest as an issue for right hemisphere lesions.[12] Experimentation on those with parietal lobe damage has shown that the process of engaging one object, releasing attention, and shifting to another object is controlled in the parietal lobe, and damage to this area causes neglect.[13] Agreement across neuropsychology demonstrates that cortico-subcortical fronto-parietal connections control spatial attention, and damage to these areas causes hemineglect in the contralateral field.[14] However, a study by Benson, et. al. has shown that this may not be the case. In a single case experiment with control, it was determined that USI was an imbalance in saccadic orienting, as well as a combination of inability to perceive information visually sampled and hyper-attention in the normal field.[15] This single case, however, is not significant enough to make any conclusions.

Table 1: Location of Cerebral Vascular Accidents in the Brain and Resulting Functional Defects

Major Artery Origin	Site of Cerebral Vascular Accident	Effect on Function
Internal Carotid Arteries	Anterior Cerebral Artery	Frontal lobe: possible USI, junctional defect, sector defect, superior defect, apraxia of gait, cerebral dementia, confused language
Internal Carotid Arteries	Left Middle Cerebral Artery	Hemianopsia, aphasia, apraxia, hemiplegia or hemiparesis, contralateral hypoesthesia, dysphagia
Internal Carotid Arteries	Right Middle Cerebral Artery	Hemianopsia, USI, prosopagnosia, hemiplegia or hemiparesis, contralateral hypoesthesia, dysphagia
Vertebro-Basilar Arteries	Posterior Cerebral Artery	Cortical blindness, alexia, visual agnosia, thalamic aphasia, ataxic (cerebellar) or flaccid (lower motor neuron) dysarthria
Circle of Willis	Anterior Communicating Arteries	Do not cause functional loss, can cause secondary flow issues due to other blocked arteries or incomplete circle (25% of population)

If an individual sustains an acquired brain injury, it is crucial to understand basic anatomy and how this affects the visual field so that it is easier to differentiate USI from true visual deficits. Based on the site of the brain injury, one can predict what visual outcome should occur. How a cerebral vascular accident affects vision can be seen in Table 1.[16]

Analysis of Visual Field Testing

There are many ways in which an individual's visual field can be tested, including confrontation, tangent screen, and automated perimetry (Tables 2 & 3). First, there are variables in target. In confrontations, one can use counting fingers, finger movement,

Table 2: Comparison of Visual Field Testing

Field Type	Positives	Negatives
Confrontation	Quick & Easy Picks up large defects (good for USI and hemianopsia) Can test static and kinetic	Does not detect small defects Background can affect testing (color of shirt, room)
Tangent Screen	Sensitive to small defects Accurate for brain localization	Time Only kinetic cues (arm, wand)
Automated Perimetry	Static Picks up small defects Computer analysis comparison to age normals	Can have tunneling (MIB effect) Kinetic programs not very good Poor accuracy in brain localization Defects are greater than they may actually be

or a confrontation wand. Variation in color is also used, commonly white or red targets. Counting fingers requires form recognition. Finger motion and confrontation wand are kinetic and incorporate motion detection, which could bypass V1 and be detected by extra-striate areas. This can be the case if the person is counting fingers and there is motion in presentation. In general, confrontation fields are completely unreliable for small defects and only find well-defined large defects.[17,18] Confrontation testing only seems to detect a defect about 38-50% of the time if it is not a defined defect such as an altitudinal defect or homonymous hemianopsia.[18,19] Regardless of this fact, it is still commonly used in vision care as a visual field screener. Because it does pick up large defects, such as hemianopsia and quadrantanopsia, it is perfectly acceptable as an initial test to identify a substantial loss.[20] This makes this test a quick, easy test for ABI patients and in visual neglect.

It has been cited that automated perimetry is more accurate in diagnosing neurological disease than kinetic perimetry, making static Humphrey perimetry the standard of care.[21] Humphrey specifically is more accurate than Matrix or FDT due to the fact that it is a solid flash of light as opposed to a frequency doubling image, which can induce a motion effect. It is more accurate because it will not activate motion sensing and bypass V1.[21] However, it is not as accurate in giving a true localization of the defect in the brain based on the field detected. A Humphrey visual field is more likely to show a more severe defect than is truly present compared to tangent screen fields and Goldmann perimetry.[22] Advantages are the precise repeatability of this testing method and the decrease in testing time. It also has the advantage of removing cues like a tangent rod.[23]

There are several issues with using the above-mentioned tests when dealing with determination of a unilateral spatial inattention vs. hemianopsia. Each element of discord is in relation to changes to attention based on the position of the individual, the task they are asked to perform, proprioceptive influences on performance, and various inter-related neurological issues. First, there is the question of the accuracy of the above-mentioned tests due to posture. If the patient is sitting, this is not a true gauge of how the person will perform during activities of daily living or when ambulatory in physical therapy and occupational therapy. When an individual stands, they must

Table 3: Results of Confrontation Visual Fields in Various Conditions

Condition	Static presentation of confrontation finger counting	Motion presentation of confrontation finger counting	Static dual presentation	Motion dual presentation	neck vibration/ vestibular input
Normal	Normal	Normal	Normal	Normal	Normal
Hemianopsia	Abnormal	Normal* or Abnormal	Normal* or Abnormal	Normal	No change
USI	Abnormal	Abnormal	Abnormal	Abnormal	Improved
Extinction	Normal	Normal	Abnormal	Abnormal	Improved

*Riddoch Phenomenon

attend to balance, and cognitive abilities must now be split between visual awareness and balance. Since balance is tightly intertwined with peripheral vision, it is harder for someone to attend to visual stimuli when using vision for balance, making USI more pronounced. A study on the effects of a cell phone conversation on visual field using Goldmann visual fields demonstrates this well. The investigators tested normal individuals with and without visual complaints and compared their visual fields to ones performed while on a cell phone. There was a significant decrease in visual field when attending to a phone conversation.[24] When this idea is translated to visual field testing, it would be advantageous to have the patient both sit and stand while being tested for confrontation visual fields. During USI, one is likely to show differences, while with a hemianopsia, the loss would be constant. However, some patients may have visual field loss coupled with USI, making diagnosis difficult.

Another aspect that needs to be considered is the MIB effect (motion induced blindness). MIB causes extinction of peripherally viewed objects when simultaneously viewing a central fixation point when these peripheral targets are presented in a pattern that causes apparent motion. The suppression of microsaccades changes the gain in the visual system and causes disappearance of the peripheral targets. A study by Gorea and Caetta[25] showed that this extinction of peripheral objects is not isolated to the MIB effect alone. In their experiment, they also used static mask and absent mask targets to test the extinction phenomenon and how it varied under these three conditions. They found that although the MIB is a much stronger extinction of the peripheral object(s) being viewed, there was also temporary suppression found in the other two conditions. An example of a visual stimulus for MIB would be a central target and 3 targets in a triangular configuration surrounding the central point, with a grid rotating over the field. The grid is a moving mask, which causes the 3 paracentral points to disappear while fixating the central point. In static mask, the grid does not move. In absent mask, there is no grid. These latter 2 conditions would mimic an automated visual field because of the extent to which the patient has to hyper-focus on a central fixation target. In clinic, some patients will complain of tunneling or darkening of vision while trying to do a visual test, and they have to blink excessively to bring back their peripheral vision. They are experiencing this variant of the MIB effect. This may be why the extent of visual field severity is usually more pronounced in automated static perimetry than in tangent screen fields.

