Article

Rehabilitation of Persons with Visual Sequelae Resulting from Traumatic Brain Injury

COVD Committee on Rehabilitative Optometry
Claude Valenti, OD, FCOVD, Chairperson

INTRODUCTION

The College of Optometrists in Vision Development's (COVD) executive board in the fall of 2000 requested its Committee on Rehabilitative Optometry to draft a paper providing information specific to vision and traumatic brain injury. In conjunction with the executive members of NORA (Neuro Optometric Rehabilitation Association) the committee began a 2-year project to review the literature after 1990 and provide a summary of scientifically based understandings, guidelines and recommendations for effective rehabilitation measures for persons with visually related traumatic brain injury.

OBJECTIVE

To provide nonoptometric professionals and the lay public with the most current understanding of the diagnosis, treatment, and clinical course of events following visual insults resulting from traumatic brain injury (TBI).

TBI is broadly defined as brain injury attributable to an externally inflicted insult resulting in a clinically measured abnormality of an individual's physical, cognitive, and/or psychosocial functioning. TBI runs a wide gamut of degree, ranging from less than a few seconds of a person's blacking out with immediate and complete recovery following the insult, to near death insults resulting in protracted coma and severely affected function necessitating long-term care and rehabilitation. The gray zones of degree, referred to as mild TBI, occur when the actual impact appears inconsequential, but neurological integrity has been affected, often with mild to moderate physical symptoms and complaints but significant cognitive involvement, such as losing the ability to read as one did before the trauma. Common causes are motor vehicle accidents, work-related accidents, falls, acts of violence, war, and sports injuries. It is estimated that 1.5 to 2 million TBI cases occur each year in the United States. Of these, approximately 70,000 to 90,000 cases in a year require long-term care from substantial loss of functioning. The impact of TBI on society is great. There is a significant economic loss of work productivity of the TBI individual, as well as great costs for diagnoses, treatment, and care. Families are disrupted and torn. Once witnessed, the aftermath of TBI tears at the threads of society's moral conscious as we search for more effective means of prevention. When prevention efforts fail, there is a concerted effort on the part of professionals from varied disciplines working together to rebuild the world of the affected population. Therapeutic interventions include, surgery, pharmacologic management, rehabilitative therapies, assistive compensatory technology and environmental manipulation, educational services, and counseling. These interventions are administered in hospitals, day treatment and residential programs, skilled nursing facilities, private professional practices, schools and colleges, community centers, and the home. An integral part of the overall management of TBI is the administration of care specifically targeted to help with the visual sequelae resulting from TBI.

WHAT ARE THE IMPORTANT VISION FUNCTIONS?

The human visual system is neurologically ubiquitous. This makes for a system that is very resilient and sophisticated, but at the same time very susceptible to injury because it is neurologically spread over a large morphological area. The neurology involved is both sensory and motor. The retina of each eye carries 1 million neurons in each optic nerve trunk to the boney area of the sella turcica, situated in the center of the skull, where partial decussation of optic nerve fibers occur. The occipital cortex (primary visual cortex), located at the lower back of the cranium, is one of the fire major areas for visual information processing. Following this initial extraction of neurological information, visual projections leave occipital cortex to be relayed to an estimated thirty distinct visual areas in the human brain, each of which receives a complete or partial map of the visual world. These projections are made in temporal, parietal and frontal areas, as well as the amygdala in the limbic system. An example of one of these projections is middle temporal (MT), where visual motion is processed and area V4, where color vision is processed. It is with these higher-level visual projections that neurological integration occurs with audition, language, balance and posture, cognition and emotions.

A broad look at the neurological mapping of the sensory aspects of the visual system reveals two parallel neurological streams, thalamic (cortex), and collicular (brain stem). The thalamic pathway diverges into two more pathways; fiber projections to the parietal lobe carrying information concerning navigation, depth perception, spatial orientation, and visual direction sense, and projections to the temporal lobe carrying information concerning object recognition, identification, and qualities of detail and color. The collicular pathway, more primitive phylogenetically than the thalamic, carries information concerned with orienting eye movements. The cerebellum plays a role in refining oculomotor movements.

