1040-5488/13/9002-0105/0 VOL. 90, NO. 2, PP. 105–112
OPTOMETRY AND VISION SCIENCE

FEATURE ARTICLE ON LINE

Mechanisms of TBI and Visual Consequences in Military and Veteran Populations

Gregory L. Goodrich*, Heidi M. Flyg†, Jennine E. Kirby‡, Chea-Yo Chang†, and Gary L. Martinsen§

ABSTRACT

Purpose. Blast-related (BR) traumatic brain injuries (TBIs) occur secondary to explosive blasts. Blast-related TBIs can be caused by the blast wave itself or by direct head trauma caused by events surrounding the blast. Non–blast-related (NBR) TBIs are caused by direct head trauma. Recent evidence shows that TBIs are associated with vision problems, particularly binocular system problems. The purpose of this study was to determine if similar types and amounts of vision problems are present in patients with BR TBIs and NBR TBIs.

Methods. A retrospective analysis of eye examination records of 50 NBR TBI and 50 BR TBI patients was conducted. Frequencies of visual symptoms and abnormal vision function measurements were computed and compared for the two patient groups.

Results. More than 65% of NRB TBI and BR TBI patients reported vision problems. Reading complaints were found in approximately 50% of the patients. Light sensitivity was reported significantly more often in BR TBI patients (67%) than in NBR TBI patients (33%) ($p < 0.01$). Saccadic dysfunction was measured more often in NBR TBI patients (85%) than in BR TBI patients (58%) ($p < 0.01$). High rates of accommodative dysfunction and convergence insufficiency were also found, but the group differences were not significant. Strabismus, pursuit abnormalities, fixation defects, and visual field defects were also common in both groups.

Conclusions. For most findings, the mechanism of injury (NBR vs. BR) did not result in different frequencies or types of visual dysfunction. The reasons for finding higher frequencies of light sensitivity in the BR TBI group and saccadic dysfunction in the NBR TBI group are unknown, and further research is needed. Overall, the rates of vision complaints and oculomotor defects were high in both groups, indicating a need for a thorough eye examination for any patient with a history of TBI.

(Optom Vis Sci 2013;90:105–112)

Key Words: traumatic brain injury, blast injuries, binocular vision, light sensitivity, oculomotor system

On average, 1.7 million people incur a traumatic brain injury (TBI) annually in the United States according to Centers of Disease Control and Prevention statistics.[1] The leading causes of TBI in the US population are falls, running into objects (struck by/against events), motor vehicle accidents, and assaults. Traumatic brain injuries can take place in any setting and can happen to anyone. The recent wars in Iraq and Afghanistan have brought to the forefront another type of TBI that is caused by proximity to a blast or explosion. Blast-related (BR) injuries occur most often on the battlefield and frequently to military personnel. Blast-related TBIs are usually caused by blasts from improvised explosive devices, mines, rocket-propelled grenades, mortars, and other high-explosive munitions.[2] From the year 2000 through the fourth quarter of 2011, more than 233,000 military members were reported to have sustained a TBI.[3] Because blasts have been the leading cause of injury in the conflicts in Iraq and Afghanistan,[2,4] a large number of veterans and US military service personnel have BR TBIs.

Traumatic brain injury is known to cause a myriad of neurologic health problems and related symptoms.[5] Not surprisingly, TBI is also associated with numerous deleterious changes affecting the visual system. Dysfunctions of the oculomotor and binocular vision systems are some of the most widely reported visual problems in individuals with TBI.[6–8] Oculomotor deficits in fixation, pursuits, saccades, vergence, and accommodation have all been described. In addition, some studies have shown the prevalence of strabismus to be high in TBI patients.[9,10] Other areas of TBI-related visual impairment include visual acuity (VA),

*PhD, FAAO
†OD, FAAO
‡OD
§OD, PhD, FAAO
Veterans Affairs Palo Alto Health Care System, Palo Alto, California (GLG, HMF, JK, C-YC, GLM).

visual fields (VFs), reading abilities, and dark adaptation.[9,11] Subjective complaints about vision are also extremely common and include complaints of general visual disturbances, reading difficulties, diplopia, and light sensitivity.[12–14]