There are also variations that can be made in proprioception that can be positive in the detection of visual fields. According to Riddoch and Humphreys,[26] there is a distinction between extinction and unilateral spatial inattention. This distinction is important to understand, as this would change the results of testing. Often, these are considered to be one and the same. If a patient is presented with bilateral targets, and one is extinguished consistently on the left side, this is labeled left neglect (USI). However, when presented with a single target on the same side as visual extinction, they may actually see the target. Also, with USI, there does not have to be a competing stimulus for the individual not to see the stimulus presented in the neglected field. The difference is in the attending ability of the brain and possible different 'anatomical substrates.'[26] In visual extinction, the patient may or may not attend to an object based on the circumstance in which it is presented. It has been found that in patients with a brain lesion, if presented with objects oriented for action, the patient always saw the active object instead of the passive object regardless of location in space. However, if the same pictures were presented without showing action toward each other, the patient would pick up the picture on the ipsilateral side. When presented with two objects that were the same, they did

not consistently extinguish the object on the contralateral side. An example was presentation of handled cups. If the handle was turned to the left (contralesional side), the patient was much more likely to attend to the object because it engaged motor planning on the left side. They also found that motor planning that required attention to the ipsilesional side and then disengagement to attend to the contralesional side showed performance benefit.[26] This is the complete opposite of USI, which has difficulty disengaging attention from the ipsilesional side.[13]

Patients with USI sometimes show signs of varied symptoms based on whether the targets are in peri-personal space (arm's reach) or extra-personal space. Patients who perform tests in peri-personal space can show neglect while having normal responses in extra-personal space.[27] However, when the person wields a tool, this makes that extra-personal space become part of the peri-personal space schema, and the neglect reappears.[28] The tool is detected as part of the body, which expands the peri-personal space. An example would be how an individual becomes aware of the extent of his or her vehicle on the road when driving. The car becomes part of the peri-personal space. Another example would be a baseball player knowing where the bat is in space when playing baseball. This evidence shows that neglect may not just be attention but an alteration of body schema. Maravita and colleagues suspect that wielding of tools relates to properties of intra-parietal neurons.[29] Considering that neglect comes from damage in the parietal lobe, this would coincide with the idea that if something is in the peri-personal space, it requires use of the parietal lobe and would exhibit neglect; however, outside this area would not be using damaged areas of the brain.[29] Bisley and Goldberg have found specifically that the medial intraparietal area responds to reaching, the anterior intraparietal area responds to grasping, and the lateral intraparietal area responds to eye movements.[4] Based on these ideas, treatment can then be manipulated to work on correcting body schema to increase awareness on the neglected side.

One method used to alter this body schema is neck vibration. Neck muscle vibrations stimulate alpha motor neurons, creating an illusion of head-on-trunk rotation to the side of the vibrations. This works by tricking the brain into thinking that the vibrations are lengthening or shortening the muscles.[30] Karnath and colleagues have demonstrated this to be an effective treatment.[31] They have also tested whether this effect would work with other areas of the body (e.g., left hand, head), but this did not have any effect.[30] These results were confirmed with later studies.[32] Rotation of the trunk 15 degrees to the left also showed improvement.[32] This changes egocentricity, correcting the neglect by altering the egocentric coordinate system used in 'visuomotor coordination and exploration of space.'[32] However, Roeden and colleagues found that neck vibration was not valuable in affecting visuo-spatial attention.[30] This is the same principle commonly used in prism adaptation treatments.[33] Use of base-left yoked prism, used in USI treatment, creates a directionally biased visuo-motor and sensory motor response.[10] Recently, it has been suggested that prism adaptation is a cerebello-cortical network response in spatial cognition.[34] Another tool for improving neglect is with vestibular input. Karnath[35] found that spatial neglect and vestibular processing at a cortical level involve common brain areas. Functional imaging studies have revealed the superior temporal cortex, the insulate, and the temporo-parietal junction to be important components of the multi-sensory system affected by neglect. With this in mind, clinics like Vision Northwest have also applied vestibular input to patient therapy programs. Karnath found that by using caloric vestibular stimulation, he was able to improve the neglect.[36] Vision Northwest has found similar improvements in patients with

Table 4: Case Example--Progression of Results Over Time

Visit	Static Confrontation Finger Counting	Kinetic Confrontation Finger Counting	Dual Presentation (static and motion unless otherwise indicated)	Full Threshold Visual Field (Automated)
Sept 2012	----------	----------		OD & OS complete left hemianopsia with enlarged blind spot and decreased detection in the right field
Oct 2012	Left defect	Normal	Left extinction (static & motion count fingers): No improvement with cervical stimulation Then, had patient hold out own hands and be aware of them—she sees them both. Self-present dual targets: no extinction no effect with neck vibration	Incongruous left hemianopsia
Dec 2012	----------	----------		OD: left hemianopsia OS: left inferior quadrantanopsia
Feb 2013	Left defect	Normal	Left extinction (static & motion count fingers) Had patient throw beanbags up in each hand simultaneously Post-beanbag: no extinction Added turn-and-clap therapy	OD: left hemianopsia OS: left inferior quadrantanopsia
March 2013	Left defect	Normal	Left extinction (static count fingers) Static right target/kinetic left target, count fingers: no extinction	

rotational vestibular stimulation. The patient is rotated 5-10 times to the left on a rotational chair. This provides a post-rotary nystagmus to the left and an increase in attention to the left visual space. Experimentation with these principles is demonstrated in case example 1, below. Rotational vestibular, cervical, and bi-hemispheric activities need further investigation via formal study.

Case Example

The case example is an 80-year-old female who suffered a middle cerebral artery CVA in the M2 branch in July 2012 and was given tissue plasminogen activator (tPA) to improve her outcomes. This patient was unaware of her neglect. She was tested for simultagnosia, which was negative. After receiving therapy, she was cleared in March 2013 by the rehabilitative service for driving, and the examiner (an occupational therapist) noted no signs of left neglect. Table 4 shows a review of her progress and the therapies performed. Treatment at Vision Northwest began in October 2012.

As exemplified in the case given, a patient may show neglect initially in a static and kinetic presentation. However, as attention improves, kinetic fields generally improve before static fields. Since real-world situations are rarely static, it suggests that she would be safe to drive because all targets would be either moving or have a motion parallax effect when in a moving vehicle. Therefore, Humphrey visual fields don't truly measure how this patient is functioning. It is important, however, to have these patients examined by an occupational therapist for driving rehabilitative services because they investigate additional aspects of these tasks.

Discussion

We have found that patients respond best by using cervical and/or vestibular input, followed immediately by increased spatial awareness activities, especially tasks that are multisensory and bi-hemispheric in nature. An example would be using a pencil and drawing a clock with appropriate numbers in order. The patient is required to recall the number sequences (left hemisphere function) with the spatial distribution on the clock (right hemisphere function). Figure 1A shows a typical left neglect response with the numbers primarily on the right side from a patient who suffered a right middle cerebral artery stroke. When the patient

Figure 1A

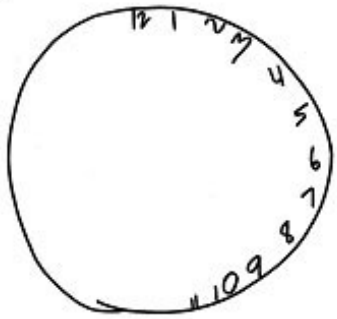

Figure 1B

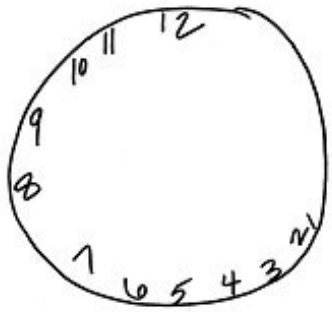

Figure 1. Comparison of Draw-A-Clock Test when starting clockwise vs. counterclockwise. *The images shown were drawn on the same day by the same individual. This individual experienced a massive Right ICH secondary to HTN (X 10 yrs). Note, this is not patient example. This figure demonstrates how the draw-a-clock test is not really a test of neglect, but rather a test of hemisphere communication in general. It shows that this particular patient, if only dependent on results from Figure 1A, would be diagnosed with USI. However, when asked to repeat the process drawing counterclockwise, the patient did not draw the numbers in a fashion that neglected the left visual field, as shown in Figure 1B. This patient cannot attend to sequencing and spacing simultaneously.*

is asked to draw the numbers in reverse order from 12 to 1, he has then shifted well to the left side (Figure 1B) because he is unable to alternate his attention between right and left hemispheres.[37] In this case of right hemisphere brain damage, the patient dominantly attends with the left hemisphere. The patient cannot attend alternately to both spatial awareness (right hemisphere) and sequencing (left hemisphere). As recovery improves, the patient is able to increase right hemispheric processing (spatial distribution of numbers) and draws the clock better.

Taking the above-mentioned principles into account, this alters the way one would view the application of visual fields in the diagnosis and treatment of those with a suspected USI vs. hemianopsia, as well as influences the treatment. The easiest means to diagnose an individual with ABI for these conditions is using confrontation visual fields with a vibration device. This is easy, requires no equipment that cannot be easily transferred, and is therefore hospital-friendly.

Conclusion

Testing of visual fields and USI can be done by confrontation, tangent screen, and threshold testing. This can be used with imaging to develop a good understanding of the type of loss (i.e., hemianopsia/quadrantanopsia vs. USI). Each test has different demands that need to be shared with the multidisciplinary team. An important consideration is testing for USI while standing compared to sitting. Confrontation fields counting fingers should include both static and kinetic presentation. Testing can also include variable probes, including (A) standing vs. sitting, (B) cervical stimulation, (C) vestibular stimulation, and (D) peri-personal vs. extra-personal space. The use of the above probes, integrated with traditional USI therapy, should help speed the recovery and overall outcomes of USI. The use of these probes with the multidisciplinary team may provide a process of rehabilitation and treatment that is more efficacious to improving a variety of different daily living activities.

