Clinical Management of Traumatic Brain Injury Induced Intermittent Exotropia in a Primary Care Practice

Travis M. Johnson, OD
Stillwater, Minnesota

Abstract

Background: *Traumatic brain injuries are unfortunately common occurrences. In a healthy individual with a normally functioning nervous system, a sudden disruption will change their life in an instant. It is often difficult for patients that have experienced a traumatic brain injury to rehabilitate. The severity of the brain injury is significantly related to the level of sensory and motor deficit.*

Case Report: *This case report is an example of a patient who experienced a motor vehicle accident that immediately changed his binocular vision status. The patient's chief complaint was described as "difficulty keeping his eye lined up." Other complaints included glare, tracking images, and trouble concentrating. His job as an Information Technology (IT) professional was hindered by this sudden-onset vision disturbance. The patient suffered a choroidal rupture in the left eye secondary to the accident. Through a plan of home vision support activities in free-space and with computers, the patient was able to return to work free from the symptoms that had been plaguing him.*

Conclusions: *Correcting refractive error, prescribing prism, occlusion therapy, ocular calisthenics, and sensory/motor training are necessary in the management of acquired noncomitant deviations. Whether or not your practice provides in-office vision therapy should not be a limiting factor in your treatment of patients that have suffered a traumatic brain injury. This case report serves as a prime example of how it can be accomplished.*

Key Words

acquired noncomitant deviations, ocular calisthenics, strabismus, traumatic brain injury, vision therapy

Introduction

A traumatic brain injury (TBI) is an accident involving the head that results in an insult to the white matter and/or gray matter.[1-6] Open head trauma occurs when a penetration of the skull directly invades brain tissue. Closed head trauma involves a combination of forces (acceleration, deceleration, and torsion) as a result of sudden contact with the bony prominences. Acceleration forces occur when a steady head is struck by a rapidly moving object. Deceleration forces occur when a head in motion strikes a motionless object. Torsional forces are shearing forces that occur when the head is forcibly rotated on axis, damaging the cranial nerves and midbrain.[1,2,7,8] The traumatic event is termed the primary injury. Secondary injuries include pressure on the brain from bleeding, leaking fluids, and the swelling that occurs as the body tries to heal itself.[2] This pressure then further damages the brain tissue since the brain is enclosed in the bony structure of the skull.

Most visual deficiencies rarely last longer than six months; long-standing disconjugate eye movements and pupil abnormalities are more likely due to damage within the cavernous sinus, hemorrhages in the circle of Willis, or other cortical or sub-cortical involvements in the midbrain and brainstem rather than from direct damage to the nerves. Midbrain and brainstem lesions can also lead to disruption of the accommodative/vergence relationship, conjugate gaze palsies, nystagmus, and a variety of disturbances of resting fixation.[1-6, 9-11]

Sub-cortical elements referencing the cerebellum and the superior colliculus are other areas to consider in cases of TBI. The cerebellum is involved in the near triad of accommodation, vergence, and pupillary constriction. The superior colliculus is the primary oculo-motor control center. Cortical elements of the brain are the frontal, parietal, temporal, and occipital cortices. The frontal cortex holds Brodmann's area #8. This area of the brain contains the frontal eye fields and is involved in the voluntary control of shift of gaze. A significant amount of sensory input and motoric outflow exist in the frontal cortex. The ramifications of injury to this area of the brain are unpredictable. The parietal cortex is responsible for motion detection, ego-centric location, and voluntary pursuit eye movements. The temporal cortex is the "what" pathway. It helps identify and inspect images transmitted to the occipital cortex. The occipital cortex is the processing center with outflow to virtually every part of the visual system. Damage to the occipital cortex can follow obvious patterns that are measurable; however, damage to the occipital cortex

Table 1: Examination findings from the initial examination and all follow-up evaluations.

