OPO OPHTHALMIC & PHYSIOLOGICAL OPTICS
THE JOURNAL OF THE COLLEGE OF OPTOMETRISTS

Ophthalmic & Physiological Optics ISSN 0275-5408

REVIEW

Vergence dysfunction in mild traumatic brain injury (mTBI): a review

Preethi Thiagarajan, Kenneth J Ciuffreda and Diana P Ludlam

SUNY State College of Optometry, Department of Vision Sciences, New York, USA

Citation information: Thiagarajan P, Ciuffreda KJ & Ludlam DP. Vergence dysfunction in mild traumatic brain injury (mTBI): a review. *Ophthalmic Physiol Opt* 2011, **31**, 456–468. doi: 10.1111/j.1475-1313.2011.00831.x

Keywords: accommodation, acquired brain injury, eye movements, oculomotor rehabilitation, traumatic brain injury, vergence, vergence dysfunction, vision rehabilitation, vision therapy, visual system plasticity

Correspondence: Kenneth J Ciuffreda
E-mail address: kciuffreda@sunyopt.edu

Received: 5 November 2010; Accepted: 31 January 2011

Abstract

Vergence eye movements are used to track objects that move in depth in one's binocular visual field to attain and maintain a fused and single percept. The mechanism and control of vergence eye movements involves complex neurological processes that may be compromised in individuals with traumatic brain injury, thus frequently resulting in a wide range of vergence dysfunctions and related near-work symptoms, such as oculomotor-based reading problems. This paper presents a review of the vergence system and its anomalies in mild traumatic brain injury, as well as their diagnostic and therapeutic clinical ramifications. Implications related to brain imaging and human neuroplasticity are also considered.

Background

Oculomotor dysfunctions are common among the general population, with a range from 20% to 30% found in the young–adult clinic population.[1–4] These dysfunctions are also found in individuals with traumatic brain injury (TBI), but with an even greater frequency of occurrence.[5,6] For example, approximately 90% of individuals with a mild traumatic brain injury (mTBI) examined in a clinic setting and having vision-related symptoms were diagnosed with one or more oculomotor dysfunctions following their acute care phase and natural recovery period.[5] Due to the pervasive nature of a brain injury (e.g., coup–contrecoup in TBI), this is not surprising, as numerous vision-related areas can be adversely affected.[6] Moreover, six of the 12 cranial nerves directly bear on the visual process. Hence, a range of oculomotor-based visual deficits and related symptoms would be expected.

One such oculomotor subsystem that is frequently adversely affected is vergence. It is comprised of sensory, motor, and perceptual areas involving multiple neuronal pathways.[7,8] Injury to any of these or related brain regions would likely result in response abnormality. In addition, presence of any such oculomotor dysfunction will negatively impact on progress in other forms of therapy (e.g., cognitive therapy).[9,10] Thus, presence of a vergence oculomotor abnormality will hinder the patient's vocational and avocational goals, and therefore delay their return as a productive member of society.

This review paper describes the range of static and dynamic vergence abnormalities found in the mTBI population, as well as related aspects. First, the basic concepts/terms and the pathophysiology of brain injury will be discussed. Then, important retrospective studies, clinical case series, and laboratory findings will be reviewed. Lastly, current treatment of these oculomotor deficits, as well as the scope of future diagnostic and treatment aspects based on recent basic and clinical research, will be considered.

Overview of brain injury definitions and pathophysiology

Traumatic brain injury (TBI) is caused by an external insult to the head following motor vehicle accidents, falls, assaults, etc. Approximately 8 million people per year suffer a TBI in the United States.[5,6,11] It is a major optometric, medical, social, economic, national, and public health priority issue in the United States. Furthermore, TBI and its rehabilitative aspects have been a national priority in the United States due to the recent military encounters in Iraq and Afghanistan.[12] TBI patients, including those with blast overpressure injury from recent military

encounters,[12] incur a global brain injury frequently resulting in more encompassing diffuse axonal injury (DAI) due to its coup–contrecoup nature and resultant global anatomical pervasiveness.[13,14]

The key pathologic feature of TBI is DAI, also known as an axonal shear injury, caused by shear–strain injury from rotational acceleration forces.[14–16] These shear-related injuries commonly occur at the white–gray matter junction, corpus callosum, and superior colliculi, as well as other brain regions.[14,17] DAI has been a challenge to image, especially in mTBI since CT and MRI scans are usually normal despite the presence of more general neurologically-based symptoms such as poor concentration, vision and balance problems, memory deficits, etc.[14,18] However, recent advances in diffuse tensor imaging (DTI) and single photon emission computerized tomography (SPECT) show great promise.[19,20]

Based on the underlying mechanisms and timeframe involved, TBI has been classified into *primary* and *secondary* injuries.[21,22] *Primary injury* occurs as a result of the mechanical forces, such as acceleration, deceleration, and rotational forces acting upon the brain at the initial insult.[15] Two inertial forces, namely linear acceleration and rotational head movement, have been proposed to cause damage to brain tissues.[22] While linear acceleration is believed to produce superficial brain damage (such as to gray matter) that results in contusions and hemorrhages,[23] the rotational forces are believed to cause deeper cerebral white matter disruption leading to DAI.[24] In contrast, the *secondary injuries* occur as a result of a cascade of biomolecular, biochemical, and physiological events that are triggered by the primary injury at the cellular level.[21,22] It involves cellular excitotoxicity, altered calcium homeostasis, and oxygen depletion that cause inflammation, and cell death. In contrast to the primary injuries, *secondary injuries* are of a non-mechanical nature, and furthermore occur with delayed clinical presentation (i.e., weeks or months later). The recovery from TBI is mainly determined by the severity of the secondary injuries.[21,22]

