

REVIEW ARTICLE

## The consequence of spatial visual processing dysfunction caused by traumatic brain injury (TBI)

William V. Padula<sup>a,b</sup>, Jose E. Capó-Aponte<sup>c</sup>, William V. Padula<sup>d</sup>, Eric L. Singman<sup>e</sup>, and Jonathan Jenness<sup>b</sup>

<sup>a</sup>Salus University College of Optometry, Philadelphia, PA, USA; <sup>b</sup>Padula Institute of Vision Rehabilitation, Guilford, CT, USA; <sup>c</sup>Department of Optometry Womack Army Medical Center, Fort Bragg, NC, USA; <sup>d</sup>Department of Health Policy and Management, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, USA; <sup>e</sup>Department of Ophthalmology, Wilmer Eye Institute, Johns Hopkins Medicine, Baltimore, MD, USA

### ABSTRACT

**Objective:** A bi-modal visual processing model is supported by research to affect dysfunction following a traumatic brain injury (TBI). TBI causes dysfunction of visual processing affecting binocularity, spatial orientation, posture and balance. Research demonstrates that prescription of prisms influence the plasticity between spatial visual processing and motor-sensory systems improving visual processing and reducing symptoms following a TBI.

**Rationale:** The rationale demonstrates that visual processing underlies the functional aspects of binocularity, balance and posture. The bi-modal visual process maintains plasticity for efficiency. Compromise causes Post Trauma Vision Syndrome (PTVS) and Visual Midline Shift Syndrome (VMSS). Rehabilitation through use of lenses, prisms and sectoral occlusion has inter-professional implications in rehabilitation affecting the plasticity of the bi-modal visual process, thereby improving binocularity, spatial orientation, posture and balance

**Main outcomes:** This review provides an opportunity to create a new perspective of the consequences of TBI on visual processing and the symptoms that are often caused by trauma. It also serves to provide a perspective of visual processing dysfunction that has potential for developing new approaches of rehabilitation.

**Conclusions:** Understanding vision as a bi-modal process facilitates a new perspective of visual processing and the potentials for rehabilitation following a concussion, brain injury or other neurological events.

### ARTICLE HISTORY

Received 25 March 2016

Revised 6 December 2016

Accepted 1 February 2017

### KEYWORDS

Vision; post trauma vision syndrome (PTVS); visual midline shift syndrome (VMSS); egocentre; prisms; traumatic brain injury (TBI); concussion; cerebrovascular accident (CVA); spatial visual process; risk of fall (RoF)

## Introduction

The diagnosis and treatment of visual dysfunctions induced by a brain injury are becoming more important with the increasing number of persons affected by cerebrovascular accident (CVA) and traumatic brain injury (TBI) (Table I). The annual incidence of CVA is 700 000 nationally, with a mortality rate of 20% during the first year [1,2]. The US Center for Disease Control and Prevention (CDC) reported that approximately 3 million Americans are currently permanently disabled from a CVA [1]. On the other hand, 1.1 million cases of TBI are reported each year of which nearly 75% are concussions or other forms of mild TBI [3]. The leading causes of TBI include: falls (28%); motor vehicle accidents (20%); concussion (19%) and assaults (11%). In addition, an estimated 1.6–3.8 million of sports-related concussions occur in the USA each year [4]. American football is believed to account for >60% of concussions [5].

During the course of the current military conflicts in Afghanistan and Iraq, TBI has emerged as an increasing cause of morbidity [6,7]. Soldiers incur TBI as the result of contact with enemy forces or weapons systems, improvised explosive devices (IEDs), vehicle-borne IEDs, mortars, rocket-propelled grenades, as well as from head impact accidents

caused by enemy action, equipment failure or human factors. Blasts are the leading cause of TBI for active duty military personnel in war zones. Between 2000 and 2015, the Department of Defense reports that there have been 344 030 cases of TBI. Mild TBI (mTBI) accounts for 82.3 percent of all cases [7].

There are a multitude of specific pathologies resulting from TBI. However, the visual and ocular system can serve as a window into the brain to evaluate the extent of brain injury and identify brain structures affected by the trauma, which can map a treatment regime for the injured patients. Between 30% and 35% of Americans diagnosed with mild-to-moderate TBI have associated visual dysfunctions [8]. Severity of a brain injury has not been correlated with the severity of visual dysfunction. According to the information provided by the Department of Defense and Veteran Affairs (VA), over 70% of TBI patients at military medical facilities have reported visual complaints [6]. In addition, more than 74% of all TBI patients receiving medical treatment in some of the VA poly-trauma care facilities have reported visual complaints. Oculomotor, binocular, perceptual and reading problems were amongst the most common visual deficits identified in service members with TBI (Table II) [9].

**Table I.** Table of abbreviations.

Abbreviation	Definition	Abbreviation	Definition
CDC	Center for disease control	NVPR	Neuro visual processing rehab
CN	Cranial nerve	PTVS	Post trauma vision syndrome
COM	Center of mass	TBI	Traumatic brain injury
CVA	Cerebrovascular accident	VA	Veterans affairs
DoD	Department of defense	VEP	Visual evoked potential
fMRI	Functional magnetic resonance image	VMSS	Visual midline shift syndrome
IED	Improvised explosive device	VT	Vision therapy
MS	Multiple sclerosis	WVFO	Wide field of view

A new understanding is emerging that provides insight into visual processing dysfunction caused by a concussion, TBI or other neurological events called neuro-visual processing rehabilitation (NVPR). NVPR provides clinical assessment and rehabilitative treatment options that affect plasticity in visuo-spatial processing [10–13]. NVPR involves the use of prisms as non-compensatory instruments affecting dysfunction of visual processing.

Following a neurological event, persons will often experience a wide range of symptoms related to vision such as headaches, diplopia, vertigo, asthenopia, inability to focus, movement of print when reading, difficulty with tracking and fixations, and photophobia [14]. Binocular dysfunction is prevalent following mTBI [15]. Exotropia and exophoria are common characteristics of a neurological event [16,17]. In addition, visual motor function, posture and balance can often be affected following a neurological event. There is new evidence that demonstrates that neuro-motor function contributes to spatial mapping and the effects of egocentric concepts of visual midline. The definition of visual midline is ‘the perceived visual/sensorimotor concept of one’s lateral anterior-posterior ego centre or centre of the vertical axis within the body’ beyond the role of acuity [18,19]. The influence of Homonymous hemianopsia —bilateral visual field loss on the same side—also affects the preconscious perception of visual midline, thereby affecting balance and posture. Vertigo, dizziness and other balance disorders are common following the neurological event [10]. Loss of half of the visual field causes the visuo-spatial egocentre to orient towards the remaining portion of the visual field.

The purpose of this paper is to bring forward research to support a new model of vision and visual processing that potentially provides a new framework for understanding the complex constellation of symptoms and binocular dysfunction that can occur following an TBI. This is done to engage a new level of thinking pertaining to both the symptoms and dysfunction that occur visually following an TBI as well as to

stimulate thinking about what this new model of vision may offer regarding rehabilitation.

The authors propose that based on the review of the literature discussed in this paper, a new perspective of vision is possible that may offer insight into the symptoms presented following an TBI as well as potential means to affect the dysfunction in visual processing that occurs.

### Pathophysiology of Neurological Oculomotor Dysfunctions

Nearly 70% of sensory processing in the brain is vision-related [20]. The retina, in particular, is a direct extension of the brain, and the oculomotor system is intimately linked to the brain. Many of the structures within the brain that are most vulnerable to TBI and CVA are vision-related (i.e. the frontal, occipital, temporal and parietal lobes and the long axonal fibres connecting midbrain structures to cortex). Consequently, it comes as no surprise that the incidence of oculomotor and binocular dysfunctions resulting from a neurological event is very high.