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References

1. Dragoi V. Visual Processing: Cortical Pathways (Section 2, Chapter 15). In: Neuroscience Online: An Electronic Textbook for the Neurosciences. Department of Neurobiology and Anatomy: The University of Texas Health Science Center at Houston (UTHealth). 1997. Available at: http://bit.ly/1Q1c148 Accessed: September 14, 2014.
2. Huk A. Visual Pathways: Lecture Notes. In: Psychology 115; 29 June 1999 & 1 July 1999; Stanford University: California, 1999. Available at: http://stanford.io/1RnrGtS Accessed February 4, 2013.
3. Remington LA. Clinical Anatomy of the Visual System, 2nd Edition. Waltham, Mass: Butterworth-Heinemann, 2004. http://1.usa.gov/1jYYLie
4. Bisley JW, Goldberg ME. Attention, intention, and priority in the parietal lobe. Annu Rev Neurosci 2010;33:1-21. http://1.usa.gov/1NlTj3F
5. Wash T. Ophthalmology Monographs 2: Visual Fields Examination and Interpretation. American Academy of Ophthalmology. La Jolla, CA: Palace Press, 1990. http://bit.ly/1mkxgla
6. Zeki S, Ffytche DH. The Riddoch syndrome: Insights into the neurobiology of conscious vision. Brain 1998;121(1):25-45. http://1.usa.gov/1QjdF1m
7. Coway A. Visual System: How does blindsight arise? Current Biology 201020(17):R702-4. http://bit.ly/1Ybd4mL
8. Seemungal BM, Guzman-Lopez J, Arshad Q, Schultz SR, et al. Vestibular activation differentially modulates human early visual cortex and V5/MT excitability and response entropy. Cerebral Cortex 2013;23:12-9. http://1.usa.gov/1NQ4kMe
9. Wortzel HS. Forensic Neuropsychiatry Committee Course: Review of Clinical Neuroscience for Forensic Psychiatry. In: Forensic Neuropsychiatric Assessment of Cognition VISN 19 MIRECC; 2010; Denver: Denver Veterans Affairs Hospital. Available at: http://1.usa.gov/1Xzld44. Accessed January 28, 2013.
10. Jacquin-Ciourtois S, O'Shea J, Luaute J, Pisella L, et al. Prism Adaptation and The Rehabilitation of Special Neglect. In: Systems Neuroscience and Rehabilitation. Japan: Springer Press, 2011:81-104. http://bit.ly/1Qje1oE
11. Mesulam MM. Spatial attention and neglect: parietal, frontal and cingulate contributions to the mental representation and attentional targeting of salient extrapersonal events. Phil Trans R Soc Land B 1999;354(1387):1325-46. http://1.usa.gov/1NlTCeF
12. Windsor RL, Ford CA, Windsor LK. Visual Neglect–Hemianopsia.net. The Low Vision Centers of Indiana [Internet] 2010. Updated 2010 May 10. Available at: http://bit.ly/1IpyIfO. Accessed September 15, 2014.
13. Posner MI, Walker JA, Friedrich FJ, Rafal RD. Effects of parietal injury on covert orienting of attention. J Neurosci 1984;4:1863-74. http://1.usa.gov/1MdN9yu
14. Dominguez-Borras J, Saj A, Armony, JL, Vuilleumier P. Emotional processing and its impact on unilateral neglect and extinction. J Neuropsychologia 2012;3:1054-71. http://1.usa.gov/1RkUX9s
15. Benson V, Letswaart M, Milner D. Eye movements and verbal report in a single case of visual neglect. PLOS one. 2012;7(8) e43743:1-11. http://1.usa.gov/1P3NliQ
16. McCaffrey P. Chapter 4: Medical Aspects: Blood Supply to the Brain. In: The Neuroscience on the Web Series: SPPA 636 Neuropathologies of Language and Cognition. California State University Chico: Chico, California. 1999-2011. Available at: http://bit.ly/1Npiqzu. Accessed September 14, 2014.
17. Lee SM, Balcer LJ, Volpe NJ, Liu GT, et al. Laser pointer visual field screening. J Neuro-Ophthalmol 2003;23:260-3. http://1.usa.gov/1IR1h5U
18. Johnson LN, Baloh FG. The accuracy of confrontation visual field test in comparison with automated perimetry. J Nat Med Assoc 1991;83:895-8. http://1.usa.gov/1TLXabN
19. Shahinfar S, Johnson LN, Madsen RW. Confrontation visual field loss as a function of decibel sensitivity loss on automated static perimetry: Implications on the accuracy of confrontation visual field testing. Ophthalmology 1995;103:872-7. http://1.usa.gov/1Z8Ftqy
20. Ranjeet JP, Gales K, Griffiths PG. Effectiveness of testing visual fields by confrontation. The Lancet 2001;358(9290):1339-40. http://bit.ly/1Ut2iCE
21. Heijl A, Patella VM. Essential Perimetry: The Field Analyzer Primer. Dublin, CA: Carl Zeiss Meditec Inc, 2002. http://amzn.to/1P4xh6a
22. Wong A, Sharpe J. A comparison of tangent screen, Goldmann, and Humphrey perimetry in the detection and localization of occipital lesions. Ophthalmology 2000;107:527-44. http://1.usa.gov/1SYQh79
23. Buchanan WM, Gloster J. Automatic device for rapid assessment of the central visual field. Brit J Ophthal 1965;49:57-70. http://bit.ly/1Z9Byd3
24. Maples WC, DeRosier W, Hoenes R, Dendure R, Moore S. The effects of cellphone use on peripheral vision. Optometry 2008;79:36-42. http://bit.ly/1YcvlA2
25. Gorea A, Caetta F. Adaptation and prolonged inhibition as a main cause of motion-induced blindness. J Vis 26;9(6):16.1-17. doi: 10.1167/9.6.16. http://bit.ly/1jZHJ3I
26. Riddoch JM, Rappaport SJ, Humphreys GW. Extinction: A Window Into Attentional Competition. In: 176 Progress in Brain Research: Attention, 1st ed. Amsterdam, Netherlands: Elsevier, 2009:149-57. http://amzn.to/1J8Ea1z
27. Ferber S, Karnath HO. How to assess spatial neglect–line bisection or cancellation tasks? J Clin Exp Neuropsychol 2001;23:599-607. http://bit.ly/1Ut2YYW
28. Maravita A, Iriki A. Tools for the body (schema).Trends Cog Sci 2004;8(2):79-86. http://bit.ly/1I6d2pc
29. Maravita A, Husain M, Clarke K, Driver J. Reaching with a tool extends visual-tactile interactions into far space: evidence from cross-modal extinction. Neuropsychologia 2001;39:580-5. http://bit.ly/1SYQEyk
30. Rorden C, Karnath HO, Driver J. Do neck proprioceptive and caloric-vestibular stimulation influence covert attention in normals, as they influence visual neglect? Neuropsychologia 2001;29:364-75. http://bit.ly/1SYQFlU

31. Schindler I, Kerkoff G, Karnath HO, Keller I, Goldenberg G. Neck muscle vibration induces lasting recovery in spatial neglect. J Neurol Neurosurg Psychiatry 2002;73:412-9. http://1.usa.gov/1MeBBen

32. Karnath HO, Christ K, Hartje W. Decrease of contralesional neglect by neck muscle vibration and spatial orientation of trunk midline. Brain 1993;116:383-96. http://bit.ly/1Ut3oOM

33. Luaute J, Halligan P, Rode G, Rossetti Y, Boisson D. Visuo-spatial neglect: A systematic review of current interventions and their effectiveness. Neurosci Biobehavioral Reviews 2006:30:961-2. http://bit.ly/1NQQKrG

34. Jacquin-Courois S, O-Shea J, Luate J, Pisella L, et al. Rehabilitation of spatial neglect by prism adaptation: a peculiar expansion of sensorimotor after-effects to spatial cognition. Neurosci Behavioral Reviews 2013;37:594-609. http://1.usa.gov/1P4kd2k

35. Kamada K, Shimodozono M, Hamada H, Kawahira K. Effects of 5 minutes of neck vibration immediately before occupational therapy on unilateral spatial neglect. Disabil Rehab J 2011;33:2322-8. http://1.usa.gov/1lL652J

36. Karnath HO, Dieterich M. Spatial neglect–A vestibular disorder? Brain 2006;129(2):293-305. http://1.usa.gov/1Otj3ZD

37. Johannsen L, Ackerman H, Karnath HO. Lasting amelioration of spatial neglect by treatment with neck muscle vibration even without concurrent training. J Rehab Med 2003;35:249-53. http://bit.ly/1l6dd3S

Correspondence regarding this article should be emailed to Tonya Marie Smith Umbrel, OD at Lastsmitty86@gmail.com. *All statements are the authors' personal opinions and may not reflect the opinions of the representative organizations, ACBO or OEPF, Optometry & Visual Performance, or any institution or organization with which the authors may be affiliated. Permission to use reprints of this article must be obtained from the editor. Copyright 2015 Optometric Extension Program Foundation. Online access is available at* www.acbo.org.au, www.oepf.org, and www.ovpjournal.org.