The primary focus of the present paper will be mTBI, as it accounts for 70–80% of the TBI in the United States.[11,14,25,26] The criteria for mTBI are: (1) either loss of consciousness for <30 min or an altered state of consciousness, (2) 13 or greater score on the Glasgow coma scale (GCS), and (3) post-traumatic amnesia (PTA) lasting <24 h.[27]

Retrospective and prospective clinical studies on mTBI[a]

Retrospective studies

There have been five recent retrospective studies that have determined the prevalence of oculomotor abnormalities in mTBI patients in a clinic population[5] and in Veteran's Administration (VA/military) populations.[28–31]

Ciuffreda *et al.*[5] determined the frequency of occurrence of oculomotor dysfunctions encompassing vergence, accommodation, version, strabismus, and cranial nerve palsy in 160 individuals [between 8 and 91 years of age, mean (±1 S.E.M.) age of 44.9 (1.25) years] with mTBI and reporting vision-based symptoms. Ninety per cent of these patients were found to have an oculomotor dysfunction based on the above categorization. A vergence system abnormality was the most common dysfunction: 56.3% of the population had one or more vergence-related abnormalities. While convergence insufficiency (CI) was the main vergence dysfunction (42.5%), other vergence deficits also found with high frequency included binocular instability, convergence excess, basic exo, and divergence insufficiency. In addition, 51.3% of the population manifested one or more versional dysfunctions, with saccadic deficits (e.g., saccadic dysmetria) being the most common anomaly. Among those who were below 40 years of age (51 out of the 160 subjects), 41.1% exhibited an accommodative dysfunction, with accommodative insufficiency (AI) being the most common problem. Strabismus in the form of constant/intermittent deviations was present in 25.6% of the population, with strabismus at near being the main dysfunction. Lastly, third and fourth cranial nerve palsies were found in approximately 6.9% of the population. The frequency of occurrence of these five categories of oculomotor dysfunctions, and their subgroups, are typically 5–10 times greater than found in the general adult visually-normal population. The frequency of occurrence (%) of the different categories of oculomotor dysfunctions from the Ciuffreda *et al.*[5] study is presented in *Figure 1*.

In addition, there have been four subsequent retrospective studies in mTBI, with all being in the VA/military populations.[28–31] Their basic findings are presented in *Table 1*, along with the more detailed findings of the Ciuffreda *et al.* study.[5] The results are remarkably similar across the civilian and VA/military populations, most notably in the Goodrich *et al.*[28] and Brahm *et al.*[31] studies in which the aetiology of the mTBI included both blast and non-blast injuries. Of particular significance is the very high frequency of those having an oculomotor problem across studies (~50–90%), with the most common symptom related to reading (~50–90%). Vergence

[a]While not specified in all studies, based on the descriptions of the patients and their visual and other characteristics, most/all appear to be mTBI. Secondly, approximately 70–80% of all TBI patients are classified as mTBI.[11,14,25,26]

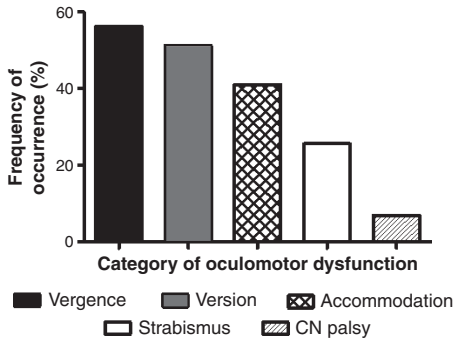

Figure 1. Frequency of occurrence (%) of oculomotor dysfunctions in a clinic population (n = 160) of mTBI, from Ciuffreda *et al.*[5]

dysfunctions ranged from 24% to 48%. These similarities in frequency of occurrence across studies suggest that the resultant visual dysfunction is relatively transparent to the aetiology of the brain injury, at least in mTBI. Furthermore, it suggests that similar vision therapies can be implemented and likely prove successful in these two populations. The high frequency of oculomotor problems and reading dysfunctions is not very surprising, as three of the 12 cranial nerves deal directly with fine oculomotor control, and a fourth one deals with vision-vestibular function.

Prospective clinical studies

Although the earlier section on 'retrospective studies' considered general oculomotor anomalies, such as vergence and accommodative disorders, strabismus, CN palsies, etc., the main purpose of the present paper is to review and focus upon non-strabismic vergence dysfunctions. Henceforth, the sections on clinical studies and laboratory investigations will consider only non-strabismic vergence disorders in the mTBI population.