Procedure	17 Sept 2005	6 Oct 2005	5 Nov 2005	6 Jan 2006	9 Jan 2007
Visual Acuity Uncorrected OD, OS	Distance 20/20 Near 20/60	Distance 20/20 Near 20/60	Distance 20/20 Near 20/60	Distance 20/20 OD and 20/30 OS Near 20/40	Distance 20/20 Near 20/80
Phoria/Tropia	6 m 10 IAXT 55 cm 25 IAXT	6 m 10 IAXT 55 cm 25 IAXT	6 m 8 IAXT 55 cm 25 IAXT	6 m 7 XP 55 cm 14 IAXT	6 m 5 XP 55 cm 12 IAXT
Base In	X/7/4 Risley Prism X/13/9 HTS	X/8/5 Risley Prism X/20/22 HTS	X/11/9 Risley Prism X/36/33 HTS	X/11/9 Risley Prism X/36/33 HTS	X/10/8 Risley Prism X/28/24 HTS
Base Out	X/16/11 Risley Prism X/35/35 HTS System	X/21/13 Risley Prism X/55/55 HTS System	X/23/16 Risley Prism X/55/55 HTS System	X/26/19 Risley Prism X/55/55 HTS System	X/22/14 Risley Prism X/55/55 HTS System
Least Plus to 20/20 Near	+1.00 over subj dist	+1.00 over subj dist	+0.75 over subj dist	+0.75 over subj dist	+1.25 over subj dist
Pursuits HTS Response Time	0.82 seconds	0.75 seconds	0.72 seconds	0.69 seconds	0.77 seconds
Saccades HTS Response Time	1.04 seconds	0.92 seconds	0.83 seconds	0.79 seconds	0.91 seconds
Accommodative Facility with Add	11 cycles per minute	14 cycles per minute	21 cycles per minute	29 cycles per minute	14 cycles per minute

can manifest in random consequences that are also unpredictable.[1-6, 11]

The examination of the TBI patient should include a thorough case history with emphasis on the details of the injury itself. The date and time of the trauma, the initial symptoms, the level of consciousness at the time of the injury, and the treatment provided should be queried. Visual acuities, ocular motilities, and pupil, binocular, and accommodative testing are typical entrance tests that are necessary. These results will help guide the remainder of the examination. Refraction, ocular health, and visual fields are also a part of a standard battery of tests in any comprehensive vision examination, but deviation from normal protocols may be necessary to elicit the information necessary for a TBI workup.[3-7, 9, 10]

Case Report

A 51-year-old white male presented to the clinic on 17 September 2005 for an examination and evaluation of an intermittent eye turn OS. The patient had been involved in a car accident six months prior. He was evaluated for traumatic injuries to the left side of his body and head, including the orbit, immediately after the accident. He was diagnosed by a local ophthalmology clinic with a traumatic choroidal rupture. The patient's complaint of intermittent diplopia was not addressed at that visit. The patient was prescribed reading glasses and released with an annual examination recall.

The patient's chief complaint was what he described as "difficulty keeping his eye lined up." He was aware that his left eye was problematic but reported no previous alignment issues prior to the car accident. He also noticed a "blank spot" with his left eye when he covered his right eye. The ophthalmologist had described this to him as a bruise on the retina from the car accident. The patient experienced the phenomenon of tracking an image and having that image disappear before he felt it was past his field of vision. The patient also complained of glare and light sensitivity, tired eyes, difficulty sustaining focus at near, and intermittent double vision throughout the day with spots and floaters in his left eye. The patient was not taking medication and was not aware of allergies to medicines. He showed normal affect and responsiveness to questioning. His family's ocular history was negative for eye disease; however, his father was diabetic and dealt with some random vision issues from the diabetes.

The patient's uncorrected distance acuity was 20/20 OD, OS, and OU. The uncorrected near acuity was 20/60 OD, 20/80 OS, and 20/60 OU. Table 1 shows the examination findings from the initial examination as well as all subsequent evaluations. The saturation color on red and blue cap tests revealed a mildly decreased degree of saturation for the left eye. Ocular motilities were full OU. Pupils were equal, round, and reactive to light with no afferent pupillary defect. Threshold visual field was normal for the right eye, but showed an area of significantly reduced sensitivity in the upper nasal quadrant OS. Amsler grid was normal OD and distorted OS consistent with the area of defect on the visual field. Ishihara color test was normal OU; however, the colors were not as brilliant OS. Fusion was easily broken on cover testing. The distance and near cover test showed an alternating intermittent exotropia greater at near. The vergence information obtained was measured with the HTS home therapy system. The anaglyphic nature of the program encourages binocular vision with regard to the ductions. The ductions showed greater than expected results for the patient.