The ability of the visual process to coordinate the two eyes to precisely align, fixate and track is critical to forming a three-dimensional perception of the world. The ability to effectively read, fuse images mediated by the two eyes, follow moving objects, detect targets and determine distance all depend on oculomotor functions [21].

Pursuits and saccades are the conjugated eye movements coordinated by the visual process and are most frequently affected by neurological events [9,22–25]. Smooth pursuits are slow conjugated eye movements made to keep the image of a moving object, or a stationary object in view during head movements with focal fixation. In contrast, saccades are very accurate, quick eye movements to align the retinal image of an object of interest onto the fovea. Saccades are initiated by the frontal lobes in conjunction the parietal lobes and mediated by the superior colliculus located in midbrain and the brain stem. The frontal lobes are often affected and vulnerable to concussive brain injury [24]. The visuo-spatial process through spatial mapping prior to conscious attention establishes a framework for accuracy and efficiency of saccades and pursuits. Frontal lobe injuries affect feedback orientation and initiation intent for accuracy of the saccades and pursuits.

Saccadic eye movements are critical for normal reading. A retrospective analysis conducted by Ciuffreda et. al. in a large outpatient civilian population diagnosed with TBI showed that over 51% of these patients had saccade deficits [22]. These data can be correlated with the high incidence of near vision problems affecting reading (61%) found in TBI

**Table II.** Most common binocular, perceptual and reading deficits resulting from war-related injury.

Deficit	Incidence (%)
Reading problem	60.9
Visuo-spatial deficit	30.4
Convergence dysfunction	30.4
Accommodative dysfunction	21.7
Pursuits/saccades dysfunction	19.6
Suppression	15.2
Neglect	8.7
Diplopia	6.5
Fixation/nystagmus	2.2

Veterans receiving care in the Palo Alto VA Polytrauma care facilities (i.e. loss of place, blurred near vision, diplopia) [9].

Accommodative dysfunctions (i.e., accommodative spasm, accommodative insufficiency, and accommodative infacility) may result in either intermittent or constantly blurred vision depending on the severity of the damage. These anomalies are also common sequelae of neurological events and can further aggravate other oculomotor functions [26]. Since vergence movements are accompanied by accommodation of the lens in order to prevent the blur induced from a poorly focused image, many binocular vision dysfunctions occur in association with defective accommodative convergence [27,28].

Oculomotor deficits resulting in accommodative and binocular vision dysfunctions may occur from damage to the cranial nerves (CN), including: CN III (oculomotor nerve); CN IV (trochlear nerve) and CN VI (abducens nerve) [29,30]. However, determination that there is damage to the cranial nerve and/or motor neuron is often not evident and only assumed. It has been stated in the literature that computed tomography (CT), magnetic resonance imaging (MRI) and magnetic resonance angiography of the brain are often normal [31].

Additionally, TBI-induced damage can affect ocular motility as well as binocular and accommodative functions [24,29]. Specifically, the motor fibres innervating accommodation are particularly susceptible to axonal injury because of the anatomical structure of the accommodative pathway and its multiple stages of motor innervations [32].

While binocular function has been studied and reviewed relative to pursuits, saccades, vergence, accommodation, phoria and strabismus, research demonstrates a visual process actively and dynamically coordinates and organizes these binocular functions.

Research with visual evoked potentials (VEP) pattern-reversal documents changes in the P-100 using prisms [11,33,34]. This will be discussed in greater depth later in this paper.

The balance of the bi-modal visual process is necessary for plasticity of the visual process. The ambient spatial visual process is 'gravity specific' and is the first process that we are born with. [10,20]. The ambient process provides organization to integrate the primitive reflexes enabling postural alignment upright against gravity and controlled movement. This establishes a platform base of vision that is preconscious and that matches information with the proprioceptive base of support (BOS). The feed-forward to the cortices provides spatial mapping and orients for anticipation of change in postural orientation through visual intent that becomes feedback to the thalamus, midbrain and brainstem.

This spatial component of vision serves as a platform to enable disassociation of the focal process occurring at approximately 4–6 months of age in relationship with the righting reflex. This begins the relationship between the processes of vision that enables plasticity so long as the feed-forward component of the platform base of vision provides the opportunity for the focal 'non-gravity specific' visual process to disassociate for interest and curiosity.

An TBI affects the balance between the bi-modal visual process. Research has demonstrated that the feed-forward and

feedback relationship between the spatial mapping of the higher cortices and the organization of the proprioceptive BOS becomes affected [31]. This compromise affects the plasticity of the visual process. The nature of plasticity of the visual process affects the ability of feed-forward to the frontal eye fields (FEF), supplementary eye fields (SEF) parietal and occipital lobes for spatial mapping affecting intent of oculomotor movement as well as initiation of the action.

Compromise of the spatial visual process leads to isolation on detail and disruption of the plasticity of visual processing. This begins the cascade effect that interferes with adaptability to environmental change. A compromise of the spatial visual process leaves the higher visual process without spatial context, and in turn, the bi-modal visual process becomes compromised.

Barnett and Singman discuss the effect of mTBI and the condition of 'tunnel vision' or isolation on detail. Reference is made to the use of prism lenses to affect this over-focalization on detail, leading to the characteristic of 'tunnel vision' [35].

Diffuse axonal injury can affect afferent and efferent visual function as well as the process that organizes the balance between visual processes. Since the etiology of the binocular dysfunction is from a neurological event, it provides a basis for the understanding that binocular dysfunction may be caused not only by injury to CN but also by compromise of visual processing in the brain. In turn, the means of rehabilitation through the evolving non-invasive NVPR provides a means of treating the visual processing disorder causing the binocular dysfunction and a means of re-establishing balance within the bi-modal visual process potentially affecting binocularity and visual skills.

NVPR incorporates the use of rehabilitative lenses, prisms, sectoral occlusion, etc., to affect the visual processing disorder causing the binocular dysfunction. This has implications for use in rehabilitation to affect visual processing in physical, occupational, speech and neuro-cognitive therapies.

## Vision: A Bi-Modal Process

The dichotomy of visual processing has been recognized and demonstrated by models of vision since the early part of the 20th century. Attempts to explain this dichotomy are represented in description of vision as a 'what/where', 'attention/pre-attention' and 'perceptual/motor' theories. These dichotomies are based on a visual process that is conscious. This has been the standard thinking throughout the 19th and 20th centuries. The 'what/where' concept is a generality applied to vision relating the 'what' component to the focal process delivering information about detail for identification and the 'where' component to the portion of vision associated with relationships of objects spatially to oneself and other objects. This concept does not account for the preconscious nature of the ambient process with proprioception and the BOS. The 'attention/pre-attention' concept orients towards the conscious seeing part of vision that provides concentration, identification and higher cognitive processes during the time of attention and the before. However, the emphasis is on attention, and it does not encompass the 'gravity specific' spatial visual process and its organization with the BOS. The

'perceptual/motor' concept involves the relationship of higher perception with balance and movement. This is important but as feedback orientation. Perceptual-motor is a representation of incomplete understanding of the ambient process and is oriented towards conscious perception.

These concepts can be used to describe relationships about vision but do not provide for the nature of preconscious spatial visual processing that is 'gravity specific' and related in development to the proprioceptive BOS.

According to Trevarthen, Liebowitz, Post and other researchers, there are two documented modes of visual processing: (1) the focal process; and (2) the ambient process [36,37]. While the focal process delivers information for the purpose of attention, concentration and higher cognitive visual processing, the ambient visual process delivers information for visuo-spatial awareness and is transmitted through fibres from approximately 20% of the peripheral retinas of both eyes. The latter is delivered to the area of the midbrain [10,37]. In the midbrain, visuo-spatial information is matched with information gathered from the kinesthetic, proprioceptive, tactile and vestibular systems. This function has been labelled the sensorimotor feedback loop [38,39]. The ambient visual process serves to provide visuo-spatial relationships to be matched with spatial information received from the sensorimotor systems [15,17]. A mismatch between the focal and ambient processes causes distortion in the relationship between the BOS and the egocenter relative to the BOS through the visual midline affecting postural alignment [18,40].