One of the earliest formal studies on the presence of binocular vision abnormalities following head injury was by Cross in 1945.[32] Observations were made from several hundred cases examined at a military hospital with either closed-head injury or open-head gunshot wounds. Convergence dysfunction, with or without accommodative abnormality and other type of eye movement problem, was found to be one of the most common neuromuscular anomalies. While closed-head injury was typically associated with convergence abnormalities, the open-head ones were not, especially when there was either no loss of consciousness (LOC) or only short post-traumatic amnesia (PTA). General fatigue following head injury was attributed to be the cause of their reported 'ocular muscle fatigue', thus resulting in 'defective convergence' in these individuals.[32,33]

There have been a number of more recent studies conducted in clinic populations that have evaluated vergence function following head trauma. One of the earlier studies was by Krohel *et al.*[34] It was conducted in 23 patients who reported reading difficulty (26%) and/or diplopia at near (52%) as their main symptoms. CI manifested as a receded near point of convergence (NPC) (74%) and reduced fusional vergence reserves (52%) in these patients. This result is consistent with the later study of Cohen *et al.*,[35] who found CI in two different populations tested based on time elapsed after their head trauma. That is, while 42% of the patients tested 3 years after trauma suffered from long-standing CI, it was similarly found in 38% of the patients tested only 3 months after their injury. Thus, time after the insult appeared to have no influence on the frequency of this specific vergence dysfunction. Presence of CI was also associated with longer periods of coma (>30 days), cognitive disturbance,

Table 1. Summary of data from the retrospective studies showing frequency of occurrence (%) of the different types of oculomotor dysfunctions

	Ciuffreda *et al.*[5]	**Goodrich *et al.*[28]**		**Lew *et al.*[29]**	**Stelmack *et al.*[30]**	**Brahm *et al.*[31]**	
Sample size (n)	160	Non-blast 25	Blast 21	62	88	Non-blast 12	Blast 112
Percent of war fighters	0	100	100	94	88	100	100
Reading problem	75 (est)	60	62	70	50	83.3	87.5
Vergence	56	36	24	46	28	63.6	46.8
Version	51	32	5	25	6	16.7	24.1
Accommodation	41	20	24	21	47	71.4	45.7
Strabismus	26	50 (est)	30 (est)	11	8	8.3	7.1
CN palsy	7	50 (est)	30 (est)	Not listed	0	–	–
Nystagmus	0.6	4	0	5	Not listed	0	7.1
General oculomotor dysfunction	90	At least 50 (est)	At least 50 (est)	70	50 (est)	40 (est)	40 (est)

est, estimate; –, data not available. Actual percentages are rounded off for simplicity. Nystagmus - includes unidentified fixation instability.

and dysphasia, but not with behavioural problems. Similarly, 42% of the ABI population (including TBI and CVA) in nursing care centres were found to have abnormal exo deviations including CI, and either constant or intermittent exotropia.[36] Vergence dysfunctions such as abnormal NPC break and recovery, and abnormal near cover test, were also commonly found in a group of symptomatic patients ($n = 16$), along with reduced stereoacuity.[37] Receded NPC (63%) and reduced fusional range (100%), along with associated accommodative problems (36%), were reported in a group of TBI patients ($n = 11$) that suffered frontal and mid-facial trauma.[38] In addition to the receded NPC and reduced fusional vergence reserve, the near phoria was found to be abnormal (i.e., large exophoria) in this population. Lastly, in a hospital-based study of 51 patients with unspecified TBI, Schlageter *et al.*[39] found three vergence abnormalities present as related to the phoria: 38% exhibited an abnormal horizontal phoria at near, 18% exhibited an abnormal vertical phoria at near, and 26% manifested an abnormal horizontal phoria at far.

Other than the aforementioned clinic population studies, numerous clinical case series have been presented in the literature reporting vergence system abnormalities following mTBI. Again, the most common finding was convergence insufficiency, typically causing symptoms related to reading.[40,41] Complete or partial motor-based 'loss of fusion' was also a common finding in a series of ophthalmologically-based studies.[42–45] In addition, sensory-based fusion disruption syndrome has also been reported.[46,47]

From these clinical studies, it is evident that vergence system abnormalities are common in mTBI patients. *Figures 2 and 3* provide a global overview of the most common clinical symptoms[34,37,38] and signs[34–38,48] reported in the literature following mTBI, respectively.

Laboratory investigations

A wide range of static and dynamic vergence parameters were tested in a group of visually-symptomatic mTBI patients [mean (±1 S.E.M.) age of 45.7 ± 3.1 years; $n = 21$] as related to nearwork by our SUNY acquired brain injury research group (D. Szymanowicz, K.J. Ciuffreda, P. Thiagarajan, W. Green, W. Ludlam and N. Kapoor, unpublished data).[49] None of the previous studies assessed such a wide range of static and dynamic horizontal vergence functions in the same relatively large mTBI patient population. Static parameters at near included the cover test and the von Graefe heterophoria, near point of convergence, positive and negative fusional vergence ranges (PFV/NFV), convergence-accommodation to convergence stimulus (CA/C) ratio, prism adaptation, and horizontal fixation disparity and the associated phoria, as well as stereoacuity (per its relation to vergence error). They were assessed using standardized clinical test procedures.[1,50] Symmetric vergence (convergence and divergence) dynamics to a 6.5° step stimulus (temporally randomized) was determined using the Power Refractor II (Plusoptix, Nuremberg, Germany); its sampling rate was 12.5 Hz with an effective resolution of ≤0.9°. Oculomotor parameters included peak velocity, time constant, latency, and steady-state variability, as well as clinical prism facility. All of the above measures were compared with a group of visually-normal asymptomatic individuals (mean age of 36.7 ± 5.4 years; $n = 10$).