Refraction revealed -0.25D /+2.00 add OU. The base out (BO) ranges at near for this patient were measured at the maximum level with the HTS home therapy system, thus it was not useful to employ Sheard's criterion or Percival's criterion for prescribing prism. Several prism amounts were demon-

strated to the patient. Forced choice was used to find a prism amount that was most comfortable. The patient preferred 4 prism diopters base-in (BI) which was evaluated in a trial frame for 20 minutes. He reported a pleasant sense of stability and clarity with this prescription.

Slit lamp biomicroscopy of the anterior segment was not significant except for two plus two cells with a posterior vitreous detachment in the left eye. The posterior segment was examined with dilation. The cup to disc ratio was .2/.2 OU. The OD peripheral fundus examination showed a peripheral retinal atrophic scar superotemporally, with no tears or peripheral cystoid changes. The peripheral fundus exam OS showed peripheral cystoid changes and a pigmented choroidal lesion inferonasally measuring two disc diameters in size. There was no subretinal fluid around the inferonasal lesion and a choroidal rupture along the inferotemporal arcade within the macular region. There was a small nevus superonasally.

The differential diagnosis included cerebral vascular accident/aneurysm, intracranial mass lesion, cranial nerve III palsy, myasthenia gravis, and TBI. Cerebral vascular accident/aneurysm are both conditions that are typically associated with high blood pressure and/or diabetes.[12] Intracranial mass lesion has associated symptoms of headache, vomiting, memory loss, gait ataxia, urinary incontinence, papilledema, and other neurological issues. A cranial nerve III palsy will present with a ptosis. The ptosis will often mask the motility restriction; however, when the lid is lifted, the affected eye will have difficulty in adduction, elevation, and depression. The most common ocular findings in myasthenia gravis include droopy eyelids that appear worse at the end of the day, ptosis, orbicularis weakness, Cogan's lid twitch (transient eyelid retraction following an extended period of downgaze), limitation of ocular motility, paradoxical lid retraction, exposure keratitis, and intermittent diplopia.[13]

The diagnoses were as follows:

- Myopia OU
- Presbyopia OU
- Noncomitant, Intermittent alternating exotropia in primary gaze
- Choridal rupture OS

Options were discussed regarding what type of spectacle correction to prescribe. Given the cost involved for two separate pairs of glasses, the patient elected to try progressive addition lenses. The prescription was -0.25 sphere OU with a +2.00 add OU. A total of 4 pd BI was split evenly between the two eyes. The patient was given a regimen of ocular calisthenics, vision training activities, and a computer-based accommodation/vergence program. This regimen consisted of pursuit eye movement training in a monocular setting. The Marsden ball and Groffman visual tracing were used. The patient was also prescribed saccadic training activities in a monocular setting. The Developmental Eye Movement test and the Wayne saccadic star were used. Fusion activities were also prescribed. The patient used vectograms and anaglyphic materials to increase vergence ranges.

The patient was dispensed the HTS computer program as part of his therapy. The activities of the computer program included training pursuits, saccades, accommodation, and vergence ranges. The anaglyphic nature of the program also trained anti-suppression and simultaneous perception.

The retinal defect was photodocumented with Optomap technology (Figure 1). The patient was given an Amsler grid and trained to evaluate the area of concern in the left eye.

Follow up #1

The patient returned on 6 October 2005 for a vision therapy evaluation. He explained that he was having difficulty adjusting to progressive lenses. His distance vision was 20/20 OD, OS, and OU, and his ability to sustain alignment was better; however, his near activities were restricted. Full examination data can be found in Table 1. He elected to wear two separate pairs of glasses as a solution to this problem. The patient did not notice a change to the contrast sensitivity or Amsler grid OS. He was also working with the vision therapy regimen as prescribed previously. Distance spectacle prescription was -0.25D 2 BI OU. Near spectacle correction was +1.50D 2 BI OU. The patient was asked to return to the clinic in four weeks for an evaluation.