The intracranial mechanisms of injury during a TBI and the subsequent pathophysiologic changes are complicated. Generally, a non–blast-related (NBR) TBI is caused by an object in motion hitting the head, as might occur from being punched, or a head in motion hitting an object, as might occur during a fall. Injurious events can also cause a rapid acceleration and deceleration of the head, thereby causing the brain to move within the skull.[15,16] A classically defined ''coup'' and ''contrecoup'' injury happens when opposite areas of the brain are injured from a single event. The coup injury occurs when the head impacts a solid object (or vice versa), causing the brain to impact the skull with neural damage at the point of impact. The contrecoup injury occurs opposite the coup site caused by the brain moving within, and making contact with, the skull opposite the point of initial impact. Both focal and diffuse brain injuries arise from NBR events and often occur together.[17] Focal injuries include contusions, hematomas, and lacerations and are typically caused by direct head trauma. Diffuse injuries often arise from motor vehicle accidents, falls, and trauma and are characterized by shearing or diffuse axonal injuries and brain swelling caused by acceleration/deceleration and inertial forces.[17,18]

Blast-related injuries have three principal causes that are termed ''primary,'' ''secondary,'' and ''tertiary.'' As in NBR cases, BR TBIs cause both focal and diffuse brain injuries. A primary blast injury results directly from the blast wave and the subsequent changes in atmospheric pressure.[19] As a result, the brain and skull are accelerated at different density-dependent rates. In such an event, the brain can be displaced and impact the skull, causing contusions and other injuries. Brain tissues can also undergo shearing and stretching forces that injure axons and other cellular elements.[20,21] Objects put into motion by a blast cause secondary blast-induced TBI. Shrapnel, debris, and other flying objects can cause penetrating or nonpenetrating TBI and other injuries. A tertiary blast injury occurs when a body, put into motion by a blast, strikes an object. Most BR TBIs are probably not the result of an isolated mechanism, and many are caused by primary blast exposure in addition to secondary and/or tertiary injury mechanisms.[20,22]

Both nonblast and secondary/tertiary blast TBIs are caused by the head contacting solid objects. Thus, the brain injuries caused by these mechanisms might be expected to be similar. By contrast, primary blast injuries are caused by pressure waves hitting the head and body, and primary blast TBI does not have a nonblast correlate. Because the injury mechanisms of primary BR TBI and NBR TBI differ, the manifestations of each might also be expected to differ.[21] Unfortunately, information on neuroanatomical damage differences from NBR TBI and BR TBI is sparse.[19] Altogether, these observations lead to several questions: Are similar health and psychological problems expected after NBR TBI versus BR TBI? How many of those affected by each condition are expected to experience problems? Finally, what vision function sequelae arise from NBR TBI and BR TBI? The answers to these questions are still being investigated. With respect to the visual system, more research is needed to determine the types and amounts of visual dysfunctions that occur in individuals with NBR TBI compared with those with BR TBI. This knowledge will provide improved diagnostic and treatment strategies for all patients with a history of TBI, regardless of type.

METHODS

Approval was obtained from the Institutional Review Board and the Veterans Affairs Palo Alto Health Care System Research and Development Committee before initiation of this research. A retrospective record review of 100 patients admitted to the Palo Alto Polytrauma Rehabilitation Center (PRC) was conducted for this study. The PRC is primarily an inpatient subacute rehabilitation service for polytrauma patients. A ''polytrauma patient'' is defined as an individual who has sustained injuries to multiple body parts and organs, one of which may be life threatening.[23] Traumatic brain injury often occurs in combination with other disabling conditions, such as amputation, posttraumatic stress disorder, visual impairment, auditory impairment, and other medical conditions. For the current study, the records of 50 patients with a history of NBR TBI and 50 patients with BR TBI were reviewed. All patients had documented eye examinations with optometry. Subjective and objective ocular and vision data from eye examinations nearest in date to the injury date were collected. For many reasons, not all data were available in every record reviewed. For example, convergence was not assessed in patients with constant strabismus. In addition, some severely injured patients were unable to complete all of the testing. Therefore, the number of patients included in each data category is listed along with the data in each figure and table.

TBI Severity

Traumatic brain injury severity was assigned to one of two categories as either mild or moderate/severe. When available, TBI severity was obtained from a Defense and Veterans Brain Injury Center evaluation record. If an evaluation was unavailable, TBI severity was determined from entries by other physicians who evaluated the patient. If this information was unavailable, a determination was made from data in the patient's record and by following the American Congress of Rehabilitation Medicine definition of mild TBI.[24] Traumatic brain injury patients with signs or symptoms that exceeded the mild TBI definition were classified as moderate/severe.