Five static parameters revealed a significant difference between the mTBI and the normal groups: NPC break

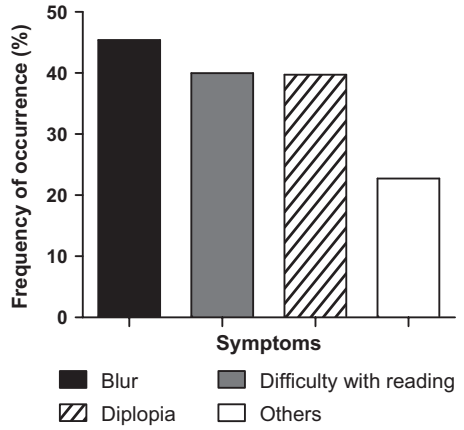

Figure 2. Commonly reported clinical symptoms in non-strabismic vergence disorders in mTBI. The category 'Others' includes symptoms such as headache, dizziness, ocular pain, and poor visually-based concentration.

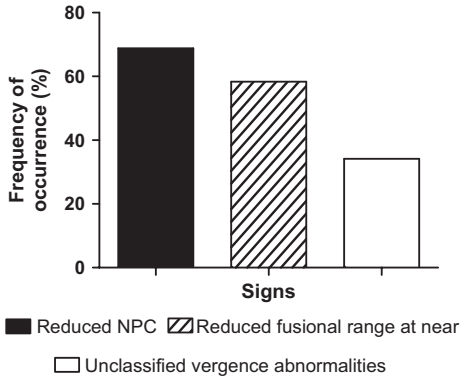

Figure 3. Commonly reported clinical signs in non-strabismic vergence disorders in mTBI. Unclassified vergence abnormalities include studies that have not differentiated between NPC, reduced fusional reserves, and exo deviations.

Table 2. Static parameters (mean ± 1SEM) in the mTBI and normal groups

Static parameters	mTBI	Normal	Statistically significant ($p < 0.05$)?	Predicted abnormal directionality?
Cover Test (PD)	5.75 ± 1.00	4.43 ± 0.84	No	Yes
Von Graefe (PD)	7.15 ± 1.40	4.13 ± 1.00	No	Yes
NPC break (cm)	13.98 ± 2.06	7.03 ± 0.33	Yes	
NPC recovery (cm)	19.46 ± 2.81	9.56 ± 0.46	Yes	
PFV break (PD)	22.03 ± 2.39	30.10 ± 1.18	Yes	
PFV recovery (PD)	11.30 ± 2.28	18.70 ± 1.48	Yes	
CA/C (D/PD)	0.42 ± 0.08	0.37 ± 0.11	No	No
NFV break (PD)	16.40 ± 1.36	17.00 ± 1.83	No	No
NFV recovery (PD)	10.20 ± 1.30	11.10 ± 1.96	No	No
FD (min arc)	2.60 ± 2.45	0.70 ± 1.98	No	Yes
AP (PD)	1.76 ± 2.01	2.70 ± 1.29	No	Yes
Adaptation (PD)	1.45 ± 0.70	2.70 ± 0.89	No	Yes
Ampl conv (°)	6.43 ± 0.28	6.21 ± 0.15	No	No
Ampl div (°)	6.54 ± 0.21	6.57 ± 0.19	No	No
Stereoacuity (s arc)	38.8 ± 3.87	20.5 ± 0.50	Yes	

NPC, near point of convergence; PFV, positive fusional vergence; NFV, negative fusional vergence; FD, fixation disparity; AP, associated phoria; conv, convergence; div, divergence; CA/C, convergence accommodation/convergence ratio; PD, prism diopter; D, diopter; Ampl, amplitude. Adapted from D. Szymanowicz, K.J. Ciuffreda, P. Thiagarajan, W. Green, W. Ludlam and N. Kapoor, unpublished data.[49]

and recovery values were receded, PFV break and recovery values were reduced, and the stereoacuity threshold was increased in the mTBI group. In addition, there were five parameters which exhibited predicted directionally-abnormal effects between the mTBI group and the normals: von Graefe phoria test (exophoric values only), cover test (exophoric values only), base-out prism adaptation, associated phoria, and horizontal fixation disparity. The mean values (±1 S.E.M.) of the 14 static parameters tested, as well as stereoacuity, in both the mTBI and in the normal groups are presented in *Table 2*.

Table 3 presents the mean dynamic parameter values found in the mTBI and in the normal groups. While the response amplitudes for convergence and divergence did not differ significantly between the normal and the mTBI groups (see *Table 2*), *all* of the dynamic parameters were significantly different ($p < 0.05$) between the two groups for both convergence and divergence. They were all slowed, delayed, and more variable in the mTBI group as compared with the normal group.

Table 3. Dynamic parameters (mean ± 1SEM) in the mTBI and normal groups

Dynamic parameters	mTBI	Normal
Prism facility (cpm)	12.02 ± 1.10	16.35 ± 0.90
PV conv (° s^{-1})	14.35 ± 0.78	28.69 ± 1.12
PV div ((° s^{-1})	14.60 ± 0.77	24.81 ± 1.24
Latency conv (ms)	323.00 ± 26.83	216.00 ± 17.08
Latency div (ms)	343.70 ± 22.30	258.70 ± 20.30
TC conv (ms)	458.70 ± 25.67	220.90 ± 9.66
TC div (ms)	489.30 ± 27.06	273.40 ± 19.08
SS variability conv (°)	0.78 ± 0.04	0.52 ± 0.02
SS variability div (°)	0.83 ± 0.04	0.5 ± 0.02

PV, peak velocity; TC, time constant; conv, convergence; div, divergence; cpm, cycles per minute; SS, steady-state response. Adapted from D. Szymanowicz, K.J. Ciuffreda, P. Thiagarajan, W. Green, W. Ludlam and N. Kapoor, unpublished data.[49]

Figure 4 presents the best fit exponential for dynamic convergence and divergence responses in a typical normal control subject (N-V-9), and in a typical patient with mTBI (TBI-V-16), with the mTBI patient exhibiting slowed and variable responses. For example, peak velocity for convergence and divergence in the normal subject was 47.5 and 43.6° s^{-1}, respectively, whereas they were only 14 and 15.1° s^{-1} for convergence and divergence in the mTBI subject, respectively, thus representing about a threefold decrease in peak velocity.