Umbel TMS, Baxstrom C. Multi-sensory factors when examining visual fields in unilateral spatial inattention and its effects on treatment. Optom Vis Perf 2015;3(6):475-84.

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OPTOMETRIC EXTENSION PROGRAM FOUNDATION

Article ▸ Treatment of Traumatic Brain Injury-Induced Oculomotor and Binocular Vision Dysfunctions with Vision Therapy

Greg R. Waldorf, OD, Lincoln, Rhode Island

ABSTRACT

Background: Patients with traumatic brain injury (TBI) commonly experience a collective group of oculomotor dysfunctions and visual disturbances that impact their activities of daily living. This includes deficits in accommodation, versions, vergence, and secondary strabismus due to a cranial nerve III, IV, or VI palsy. Photosensitivity, visual field loss, and ocular health complications may also result. TBI patients are likely to present to an optometrist for evaluation, treatment, and management of the oculomotor-related symptoms that other health care providers are unable effectively to resolve. Vision therapy (VT) can be used to treat the oculomotor disorders, binocular vision dysfunctions, and symptoms that a patient with TBI experiences. VT may be used in conjunction with optical correction involving prisms or tints to treat the symptoms of diplopia, field loss, or photosensitivity.

Case Report: An 18-year-old male college student endured a severe TBI while skiing. After three months of recovery, the patient suffered from constant double and blurred vision due to a partial cranial nerve (CN) III palsy and CN IV palsy in his left eye as well as an intermittent upbeat nystagmus in his right. After undergoing 12 weeks of active office-based vision therapy, the patient achieved clear and single vision at all ranges and was able to return to college the following semester.

Conclusion: This case demonstrates how the optometrist is an important part of the TBI-related rehabilitation team and can make unique management and treatment contributions that improve both visual function and quality of life for those suffering from a TBI.

Keywords: binocular vision, Glasgow Coma Scale, oculomotor dysfunction, traumatic brain injury, vision therapy

There are an estimated 1.7 million traumatic brain injuries (TBIs) in the United States annually; approximately 5.3 million Americans live with a TBI-related disability.[1] A TBI occurs when there is an external mechanical force (a bump, blow, or jolt to the head or body) that causes coup-contrecoup injuries and axonal shearing. TBIs are widely characterized as mild, moderate, or severe based on the Glasgow Coma Scale[2] (GCS) score that a patient is given upon presentation to the emergency department. The GCS score grades a person's ability to open their eyes, to respond verbally, or to make motor responses. The highest GCS score would be in a patient who could spontaneously open their eyes, respond verbally in an oriented sense, and obey motor commands; the lowest score would be an inability to open the eyes and no verbal or motor responses present.

A GCS score between 15 and 13 is considered a mild TBI (mTBI) and may cause temporary changes in brain function, including a change in mental status or consciousness. About 75% of TBIs that occur each year are concussions or other forms of mTBI.[3] Mild TBI does not always cause long-term disability; however,

mTBI patients who experience symptoms such as headaches, dizziness, and nausea in the emergency department are more likely to have post-traumatic complaints such as headaches, dizziness, and drowsiness six months after their injury.[4] Most of the symptoms of mTBI resolve completely within three months of the brain injury, but up to 10% are symptoms that are persistent or permanent.[5] Many patients who suffer from persistent symptoms report improvements by one year post-injury but still suffer disabilities at work such as finding their work more tiring and an inability to maintain previous workloads. Mild TBI patients also report lower levels of life satisfaction one year post-injury.[6]

A GCS score between 12 and 8 is considered a moderate brain injury, and a score below 8 is considered severe. Moderate and severe traumatic brain injuries can result in bruising, torn tissues, bleeding, and other physical damages to the brain that can result in long-term complications or death. Severe TBIs are either closed or penetrating. Closed TBIs cause injury to the brain by movement of the brain within the skull, and penetrating TBIs occur when the brain is injured by a foreign object entering the skull. Sixty-five percent of individuals with moderate brain injury and 100% of patients with severe TBI will have a permanent disability.[7] One-third of severe TBI victims, or 52,000 people, die each year.[1]

The three groups at highest risk of incurring TBI are children aged 0 to 4 years, older adolescents aged 15 to 19 years, and adults aged 65 years and older. In all three age groups, males have higher TBI rates than females, particularly in males aged 0 to 4 years. Falls are the leading cause of TBI in children aged 0-4 years and in adults over 65 years. Motor vehicle injury is the leading cause of TBI-related deaths and is most common for adults aged 20 to 24 years.[1] Racial minorities and those of lower socioeconomic status suffer TBI rates 35% higher than other individuals.[8] Ethnic minorities have worse long-term outcomes and a higher mortality rate after TBI due to a lack of insurance. Typically, younger Caucasian patients with insurance, with a less severe TBI, and with fewer concomitant injuries will have better outcomes.[5,9]

Patients with a TBI may present to the optometric practice with variable symptoms and clinical findings.[10] Common oculomotor/binocular vision disorders associated with TBI are accommodation deficiencies, version and vergence dysfunctions, and strabismus and cranial nerve palsies.[11] Patients can also experience problems with visual field loss, photosensitivity, and eye health issues. While it is possible for oculomotor disorders to occur individually, it is more common for simultaneous visual dysfunctions to be present, complicating diagnosis, management, and treatment.[12] TBI-related oculomotor dysfunctions frequently lead to complaints of blurred vision, asthenopia, and diplopia; problems reading; or a combination of these symptoms. These symptoms make it difficult successfully to complete work, leisure activities, or activities of daily living efficiently and comfortably, causing loss of productivity and a reduced quality of life.

Vision therapy (VT) has long been established as an effective treatment option for oculomotor dysfunctions and binocular vision disorders in the general population as well as in those who have suffered a TBI. Office-based VT can effectively treat oculomotor dysfunctions that occur after a TBI event and can be an effective tool in helping TBI patients regain visual function and comfort for activities of daily living.[13-21]

Case Report

Patient History

AC, an 18-year-old white male, was referred to my office on March 8, 2011 by a rehabilitation center for a VT program to treat his diplopia and blurred vision. AC had suffered a severe TBI when he skied into a snow machine on December 4, 2010. AC was a college freshman, and, being highly motivated to recover, had

established a goal of returning to school for the fall 2011 semester. AC had actively participated in adventure sports such as skateboarding, skiing, and snowboarding for years and hoped to return to them in a safer, reduced capacity.

When AC was admitted to the emergency department, his GCS score was 3. A computed tomography scan of the head revealed punctate contusions in his left temporal lobe and left tectum and a small intracranial hemorrhage in the interpeduncular cistern. Brain magnetic resonance imaging findings were reported to be consistent with diffuse axonal injury, with the largest lesion along the course of the left CN III nucleus. Innumerable foci of susceptibility were scattered throughout the gray/white matter junction and were consistent with the micro-hemorrhages.

A neuro-ophthalmological examination was performed on 1/24/2011 while AC was at a rehabilitation center. The neuro-ophthalmologist reported the following: AC was reported to be alert, oriented, communicative, and cooperative for testing. Corrected visual acuities were 20/70 with pinhole improvement to 20/50^{-} OD, 20/20 without pinhole improvement OS. Color vision (Ishihara) testing showed correct identification of 8/8 plates OD, 8/8 plates OS. Amsler grid testing was normal OU. Pupils were unequal in size; the left pupil was fixed and dilated. There was no APD by reverse testing. External examination of the eyes and orbits was normal. The lids showed a 2-3 mm ptosis on the left. Examination of extraocular motility revealed -0.5 limitation adduction OS, -1 limitation elevation OS, and -3 limitation depression OS. There was also slight limitation of incyclotorsion OS. Pursuit and saccadic functions were normal. There was moderate amplitude, moderate frequency dissociated upbeat nystagmus OD. External ocular health was normal. Applanation tonometry revealed pressures of 15 mm OD and OS. Dilated fundus examination revealed normal optic nerves, maculae, and mid-peripheral retinae OU. The cup-to-disc ratios were 0.3 OU. Automated (Humphrey) visual field testing was performed reliably and found no field defects OU.