Vision and Ocular Data

The techniques used to measure vision and oculomotor functions have been previously described.[14] All eye examinations were conducted by optometrists with extensive experience in the evaluation and treatment of brain-injured patients. Each patient's record was used to gather self-reported complaints of blurred vision, reading problems, diplopia, light sensitivity, and any other visual symptoms. Visual acuity was measured with an ETDRS (Early Treatment Diabetic Retinopathy Study) eye chart when possible. A Feinbloom distance low-vision flip chart was used to measure acuity in bedridden patients and in eyes with very poor vision. Fixation was assessed by having the patient fixate a 20/50

near target and noting any unsteadiness or nystagmus. Pursuits were evaluated by having the patient follow a target that was moved into the cardinal positions of gaze. Saccades were assessed by having a patient switch fixation between two targets located approximately 10 cm apart and 40 cm in front of the patient's midline. Saccadic function was rated as normal or deficient following Northeastern State University College of Optometry oculomotor test criteria.[25] Cover testing was conducted in primary gaze at distance and near when fixation was adequate to evaluate ocular alignment. Otherwise, Hirschberg corneal reflexes were assessed to determine whether the eyes were aligned. The near point of convergence was measured with the patient fixating a single 20/50 letter. A near point of convergence of more than 8 cm was classified as convergence insufficiency. The amplitude of accommodation was measured monocularly on patients aged 40 years or younger using the pull-away technique. Established norms were used to determine if a patient had accommodative insufficiency.[26]

Reading

Reading ability in these brain-injured patients was tested using internally developed reading materials. The test consisted of continuous text (10-point Times New Roman font), and attention was paid to reading facility, speed, and comprehension. Using these criteria, the examiner determined if the patient's reading ability was normal or deficient.

Visual Fields

Visual fields were performed using a method suitable to the patient's condition. Confrontation fields, tangent screen, arc, and Goldmann perimetry were all used to determine the presence and characteristics of any VF defects.

Ocular Injuries

Ocular injury determination was made by patient history and the results of a complete anterior and posterior segment examination.

Statistical Analyses

Fisher exact test was used to examine NBR TBI versus BR TBI group differences for all measurements of vision function and visual symptoms. A *t* test was used to evaluate logMAR (logarithm of the minimum angle of resolution) VAs and elapsed times between injury and vision examination between the two patient groups.

RESULTS

The average age of the 50 NBR TBI patients was 29 years (median, 24 years; range, 19 to 63 years) at the time of their optometric examination. Two of the 50 NBR TBI patients were women. The most common cause of NBR TBI in this population was motor vehicle accidents, with 30 (60%) of 50 patients sustaining TBI in this manner. Motor vehicle accidents occurred while riding in automobiles, motorcycles, all-terrain vehicles, and snowmobiles. Seven patients (14%) had falls. Falls from trees and ladders and while rock climbing were all documented in patients' records. Six patients (12%) incurred NBR TBIs from assaults, and seven (14%) had TBIs because of other causes. Other causes included bicycle accidents, pedestrian versus automobile collisions, and a snowboarding accident. Two sustained NBR TBI in Iraq, 40 in the United States, and eight in other overseas locations. These other locations include Okinawa in Japan, Germany, Italy, Guam, and Turkey.

The average age of the 50 BR TBI patients was also 29 years (median, 26 years; range, 19 to 55 years), and three were women. All 50 of the BR TBI patients were involved in a blast event in either Iraq or Afghanistan. The sources of the blasts included improvised explosive devices, mines, rocket-propelled grenades, and mortars. Sixteen (32%) of the 50 BR TBI patients had documented secondary/tertiary head trauma that accompanied their blast exposure. Of these, nine had penetrating TBI from shrapnel, flying debris/fragments, and other causes. Seven patients hit their heads on vehicle windshields, dashboards, or on other solid objects in the events that accompanied the blast. Most of the BR TBI patients (33 of 50) had no documentation of secondary or tertiary injury related to the blast event that caused their TBI. This does not mean that all 33 had only primary blast injuries. Secondary or tertiary injuries may not have been realized because loss of consciousness and amnesia frequently occur with TBI. Sixteen of the 50 BR TBI patients had exposure to more than one blast event during their time in theater. In addition, four had incurred NBR head injuries in the past.

The TBI and ocular injury characteristics for the 50 NBR TBI and 50 BR TBI patients are given in Table 1. Traumatic brain injury severity and ocular injury data were only available for 49 of 50 patients in each group. Significantly more BR TBI patients had TBIs rated as mild compared with those in the NBR TBI group ($p = 0.0001$). In fact, only one of 49 NBR TBI patients had a TBI rated as mild. Alternatively, 26 of 49 BR TBI patients had mild TBI, and 23 of 49 had TBI rated as moderate/severe. Table 1 also shows that 2% of NBR TBI and 16% of BR TBI patients had penetrating brain injuries. This between-group difference was significant ($p = 0.031$). The frequency of ocular injuries was approximately equal in the two groups: 29% in the NBR TBI patients and 31% in the BR TBI patients ($p = 1.0$). Ocular injuries in the NBR TBI group included orbital fractures, lid lacerations, traumatic cataracts, traumatic maculopathy, retinal hemorrhages, and optic neuropathy. Recorded cases of ocular injuries in the BR TBI patients were globe ruptures, optic neuropathy, angle

TABLE 1.