The sensory visual system is also classified according to its scope. That is, according to central and peripheral vision. Central vision encompasses the macular point of fixation and no more than 10 degrees off fixation. Peripheral vision is all of the vision remaining in superior, inferior, and lateral quadrants.

The motor visual system includes all the major external oculomotor recti muscles for each eye, as well as the cranial nerves IIIn, IVn, and VIn, their nuclei located in pons, medulla oblongotta, and midbrain, and the cortical representation for control of coordinated eye movements. Eye movements are classified into; *conjugate,* (saccadic and pursuit movements); *position maintenance,* (oculomotor reflexes); and *disconjugate,* (binocular movements for convergence and divergence).

The ciliary muscle is located inside the eye and is responsible for accommodative control. This motor system is mediated through the autonomic nervous system, taking its origin in the T1, T2 sympathetic chain and the parasympathetic portion of the IIIn nuclei.

The important visual functions can be summarized into three categories.

1. Functions for clear central imaging and wide peripheral scope.
   These are, visual acuity, accommodation, and peripheral visual fields.
2. Functions for interpretation of visual information for meaning, understanding, and integration with other major neurological systems in the body.
   These are perceptual visual functions.
3. Functions for motor movement of the eyes, navigation and balance of the body.
   These are saccadic and pursuit eye movements, visual ocular reflex eye movements, convergence and divergence, binocular fusion and stereopsis.

WHAT IS THE EXTENT TO WHICH TBI RESULTS IN VISUAL FUNCTION SEQUELAE AND WHAT ARE THE SPECIFIC VISUAL FUNCTIONS THAT MAY BE AFFECTED FROM TBI?

Visual functions are affected to a great degree in the TBI population. A review of many studies revealed that several visual functions frequently are associated with TBI. Disorders of binocular coordination occur in approximately 40% of the TBI population. These include exo, eso, and vertical deviation, with exo deviations occurring more than three times as much. Disorders of oculomotor function occur

in approximately 40% of the TBI population. These include disorders of pursuit, saccadic, and ocular fixation. An increase in nystagmus is seen in the TBI population. Disorders of accommodative function occur in approximately 10% of the TBI population. Refractive disorders are present in this population, and it is not uncommon for refractive status to change significantly following TBI. Visual field disorders occur in approximately 30% of the TBI population. These include hemianopsias and visual field neglect.

There are numerous reports of perceptual visual disturbances resulting from TBI. These are typically reported in the literature with case histories. These include, but are not limited to, disorders of visual motion recognition, interferences with reading and letter and object recognition, disorders of visual memory and visual imagery, disorders of visual attention, disorders of color perception, disorders of face recognition, disorders of visual direction awareness, disorders of visual recognition of emotion, and disorders of figure ground distinctions.

Post-traumatic vision syndrome has been reported in the literature. This syndrome, associated with TBI, defines a cluster of abnormal visual findings characterized by exophoric or exotropic binocular coordination, reduced accommodation ability, altered central and peripheral visual functions, and altered spatial awareness as measured with midline shifts.

Although each individual suffering a TBI incident may be diagnosed with depressed visual functions in specifically limited defined areas, the profile of overall visual disturbance for each particular individual can be quite varied. The specific combination of visual function disturbance is unique to each individual. The extent to which a particular visual dysfunction may impair an individual's abilities is determined by the presence of other associated visual dysfunctions, as well as dysfunctions in other neurological systems. The visual system, being so neurologically complex, diverse, and encompassing such a variety of different functions, as well as integrating with such a variety of other neurological systems, is the cause for many varying combinations, which when looked at as a whole, ultimately defines the extent of visual sequelae resulting from TBI.