Follow up #2

The patient returned on 5 November 2005. The Amsler grid testing was consistent with previous examinations. The patient reported that he was able to have better control over his ocular alignment. His BI vergence ranges improved to 29 break/28 recovery and his BO ranges improved to 55 break/55 recovery. Improvement in pursuits, saccades, and accommodative facility were documented. The ocular motilities showed an improvement in range of motion in the temporal quadrant OS. The current vision therapy regimen with the addition of vertical base range training was continued. He was asked to return to the clinic in four weeks.

Follow up #3

The patient returned on 6 January 2006. The contrast difference and Amsler grid testing were unchanged from previous exams. The BI vergence ranges improved to 36 break/33 recovery and the BO ranges remained at testing limits of 55 break/55 recovery. The patient was excited about his progress visually and reported that he was having fun with his new found skills of fusion. He was asked to continue his current vision training regimen and to return to the clinic.

Follow up #4

The patient returned to the clinic on 4 February 2006. He reported that his vision training was continuing to improve his visual performance; however, he was concerned that he was experiencing a new disturbance in the left eye. He described that a portion of his vision OS was "smooshed" or compressed when compared to the image OD. The Amsler grid showed an expanded area of involvement OS. The uncorrected distance visual acuity was 20/20 OD, 20/30 OS. Anterior segment evaluation showed no change in comparison to the initial examination. The posterior segment evaluation showed the presence of a choroidal rupture along the inferotemporal arcade within the macular region with subretinal fluid, hemorrhage, and exudate in the subfoveal space. In the juxtafoveal space inferiorly there were retinal pigment changes and a choroidal rupture. The Optomap retinal scan was preformed and aided in the diagnosis of this condition. The Optomap from the original visit was compared with the

Figure 1: Traumatic Choroidal Rupture-September 2005

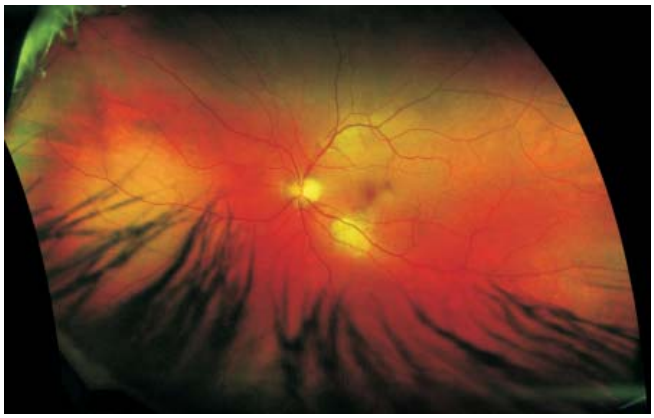
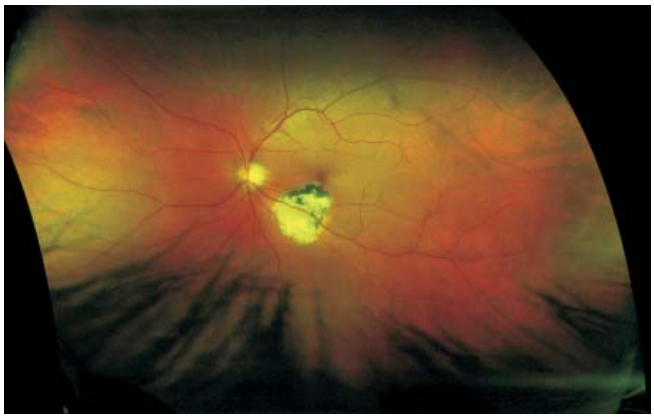


Figure 2: Choroidal rupture with developing choroidal neovascular membrane-February 2006



Optomap from this visit (Figure 2). The patient was referred to a local retinologist for evaluation and treatment.