Percentages (n/N) of TBI and ocular injury characteristics for the NBR TBI and BR TBI patients

	Mild TBI		Penetrating TBI		Ocular injury		Monocular	
	%	p	%	p	%	p	%	p
NBR	2 (1/49)	0.0001	2 (1/50)	0.031	29 (14/49)	1.0	2 (1/50)	0.112
BR	53 (26/49)		16 (8/50)		31 (15/49)		12 (6/50)	

p values from Fisher exact test.

recession, orbital fractures, hyphema, lid lacerations, and corneal injuries. The last column in Table 1 gives the number of patients who were monocular because of injuries sustained in the TBI-inducing event. The monocular designation included patients with enucleations and also included those with an eye so severely injured that VA could not be measured. One of the NBR TBI and six of the BR TBI patients were monocular. This difference was not significant (p = 0.112). The single monocular NBR TBI patient had no light perception in one eye because of a gunshot wound. There were four enucleations in the BR TBI patient group because of ruptured globes. In addition, two BR TBI patients were rendered blind in one eye, although the type of injuries sustained were not available in the medical record.

The ''best eye'' (best of the two eyes) VA might be thought of as an individual's functional or working VA, and it was very good for most of the TBI patients in this study. The mean logMAR VAs from the ''best eye'' were 0.03 and 0.02 for the NBR TBI and BR TBI groups, respectively. Both of these logMAR values equate to a Snellen VA of approximately 20/21. Only two patients in the NBR TBI and four in the BR TBI group had a best eye VA that was worse than 0.30 (20/40). The worst measured (maximum) VA from all eyes where VA could be measured was 1.30 (20/400) in the NBR TBI patients and 1.53 (20/667) in the BR TBI patients. These values do not include any data from enucleated or severely injured eyes because VA could not be measured in those eyes. To capture the data from blind or missing eyes, VA measurements from each patient's ''worst eye'' were analyzed, and the groups were compared. This was done because reduced acuity from TBI-related neural damage (e.g. optic neuropathy) might be more apparent in one eye, and reduced VA from ocular injury (e.g. globe rupture) would occur in the affected eye. When the acuity of the two eyes was equal, that value was used. In two NBR TBI eyes and two BR TBI eyes, the worst eye VA was reduced from congenital amblyopia, and the VA from the other eye was used. Worst eye acuities were grouped into two categories: good and poor. The good VA category included eyes with VAs that were better than 0.30 (20/40), and the poor VA category included VAs of 0.30 (20/40) or worse. Data from enucleated eyes and eyes where VA could not be measured were included in the poor VA category. With this method, 18% (9 of 50) of NBR TBI eyes and 28% (15 of 50) of BR TBI eyes fell into the poor category. This difference between the NBR TBI and BR TBI groups was not significant (p = 0.342).

Fig. 1 shows percentages of patients from the NBR TBI and BR TBI groups with subjective vision complaints and reading performance deficits. Overall, 69% of NBR TBI patients and 66% of BR TBI patients had a complaint about their vision. A determination of light sensitivity could not be found in all patient records, and data were available for 40 of 50 NBR TBI patients and for 46 of 50 BR TBI patients. On a percentage basis, more than twice as many BR TBI patients reported light sensitivity (67% BR TBI patients vs. 33% NBR TBI patients), and this difference was significant (p = 0.002). About 40% of both NBR TBI patients and BR TBI patients reported having diplopia at some point after their injury (p = 0.670). Reading complaints were reported at a slightly higher rate in BR TBI patients (56% BR TBI patients vs. 47% NBR TBI patients), but the difference was not significant (p = 0.404). Conversely, the frequency of reading deficits was slightly higher in the NBR TBI group (56% BR TBI patients vs. 51% NBR TBI patients; p = 0.820).

Oculomotor deficits found in the two patient groups are shown in Fig. 2. The only significant between-group difference found was for saccades (p = 0.006). Saccadic dysfunction was higher in the NBR TBI patients (85%) compared with that in the BR TBI group (58%). Convergence was measured in only 25 NBR TBI patients and 23 BR TBI patients primarily because many subjects had strabismus or were monocular. In addition, some patients' injuries were such that they could not complete the task. Seventy-eight percent of BR TBI patients had convergence insufficiency compared with 48% of NBR TBI patients, and this difference was not significant (p = 0.062).