Spatial mapping is a critical component of visual processing. This is accomplished by a preconscious organization of spatial context with the 'gravity specific' spatial visual process matching information regarding postural orientation upright against gravity with the proprioceptive "BOS" initially. This occurs by matching spatial information between the superior colliculus (SC) and proprioceptive information received from brain stem regarding postural alignment.

The SC is receiving input from brain stem, cerebellum, vestibular system, occipital cortex and thalamus. The SC provides output to the occipital cortex, FEF and SEF, parietal lobe, thalamus, cerebellum and the vestibular system. The feed-forward component of this information to the frontal lobe provides context to the FEF and SEF to create spatial mapping for intent of action. The frontal lobe provides anticipation and expectation. The frontal fields are important for the intent of pursuits and saccades. The spatial mapping is critical for spatial organization affecting the sequence of ocular motility and visual decision-making. The SC is also important for initial spatial context of fusion.

The parietal lobe is critical for vision-auditory and tactile sensory matching of information. The posterior parietal lobe provides spatial match with the FEF and the SC. The parietal lobe is important for consciousness affecting position awareness. The parietal lobe receives input from the SC, occipital lobe and temporal lobe. It provides output to the FEF, SC, occipital lobe and temporal lobe. The parietal lobe utilizes the input for organizing with the occipital lobe to initiate the motor action of the pursuit, saccade and/or vergence movement of the eyes.

The temporal lobe matches visual information with auditory. It receives input from the FEF, SEF and thalamus, cerebellum, inferior colliculus and the parietal lobe. It provides output to the FEF and SEF, parietal lobe, midbrain and cerebellum as well as the occipital lobe. It is important to the bi-modal visual process with regard to temporal relationships established with the FEF spatial context of intention of action.

The brain stem provides a proprioceptive field for sensory organization spatially. It is developmentally oriented, and it establishes the early reflex patterns from which the sensorimotor systems organize and in turn integrate the reflexes. It serves to provide trunk stability and association of trunk-on-body movement. It receives input from the spinotectal tract for sensorimotor information and control of autonomic functions. The brain stem receives input from the spinotectal tract for sensorimotor information and control of autonomic functions. It provides output to the SC, FEF, parietal and temporal lobes.

The emphasis is that the cortices require input from midbrain, brain stem and thalamus of which vestibular input and cerebellar modulation is critical for organization of intent and action. As well, midbrain, thalamus and brain stem require feedback from the cortices for refinement of preconscious organization of spatial visual processing.

The neocortex is the outermost layer of the cortex. It is 2.5-mm thick and follows the contours of the folds of the cortex. It provides 80% of the interaction of the cortical brain. There are six layers of which layers I-III are myelinated axons and layers II and III project to other areas of the neocortex. Layer IV receives input connections from outside the neocortex especially from thalamus that is oriented to feed-forward. Layers V and VI are output connections to outside neocortex. This is especially true for thalamus and brain stem for feedback.

The visual neocortex is composed of the original six sub-layers plus an additional three layers. The additional three sub-layers are devoted to the significant increase in input from thalamus, SC and brain stem.

The parietal lobe, FEF, SEF and occipital lobe in conjunction are very important for spatial mapping to initiate intent and action related to pursuits, saccades and the vergences. The temporal lobe is important to provide a temporal context for the oculomotor movements in real time and related to matching of auditory spatial processing. However, this all requires feed-forward from the SC, thalamus and brain stem [10,13].

Although the occipital cortex is emphasized as the primary area of vision for purposes of seeing (i.e., retino-geniculocortical pathway), the SC is a critical area for organizing spatially directed eye movements and body-eye coordination (i.e., retino-tectal pathway) [17,41]. Sensorimotor matching begins in the midbrain and leads to the development of body awareness for posture, balance and spatial orientation. The SC also receives fibres from the spinotectal tract that connects with sensorimotor information areas from the spinal cord and medulla. In the midbrain, the sensorimotor matching of midline, body awareness, posture and orientation are established. Research suggests that horizontal and feedback connections demonstrate the integration of information that



underlies perception, whereas receptive field tuning properties demonstrate feed-forward processing [44,45].

The authors suggest that the role of feed-forward in visual processing initially organizes with proprioceptive input through the spinotectal tract influencing a component of the visuo-spatial process beginning at midbrain. This is where the SC provides spatial context for associated spatial mapping in the frontal lobes, SEF, the parietal lobes and the occipital lobes. Nashold has demonstrated that radio-tactic lesions of the SC produced exotropia [44]. Furthermore, feed-forward of this information from the SC appears to provide information from the midbrain to the occipital cortex where it is used to provide spatial context prior to focalization. This spatial information is important to binocularity and fusion within vision. The feed-forward process from the SC is delivered to the binocular coordination cells to provide a spatial context for the fusion process and ultimately binocularity [10,13,17,44,46–49]. Lesions in the SC and/or those affecting the match of ambient visual information with sensorimotor information will affect fusion and binocularity causing convergence insufficiency, divergence excess and exotropia [16,17]. Ketchum, et. al. discuss that movement distorts at lower thresholds with the absence of vision, suggesting that vision provides feed-forward in the presence of proprioception to improve the efficiency of movement [50].

Ganglion cells travelling from the retinas can be categorized physiologically and functionally into three types: P-cells (parvocellular), M-cells (magnocellular) and K-cells (konio-cellular). The M-cells and P-cells provide the physiological substrate for the ambient and focal processes, respectively. M-cells transmit visual information about shape and movement, but without detail. P-cells transmit detail information contained in shapes, but are much slower in process [51]. Thus, M-cells are the major component of the ambient visual process, and P-cells are the main component of the focal process. Some K-cells are direction-sensitive, suggesting that the K-pathway plays a role in motion encoding [51]. The retino-cortical-geniculate pathway combines M-, P- and K-cells to the occipital cortex, whereas the retino-tectal pathway provides only M-cells with axons directed to the SC [51,52]. In the midbrain, the sensorimotor information from the spinotectal tract from neck proprioceptors matches with the M-system information. Eye and neck proprioceptive messages contribute to the spatial coding of head-on-body orientation together with the extra-ocular proprioception and visual signals in the superior colliculus of the cat neck to contribute to the spatial coding of retinal input in visually oriented activities [53].

The combination of this information causes the ambient process to become greater than the M-system alone. The 'gestalt' of this relationship becomes a spatial grounding of the visual process [36]. The M-cells, although a component of the ambient process, are not inclusive [10].

The ambient visual process is not a conscious process as is the focal visual system.[36] It is a preconscious process, and because of its developmental hierarchy with proprioception, it is a 'gravity specific' process providing orientation for spatial mapping in the higher cortices beginning with being upright against gravity. The focal visual process is 'non-gravity

specific' and provides the means for organization of higher cortices for attention and concentration. In child development, the ability to begin to disassociate the focal visual process from the spatial process occurs in conjunction with the righting reflex at approximately 6 months. This occurs in the prone position as the child pushes up away from the horizontal plane against gravity and then disassociates vision for curiosity while maintaining extension of the trunk against gravity [10,20,49]. This disassociation between spatial (ambient) and attention (focal) visual processing is critical for the organization of head-on-body processing.

A mismatch between the ambient visual process and sensorimotor information can cause a spatial disrupt [13,42,45]. This has the potential of affecting binocularity. The ambient visual process supports spatial organization, posture and balance [10]. Traditionally, there has been greater emphasis on the conscious process of vision as an afferent relay than on the relationship between the pre-conscious spatial visual process and proprioceptive information related to head-on-body processing [54]. The bi-modal process has been given little attention clinically [13]. The focal vision process has predominantly been given attention for the provision of perception of vision (i.e., acuity object recognition, etc.) without recognition of the profound importance of the ambient process to support the conscious level of vision. Additionally, there are at least two higher cortical areas that become important to the distribution of the ambient process. Studies in both humans and monkeys found that wide-field-of-view provides motion input and are critical for vection [55]. Vection refers to the perception of self-motion induced by visual stimuli. These areas are the medial parietal occipital region that processes peripheral motion and the retro-insular region that is located at the parieto-temporal junction and distinguishes motion resulting only from self-motion in contrast to object motion [56].