Follow up #5

The patient was lost to follow up until 15 January 2007. Correspondence had been sent from the Retina Center between his 6 February 2006 and the 15 January 2007 examination. The correspondence detailed the evaluation, diagnosis, and management of a choroidal neovascular membrane (CNVM) OS. The retinal specialist treated the CNVM with thermal laser macular photocoagulation on 27 February 2006. The patient was re-evaluated on 14 March 2006. Fluorescein angiogram demonstrated a small punctate recurrence of the CNVM at the superior border. The retinal specialist re-treated the CNVM with thermal laser photocoagulation and intravitreal triamcinolone acetonide. The patient was followed monthly through June of 2006. He was instructed to follow up with our office every three months thereafter. The patient returned to the clinic on 15 January 2007. He reported that his vision remained stable with no recurrence of the symptoms. He also reported that his visual skills had remained strong and that his voluntary control over his ocular alignment continued to provide him stable vision.

Discussion

The patient referenced in the case report was impacted by closed head trauma and torsional forces from his car accident. Whether the accident was the primary cause or a secondary effect is unclear. The fact that motor alignment was disrupted would point to damage in the midbrain. Cranial nerves III, IV, V, and VI must pass through a portal in order to reach their destination. Shearing torsional forces in these portals can cause subsequent damage impacting these nerves. This could result in measurable damage or deinnervation to the nerves. A variety of problems are also possible with these torsional forces. Strabismus, ptosis, lagophthalmos, pupil abnormalities, and corneal anesthesia are some possible visual consequences.[1,2,7,8]

With regard to clinical care for TBI in the primary care setting, references to general themes are helpful in classifying what areas of the brain are affected during a traumatic injury.

- Diagnosis of the damage to the supra-nuclear elements involves much discussion. The frontal cortex is responsible for motor control, motor planning, abstract thinking, foresight, mature judgment, and tactfulness. Damage to this area may cause impairment in learning tasks that require movements in response to stimuli and in voluntary saccades.
- The temporal cortex is responsible for visual recognition, object localization, memory function, and auditory function. It can be characterized as the "what" area of the brain. Damage to this area may cause oculomotor deficits and receptive aphasia.
- The parietal cortex is responsible for integration of sensory information. This integration involves sensory information that helps develop the "where" in the relationship of objects to each other. The parietal cortex is also responsible for proprioceptive, kinesthetic, and vestibular inputs that contribute to the extrapersonal space relationships of an individual. Damage to this area may cause poor figure/ground analysis, poor spatial organization, and inappropriate body and/or eye movements.
- The occipital cortex is responsible for sensory reception of visual stimuli. The occipital cortex sends information to the parietal cortex and the temporal cortex. Damage to this area may cause hemianopsias with or without macular sparing, optic apraxia, and pursuit deficits.[1,3-6,11]
- Damage to the right hemisphere, excluding frontal cortex, may result in left hemiplegia and hemianopsia, agnosia, reduced contrast sensitivity, and neglect of the left side of space and/or self. It may also result in spatial inaccuracies in judgments of relative speed or motion and distances between objects.
- Damage to the left hemisphere, excluding frontal cortex, may result in right hemiplegia and hemianopsia, neglect of the right side, and aphasias.[1,3-6]
- Injury to the thalamus and the hypothalamus has profound effects on ocular motilities. The pulse-step function in saccadic eye movements arises first in the pons and then goes on to the medulla. The pre-motor nuclei involve the III nerve nucleus, also known as the Edinger-Westphal nucleus located in the thalamus. Damage to this area would affect medial gaze, lateral gaze, and vertical gaze.
- The medial longitudinal fasciculus has motoric outflow. This motoric outflow involves cranial nerves III, IV, V, VI, VII, and VIII. This area of the brain is

often called the pre-motor final common pathway to the orbit. Damage to this area could alter the accommodation/vergence ratio, the pursuits, the saccades, the vestibular-ocular system, the phorias, the ocular motilities, the sensory input, and the motor outflow.[3-6,8]