The mTBI population also exhibited significantly increased steady-state (SS) response variability for both convergence and divergence as compared to the normal group (see *Table 3*). *Figure 5* presents the dynamic vergence step responses with a compressed time scale from a typical control subject (N-4) and from a typical mTBI patient (TBI-16). Subject N-4 exhibited little variability with respect to the two mean steady-state levels, as well as for the intervening dynamic response trajectories. In contrast, patient TBI-16 exhibited a markedly increased level of overall response variability. The mean SS convergence variability was 0.47° in N-4, whereas it was increased to 0.88° in TBI-16. Similarly, the mean SS divergence variability was 0.34° in N-4, while it was increased to 0.77° in TBI-16.

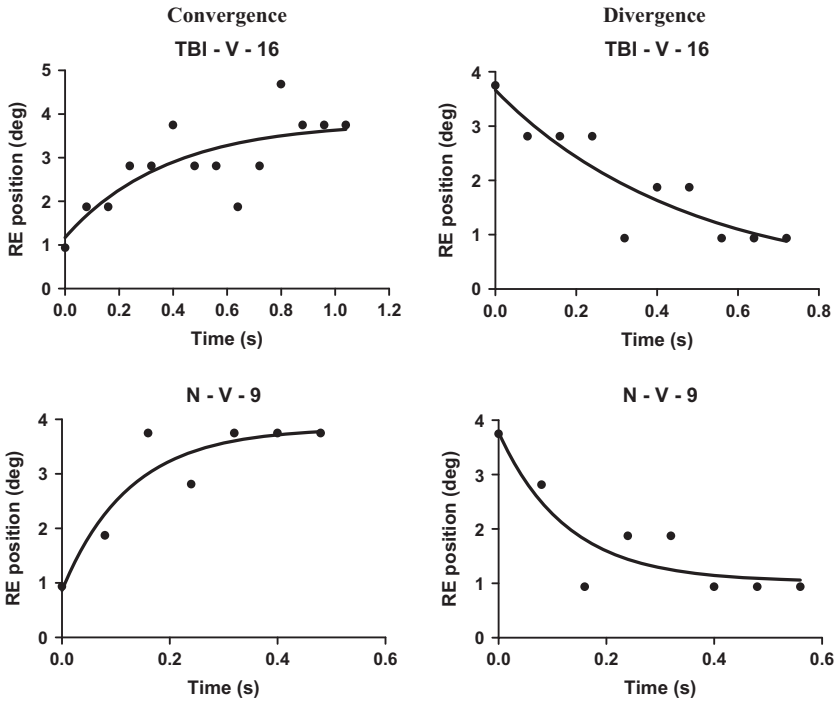

Figure 4. Convergence and divergence responses fit exponentially in a typical mTBI subject (TBI-V-16) and in a normal subject (N-V-9), with the mTBI subject exhibiting slowed responses.

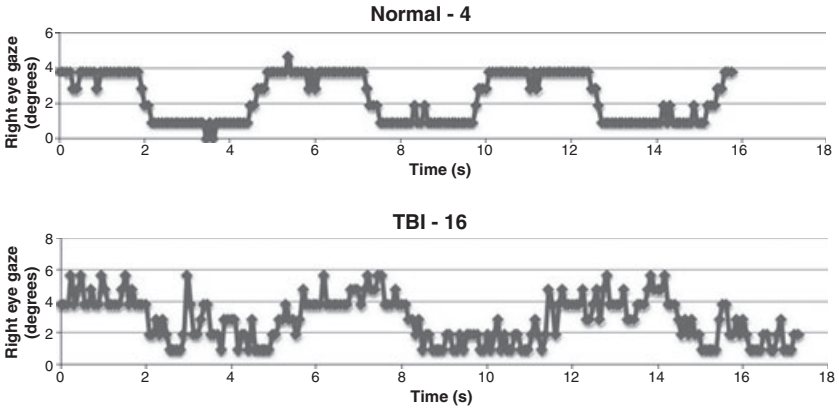

Figure 5. Unprocessed dynamic vergence step responses with an expanded time scale from a typical control subject (N-4) and in an mTBI patient (TBI-16), with the latter exhibiting a markedly abnormal and variable dynamic profile.