### ***Compromise of the bi-modal vision process: Cytotoxicity***

Cytotoxicity can occur following an injury or lesion in the brain. At the site of injury, damaged cells often can release substances that will attack cellular function and in turn compromise cellular integrity of surrounding cells. Responses of this nature can affect visual processing. Injury from a TBI or other acquired neurological insults can cause a disassociation of the focal and ambient visual process including the M- and P-cellular systems by this cytotoxic response and/or by the shearing effect that causes a physical interference with axonal relationships [10,13]. The compromise of the focal and ambient visual processes often occurs due to the ischaemic-hyperaemia resulting in metabolic imbalances, which derange neuronal transmission and cell membrane permeability, causing potential cytotoxicity even in a minor whiplash accident [57].

### ***Characteristics of visual processing compromise: Post trauma vision syndrome (PTVS)***

Following a neurological event, visual signs and symptoms may occur (Table III). Characteristically, the visual examination may demonstrate binocular dysfunction such as

**Table III.** Characteristics and symptoms associated with the post-trauma vision syndrome.

Characteristics	Symptoms
Exotropia or exophoria	Diplopia
Accommodative dysfunction	Objects appearing to move
Convergence insufficiency	Poor concentration and attention
Low blink rate	Staring behavior
Spatial disorientation	Poor visual memory
Poor fixations and pursuits	Photophobia
Spatial disorientation	Associated neuro-motor difficulties
Increased myopia	Balance and coordination; posture
Hallucinations	

strabismus, convergence insufficiency, divergence excess, oculomotor dysfunction and accommodative insufficiency. These have been documented and are prevalent with neurological events, and are frequently seen in the TBI patient [58].

As was previously discussed, the matching of ambient (spatial) information with sensorimotor information creates the spatial context that is delivered through a feed-forward process to the binocular coordination cells and occipital cortex from primarily the SC [45,59]. At the level of the binocular coordination cells, the feed-forward processes provide a spatial context used for binding visual information from each eye in the process of fusion [16,17]. This process also provides spatial reference for the detail visual information that focalization manipulates for visual perception and cognitive processing [10,13]. Feedback from these higher processes develops the dynamics by which focal and ambient processes create the intention of how we see and attend to our visual environment [31,36]. The balance of feed-forward and feedback information together with the dynamics of the focal and ambient visual process establishes our plasticity in visual processing [10,13]. The dynamics of this plasticity is different for each person. The dynamics of the focal and the ambient visual processes enable adaptation to new information in order to predetermine how we will perceive our visual world and orient to posture upright against gravity.

Vision and sight have been studied extensively relative to its afferent relationship to the brain through a Stimulus-Response (S-R) action or learning model [60]. However, the S-R model does not explain the diffuse dysfunction that can occur following a concussion or TBI. A learning model that may offer greater understanding of performance as well as the balance of bi-modal visual processing is Response-Stimulus-Response (R-S-R). The motor component for organization has been demonstrated to be a strong influence for action models of learning [61]. This is considered a readiness response prior to stimulus introduction. Furthermore, Held's classical study of the 'kitten in the cradle' demonstrates that motor action is critical for sensory learning (Figure 1) [62].

Applying the R-S-R model to vision provides a new perspective in understanding. The S-R model is only one theoretical model of vision [63]. Dynamic visual processing and the R-S-R model is supported by the understanding of the dichotomous blend of focal and ambient visual processing providing feed-forward and feed-back information [31,63]. In order to create the perception of something such as becoming aware of an object in one's path, one must first be capable of setting up the conditioned readiness to avoid it, grasp it, etc. Additionally, the ability to perceive the object as a unit of

**Figure 1.** Spatial learning occurred for the kitten permitted to move but it did not occur for the kitten restricted of active movement despite both kittens having the same visual sensory experience.

'wholeness' reflects the integration of the 'state of conditioned readiness' to match the stimulus from the object [64].

Interference with the feed-forward ambient process, for example, from a neurological event affects the needed spatial context for fusion [16,17]. Convergence and accommodation, while in the process of including the intention of focalization from higher cognitive and visual perception, are ineffective unless oriented and organized first, or at least simultaneously involved with the feed-forward of ambient context [10,13,36,65]. In turn, over-focalization causes isolation to detail. The intensity of focalization and the lack of ambient (spatial) context produce limited reference for establishing oculomotor control of convergence, accommodation and fusion along the z-axis (the near-distance axis from the eyes projected to infinity). In turn, the z-axis can potentially become compressed from compromise of ambient visual context. This can yield the characteristics of convergence insufficiency, accommodative insufficiency, as well as in some cases, exotropia [10,13]. Trevarthen offers the basis of this understanding by discussing the role of a preconscious process that precedes conscious attention [43].

The compromise of the ambient-focal balance may yield over-focalization. We propose that over-focalization produces a reduction or in severe cases, a complete collapse of the ambient spatial base of visual function. Clinically, the authors have found that following a concussion or TBI, when a patient is asked to read one letter on a chart per step while walking towards the chart, they will report afterward that they only were aware of the letters on the chart and did not see the room while performing the activity. This offers the possibility that compromise of the spatial visual process affects conscious awareness of the spatial visual field, causing over-focalization.

Research has documented that following a TBI, the amplitude of the P-100 VEP pattern reversal increases with the addition of prisms in the experimental group compared to a decrease in the control group. This research emphasizes that the prisms are affecting a component of visual processing that is not conscious. This effect has been termed 'Post Trauma

Vision Syndrome (PTVS)' [11]. The authors correlate the binocular vision dysfunction following a TBI with compromise of the spatial visual process or ambient process. PTVS is a representation of the spatial compromise producing characteristics of this dysfunction. The characteristics are represented as convergence insufficiency, accommodative insufficiency and oculomotor dysfunction. Other researchers (Sarno et al. and Cuiffreda et al.) have also studied similar relationships using VEP findings [33,34]. An example of this is that persons with this imbalance in the visual process often have difficulty scanning (e.g. searching a shelf to find an object). A compromise of the visuo-spatial process, in turn, affects the stability of the spatial visual process. Persons with this dysfunction often describe their visual world as being over-stimulating and over-whelming. This can cause a person to become very uncomfortable in a busy-crowded environment.

The balance of the bi-modal visual process is necessary for plasticity of the visual process. The ambient spatial visual process is 'gravity specific' and is the first process that the child is born with [10,20]. The spatial visual process provides organization to integrate the primitive reflexes, enabling postural alignment upright against gravity and controlled movement. This establishes a platform base of vision that is preconscious and that matches information with the proprioceptive BOS. The feed-forward to the cortices provides spatial mapping and orients for anticipation of change in postural orientation through visual intent that becomes feedback to the thalamus, midbrain and brainstem.

This spatial component of vision serves as a platform to enable disassociation of the focal process, as mentioned previously occurring at approximately 6 months of age in relationship with the righting reflex. This begins the relationship between the processes of vision that enables plasticity so long as the feed-forward component of the platform base of vision provides the opportunity for the focal 'non-gravity specific' visual process to disassociate for interest and curiosity.