WHAT IS KNOWN ABOUT THE MECHANISMS UNDERLYING AFFECTED VISUAL FUNCTIONS RESULTING FROM TBI AND HOW DOES THAT EXPAND OUR UNDERSTANDING FOR TREATMENT?

The pathophysiological effects of TBI occur at the cellular level (individual neuron), the level of connected networks of such neurons (neural networks), and the further elaboration and integration of these networks (visual perception/cognitive function). Abnormal chemical changes have been found to occur at the metabolic level of individual neurons, as well as abnormalities with communication chemicals between neurons responsible for modulating either excitatory or inhibitory responses.

TBI-inflicted visual disturbances are caused from frank neurological impairment. The mechanisms for TBI visual disturbances are unique in the overall arena of visual abnormalities in that often neurology is significantly disrupted. On the one hand, visual abnormalities typically occur from genetic propensities or poor neurological development and lack of refinement of function with experience-dependent neuronal networks, caused by poor environmental experience or practice, while on the other hand, with TBI, visual disturbances can occur from loss of the entire neurology subserving a specific or several visual functions. This distinction of mechanism typically results in more severe visual sequelae for TBI. Even in cases of mild TBI, the underlying mechanism for visual disturbance can be severe, resulting in profound loss of function.

The time course for recovery is often protracted (months to years) and reflects different stages and substages of physiologic and metabolic cellular and neuronal network changes. The injured brain does have some capacity to recover, and some lines of research are exploring the effects on cellular plasticity from exogenous agents (*e.g.,* nerve growth factors, tissue transplantation, gene therapy) in conjunction with stimulation of experience-dependent neuronal networks in controlled, complex environments.

It follows that treatment outcomes for TBI are often less than expected from treatments for non-TBI-related visual abnormalities. The

underlying mechanisms of recovery at the cellular and neuronal network level, being complex over a prolonged period of time, in some cases lifelong, necessitates a treatment regimen that monitors the individual closely to determine when and if maximal responses may be achieved. It may be that introduction of a particular treatment regimen (*e.g.,* lenses, prisms, rehabilitative vision therapy), proved ineffective at one point in time of recovery, may prove to be very effective when introduced at another point in time of recovery, or when other areas of dysfunction, seemingly unrelated to vision function, have been improved through other avenues of treatment.

HOW MAY AFFECTED VISUAL FUNCTIONS HAVE AN IMPACT ON QUALITY OF LIFE OF A TBI INDIVIDUAL?

The potential areas in an individual's life that may be affected from vision abnormalities resulting from TBI are enormous, because society demands so much from our vision functions and abilities. TBI individuals may have had no visual problems before the trauma. They may have never had any cause for concern, nor felt a need for a vision examination. After trauma their vision, which was their primary sense modality from which the stability of their environment was based, may be altered. Some individuals may have potential vision problems before the trauma (*e.g.,* intermittent strabismus, accommodative infacility), and the shock of trauma may cause these to become manifest in symptoms unnoticed before. Even individuals who experienced overt vision problems before the trauma, and may have even sought treatment for these problems, after trauma may see a different world. In each of these cases, before trauma, individuals learned over the course of their lives to rely and act upon visual information that was consistent to them, even if faulted, by anticipating and setting up expectations, usually unknowingly. What appears to be most devastating to some TBI individuals is that when visual sequelae occur, there is not only a dramatic and sudden potential loss of visual information, but also a *distortion* of visual information input and interpretation. It is akin to suddenly finding yourself driving a car in England, where right hand turns in traffic are difficult because everything is reversed from that in the US, and although visual information is present and may even *appear* to be undistorted, previously learned visual behavior habits are strained, strange, confusing, ineffective, and potentially dangerous. In addition, the TBI individual often does not have the means to understand or correct the problem. Common symptoms reported are; photophobia, blurred vision, double vision, dizziness, insecurity, visual confusion, blank spots in visual field, pain and discomfort, headache, difficulty moving, bumping into things, not responding to images in certain parts of the visual field, reduced memory, difficulty reading or recognizing letters or objects, trouble judging space, sluggish eye movements, difficulty discriminating color, and false movement of objects.