In addition to the above dynamic and static measurements, at the end of the 1.5 h test session, vergence flipper facility was reassessed. This was immediately followed by a continuous 3-min period of prism alteration in an attempt to fatigue the subject visually, as 'visual fatigue' is a common symptom in this population.[5,6] The subject was instructed to alternate the prism flipper every 10 s upon command of the examiner. During the intervening 10 s sustained period, the subject attempted to maintain the target fused and in focus at all times. Immediately after the 3-min period, the 1-min vergence flipper facility test procedure was repeated to assess for any fatigue effects. While the baseline flipper rate in the mTBI group was significantly lower than in the normal group (*Table 3*), a significant fatigue effect (manifested as a reduced flipper rate) was not found [pre – 11.6 cpm (±1.2 cpm), and post – 11.1 cpm (±1.2 cpm)] either in the mTBI group or the normal group.

In a recent pilot study,[51] objective recordings of vergence were taken in two individuals with self-reported

mTBI. Vergence dynamics were markedly slowed (i.e., reduced peak velocity) for convergence but not for divergence.

In addition to the constellation of static and dynamic vergence deficits found in individuals with mTBI, they also manifest a range of static and dynamic dysfunctions that may affect the accommodative system and its interaction with vergence.[52,53] Some of the primary and most relevant accommodative parameters are (1) reduced amplitude of accommodation, (2) reduced accommodative facility, (3) increased time constant, (4) reduced peak velocity, and (5) increased SS variability.

Oculomotor rehabilitation

The primary purpose of optometric vision therapy (i.e., vision rehabilitation) for binocular vision disorders, more specifically non-strabismic binocular dysfunctions, is to achieve an overall improvement in the speed and accuracy of the various integrated oculomotor functions, to attain clear, single, sustained, and symptom-free binocular vision at all times.[54] The efficacy of vision therapy for remediation of binocular vision anomalies, such as AI, CI, etc., is well established in the general clinical population (i.e., non-mTBI).[1,54–58] Numerous studies have demonstrated considerable normalization in the patient's clinical oculomotor parameters that are associated with amelioration of the related symptoms.[55–60] Treating these oculomotor anomalies using conventional vision therapy procedures in the mTBI population can be challenging due to complicating general factors, such as excessive fatigue, depression, memory problems, and difficulty performing the vision therapy procedures regularly due to other physical ailments, to name a few, as well as other non-oculomotor-based vision problems such as visual field defects and photosensitivity.[6,61,62] However, and very importantly, improved oculomotor coordination and visual-perceptual skills can hasten progress in the patient's other rehabilitative programs.[9,10] This would include cognitive therapy which requires complex visual scanning and fine detail discrimination.

Several clinical case studies and a few population studies have evaluated the effect of vision therapy in individuals with mTBI. This section below summarizes the results from these important investigations.

One of the earliest studies involved with the treatment of accommodative and vergence disorders was conducted by Candler[63] in a series of World War II related head injury cases. Orthoptic treatment (unspecified) commenced anywhere from 3 weeks to 5 years post-injury. While 73% (24/33) of the patients treated were either fully remediated or markedly improved, 12% (4/33) failed to improve, and only 6% (2/33) exhibited spontaneous recovery. Of those having convergence and accommodative deficits (with a monocular and binocular component), 78% showed a complete cure/improvement; of those having convergence and binocular accommodative problems (without a monocular accommodative component), 100% exhibited considerable improvement. Cohen[64] demonstrated significant improvement of oculomotor function in two head trauma cases following optometric vision therapy administered in the form of lenses, prisms, fusional procedures, and versional eye movement training (i.e., saccades and pursuit). Cohen's[64] results are consistent with the findings of Hellerstein and Freed,[65] as well as Ludlam.[66] The impact of optometric vision therapy in improving oculomotor function was also reported by Berne[40] in three cases with mTBI. Each patient demonstrated improved NPC and PFV reserves, along with reduced exophoria, following 6 months of vision therapy (1 h session per week). Although the above case studies consistently reported marked improvement in vergence function following vision therapy, long-term follow-up data were generally not available. Such information is important to evaluate the long-term efficacy of vision therapy. However, one such case study with follow-up of 2–6 months reported no regression in the improved visual performance of three mTBI patients after having received 6 weeks of vision therapy (two sessions/week; 50 min each session) which emphasized fusional abilities.[67] Furthermore, each patient also exhibited an overall improvement in reading ability, and two demonstrated an improvement in stereoacuity. More recently, Scheiman and Gallaway[41] reported results following optometric vision therapy in nine cases (eight with mTBI, one with cerebral aneurysm) who suffered mainly from convergence and accommodative insufficiencies (6/9), as well as other vision problems such as visual field defects (2/9), sensory fusion disruption (1/9), and IV nerve palsy (1/9). While isolated convergence/accommodative problems responded very well to vision therapy, treatment success was not as effective if the patient concurrently had visual field defects, cognitive and perceptual problems, sensory fusion disruption, and/or cyclophoria.

Evidence to support the fact that programmed vision therapy remediates binocular vision anomalies in mTBI patients also comes from several clinical population studies.[34,41,68] In each study, reading difficulty was one of the most common symptoms. Krohel *et al.*[34] employed primarily vergence training procedures. They reported improvement in 65% of patients ($n = 23$) with CI following closed-head trauma. As per the Scheiman and Gallaway[41] findings, patients without any serious neurologic consequences exhibited more benefit from the therapy than those that did. In a recent retrospective analysis which assessed the effect of conventional broad-based

optometric vision therapy in 33 mTBI patients,[68] the majority demonstrated significant improvement: 90% (30/33) exhibited reduction in at least one of their primary symptoms (e.g., difficulty when reading). Furthermore in those 30 patients, 27/30 (90%) showed significant improvement in their primary clinical sign (e.g., receded NPC). More recently, a laboratory pilot study was performed in two individuals with self-reported mTBI, CI, and related nearwork symptoms. Convergence, but not divergence, was slowed before vision therapy, and it normalized following 6 weeks (a total of 18 h) of combined office- and home-based vision therapy. In addition, near-vision symptoms reduced markedly.