A TBI affects the balance between the bi-modal visual process. Research has demonstrated that the feed-forward and feedback relationship between the spatial mapping of the higher cortices and the organization of the proprioceptive BOS becomes affected [31]. This compromise affects the plasticity of the visual process. The nature of plasticity of the visual process affects the ability of feed-forward to the FEF, SEF, parietal and occipital lobes for spatial mapping affecting intent of oculomotor movement as well as initiation of the action. This compromise is the basis of PTVS that is demonstrated in the research that shows a reduction in the amplitude of the P-100 VEP and that is affected by the introduction of prisms. The prisms can affect the spatial visual process by creating improved balance between the bi-modal visual process. The effect causes an increase in the amplitude of the P-100 that was found to be statistically significant for those with TBI in the experimental group compared to the control group [11]. The importance of this is that the amplitude increase represents a re-balancing of the bi-modal visual process and an increase in plasticity of the visual process [11,33,34].

This is the condition of visual processing dysfunction that exists in PTVS. Specifically, the spatial visual process

represents preconscious expansion of the platform base of the bi-modal visual process, and the focal process represents compression or isolation on detail. Compromise of the spatial visual process leads to isolation on detail. This begins the cascade effect that interferes with adaptability to environmental change. The visual adaptability requires an R-S-R action system that organizes and processes spatial information with the proprioceptive BOS prior to interaction with detail. A compromise of the spatial visual process leaves the higher visual process without spatial context, and in turn, the bi-modal visual process becomes compromised.

The symptoms and characteristics of PTVS are not limited to concussion or TBI and can occur from various neurological conditions such as Parkinson's disease, multiple sclerosis, cerebrovascular accident, to name several [10,50].

PTVS may be present in autism. Autistic individuals describe a 'fragmentation' of their vision and isolation of details [66,67]. This appears to be an over-focalization with lack of ambient spatial reference.

By considering that vision problems such as convergence insufficiency, accommodative insufficiency, oculomotor dysfunction, exotropia represent compromise of the spatial visual process, it allows for consideration of a preconscious processing system that has been compromised as well as the potential for a new model to emerge for rehabilitation that includes the R-S-R concept of organization.

Evidence demonstrates that visual processing dysfunction is present with compromise of binocularity and visual skills. Evidence is also presented to demonstrate that prisms and sectoral occlusion can affect the amplitude of VEP while also causing a reduction in symptoms and improved visual skills. This suggests the possibility that the compromise of binocularity and visual skills occurring following a TBI are characteristics of a visual processing dysfunction. Further, this permits a new concept of understanding vision dysfunction to include bi-modal visual processing dysfunction as well as the potential for a new model of vision rehabilitation to be introduced.

It also engages new thinking that the neurological conditions causing the characteristics and symptoms of PTVS are a result of visual processing dysfunction as well as the potentials for rehabilitation to improve visual processing dysfunction [11,13].

The proposed protocol for diagnosis of PTVS is the first to determine the characteristics of PTVS through the neuro-optometric rehabilitation evaluation. The next step is to rule out PTVS by a P-100 pattern-reversal binocular VEP. The protocol for using prisms and bi-nasal occlusion has been reported by Padula and Argyris [68]. Evidence suggests that before applying the techniques of vision therapy to affect fusion, pursuits and saccadic fixations, clinicians are cautioned to rule out PTVS until the visual processing dysfunction has been treated through the use of lenses, base-in prism and possibly bi-nasal occlusion [10,13].

Postural adaptations that compensate for vision dysfunction have been reported in past research [49,69]. These adaptations have included secondary somatic muscular contracture and/or spasms [13]. As discussed previously, over-focalization causes intensity in vision without an adequate ambient spatial

match with sensorimotor information. Furthermore, clinical findings have demonstrated that over-focalization can produce an overflow of high abnormal postural tone particularly in the neck and shoulders. Thus, the lack of ambient spatial orientation can affect posture and may contribute to neck strain and headaches.

### **Visual postural imbalance: Visual midline shift syndrome (VMSS)**

The ambient visual process is primary in the establishment of postural orientation [13,18,19,65]. This process must match information with the sensorimotor systems. This is essential in order to establish spatial organization from which the relationships of visual midline are developed. This latter entity is important in optimal head and neck positioning, as well as postural organization [18,65,70]. Following a neurological event, a person may have postural difficulties affecting standing, ambulation, and/or position in a wheelchair, thereby causing spatial disorganization. Often the person is not consciously aware of this, and this may increase their risk of fall (RoF). The balance and postural difficulties may be the result of ambient mismatch with the sensorimotor systems, resulting in a shift in visual egocenter or what is referred to as the spatial experience of visual midline [13,18,19,65].

The compromise of the bi-modal visual process by a TBI underlies the characteristics of binocular dysfunction and disruption of the visual skills associated with pursuits, saccades and vergences.

The interruption of matching between the spatial 'gravity specific' visual process and the proprioceptive BOS following a neurological event affects the nature of the bi-modal visual process established from the earliest moments of development related to postural alignment upright against gravity. Once interrupted, a compromise of the feed-forward process creating accurate spatial mapping in the cortices associated with feedback to the midbrain, thalamus and brain stem for maintenance of postural alignment and weight shift during ambulation often occurs.

This interruption or compromise in bi-modal visual processing affects the egocentric organization of matching the centre of mass, located approximately an inch below the navel in the adult, and becomes shifted in relationship to the geometric centre associated with the step length and step width. The shift in centre of mass is affected by the mismatch of spatial information between the spatial visual process and the proprioceptive BOS. This mismatch has been termed 'Visual Midline Shift Syndrome (VMSS)' [18,19]. Research has documented that yoked prisms are effective in altering the relationship of this mismatch in the spatial visual process and the proprioceptive BOS. The effective change is a realignment of the centre of mass with the geometric centre of the length and width of corresponding footfalls during ambulation. The research has demonstrated improved balance and reduction in RoF amongst subjects with a neurological event affecting balance and posture.

Persons with a neurological dysfunction such as a hemiplegia or hemiparesis will predominantly demonstrate difficulty with weight shift to the affected side. This dysfunction

affecting motor function and postural orientation has been traditionally thought to be the inability to weight-bear on the affected side or, in the paradoxical case, to collapse into the affected side [40]. However, research has demonstrated that there is a corresponding shift of the visual midline most frequently away from the affected side that shifts the centre of mass (COM) away from the affected side [10,13,18]. The shift of visual midline essentially reinforces a lack of weight bearing on the affected side. Rehabilitation of this condition with yoked prisms has been described in a randomized-controlled trial [18]. Yoked prisms are utilized to centre the visual midline by countering the distortion within the ambient and sensorimotor mismatch. This distortion is caused by the relative compression and expansion of perceived visual space [10]. A shift of visual midline may occur laterally or in the anterior-posterior axis. Often this shift is in the anterior-posterior and medial-lateral axes simultaneously and will require treatment with oblique axes yoked prisms [10,13,18].

Clinical results have demonstrated that an anterior shift of VMSS often occurs with children who are autistic or 'on the spectrum'. This shift produces the behaviour of 'toe-walking' [10,13]. It has been clinically observed that many children respond to specifically oriented yoked prisms and can develop a 'heel-to-toe' walking pattern. It is suggested that the improvement occurs by establishing a balance between the ambient process and the focal process, causing a reduction or an elimination of the VMSS. Clinically, it has been found that the prisms can be reduced and often eliminated over time.

A prism has traditionally been used to treat strabismus by applying the specific amount of prism diopters to shift the image to align with the deviating eye. The prism shifts the image because the wedge of optical material compresses space and expands space [71]. Considering the bi-modal visual process, only the conscious or focal process sees the image shift. It was demonstrated in the research by Padula and colleagues that the yoked prisms had an effect on posture and balance by causing subjects to weight-bear on their affected side [18]. The potential explanation to this is that it is not the conscious portion of the bi-modal visual process that was involved in affecting this improved weight shift but the spatial or ambient visual process. The effect can be experienced if one is to wear yoked prisms (prisms with the base end in the same direction). The experience is that if, for example, 14 diopters of base right yoked prisms are worn while walking, the feeling will be of an increase in weight shift to the right or in the direction of the base end of the prisms. A possible explanation for this is that the focal process 'sees' the image shift. However, the spatial visual process is preconscious. It does not perceive the image shift but represents the expansion and compression of space as a shift by the person not the image. The result is that the spatial shift affects visual midline and motor-postural adaptation and is not produced by a conscious or higher sensory awareness. In fact, following a CVA or TBI, the person may not be aware of weight shifting abnormally. Even when the yoked prisms are introduced to improve weight shift, the person may still not be conscious of the improvement. Yoked prisms can be prescribed in conjunction with physical/occupational therapy to support the effort of these therapies in maintaining postural alignment and reducing RoF.