The neuro-ophthalmologist diagnosed AC with the following:

1. Left partial CN III palsy
2. Left CN IV palsy
3. Moderate amplitude, moderate frequency dissociated upbeat nystagmus OD

Neuro-ophthalmology recommendations were occlusion OS to eliminate diplopia and a follow-up exam in three months.

Examination Findings

At his first visit to my office in March, AC's chief complaints were intermittent diplopia that was both vertical and horizontal in nature, sensitivity to light outside, and blurred distance vision that was worse in his right eye than his left with his current glasses. AC reported that he preferred using his right eye when he was walking and his left eye when he was reading. Presenting spectacles were OD: -1.00-0.50x005 (20/100) and OS: -1.25 DS (20/20). The manifest refraction was OD: -2.50-1.00x010 and OS: -1.25 DS (20/20 OU). External examination revealed normal confrontation fields OU. His right pupil measured 4.0 mm, and his left pupil was fixed and dilated at 7.0 mm due to the partial left 3rd nerve palsy; there was no APD by reverse testing.

AC did not appreciate stereopsis on the Randot stereotest and reported 5 dots on Worth 4-dot testing at both distance and near. Extraocular muscle testing (versions) were full (no under- or overactions) in his right eye; his left eye demonstrated a full range of motion except for an underaction of the superior oblique muscle. Distance cover test through a trial frame of his manifest refraction revealed a 16$^{\Delta}$ constant left hypertropia and a 25$^{\Delta}$ constant left exotropia. Near cover test measured an 11$^{\Delta}$ constant left hypertropia and a 15$^{\Delta}$ constant left exotropia. An intermittent right upbeat

nystagmus was observed, and a partial left ptosis was noted.

Accommodative amplitudes were measured through the trial frame by push-up method to obtain a free-space assessment. AC reported blur with his right eye at 7 cm (14.28 diopters) and blur with his left eye at 33 cm (3.03 diopters). The left side reduction was due to his partial left 3rd nerve palsy. Hofstetter's formula [15-0.25x(age of patient)] estimates that the lower limit of accommodation for an 18-year-old would be 10.5 diopters; however, research by Sterner in 2004 found that Hofstetter's formula overestimates monocular accommodative amplitudes an average of 3.5-3.6 diopters.[22]

Slit lamp examination was unremarkable OU. Applanation tonometry measured intraocular pressures of 17 mm Hg OU, and fundus evaluation was normal in both eyes, with cup-to-disc ratios of 0.3 OU.

AC reported that it was easier to maintain single vision at near than at far. On near point of convergence testing, he could first obtain single vision in free space on a small bead with an accommodative target at 35 inches (91 cm) with a chin-tip down. He could maintain single vision on the target up to 10 inches (25 cm), at which point the target broke, but he had to continue to tip his chin down. When he was prompted to maintain a normal head posture with no chin-tip down, he could maintain fusion to 10 inches (25 cm), but he reported that it took much greater effort.

Diagnoses

- Diplopia at both distance and near secondary to a left hypertropia (CN IV palsy) and a left exotropia (partial CN III palsy)
- Left underacting superior oblique muscle due to CN IV palsy
- Imbalanced accommodative amplitudes secondary to the partial CN III palsy
- Left partial ptosis from the partial CN III palsy
- Blurred vision secondary to under-corrected refractive error
- Photophobia secondary to TBI as well as from the fixed and dilated left pupil

Treatment Plan and Vision Therapy

Treatment options were presented to AC to provide immediate and long-term relief of his symptoms. Glasses were prescribed without a tint for indoor use, and prescription sunglasses were prescribed to reduce sensitivity to light outside. Fresnel 3M Press-on™ prisms for AC's vertical diplopia were discussed, but AC was highly motivated by the idea of the VT and wanted as few accommodations as possible during the therapy program. AC's personal goal was to complete the VT program by mid-summer (12 weeks) so that he could return to school in the fall. Due to his eagerness to start the therapy program, AC was prescribed a -2.00 diopter loose lens, and he was instructed on how to perform monocular minus lens rock exercises with the Hart chart at 10 feet with his left eye. He was also taught how to perform gross convergence with pencil push-ups so he could start to experience the sensation of convergence until the start of his therapy program one week later.

The goals of the VT program were as follows:

- Eliminate diplopia
- Balance the accommodative system between the eyes
- Improve free-space balance and coordination during visual tasks

All therapy sessions took place on a weekly basis, with one hour of in-office therapy and a daily home-based therapy program prescribed at the end of each in-office session. An ambitious goal of completing all therapy in 12 weeks was set so that AC could return to college for the fall semester.

Therapy Sessions #1-4

The goal of the first four therapy sessions was to reduce AC's diplopia by improving

his convergence and accommodative skills. Tracking work was also introduced since he was a student and would be returning to heavy coursework in the fall. At the first therapy session, the following exercises were introduced as part of AC's in-office and home-based program:

- Ann Arbor tracking (monocular) to improve saccadic reading skills, with a goal to complete each paragraph in less than one minute without the motor support of underlining each line of letters.
- Marsden ball tracking (monocular) to improve pursuit eye movements, with a goal of completing 10 horizontal passes with smooth tracking while seated.
- Brock string to improve convergence skills.
- Minus lens rock (monocular, emphasis on left eye) to improve and to balance accommodative skills, with a goal to complete the full Hart Chart at 10 feet in less than one minute.
- +/-2.00 diopter flippers (monocular, emphasis on left eye) while reading to improve accommodative facility.
- Balance board (assisted) to increase body awareness and balance reactions.

At the end of the first four weeks of therapy, AC had met the prescribed goals for both Ann Arbor tracking and the Marsden ball. With his head in a normal position, AC was able to converge his eyes on a Brock string bead up to 2 inches (5 cm) from his nose, as well as jump between a near bead at 2 inches (5 cm) and one 18 inches (46 cm) away. He was also able to maintain clear, single vision on the Brock string bead while using +/-2 diopter flippers binocularly with the bead at 16 inches (40 cm). AC's accommodative system in his left eye was able to perform the minus lens rock with the Hart chart using a -3.00 diopter lens in less than one minute. AC had done so well on convergence and accommodative tasks that the opaque Lifesaver card for convergence was introduced on week #3, and in one week he was able to fuse and to clear the 3rd Lifesaver from the bottom but was unable to appreciate depth of the letters E and R. AC subjectively reported that monocularly using the +/-2.00 diopter flippers while reading was much easier. AC's balance improved on the balance board over the first four weeks; by week four he could keep a ball balanced in a groove in the board for 3-5 seconds as well as maintain balance while throwing and catching a 3 lb weighted ball at his midline.

AC reported that the headaches that he had experienced during the first three weeks of therapy had dissipated. His cover test measurement at distance was an intermittent 16^{Δ} left hypertropia and 14^{Δ} left exotropia. Cover test at near measured an intermittent 16^{Δ} left hypertropia and 10^{Δ} left exotropia. His accommodative amplitude (push-up) with his left eye was 8.33 D. At this point, AC requested that the Fresnel prism be placed on the left lens of one pair of his glasses to eliminate the intermittent vertical diplopia when he was working with his physical therapist on balance and gross muscular strengthening at the rehabilitation center. Based upon AC's cover test measurements and trial framing of vertical prism, AC felt most comfortable with 10^{Δ} of base-down prism over his left eye, so this Fresnel prism was placed on AC's left spectacle lens.

Therapy Sessions #5-7

The following therapy procedures were prescribed as part of AC's in-office and home-based therapy program for therapy sessions #5-7:

- Binocular Ann Arbor tracking (smaller print paragraphs) with a goal to complete each paragraph in less than one minute without the motor support of underlining each line of letters.
- Marsden ball tracking (binocular) with a goal of completing 20 smooth horizontal passes with smooth tracking while standing.