In contrast to the above positive studies, there is one negative study.[38] The effect of vision therapy vs natural recovery was tested in a group of TBI patients with frontal and/or midfacial fractures. Five out of the six patients (83%) who received therapy showed markedly improved convergence/accommodation, while the sixth patient showed only partial improvement. However, four out of the five patients (80%) in the natural recovery group also appeared to recover, and one only showed slight improvement. Unfortunately, the details regarding specifics dealing with diagnosis, treatment type, total duration of treatment, etc., were not available, and hence the results of this study are difficult to evaluate.

From the above studies, there is abundant evidence in both the optometric and ophthalmological literatures supporting the notion that targeted, specific, programmed vision therapy procedures (i.e., motor learning)[54] can remediate patients with a range of binocular vision disorders as a consequence of mTBI. Symptoms were ameliorated concurrent with normalization of clinical signs.

Discussion

The frequency and range of vergence dysfunctions revealed in our laboratory investigations, as well as in past clinical studies, readily explains the symptoms frequently reported in the mTBI population. These included intermittent diplopia (due to large exophoria and reduced fusional ability), lateral 'movement' of words/line of text (due to fusional instability), and transient blur (due to vergence–accommodative interactions), to name a few.

The greater frequency of occurrence of large exophoria at near in individuals with mTBI may be attributed to at least three factors. First, due to their reduced accommodative gain/response amplitude,[52,53] per the crosslink gain, the correlated accommodative vergence would be reduced at near. Second, individuals with binocular vision dysfunction typically exhibit reduced/impaired vergence adaptation.[69] Over time, this abnormal vergence adaptation will permit the true magnitude of exophoria to manifest itself, especially with prolonged occlusion. Third, in those with lower amounts of uncorrected hyperopia, their ability to compensate via accommodation is frequently no longer effective.[70] Any one or more of the above factors will result in increased exophoria at near.

Presence of accommodative abnormalities would produce a slowed, reduced, and variable accommodative response (AR). What might be the effect of this abnormal accommodation on the vergence system, especially at near? First, if the blur-driven AR is reduced at near, the accommodative vergence response in turn will also be reduced, and this may initially result in the perception of blur and/or diplopia when changing bifixation from far to near. Such an impoverished response would demand a greater amount of PFV to achieve eventually haplopic retinal imagery. This increased PFV would concurrently increase the amount of vergence-accommodation per the CA/C ratio, and thus likely help to obtain and maintain the target in focus. However, since many of individuals with mTBI have reduced PFV amplitude, sustained and accurate bifixation and focus may not be readily achieved. Furthermore, given the overall delayed and slowed accommodation and vergence in these individuals, eventual clarity and singleness of the target would not occur in a time-optimal manner, thus further exacerbating their near-vision symptoms and overall visual efficiency. In addition, the effect of fatigue on either or both of these two oculomotor systems would further erode their response capabilities, thus producing yet increased nearwork symptomatology.

Presence of such vergence-related oculomotor deficits, either alone or in conjunction with other frequent concomitant versional (e.g., saccadic dysmetria)[71] and accommodative (e.g., accommodative insufficiency) deficits,[52,53] would logically lead to global nearwork-related symptoms.[34,37] This is especially true as related to the complex task of reading, which is the most common symptom reported in the mTBI population.[5,28–31] During reading, there is a fine interplay between the saccadic and vergence systems as the eyes move across the line of print.[7,72] Disruption to either or both systems, as frequently found in mTBI,[5,73] would result in slowed and inefficient reading. Furthermore, any residual vergence deficits would have an adverse effect on vocational (e.g., computer data entry) and avocational (e.g., needlepoint) goals, as well as their rehabilitative progress (e.g., cognitive therapy requiring visual scanning and visual discrimination tasks at different distances).[9,10]

Neurological control implications

The dynamic vergence findings of the present study have important neurological control implications. Neurons that

control convergence and divergence have been found in the midbrain[74,75] in the mesencephalic reticular formation in the monkey, 1–2 mm dorsal and dorsolateral to the oculomotor nucleus.[74,76] Similar to saccades, the final motoneuronal controller signal for convergence consists of a small and broad pulse combined with a step.[77–79] The step component functions to maintain accurately binocular eye position (i.e., vergence angle) on the newly-acquired target, whereas the small pulse component functions to displace the eyes dynamically in a time-optimal manner to this new target position.[7,78]

Hence, based on the findings of our recent study (Szymanowicz D, Ciuffreda KJ, Thiagarajan P, Green W, Ludlam W & Kapoor N, unpublished data),[49] the primary neural deficit in the mTBI patient is the pulse. This is reflected in the consistently slowed dynamics (e.g., reduced peak velocity) for both convergence and divergence. The reduced peak velocity and related increased time constant can be accounted for by a reduction in pulse height and/or duration. Thus, the overall time course of the vergence dynamic trajectory will be slowed. Since the appropriate vergence amplitude was eventually attained accurately, this suggests that the step component had the appropriate mean height. However, the vergence steady-state level was quite variable, which suggests the presence of increased neural noise producing step component variability. Lastly, the increased latency suggests a processing delay in the afferent visual pathways related to computation of the retinal disparity signal that drives the vergence system.[80] This delay in temporal processing is consistent with other studies indicating increased reaction time in the mTBI population.[26]

In addition to the midbrain, neurons also discharge during vergence in the pons,[81–83] cerebellum,[83,84] and in areas of the cerebral cortex, such as the frontal eye fields,[83,85] parietal lobes,[83,86] middle temporal and medial superior temporal visual areas,[87] and in the primary visual cortex (V1).[88] Thus, given the complexity of the vergence pathways, it is not surprising that injury due to mTBI can have an adverse effect on its responsivity.