## Visual field dysfunction

Injuries beyond the optic chiasm can produce field defects that are always homonymous hemianopia, but are not necessarily congruous [19]. Depending upon the area of the cortex affected, the person may or may not be aware of the field loss [13,69]. Temporal lobe lesions often produce incongruous superior quadrantanopia with sloping margins. Parietal lobe lesions often cause a lack of awareness of the field loss [72]. Visuo-spatial neglect may manifest as a complete loss of visual perception on the side affected or as a relevant loss, such as in a field neglect [13].

A homonymous hemianopsia can be functionally very debilitating to the person in rehabilitation [73]. Visual field loss also affects positioning of the visual midline that can interfere with posture and balance. The individual with the homonymous hemianopia, in most cases, will shift the concept of visual midline towards the centre of the remaining field. Homonymous hemianopsia causes a VMSS and yoked prisms can be used effectively to realign the visual midline caused by this condition [74].

Enhanced sector prism systems can be used to affect function following a homonymous hemianopsia by increasing awareness of the peripheral visual field on a sensory level, whereas yoked prisms are used to affect the VMSS on a sensorimotor level [73,75]. Enhanced sector prism systems can be mounted binocularly or monocularly and positioned in spectacles to the side of the line of sight with the base end oriented in the direction of the field loss [13]. Clinically, the success in patient adaptation and use of enhanced sector prisms increases if the VMSS is treated first or simultaneously.

Research regarding spatial neglect has emphasized the visual process as an attention system [76]. Robertson, Lamb and Knight's research on the 'Effect of lesions of temporo-parietal junction on attention and perceptual processing in humans' demonstrates that the loci of a hemispheric lesion affects the subject's ability to see small (local) forms from large (global) forms. In particular, large lesions of the right hemisphere are more likely to produce the subject missing the global form than the local form. Subjects with large left hemispheric lesions are more likely to miss the global form than the local form.

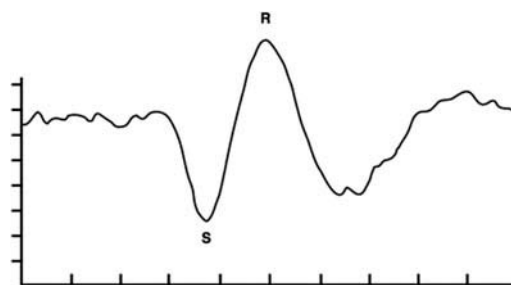
This research uses a Stimulus-Response (S-R) model, and the emphasis is on the afferent visual system to evaluate dysfunction produced hemispherically of the conscious mode of visual processing. The point of this paper is to establish that the nature of visual processing is not just for attention, recognition, concentration and identification but is also related to balance, movement, postural organization and spatial organization prior to conscious attention. In their well-designed study to assess spatial visual neglect based on hemispheric involvement, there was no discussion of postural alignment in relationship to visual processing of the subjects during testing. Rosetti et. al. has demonstrated that reduced visual processing plasticity causing spatial neglect can be rehabilitated by use of yoked prisms. This research has demonstrated that through use of yoked prisms, the effect of spatial neglect can be rehabilitated [77,78].

## Neuro-visual processing rehabilitation (NVPR)

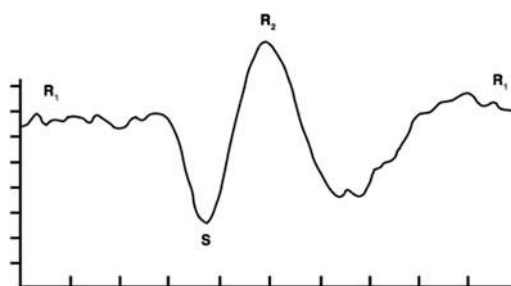
A model of vision that is bi-modal applied to the learning, and performance theory of R-S-R may provide new insights into dysfunction in vision following a neurological event. For example, the pattern reversal P-100 VEP has been considered primarily for the latency response between the stimulus presentation and the P-100 response (Figure 2).

Application of the R-S-R model to the VEP provides a new perspective to recognize that the preconscious component of the VEP occurs as a readiness or anticipation of the potential event of a stimulus presentation (Figure 3). This can be characterized as the response ( $R_1$ ) before the stimulus (S). Following the S presentation, a response ( $R_2$ ) occurs which maximizes the P-100 amplitude and is then released returning the process to the preconscious state of visual processing and readiness ( $R_1$ ) for the next stimulus event.

This model of organization of the bi-modal visual process in conjunction with the R-S-R model of performance provides a robust interpretation of visual performance as well as recognizing that the binocular dysfunction and related symptoms may be due to compromise of the bi-modal visual process and not damage to cranial nerve motor neurons. It opens the possibility that by affecting this compromise of bi-modal visual processing, the characteristics of binocular dysfunction as well as a reduction in symptoms caused by the compromised visual state can be reduced.



**Figure 2.** The traditional application of the stimulus-response presentation of the pattern change produces a P-100 representation of the response to the stimulus.



**Figure 3.** With the representation of the response-stimulus-response model, the VEP demonstrates that the readiness potential prepares for the event of the stimulus.  $R_1$  represents preconscious readiness organization of the spatial visual process. S represents the presentation of the stimulus.  $R_2$  represents the response to the stimulus (conscious).

## Discussion

The incidence of visual dysfunctions is common following a TBI.

The challenging part in understanding the ambient spatial visual process is that it comprises a preconscious organization of visual spatial information together with the proprioceptive BOS that occurs prior to 'visuo-spatial awareness'. This 'gravity specific' portion of the visual process establishes spatial mapping in the SEF of the frontal lobe as well as spatial mapping in the parietal and occipital lobes. The feed-forward nature of this process is *pre*-conscious, and it occurs prior to conscious visual spatial awareness. Since this is occurring prior to consciousness, it is important to consider what is the 'action' or 'learning theory' we are applying to understand the vision and the compromise of performance that can occur following an mTBI. The S-R model does not account for the preconscious organization of the 'gravity specific' organization of the ambient spatial visual process. The R-S-R action model provides an understanding that the preconscious organization of this spatial process with proprioception establishes a readiness that occurs as a response prior to the stimulus introduction. It readies the process to anticipate change to respond to a stimulus.

The consideration of the action or learning theory related to vision seems to have been overlooked as we have attempted to research and study vision throughout the 20th and early 21st century. However, research outcome is only interpreted by the question that we ask. If we do not ask the question regarding which action or learning model are we applying, the results will be interpreted only by the assumption that we function based on a Stimulus – Response learning style. By asking the question 'Is vision based on a response-stimulus-response model of action or learning?', our interpretation of behaviour and action can potentially be perceived in a new perspective. It is beyond the scope of this paper to expand on this concept, but the implications are profound.

This emerging model provides an understanding of bi-modal visual processing that is not S-R bound. The model also provides a framework for consideration that the dysfunction of oculomotor coordination and binocularity may not be a dysfunction of neuro-motor control of the extra-ocular muscles but in the compromise of the preconscious organization affecting higher order spatial mapping. The visual skills of pursuits, saccades and convergence must rely on the accuracy of spatial mapping as a platform for organization prior to initiation. This spatial context provides the efficiency of oculo-motor movements served by a preconscious process.

