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

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ABSTRACT_

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

Background: Patients with acquired brain injury (ABI) often experience sensorimotor

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

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

INTRODUCTION Acquired Brain Injury

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



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

Epidemiology

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

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

Common Symptoms Associated with ABI

Because of the recent advances in technology, the survival rate of patients with ABI has increased. However, this also means that more individuals may be suffering from sensorimotor visual deficits as a result of the ABI affecting their activities of daily living (ADLs) and quality of life (QOL).⁸ These visual deficits vary from mild to severe, depending on the etiology, location and severity of the ABI. The disturbance can affect the patient's ability to process visually related tasks which require higher level cortical processing, such as visual closure and figure ground, or visually guided information such as encoding visual spatial relations with the motor output system in directing themselves in a crowded environment or grasping an object.⁹ The most common problems associated with ABI have been categorized as Post Trauma Vision Syndrome¹⁰ which includes: binocular dysfunctions (specifically, convergence insufficiency), acquired strabismus, diplopia, blurred vision, ocular motility deficits, visual field loss, photosensitivity, vestibular dysfunction, and visual perceptual-motor dysfunctions.^{11,12,13}

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

According to a study by Ciuffreda et al in 2007, the most common ocular motor dysfunctions in a visually symptomatic TBI sample included convergence insufficiency (56.3%), deficits of saccades (51.3%), accommodation (41%), strabismus (25.6%), and cranial nerve palsy (6.9%).¹⁴ In terms of the effectiveness of rehabilitation, one study with nine TBI and five stroke patients suggested that both subjective symptoms and objective eye movement recordings significantly improved post vision therapy.¹⁵ A larger scale study by Ciuffreda et al in 2008 also demonstrated that 90 percent of patients had improvements in signs and reduction in symptoms after the completion of in office vision therapy targeting oculomotor dysfunctions.¹⁶

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

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

Why Is This Important for Optometrists?

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

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

Key Definitions

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

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



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

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

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

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

NEUROLOGY OF DISEQUILIBRIUM Brief Overview of the Vestibular System

The human balance system has three afferent systems: vestibular, visual, and somatosensory, while the efferent system is composed of multiple neurological pathways that partially overlap and are redundant.¹³ Maintaining a



Figure 1: Vestibular apparatus- semi-circular canals otoliths organs. Courtesy of Wikipedia Commons.

sense of balance during changing situations requires all of these pathways to be in sync in order to coordinate motor responses of the limbs, trunk and eyes to the incoming afferent information.

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

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



transmitted to the association cortex regions for decoding.¹³

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

Central processing of vestibular information takes place in two main locations: the vestibular nuclear complex and the cerebellum.³⁰ Vestibular afferent information from receptor hair cells travels to the vestibular nuclei and brainstem (central component) via cranial nerve VIII where it is combined with visual, somatosensory, and cerebellar information for maintenance of balance and equilibrium.¹³ From the vestibular nuclei, axons then split and some fibers extend and communicate with the cerebellum. The cerebellum serves as an adaptive role and modifies the VOR as needed and recalibrates the neural input from the vestibular system within the inner ears to the motor output of the EOMs.³² Additionally, in 1992 Scudder and Fuchs³¹ described eye velocity-head velocity neurons as being part of the pathways allowing vision to override or enhance vestibular information. This study sets the groundwork for neuro-optometric rehabilitation in the management of patients with vestibular dysfunction.

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



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

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

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

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

Dorsal Stream Processing Deficit

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

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

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

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

NEURO-OPTOMETRIC REHABILITATION MODEL

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

How to Avoid Sensation of Disequilibrium

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



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

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

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

TREATMENT AND MANAGEMENT

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

Table 1: 5 Components f	for Effe	ective NORT	with Examples
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Component	Description	Example	
Motivation and Active Participation	-Motivation empowers the patient to be an active participant. -Goals allow therapists and patients to measure success and keep patients engaged. -Therapy procedures are presented using various problem- solving tasks allowing for active participation.	-Set realistic goals at the beginning of therapy (both doctor and patient) -Design therapy procedures that may be incorporated into every day life	
Feedback	-Using multi-sensory alerts to recalibrate and refine encoded responses	-Anti-suppression procedures -Physiological diplopia -Polarized vectograms -Buzzers and beep -Verbal feedbacks	
Repetition	-Repetition helps to encourage synaptic strength	-Home therapy techniques performed 3 times a week for 20 minutes each duration	
Motor Match to Sensory Mismatch	-Patient visually guides motor response to specific visual input enhancing sensorimotor recalibration	-Localization with a pointer such as with vectograms -Prism lens shift -Stereoscopic cards in a stereoscope	
Intermodal Integration	 Procedures should incorporate multi- sensory tasks for patients to react to. Procedures may entail gradual multi- sensory input and output requiring visual, touch, auditory, proprioceptive and balance. Requires patients to filter extraneous sensory information while attending to stimuli important for solving the visual task. 	-Metronome -Balance board -Auditory beep -Yoked prism -Distraction	