- Brock string work continued with the addition of +/-2.50 flippers and 8 BO/2 BI prism flippers.
- Lifesaver card was continued for convergence, and the clear divergence Lifesaver card was introduced.
- Minus lens rock continued (monocular, emphasis on left eye) with a -3.50 diopter lens.
- +/-2.50 flippers (monocular, emphasis on left eye) while reading.
- Monocular pursuits with the Brock string attached to the rotating pegboard.

As therapy demands increased, AC used his prism glasses at times during in-office and home-based therapy sessions when he was tired or struggling to fuse targets (primarily during binocular Brock string activities). Binocular Ann Arbor tracking was discontinued after week 5 because it was "too easy:" AC could complete the task with both eyes open in 40 seconds. AC continued to work on Brock string push-ups and "jump" exercises from the first four sessions with an emphasis on getting the strings to cross quickly and consistently at each bead. Lens and prism flippers were introduced with the Brock string to improve and to refine accommodation/vergence skills. The Lifesaver card for convergence was continued, and the clear divergence Lifesaver card was introduced to promote balance between AC's convergence and divergence systems. The monocular minus lens rock lens power was increased to -3.50 diopters and was still emphasized for AC's left eye. Accommodative facility flipper powers were increased to +/-2.50 for monocular use (emphasis left eye) to improve and to balance AC's accommodative facility skills while reading. Monocular pursuit exercises with the Brock string attached to the rotating pegboard (6 feet away) were introduced in-office to promote slow circular pursuit skills.

At the end of the seventh therapy session, AC was able to track the Marsden Ball smoothly for 20 passes binocularly while standing. With the Brock string, AC maintained his convergence skills up to 2 inches (5 cm) from his nose, and he gained more control during "jumps," getting the strings to cross at the beads quickly and consistently. AC was able to hold clear fusion on the Brock string beads at 16 inches (40 cm) while using +/-2.50 diopter flippers, as well as to obtain quick recovery of bead fusion with the 8 BO/2 BI prism flippers. AC continued his work on the Lifesaver cards and gained the ability to fuse the fourth, most demanding, Lifesaver on the convergence card and the second Lifesaver on the divergence card; he never appreciated depth of the letters on either card. Minus lens rock and +/-2.50 diopter flipper therapy continued primarily as home-based therapy, and AC reported that he was able successfully to use the -3.50 diopter lens with the Hart chart and the +/-2.50 flippers while reading. AC's monocular circular tracking exercises with the Brock string attached to the rotating pegboard proved more difficult for his right eye than for his left. AC worked to track and to jump between beads on the Brock string at 24 inches (61 cm) and 40 inches (101 cm) away from his nose as the string rotated with the rotating pegboard. AC's ability to maintain balance on the balance board while catching a 3 lb weighted ball continued to improve, and the ball started to be thrown randomly to his right and left. By the end of week 7, AC's accommodative amplitude (push-up) measurement with his left eye was 14 diopters, and he subjectively reported fewer symptoms of diplopia.

Therapy Sessions #8-12

Before the beginning of session #8, AC was fit into the following contact lenses:

OD: Ciba Focus Dailies for Astigmatism, B.C. 8.6, DIA. 14.2, -2.50-0.75x180

OS: Ciba Dailies, B.C. 8.6, DIA. 13.8, -1.25 DS

The contact lenses provided visual acuities of 20/20 in each eye. At his one-week follow-up, AC was experiencing good contact lens comfort and vision. His acuity remained at 20/20 in each eye, the contact lens fit was determined to be good, and his prescription was finalized. AC was given the option of having plano prescription glasses prescribed with base down Fresnel prism over his left lens for intermittent use, but he declined. At this point in his treatment, AC felt that he experienced diplopia so infrequently that it did not pose a functional problem.

Therapy sessions #8-12 introduced more free-space and motor work, including core stability to round out AC's therapy program. The following exercises were continued/introduced during AC's last four therapy sessions:

- Marsden ball (binocular) tracking while standing on a balance board or standing in "tree" yoga pose.
- Monocular Brock string pursuit rotations with rotating pegboard were continued; manual large (arm's length) diameter rotations with therapist holding/rotating the string were added.
- Binocular pursuits with the Brock string attached to the rotating pegboard for small rotations as well as large (arm's length) diameter rotations with the therapist holding/rotating the string were introduced.
- Brock string convergence work was continued primarily as part of AC's home-based therapy.
- Minus lens rock work was continued only as part of AC's home-based therapy; he continued to work with the -3.50 diopter lens.
- +/-2.50 flippers while reading (binocular).
- Core stability work with therapy ball sit-ups catching a 3 lb ball.

On week 8, AC started to work on Marsden ball tracking exercises with two separate balance techniques incorporated into his Marsden ball routine. One combination technique required AC to track the Marsden ball while maintaining balance on a balance board. The second combination technique required AC to track the Marsden ball while maintaining a standing yoga "tree" pose. "Tree" pose required AC to stand on one leg while his other leg was bent and his raised foot was tucked on the inside of the opposite thigh. Initially, AC had more trouble balancing on his left leg (2 seconds) than his right (10 seconds), but after practicing at home for one week, AC could balance on each leg equally well for 10 seconds while tracking horizontal passes of the Marsden ball. Monocular Brock string rotations with the rotating pegboard (6 feet away) and the newly introduced manual rotations continued, and AC reported that his circular pursuits started to equalize between his eyes by week 11. Binocular Brock string rotations with the rotating pegboard while maintaining convergence on the Brock string were introduced, with bead placement between 24 inches (61 cm) and 40 inches (101 cm). Intermittent vertical diplopia was reported when the bead was at its lowest point of rotation on the rotating pegboard. During manual large-diameter circular rotations with the Brock string, beads were placed at 24 inches (61 cm), 40 inches (101 cm), and 60 inches (152 cm); AC continued to experience intermittent vertical diplopia when the beads were in the lower one-third of his inferior field. The intermittent vertical diplopia did not fully resolve by week 12 unless AC was wearing his prism glasses. At week 12, AC could complete the Hart chart -3.50 diopter minus lens rock exercises successfully (45 seconds right eye; 50 seconds left eye). AC's basic Brock string (push-ups, jumps, +/-2.50 diopter flippers, and 8 BO/2 BI prism flippers) and minus lens rock exercises were continued but were emphasized more at home than in office so that expanded free space motor work could be introduced. Therapy ball sit-ups while

catching a 3 lb weighted ball at his midline and to his right and left were introduced to improve AC's core stability.

Follow-Up Visit Post-Vision Therapy

AC reported to my office for his follow-up visit post-VT program on June 13, 2011. He reported that he only experienced transient vertical diplopia in downgaze and had completely discontinued wearing his Fresnel prism glasses. Cover test measurement at distance was 10^{Δ} left hyperphoria and 8^{Δ} exophoria. His near cover test revealed 10^{Δ} left hyperphoria and 7^{Δ} exophoria. An intermittent upbeat nystagmus was still noted in his right eye during cover testing at distance and near. During near point of convergence testing, AC reported a break in fusion of the target at 1 inch (2.5 cm) and could recover fusion when the target was moved back to 2 inches (5cm). AC reported 4 dots at distance and near with Worth 4-dot testing. Randot stereotest recorded 500" of global stereopsis. Accommodative amplitudes by push-up were 6 cm (16 diopters) in his right eye and 7 cm (14 diopters) in his left. AC's monocular accommodative facility measurements with +/-2.00 diopter flippers and a 20/40 target at 16 inches (40 cm) were 10 cycles per minute in the right eye and 8.5 cycles per minute in the left. The expected value for patients between 13 and 30 years of age is 11 cycles per minute (+/-5 cycles).[23] AC's pre- and post-therapy results can be seen in Table 1. AC was encouraged to continue his home-based vision therapy at least three times per week to maintain solid control over his convergence and accommodative skills.