Considering these issues, the characteristics of binocular dysfunction are understood by the nature of the processing dysfunction as well as the postural imbalance often accompanying a neurological event. Further, vision as a bi-modal process offers some advantages when developing a model of rehabilitation not just to affect binocularity and visual skills but also to consider the effect of dysfunction of the visual process on balance, posture and movement. Dysfunction of the ambient visual process from a neurological event has been demonstrated to directly interfere with functional performance by causing PTVS and VMSS [10,13,15,17,18].

The use of lenses, prisms (yoked) and sectoral occlusion may be understood not on the effect on conscious visual awareness but on the preconscious nature of visual processing and the intimate relationship with motor-sensory processing.

The treatment for visual processing dysfunction using asymmetrical prisms and bi-nasal occlusion is an effective means of supporting and re-establishing the relationship in the bi-modal visual process between the ambient and focal processes, thereby improving binocularity and spatial orientation [13,18,19]. Through use of yoked prisms, treatment of VMSS can re-establish an appropriate match between the ambient visual process and the sensorimotor system affecting the relationship between the ambient visual midline and the proprioceptive BOS. In turn, this will induce weight bearing on the affected side and provide a more stable concept of visual egocenter improving balance and posture. Yoked prisms can also be used to affect the paradoxical VMSS as well [18].

Often individuals who have a combination of PTVS and VMSS can be treated to reduce symptoms by combining treatments for both conditions through the use of asymmetrical yoked prisms [10]. The result will be directly related to outcome in the overall rehabilitation of the individual.

Recent studies with the help of functional MRI and other technologies have demonstrated the re-mapping of the damaged brain areas following neurological damage, thus dispelling the myth that the brain loses its plasticity at an early age. These new discoveries have stimulated many new ideas and research that eventually will affect future clinical interventions.

The insight regarding the bi-modal process has the potential of bringing new perspectives for understanding the sequelae of visual symptoms and characteristics following an mTBI. This understanding can be applied inter-professionally. It also has the potential of new model of rehabilitation applied inter-professionally for those with an mTBI. The application of this new understanding to rehabilitate bi-modal visual processing and to affect the compression and expansion in visual space resulting from compromise to the ambient visual process can be made through the use of lenses, prisms and sectoral occlusion [12,13]. There is initial clinical evidence that treatment for visual conditions resulting from a neurological event is not time dependent. However, clinically, it has been found that the earlier the treatment is provided, the less the possibility of having to deal with habitual compensations.

## Acknowledgments

The authors would like to express their appreciation to Amy Frey for her assistance.

## Declaration of Interest

The authors declare that they have received no funding for this work or any related research.

## ORCID

William V. Padula  <http://orcid.org/0000-0001-8589-9606>

## References

1. National Center of Health Statistics. United States. Public Health Service Hyattsville. [Cited 2001] Available from: <http://www.cdc.gov/nchs>.
2. Scott P, Barsan WG. Stroke, transient ischemic attack, and other central focal conditions. In: Tintinalli JE, Kelen, GD, Stapczynsky JS, editors. Emergency medicine: A comprehensive study guide. 5th ed. New York: McGraw-Hill Companies; 2000.
3. Center of Disease Control and Protection (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 2003 [cited 2003] Available from: <http://www.cdc.gov>.
4. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. *J Head Trauma Rehabil*. 2006;21(5):375–8.
5. Powell JW, Barber-Foss KD. Traumatic brain injury in high school athletes. *JAMA*. 1999;282(10):958–63.
6. AOA supports efforts on Capitol Hill highlighting combat-related eye trauma. *Am Optometric Assoc News*. 2007;46(4):1.
7. Defense and Veteran Brain Injury Center. Washington, DC Walter Reed Army Medical Center. 2015 [cited 2007] Available from: <http://dvbic.org>.
8. Clark G, editor Incidence of neurological vision impairment in patients who suffer from an acquired brain injury. International Congress Series; 2005.
9. Goodrich GL, Kirby J, Cockerham G, Ingalla SP, Lew HL. Visual function in patients of a polytrauma rehabilitation center: A descriptive study. *J Rehabil Res Dev*. 2007;44(7):929–36.
10. Padula W. Neuro-visual processing rehabilitation: An integrated model of service. Santa Ana, CA: Optometric Extension Program Foundation Press; 2012.
11. Padula WV, Argyris S, Ray J. Visual evoked potentials (VEP) evaluating treatment for post-trauma vision syndrome (PTVS) in patients with traumatic brain injuries (TBI). *Brain Inj*. 1994;8(2):125–33.
12. Posner M, Rafal R. Cognitive Theories of attention and the rehabilitation of attentional defects. In: MJ Meier DL, Benton AC, editors. Neurophysiological rehabilitation. New York: Churchill Livingstone; 1987.
13. Padula W, Singman E, Magrum M, Munitz R. Evaluating and Treating Visual Dysfunction. In: Zasler N, Katz D, Zafonte R, editors. Brain injury medicine. New York: Demos Medical Publishing; 2013.
14. Hellerstein LF, Fishman B. Vision therapy and occupational therapy, an integrated approach. *J Behav Optom*. 1990;1:122–6.
15. Hart C, editor Disturbances of fusion following head injury. Proceedings of the Royal Society of Medicine, London; 1964.
16. Carrol R. Acute loss of fusional convergence following head trauma. *Arch Ophthalmol*. 1984;88:57–9.
17. Stanworth A. Defects of ocular movement and fusion after head injury. *Br J Ophthalmol*. 1974;58(3):266–71.
18. Padula WV, Nelson CA, Padula WV, Benabib R, Yilmaz T, Krevisky S. Modifying postural adaptation following a CVA through prismatic shift of visuo-spatial egocenter. *Brain Inj*. 2009;23(6):566–76.
19. Padula WV, Subramanian P, Spurling A, Jenness J. Risk of fall (RoF) intervention by affecting visual egocenter through gait analysis and yoked prisms. *NeuroRehabilitation*. 2015;37(2):305–14.
20. Gesell A, Ilg F, Bullis F. It's Development in infant and child. Santa Fe, CA: Optometric Extension Program Publishers; 1998.
21. Ogle KN. Research in binocular vision. Philadelphia (PA): Saunders; 1950.
22. Ciuffreda KJ, Kapoor N, Rutner D, Suchoff IB, Han ME, Craig S. Occurrence of oculomotor dysfunctions in acquired brain injury: a retrospective analysis. *Optometry*. 2007;78(4):155–61.
23. Ciuffreda KJ, Rutner D, Kapoor N, Suchoff IB, Craig S, Han ME. Vision therapy for oculomotor dysfunctions in acquired brain injury: a retrospective analysis. *Optometry*. 2008;79(1):18–22.
24. Kapoor N, Ciuffreda KJ. Vision problem. In: Silver JM, Mcallister TW, Yudofsky SC, editors. Textbook of traumatic brain injury. Washington, DC: American Psychiatric Publishing, Inc; 2005.
25. Lew HL, Poole JH, Vanderploeg RD, Goodrich GL, Dekelboum S, Guillory SB, Sigford B, Cifu DX. Program development and defining characteristics of returning military in a VA Polytrauma Network Site. *J Rehabil Res Dev*. 2007;44(7):1027–34.
26. Daum KM. Predicting results in the orthoptic treatment of accommodative dysfunction. *Am J Optom Physiol Opt*. 1984;61(3):184–9.
27. Cooper J, Feldman J, Selenow A, Fair R, Buccerio F, MacDonald D, Levy M. Reduction of asthenopia after accommodative facility training. *Am J Optom Physiol Opt*. 1987;64(6):430–6.
28. Cooper J, Selenow A, Ciuffreda KJ, Feldman J, Faverty J, Hokoda SC, Silver J. Reduction of asthenopia in patients with convergence insufficiency after fusional vergence training. *Am J Optom Physiol Opt*. 1983;60(12):982–9.
29. Baker RS, Epstein AD. Ocular motor abnormalities from head trauma. *Surv Ophthalmol*. 1991;35(4):245–67.
30. Suchoff IB, Gianutsos R. Rehabilitative optometric interventions for the acquired brain injury adult. In: Graboys M, Garrison SJ, Hart KA, editors. Physical medicine and rehabilitation: The complete approach. New York (NY): Blackwell Scientific; 2000.
31. Kafaligonul H, Breitmeyer BG, Ogmen H. Feedforward and feedback processes in vision. *Front Psychol*. 2015;6:279.
32. Ciuffreda K. Accommodation, the pupil, and presbyopia. In: Benjamin WJ, editor. Borish's clinical refraction. Philadelphia: WB Saunders; 1998.
33. 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;27(1):41–7.
34. Sarno S, Erasmus LP, Lippert G, Frey M, Lipp B, Schlaegel W. Electrophysiological correlates of visual impairments after traumatic brain injury. *Vision Res*. 2000;40(21):3029–38.
35. Barnett BP, Singman EL. Vision concerns after mild traumatic brain injury. *Curr Treat Options Neurol*. 2015;17(2):329.
36. Liebowitz HW, Post RB, editor. The two modes of processing concept and some implications. Mahwah, NH: Erlbaum; 1982.
37. Trevarthen C, Sperry RW. Perceptual unity of the ambient visual field in human commissurotomy patients. *Brain*. 1973;96(3):547–70.
38. Borish I. Paralytic strabismus. Clinical refraction. Chicago (IL): The Professional Press, Inc; 2006. p. 1253–82.
39. Padula W, Wu L, Vicci V, Thomas J, Nelson CA, Gottlieb D, Suter P, Politzer T, Benabib R. Evaluating and treating visual dysfunction. In: Zasler N, Katz D, Zafonte RD, editor. Brain injury medicine. New York: Demos Medical Publishing; 2007. p. 511–28.
40. Nelson. C. Improving movement and postural control in children with neuromotor dysfunction. *Clinician's View*. 2002.
41. Carroll RP, Seaber JH. Acute loss of fusional convergence following head trauma. *Am Orthopt J*. 1974;24:57–9.
42. Liebowitz H, Post R. The two modes of processing concept and some implications.. Mahwah, HH: Erlbaum; 1982.
43. Trevarthen CB. Two mechanisms of vision in primates. *Psychol Forsch*. 1968;31(4):299–348.
44. Lamme VA, Roelfsema PR. The distinct modes of vision offered by feedforward and recurrent processing. *Trends Neurosci*. 2000;23(11):571–9.
45. Lamme VA, Super H, Spekreijse H. Feedforward, horizontal, and feedback processing in the visual cortex. *Curr Opin Neurobiol*. 1998;8(4):529–35.
46. Nashold BS, Jr., Seaber JH. Defects of ocular motility after stereotactic midbrain lesions in man. *Arch Ophthalmol*. 1972;88(3):245–8.
47. Hart C. Disturbances of fusion following head injury. Paper presented at: Proceeding of the Royal Society of Medicine. 1964. London.
48. Moore J. Brain Atlas and Functional Systems.. Rockville, MD: American Occupational Therapy Association; 1993.