Neural plasticity

The hallmark feature of the brain is to modify continuously both its structure and function per its range of dynamic multi-sensory experiences: hence the term neural 'plasticity'.[89–91] Neural plasticity allows the brain to acquire new knowledge, store this information, adapt to both external and internal environmental changes, and even attempt to recover functionally following neuronal injury.[90–92] In a developing brain, neural plasticity likely involves the formation of new synapses, the strengthening/altering of existing synapses, activity-dependent synaptic plasticity, altered synaptic firing, neuronal cell death, etc.[89] A balance between the excitatory and inhibitory synapses involving a number of neurotransmitters determines the stabilization of synapses and their neuronal circuits.[93] Repeated stimulation of synapses to a particular stimulus induces long-term potentiation (LTP) mediated by the activation of *N*-methyl-D-aspartate (NMDA) receptors that trigger a cascade of cellular mechanisms resulting in learning and memory.[92] However, this neural plasticity is not just limited to the developing brain; it is also present in the adult brain,[89] even in the anatomically, physiologically, and functionally compromised brain of the adult mTBI patient.[90] Adult brain plasticity forms the basis for any learning/rehabilitation process. For the purpose of this paper, general motor learning, and then oculomotor learning, are briefly considered.

Motor and oculomotor learning

Motor learning involves the acquisition of a coordinated sensory, motor, and perceptual skill through a repeated stimulation (i.e., practice) protocol. Basically, the process involves three stages: (1) the new skill is learned via a trial-and-error method with constant feedback, (2) this newly-learned task is repeated many times and refined; then task difficulty is increased to ensure attention and skill efficiency, and this too requires feedback, and (3) the motor skill becomes automatic (pre-programmed), accurate, and precise without involving feedback control.[54,94] These same steps are involved in any training of the brain-injured patient.

The basic underlying principle of oculomotor training is a subset of motor learning, wherein targeted, specific, programmed oculomotor-based paradigms improve related visual function. It likely involves enhancing neuronal connections and synaptic strength through repetition producing LTP,[89] as mentioned earlier. Age is not a crucial factor that determines rehabilitation success, with attention playing an essential role.[95,96]

Targeted diagnostic protocol

Lastly, the results of our clinical and laboratory studies on vergence (Szymanowicz D, Ciuffreda KJ, Thiagarajan P, Green W, Ludlam W & Kapoor N, unpublished data),[49] accommodation,[52,53] and version[71,73,97,98] in the mTBI population have provided the basis for developing a 'targeted' and rapid overall oculomotor-based diagnostic clinical test protocol in this population (*Table 4*), but with an emphasis on vergence.[62] Such a protocol would result in a 'high yield' with few false positives. Those clinical oculomotor parameters found with the greatest frequency of

Table 4. Targeted clinical oculomotor parameters

Vergence
NPC break (especially with repetition)
NPC recovery (especially with repetition)
PFV break
PFV recovery
Vergence facility (prism flipper baseline)
Vergence facility (prism flipper fatigue)
Horizontal near dissociated phoria
AC/A ratio
Fixation disparity at near
Associated phoria at near
Stereoacuity (per its relation to vergence error)
Accommodation
Accommodative amplitude
Accommodative facility (lens flipper fatigue)
PRA/NRA
Version
Fixational stability
Saccadic accuracy
Pursuit accuracy
DEM

Adapted from Ciuffreda *et al.*[62]

occurrence of abnormality in the mTBI population are listed. Fortunately, most/all of these targeted abnormal parameter values can be normalized,[68] and their correlated symptoms reduced,[60,98] with relatively simple optometric vision therapeutic intervention.[1] In addition, lenses and prisms are typically incorporated into the oculomotor therapeutic intervention, as well as vestibular therapy.[98] This protocol would be especially helpful in the vision screening of our war fighters, as well as in the general vision therapy clinical practice. Use of the targeted oculomotor diagnostic test protocol,[62] in conjunction with a recent conceptual model of vision testing in the mTBI population,[61] should result in more effective quality of vision care.

Conclusions

Mild traumatic brain injury produces a wide range of static and dynamic vergence dysfunctions in the adult human due to the pervasiveness of the brain injury. Presence of vergence deficits can have a negative impact on the individual's vocational and avocational goals, as well as on the progress with other types of therapy. Fortunately, these oculomotor deficits can be remediated to some extent by optometric vision therapy involving the basic tenets of neural plasticity and motor learning, with correlated symptom reduction. Future studies should be directed to determine the most efficient and long-lasting therapeutic protocol, in conjunction with brain imaging to reveal the underlying neural correlates.

Acknowledgement

We thank Wesley Green and Dora Szymanowicz for help with data acquisition in the vergence experiments.

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