Table 1: Pre- and Post-VT Results

	Pre-VT	Post-VT
Cover Test Distance	16^{Δ} constant left hypertropia 25^{Δ} constant left exotropia	10^{Δ} left hyperphoria 8^{Δ} exophoria
Cover Test Near	11^{Δ} constant left hypertropia 15^{Δ} constant left exotropia	10^{Δ} left hyperphoria 7^{Δ} exophoria
Randot Stereo Test	No forms	500" global stereopsis
Worth 4-Dot	5 dots at distance and near	4 dots at distance and near
Accommodative Amplitudes	OD: 14.28 diopters OS: 3.03 diopters	OD: 16 diopters OS: 14 diopters
Near Point of Convergence	Break/diplopia at 2.5 cm	Break/diplopia at 2.5 cm

Discussion

Vision therapy has been shown to benefit patients experiencing oculomotor symptoms from a TBI. Even when VT is implemented years after the patient's accident, it can improve visual function.[18-21] Plasticity is the brain's ability to reorganize itself by forming new neural connections during development and after injury or disease. This process starts as soon as one hour following diminished input to a portion of the somatomotor cortex and may take weeks or years to complete.[24] Recent research has also shown that VT can evoke cortical activity changes in the brain.[25]

Patients who experience a TBI are likely to have visual or oculomotor dysfunctions that impact their activities of daily living long after their accident.[26] Severe disorders of the visual system are often discovered immediately, but less severe forms of visual dysfunction can take longer to identify and to diagnose. The symptomatology experienced by a patient after a TBI can be variable but typically presents as asthenopia, headaches, diplopia, blurred vision, problems reading, field loss, or a combination of these symptoms.[27] Immediate relief can be provided by the optometrist through optical prescriptions for some symptoms: glasses to correct an uncorrected refractive error, bifocals to assist with accommodative disorders, prism glasses to eliminate diplopia or to assist with field loss, sunglasses if the patient is photophobic outdoors, or gradient tint glasses if the patient is sensitive to overhead lighting indoors. VT can provide long-term results and eliminate many, if not all, of the ocular symptoms suffered by the TBI patient when the symptoms they experience are due to convergence insufficiency; deficits

in fixation, pursuits, and saccades; problems with steady version and vergence skills; accommodative disorders; and strabismus from cranial nerve palsies.[18,19]

During the initial assessment of a patient who has suffered a TBI, a full medical history of the TBI and post-injury problems experienced by the patient should be obtained. Clinicians should consider collecting as much medical history as possible prior to the evaluation of the TBI patient and book an extended appointment based on the patient's history and level of function. Extra time allows the examiner to prepare a clear direction for the examination and reduces scheduling conflicts when TBI patients are unable efficiently to perform the required visual tests due to poor understanding of directions or variable exam findings; less time may be necessary for patients with moderate to severe TBIs that are more medically involved and who are unable to sit and visually concentrate for extended periods of time.

A therapy program to address the oculomotor problems resulting from a TBI starts by working to improve monocular fixation, monocular pursuits, and monocular saccades, much like those undergoing VT without brain injury. A patient's physical status must be considered before starting fixation, pursuit, and saccade therapy. Patients who have generalized gross motor weakness, who fatigue easily, or who have partial paralysis may need extra support and should start this level of therapy supine on the floor where their body is well supported and eye-related exercises are isolated. As a patient's gross motor skills improve, the patient can start performing VT while sitting supported in a chair before finally moving to therapy while standing.

Muscle limitations due to paresis that result in diplopia can be treated initially with Fresnel prisms or prism ground into the patient's lenses. If the patient's ocular misalignment requires higher amounts of prism (20^{Δ} or higher), the level of reduced acuity and contrast sensitivity can be problematic, so the clinician should always try to split the prism between the lenses. This is especially true with Fresnel prisms that have been found to degrade acuity and contrast sensitivity in higher prismatic values.[28,29]

Disorders of accommodation occur in 41.1% of patients with TBI.[11] As with disorders of fixation, pursuits, and saccades, accommodation disorders should be treated monocularly until both eyes can accommodate equally before introducing binocular accommodative work. Convergence insufficiency is present in 42.5% of patients with TBI and can be addressed in tandem with binocular accommodation therapy.[11] Vergence work can begin when the patient can fuse an image seen by both eyes without diplopia; this may require temporary use of prisms to assist fusion.

As basic oculomotor and binocular vision skills of the patient improve, free space activities can begin to make vision skills more habitual as the patient's gross motor system is incorporated into therapy. In AC's case, Marsden ball work, Brock string activities, general balance work, and core stability exercises combined gross motor work into his VT.

Mild TBI is eight times more common than moderate or severe TBI, and 70-90% of patients who present to emergency departments have mTBI.[7] AC's recovery is profound in that he presented to the emergency department with a GCS score of 3, representing a severe TBI. There are many factors that contributed to AC's success: AC was young, healthy, and highly motivated to succeed; he had a strong family support network; and he had a complete medical/rehabilitation team including occupational therapists, physical therapists, a neuro-ophthalmologist, and an optometrist providing a VT program. AC also had excellent primary and secondary insurance that helped pay for most of the medical services, reducing the financial stress on his family and allowing them to focus on his recovery. AC spent hours on his home-based therapy each week (sometimes hours each day) working to improve his

oculomotor skills. AC also demonstrated a high level of visual adaptability. When he presented at his first exam, he had already developed an adaptive visual suspension mechanism to cope with his diplopia: he used his right eye when he was walking and his left eye when he was sitting down. AC's left hyper deviation never fully resolved, and after his 12th therapy session, he reported that he experienced vertical diplopia when he looked "35 degrees below horizontal." AC was aware and could clearly verbalize that he maintained fusion with both eyes during activities like sports and driving, but he still preferred using his left eye for homework and reading. AC's therapy program maximized his abilities, but it did not directly resolve the nerve pathology. He was left with the option of using glasses with vertical prism as needed post-therapy, but he chose not to pursue the option.

This case demonstrates that the optometrist can play a major role in the rehabilitation of patients who have suffered a TBI. Additionally, it shows how VT can improve functional vision skills in patients with TBI, reduce their symptoms, and help them with their activities of daily living.

Conclusion

When the health of the patient has stabilized after a TBI, they can be good candidates for an office-based program of VT to reduce symptoms from their impacted oculomotor system. Oculomotor dysfunctions associated with TBI may be more severe and complex than what are treated in traditional VT patients, but the symptoms these patients experience are similar and may be noticed immediately or months after the accident. Deficits in fixation, pursuits, saccades, accommodation, versions, or vergence, as well as the development of strabismus or cranial nerve palsies, are common post-TBI and should be addressed by the patient's optometrist. There are many factors that contribute to the success of a patient's therapy program, including the severity of the brain injury, the patient's level of commitment to the therapy program, family support, the quality of the patient's insurance, and their socioeconomic status. This case demonstrates how VT can be used effectively as a part of a comprehensive rehabilitation program to treat the oculomotor symptoms associated with TBI and to help a patient return to a highly functional quality of life.

References

1. Faul M, Xu L, Wald MM, Coronado VG. Traumatic brain injury in the United States: Emergency department visits, hospitalizations, and deaths 2002-2006. Atlanta (GA): Centers for Disease Control and Prevention, National Center for Injury Prevention and Control; 2010:2-70. Available at http://1.usa.gov/1PwMAHA. Accessed March 15, 2013.
2. Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. Lancet 1974;2:81-4. http://bit.ly/1YhfpHD
3. 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. http://1.usa.gov/1YcGpgE
4. De Kruijk JR, Leffers P, Menheere PP, Meerhoff S, et al. Prediction of post-traumatic complaints after mild traumatic brain injury: Early symptoms and biochemical markers. J Neurol Neurosurg Psychiatr 2002;73:727-32. http://1.usa.gov/1J8JgLf
5. Crandall M. Epidemiology of Traumatic Brain Injury. In: Zollman FS, ed. Manual of Traumatic Brain Injury. New York, NY: Demos Medical, 2011:25-9. http://bit.ly/1jZMhqD
6. Stålnacke BM, Björnstig U, Karlsson K, Sojka P. One-year follow-up of mild traumatic brain injury: Post-concussion symptoms, disabilities and life satisfaction in relation to serum levels of S-100B and neurons-specific enolase in acute phase. J Rehabil Med 2005;37:300-5. http://bit.ly/1Qqax3B
7. Stipler M. Trauma of the Nervous System: Craniocerebral Trauma. In: Daroff RB, Fenichel GM, Jankovic J, Mazziotta JC, eds. Neurology in Clinical Practice. 6th ed. Vol. 2. Philadelphia, PA: Elselvier-Saunders, 2012:942-56. http://bit.ly/1QJ3WzH
8. Jager TE, Weiss HB, Coben JH, Pepe PE. Traumatic brain injuries evaluated in U.S. emergency departments, 1992-1994. Acad Emerg Med 2000;7:134-40. http://1.usa.gov/1Z9Iy9T
9. Shafi S, De La Plata CM, Diaz-Arrastia R, Shipman K, et al. Racial disparities in long-term functional outcome after traumatic brain injury. J Trauma 2007;63:1263-8. http://wb.md/1UthkIH
10. Kapoor N, Ciuffreda KJ. Vision disturbances following traumatic brain injury. Cur Treat Options Neurol 2002;4:271-80. http://1.usa.gov/1NQXztw