49. Benabib R, Nelson C. Efficiency in visual skills and postural control: A dynamic interaction. *Occup Ther Pract*. 1993;3:57–6.
50. Ketchum C, Natalia V, Dounskaia N. The Role of Vision in the Control of Continuous Multijoint Movements. *J Mot Behav*. 2006;38(1):29–44.
51. Casagrande VA. A third parallel visual pathway to primate area V1. *Trends Neurosci*. 1994;17(7):305–10.
52. Morand S, Thut G, de Peralta RG, Clarke S, Khateb A, Landis T, Michel CM. Electrophysiological evidence for fast visual processing through the human koniocellular pathway when stimuli move. *Cereb Cortex*. 2000;10(8):817–25.
53. Roll R, Velay JL, Roll JP. Eye and neck proprioceptive messages contribute to the spatial coding of retinal input in visually oriented activities. *Exp Brain Res*. 1991;85(2):423–31.
54. Paulsen L. *Neurobiology and treatment of traumatic disassociation*. New York, NY: Springer Pub Co; 2014.
55. Previc FH, Beer J, Liotti M, Blakemore C, Fox P. Is “ambient vision” distributed in the brain? Effects of wide-field-view visual yaw motion on PET activation. *J Vestib Res*. 2000;10(4–5):221–5.
56. Cheng K, Fujita H, Kanno I, Miura S, Tanaka K. Human cortical regions activated by wide-field visual motion: an H2(15)O PET study. *J Neurophysiol*. 1995;74(1):413–27.
57. Barkhoudarian G, Hovda DA, Giza CC. The molecular pathophysiology of concussive brain injury. *Clin Sports Med*. 2011;30(1):33–48, vii–iii.
58. Suchoff I, Gianutsos R. *Rehabilitative optometric interventions for the acquired brain injury adult*. New York, NY: Blackwell Scientific; 2000.
59. Posner M, Raichel M. *Images of the mind*. New York, NY: Scientific American. New York Library; 1994.
60. Shunk D. *Learning theories an educational perspective*, 6th ed. Boston, MA: Pearson; 2012.
61. Hommel B. Toward an action concept model of stimulus-response learning. *Theoretical issues of stimulus response compatibility*. North Holland: Elsevier; 1997.
62. Held R. Dissociation of visual functions by deprivation and rearrangement. *Psychol Forsch*. 1968;31:338–48.
63. Holland PC. Cognitive versus stimulus-response theories of learning. *Learn Behav*. 2008;36(3):227–41.
64. Mackay DM. A mind’s eye view of the brain. *Prog Brain Res*. 1965;17:321–32.
65. Nelson C. Improving movement and postural control in children with neuromotor dysfunction. *Clinician’s View*. 2002.
66. Bach-y-Rita P. *Brain mechanisms in sensory substitution*. New York (NY): American Press; 1972.
67. Grandin T. *Thinking in pictures: My life with autism*. New York, NY: Random House; 1996.
68. Padula WV, Argyris S. Post trauma vision syndrome and visual midline shift syndrome. *J Neuro Rehab*. 1996;6:165–71.
69. Bobath B. *Abnormal postural reflex activity caused by brain lesions*. London: Westworth; 1983.
70. Nudo R. Neuroscientific bases for occupational and physical therapy interventions. In: Zasler ND, Katz DI, Zafonte RD, editors. *Brain injury medicine: Principles and practice*. New York, NY: Demos Publisher; 2007.
71. Streff J. Optical effects of plano prism with curved surfaces. *J Am Optom Assoc*. 1972;44:717–21.
72. Streff JW. Visual rehabilitation of hemianoptic head trauma patients emphasizing ambient pathways. *NeuroRehabilitation*. 1996;6(3):173–81.
73. I Suchoff KC, N Kapoor. *Visual and vestibular consequences of acquired brain injury*. Santa Ana, CA: Optometric Extension Publishers; 2001.
74. Perennou D. Postural disorders and spatial neglect in stroke patients: a strong association. *Restor Neurol Neurosci*. 2006;24(4–6):319–34.
75. Gottlieb DD, Fuhr A, Hatch WV, Wright KD. Neuro-optometric facilitation of vision recovery after acquired brain injury. *NeuroRehabilitation*. 1998;11(3):175–99.
76. Roberson L, Lamb M, Knight R. Effects of lesions of temporal-parietal junction on perceptual and attentional processing in humans. *J Neurosci*. 1988;10:3757–69.
77. Kerkhoff G, Rossetti Y. Plasticity in spatial neglect: recovery and rehabilitation. *Restor Neurol Neurosci*. 2006;24(4–6):201–6.
78. Rossetti Y, Rode G, Pisella L, Farne A, Li L, Boisson D, Perenin MT. Prism adaptation to a rightward optical deviation rehabilitates left hemispatial neglect. *Nature*. 1998;395(6698):166–9.



Copyright of Brain Injury is the property of Taylor & Francis Ltd and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.