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

- 1. Motivation and active participation: It is crucial to set realistic goals at the start of NORT. These goals allow therapists to measure success post therapy and allow the patient to stay engaged and focused on the tasks to be completed. Therapy procedures are presented in various problem-solving tasks allowing for active participation. The level of difficulty is also gradually increased.
- 2. Feedback: Feedback is achieved via antisuppression procedures, physiological diplopia, stereopsis, buzzers and other alerting systems. Verbal feedback is also important in enabling the patient to know when they have done well or improve in areas where they are not.
- 3. Repetition: It is important to use different therapy procedures in order to keep patients engaged. Additionally, home visual therapy should be performed approximately three times a week for about 20 minutes each time. It is more beneficial to break up home therapy in smaller duration of time than to complete all tasks in one sitting.
- 4. Motor Match to a Sensory Mismatch: Functioning in a multi-visually stimulating environment requires integration of various streams in the cortex. However, traumatized brain, the speed а in of processing is often reduced and visually-guided motor skills are often compromised.³⁹ Therapy procedures that incorporate various lens types, such as prism, for example, can manipulate how visual information is perceived (sensory mismatch). If the therapy procedure requires a motor response (motor match)

to this mismatch, at the same time providing a neural feedback (a beep or buzz) as to whether the response is correct or incorrect will further facilitate the recalibration and enhancement of this sensorimotor response. An example of this concept can be illustrated with a common therapy procedure utilizing polaroid filters and vectograms, in which localization of image is the motor match and the "Small In, Larger Out" (SILO) phenomenon is the sensory mismatch. Other procedures utilizing this concept can include the use of yoked prism lenses, polarized lenses and red/green glasses.

5. Intermodal integration: NORT procedures should incorporate multi-sensory tasks. Procedures may entail gradual multisensory input and output requiring visual, touch, auditory, proprioceptive and balance. ABI patients often have filtering difficulty and are easily overwhelmed with information. Thus, this top-down processing component requires patients to filter extraneous sensory information while attending to stimuli important for solving the visual task.

Lenses, Prisms and Tints

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



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

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

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

Phase 2 of NORT: Enhance Binocular Control Alignment and Sustenance

There are two main goals in this phase. The first is to develop adequate binocular control and second is to enhance speed of fusion recovery. The former is achieved as a result of neuromuscular and visuo-motor control of sustaining ocular alignment with clear and comfortable single vision. Various procedures are used to increase the facility of the accommodativeconvergence system. Procedures may include vectograms with emphasis on localization, stereopsis and perception of SILO; Brock string with emphasis on physiological diplopia; computerized equipment with emphasis on integration of vision with other sensory inputs. Enhancement of fusional recovery utilizes both static and dynamic therapy procedures such as headshake and walking with Brock string. Procedures often incorporate prisms and lenses to provide feedback and to quickly re-establish single vision with changes in gaze. An excellent procedure is performed with the Brock string in which the patient closes his or her eyes and visualizes where the bead is located. As soon as they open their eyes, they must refuse the bead back to one as quickly as possible.

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

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

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

CONCLUSION

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



Phase 1: Enhance Stability of Visual Input System

-Enhance the accuracy of fixation, pursuits, and saccades -Enhance the facility of accommodation

-Symptoms addressed: blurred near/distance, slow to shift focus (near to far to near), difficulty taking notes, pulling or tugging sensation around eyes, loss of place while reading

Phase 2: Enhance Binocular Control Alignment and Sustenance

-Develop adequate compensating fusion ranges, enhancing facility and speed of accommodativeconvergence, and increasing speed of fusion recovery

-Symptoms addressed: poor eye-hand coordination, face or head turn, head tilt, discomfort general fatigue while reading, diplopia

Phase 3: Develop Speed of Visual Information Processing & Stability of Output

-Stabilize binocularity and fusion facility

-Enhance dynamic fusion associated with top-down control of saccadic and convergence

- -Vestibular and multi-sensory integrations with a focus on recalibration of the VOR
- -Loading procedures

-Symptoms addressed: loss of balance and disequilibrium, poor posture, dizziness, poor coordination, disorientation, bothered by movement in spatial world, bothered by noises in environment, decreased attention span, reduced concentration ability

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

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

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