11. Ciuffreda KJ, Kapoor N, Rutner D, Suchoff IB, et al. Occurrence of oculomotor dysfunctions in acquired brain injury: A retrospective analysis. Optometry 2007;78:155-61. http://1.usa.gov/1mknGP0
12. Alvarez TL, Kim EH, Vicci VR, Dhar SK, et al. Concurrent vision dysfunctions in convergence insufficiency with traumatic brain injury. Optom Vis Sci 2012;89:1740-51. http://1.usa.gov/1SYZcFE
13. The 1986/87 future of visual development/performance task force. The efficacy of optometric vision therapy. J Am Optom Assoc 1988;59:95-105. http://bit.ly/1QqaTXU
14. Scheiman M, Mitchell GL, Cotter S, Kulp MT, et al. A randomized clinical trial of vision therapy/orthoptics versus pencil pushups for the treatment of convergence insufficiency in young adults. Optom Vis Sci 2005;82:583-95. http://bit.ly/1cRx8y6
15. Convergence Insufficiency Treatment Trial Study Group. Randomized clinical trial of treatments for symptomatic convergence insufficiency in children. Arch Ophthalmol 2008;126:1336-49. http://1.usa.gov/1YcHlS9
16. Ciuffreda KJ. The scientific basis for and efficacy of optometric vision therapy in nonstrabismic accommodative and vergence disorders. Optometry 2002;73:735-62. http://1.usa.gov/1NuxWLu
17. Scheiman M, Cotter S, Kulp MT, Mitchell GL, et al. Treatment of accommodative dysfunction in children: Results from a randomized clinical trial. Optom Vis Sci 2011;88:1343-52. http://1.usa.gov/1IRG4sN
18. Ciuffreda KJ, Rutner D, Kapoor N, Suchoff IB, et al. Vision therapy for oculomotor dysfunctions in acquired brain injury: A retrospective analysis. Optometry 2008;79:18-22. http://1.usa.gov/1mknGP0
19. Thiagarajan P, Ciuffreda KJ. Effect of oculomotor rehabilitation on vergence responsivity in mild traumatic brain injury. J Rehabil Res Dev 2013;50:1223-40. http://1.usa.gov/1NmQjUP
20. Ciuffreda KJ, Ludlam DP. Conceptual model of optometric vision care in mild traumatic brain injury. J Behav Optom 2011;22:10-2. http://bit.ly/opstudents
21. Fishman Hellerstein L, Freed S. Rehabilitative optometric management. J Behav Optom 1994;5:143-8. http://bit.ly/1uv5DSC
22. Sterner B. Ocular accommodation studies of amplitude, insufficiency, and facility training in young school children. Department of Ophthalmology, Institute of Clinical Neuroscience. Göteborg University, Sweden: Vasastadens Bokbinderi AB, 2004. http://bit.ly/1lLi2pj
23. Scheiman M, Wick B. Diagnostic Testing. In: Scheiman M, Wick B, eds. Clinical Management of Binocular Vision: Heterophoric, Accommodative, and Eye Movement Disorders. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2008:3-32. http://bit.ly/1NlzYQa
24. Miltner HR, Weiss T, Wolfgang JL, Meissner W, Taub E. Rapid functional plasticity in the primary somatomotor cortex and perceptual changes after nerve block. Eur J Neurosci 2004;20:3413-23. http://bit.ly/1RlX50K
25. Alvarez TL, Vincent R, Vicci YA, Kim EH, et al. Vision therapy in adults with convergence insufficiency: Clinical and functional magnetic resonance imaging measures. Optom Vis Sci 2010;87:985-1002. http://1.usa.gov/1lLijZp
26. Greenwald BD, Kapoor N, Singh AD. Visual impairments in the first year after traumatic brain injury. Brain Inj 2012;26:1338-59. http://1.usa.gov/1NmQBuM
27. Ciuffreda KJ, Neera K. Oculomotor dysfunctions, their remediation, and reading-related problems in mild traumatic brain injury. J Behav Optom 2007;18:72-7. http://bit.ly/1NQYiuM
28. Katz M. Visual acuity through Fresnel, refractive, and hybrid diffractive/refractive prisms. Optometry 2004;75:503-8. http://1.usa.gov/1O2hWQ1
29. Katz M. Contrast sensitivity through hybrid diffractive, Fresnel, and refractive prisms. Optometry 2004;75:509-16. http://1.usa.gov/1O6CGwh

Correspondence regarding this article should be emailed to Greg R. Waldorf, OD at grwaldorf@verizon.net. *All statements are the author's personal opinions and may not reflect the opinions of the representative organizations, ACBO or OEPF, Optometry & Visual Performance, or any institution or organization with which the author may be affiliated. Permission to use reprints of this article must be obtained from the editor. Copyright 2015 Optometric Extension Program Foundation. Online access is available at* www.acbo.org.au, www.oepf.org, and www.ovpjournal.org.

Waldorf R. Treatment of traumatic brain injury-induced oculomotor and binocular vision dysfunctions with vision therapy. Optom Vis Perf 2015;3(6):485-95.

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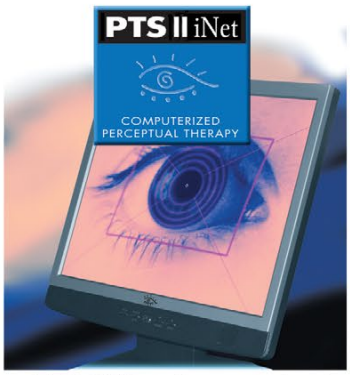

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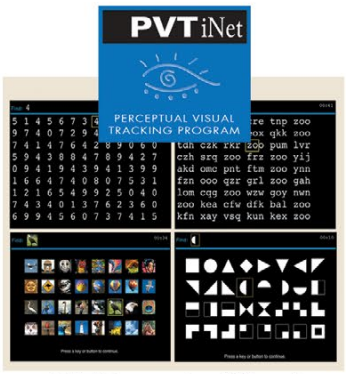

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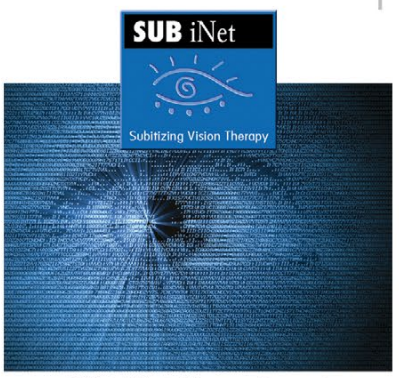

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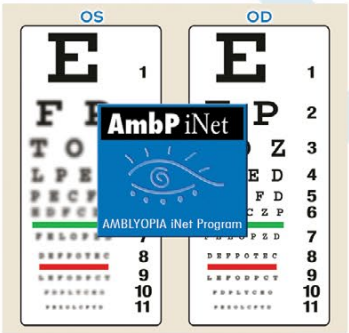

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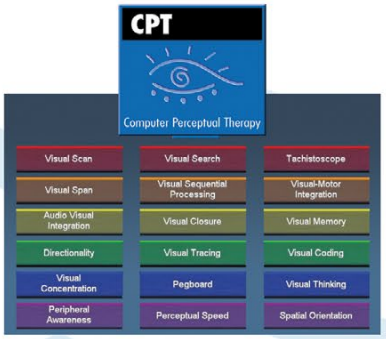

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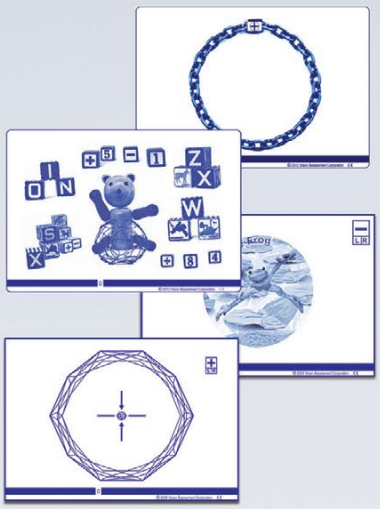

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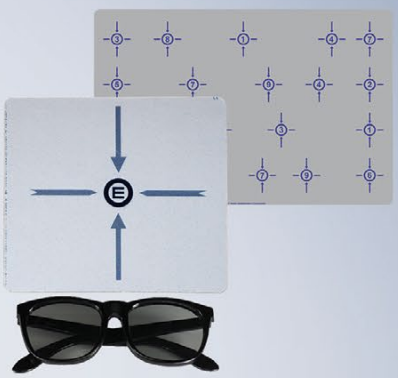

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