Visual sequelae resulting from head trauma transfer into reduced performance in activities that use specific vision functions. In some cases, the individual cannot return to prior activities because there is not enough vision function to support the activity in its simplest form. In this regard, the effects on an individual's quality of life are dependent not only on the specific vision functions affected, but consideration must also be given to what visual functions were needed to support activities the individual was engaged in before trauma. If, for example, an individual held a position as a loan officer, precisely filling out forms with long strings of numbers at a desk and working on a computer for long durations, and TBI resulted in constant double vision, blurred vision, reduced convergence, compromised saccadic ability, asthenopia, and headache with near use of the eyes, then the quality of life of this individual would be substantially affected because to carry out this position efficiently requires clear, single, comfortable near vision, with quick visual discrimination and stamina. The same individual, holding a position as a bellman, even with the same disrupted visual profile, would be most likely be less affected in his quality of life because his position has different visual demands.

Activities that are commonly compromised as a result of the visual sequelae from TBI are;

those involving reading, such as academic pursuits, desk jobs, especially those requiring visual discrimination decisions, driving, jobs and avocations demanding fine motor discriminations and manipulations, and sport-related activities.

WHAT ARE THE COMMON THERAPEUTIC INTERVENTIONS FOR AFFECTED VISUAL FUNCTIONS RESULTING FROM TBI; WHAT IS THEIR SCIENTIFIC BASIS AND HOW EFFECTIVE ARE THEY?

During the acute phase of trauma, surgery and pharmacologic therapies may be employed to restore and preserve vision function. Often these interventions are insufficient in and of themselves to prevent resulting vision anomalies. Once the individual has stabilized, further diagnoses can be made to determine what areas of vision function continue to be abnormal. At this point, other therapeutic interventions can be instituted. These begin with lenses. Lenses are an effective tool to focus light for different working distances. Lenses have been found to ameliorate abnormal refractive conditions resulting from TBI. Astigmatic, minus, and plus lens combinations have all been shown to be effective. It is not uncommon for young adult TBI individuals to benefit from bifocal prescriptions if they show presbyopic-like symptoms caused by abnormal accommodative functioning. Tints, sometimes specific in color, as well as occlusion, both monocular and partial binocular, have been found to alleviate photophobic and diplopic symptoms as well as enhance contrast awareness.

Prisms have been found to be extremely helpful in the TBI population. Monocular as well as binocular prisms help to expand field awareness in individuals with field neglect as well as hemianopsia and quandranopsia. Prisms have been shown to be helpful for increasing fusion capability with constant and intermittent strabismus, both lateral and vertical. Cyclo strabismus does not respond to prisms. Yoked prisms have been found to be helpful in moving positions of gaze to null points with nystagmus, realigning midline for individuals with midline shifts, improving head and body posture, enhancing spatial awareness, and shifting positions of gaze for optimal binocular fusion, such as with A and V binocular syndromes.

Lenses, although extremely helpful, often do not completely resolve vision sequelae resulting from TBI, especially oculomotor, binocular, and perceptual anomalies. Vision rehabilitative therapy is a common treatment approach, which may be utilized in conjunction with lenses and prisms. Vision therapy allows for the patient to be in a controlled environment with graded activities under professional supervision and utilizes specific instrumentation to stimulate and cultivate neurons and neurological networks associated with the various vision functions. Vision therapy has been found to be helpful in treating motor and sensory anomalies of vision, as well as integrative and visual perception/cognitive anomalies. Vision therapy requires the TBI individual to be seen over a period of time, (months or years), for optimal restoration of function. Although vision therapy is helpful, it is limited in its ability to restore vision function completely. Often, vision therapy begins as a rehabilitative therapy, with its goal for complete restoration of function and elimination of symptoms. Once TBI individuals reach a plateau of vision function, goals are redirected to teaching compensatory skills so that the individual can achieve improved performance even if vision function is still compromised.