Treatment and management of TBIs often require a team approach. Neurologists, occupational therapists, physical therapists, ophthalmologists, and optometrists all have a stake in restoring the quality of life to an individual that has suffered a TBI. There are significantly different manifestations of TBI and there is no one right way to approach them. Developing strategies with a team of professionals is necessary and should be coordinated as soon as a diagnosis can be ascertained. Review of the strategies developed must be performed at regular intervals in order to update the rehabilitative management. Occlusion therapy, prescribing prism, correcting refractive error, ocular calisthenics, and sensory/motor training may be necessary in the management of these patients. With respect to muscle paresis, exercises designed to stimulate the paretic eye to engage in all diagnostic action fields, especially in the field of action of the damaged muscle, may prevent contracture and help regain function of the paretic eye. Increasing the motor fusion ranges is also very important when dealing with cases of recent onset noncomitant deviations.[3, 14]

Conclusion

Optometrists have an important role in the rehabilitative team. The area of expertise over which optometry presides can improve the quality of life for these patients and will significantly affect their future. In a perfect world, the patient presented above would have been able to participate in an in-office vision therapy program. Since this treatment was not available, a home-based program along with spectacle correction, which included prism, was initiated and successfully alleviated the patient's complaints. It is the hope of the author that this case report will serve as an example for others that do not offer in-office vision therapy to perhaps jump head first into the treatment of patients that have suffered a TBI.

References

1. Vogel MS. An overview of head trauma for the primary care practitioner: Part II-ocular damage associated with head trauma. J Am Optom Assoc 1992;63:542-46.
2. Cohen AH, Rein LD. The effect of head trauma on the visual system: The doctor of optometry as a member of the rehabilitation team. J Am Optom Assoc 1992;63:530-36.
3. Ciuffreda KJ, Suchoff IB, Marrone MA, Ahmann E. Oculomotor rehabilitation in traumatic brain-injured patients. J Behav Optom 1996;7:31-38.
4. Gianutsos R, Suchoff IB. Neuropsychological consequences of mild brain injury and optometric implications. J Behav Optom 1998;9:3-6.
5. Ciuffreda KJ, Kapoor N, Rutner D, Suchoff IB, et al. Occurrence of oculomotor dysfunctions in acquired brain injury: A retrospective analysis. J Am Optom Assoc 1997;78:155-61.
6. Ciuffreda KJ, Kapoor N, Rutner D, Suchoff IB, et al. Vision therapy for oculomotor dysfunctions in acquired brain injury: A retrospective analysis. J Am Optom Assoc 2008;79:118-22.
7. Cohen AH. Optometric management of binocular dysfunction secondary to head trauma: Case reports. J Am Optom Assoc 1992;63:569-75.
8. Padula WV. Neuro-optometric rehabilitation for persons with a TBI or CVA. J Optom Vis Dev 1992;23:4-8.
9. Hellerstein LF, Freed S. Rehabilitative optometric management of a traumatic brain injury patient. J Behav Optom 1994;5:143-47.
10. Hellerstein LF, Freed S, Maples WC. Vision profile of patients with mild brain injury. J Am Optom Assoc 1995;66:634-39.
11. Aksionoff EB, Fald NS. The differential diagnosis of perceptual deficits in traumatic brain injury patients. J Am Optom Assoc 1992;63:554-58.
12. Kobashi R, Ohtsuki H, Hasebe S. Clinical studies of ocular motility disturbances: Part 2. Risk factors for ischemic ocular motor nerve palsy. Jpn J Ophthalmol 1997;41:115-19.
13. Sowka J, Gurwood A, Kabat A. Handbook of ocular disease management. Rev Optom http://cms.revoptom.com/handbook/sect7c.htm Last Accessed 3 May 2011.
14. Cook D. Optometric management of patients with incomitant strabismus. J Behav Optom. 2004;15:10-15.

Corresponding author:
Travis M. Johnson, OD
1280 Frontage Road West
Stillwater, MN 55082
austyndj@msn.com

Date submitted for publication: 27 January 2011
Date accepted for publication: 6 June 2011