Currently, given the pathophysiology of visual sequelae resulting from TBI, the majority of TBI individuals will maintain some degree of vision dysfunction even when all therapies have been utilized. It is important to be mindful that despite current limitations of therapies to remediate vision dysfunction completely, many TBI individuals will lead productive and fulfilled lives when they understand that they are now different individuals with specific limitations and visions needs. It is this understanding that often is reached by the TBI individual over the course of vision therapy treatments, as they understand first hand the how, what, and why of their vision problems, and this may be one of the yet unresearched, but very important benefits from long-term care, allowing the individual to move on.

BASED ON THE ANSWERS TO THESE QUESTIONS, WHAT CAN BE RECOMMENDED REGARDING THE CARE OF TBI INDIVIDUALS?

- Society and professionals should continue to stress prevention in all areas where the potential exists for TBI. Given that there are no known cures, communities should be educated in the unfortunate, long-lasting vision dysfunction consequences that may potentially result from TBI, in an effort to reduce the number of TBI incidents per year.
- Individuals with TBI need emergency care immediately following trauma. It must be stressed that all surgical and pharmacological interventions implemented in the initial stages of trauma have a significant impact on the degree of long-standing vision dysfunction that may result.
- Once an individual has stabilized, individuals with TBI should have access to carefully conducted and comprehensive vision evaluations to assess the visual integrity of functions for clear central imaging and peripheral scope (visual acuity, accommodation, peripheral visual fields), functions for interpretation of visual information for meaning, understanding, and integration with other major neurological systems in the body (perceptual visual functions, midline shifts), functions for motor movement of the eyes, navigation and balance of the body (saccadic and pursuit eye movements, visual ocular reflex eye movements, convergence and divergence, binocular fusion, and stereopsis).
- Individuals diagnosed with visual dysfunctions resulting from TBI should have access to a careful evaluation regarding responses to lenses. This should include a comparison of past refractive status to current refractive status, keeping in mind that refractive status may change significantly, and presbyopic-like symptoms may occur with young adults. The effect of occlusion and tints in reducing symptoms of diplopia, midline shifts, spatial disorientation, and photophobia should be carefully studied. The effect of prisms, (monocular, binocular disconjugate, and binocular yoked) should be carefully studied for their effect on improving field awareness, binocular and oculomotor capability, spatial awareness, and posture and balance.
- Individuals with visual sequelae resulting from TBI should have access to rehabilitation efforts that include vision therapy. Vision therapy should begin as soon as possible to optimize benefits, in a hospital or a private facility. It should be conducted only with trained professionals (optometrists or ophthalmologists trained in vision therapy, or certified vision therapists). Vision therapy should be coordinated with other rehabilitation efforts and carefully documented and evaluated for its effectiveness. Once vision therapy is completed, the individual should be monitored at regular intervals to determine changes and stability of improvements.
- Rehabilitation efforts should include modifications of the individual's environment to accommodate visual dysfunctions. This includes the use of assisted technologies. Individuals with visual sequelae resulting from TBI should have access to professionals who can make modification recommendations for more efficient work, home, and social environments.
- Educational programs are needed to increase the degree to which the general community's awareness of vision problems affect the lives of individuals with TBI, especially for academic, work, and home environments. Family members should have access to professionals who can help them understand the behaviors of individuals with visual sequelae resulting from TBI.

BIBLIOGRAPHY

NIH Consensus Development Panel on Rehabilitation of Persons with Traumatic Brain Injury. Rehabilitation of persons with traumatic brain injury. *JAMA.* 1999;282(10).

Suchoff I, Kapoor N, Waxman R, Ference W. The occurrence of ocular and visual dysfunctions in an acquired brain-injured patient sample. *J AOA.* 1999;70(5):301–308.