Approximately 46% of the NBR TBI patients and 29% of the BR TBI patients were found to have strabismus after their TBI. The difference between groups was not significant (p = 0.125).

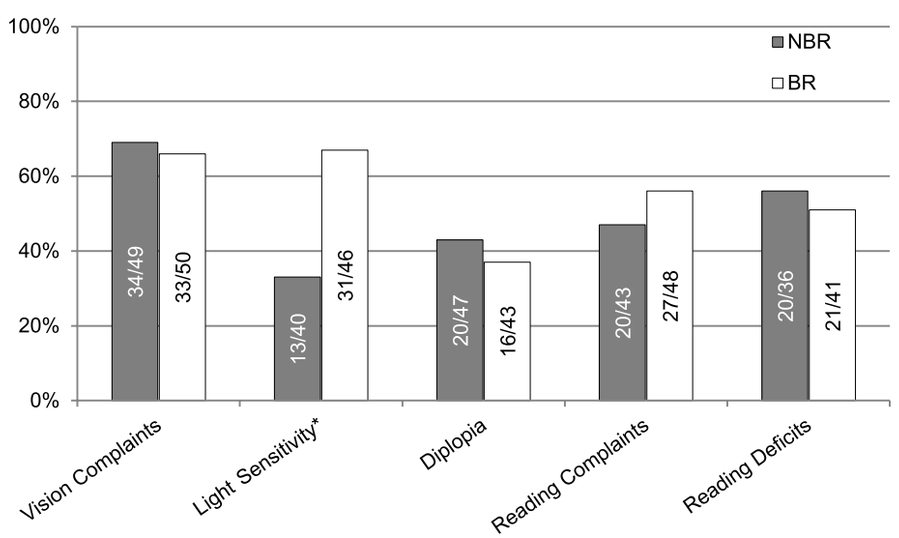

FIGURE 1.

Percentage of patients with subjective vision complaints and reading performance deficits. The number of patients with each anomaly/total number of patients measured is given in each bar. *Light sensitivity was found at a significantly higher frequency in the BR TBI group (p = 0.002).

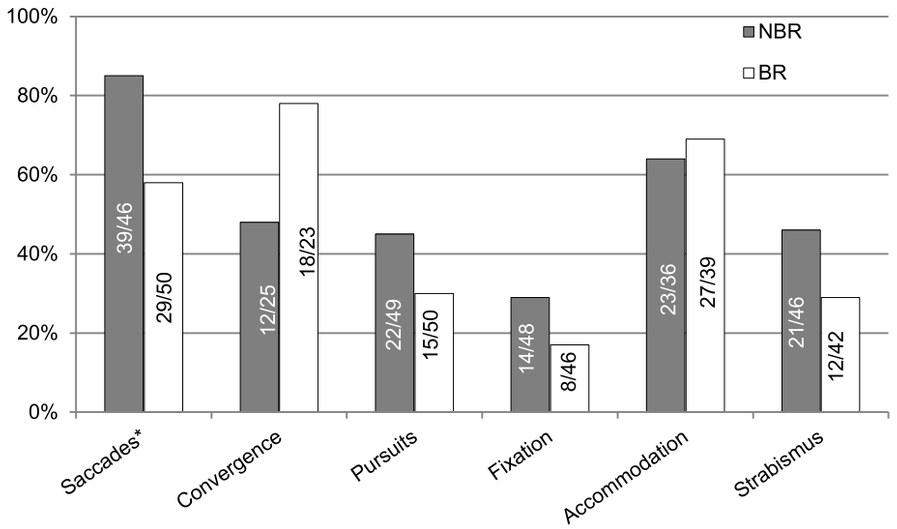

FIGURE 2.

Percentage of patients with oculomotor deficits. The number of patients with each anomaly/total number of patients measured is given in each bar. *Saccadic dysfunction was significantly higher in the NBR TBI group (p = 0.006).

The strabismus types are given in Table 2. Monocular subjects and those with congenital strabismus were excluded from the analyses. Exotropia was the most common form of strabismus in each patient group. Vertical strabismus, either alone or accompanying a lateral strabismus, was relatively common in the NBR TBI group but was only found in one BR TBI patient.

Visual field defects were classified into two categories based on the probable cause of the defect—with and without an associated eye injury. When an eye injury was present, it was determined to be the cause of the defect. Enucleated, no light perception, and light perception only eyes were not included in the analyses. When the data from the NBR TBI and BR TBI groups were pooled, 18% (18 of 99) of the TBI patients had a VF defect in one or both eyes. The occurrence of VF defects was somewhat higher in the NBR TBI group (14% BR TBI vs. 22% NBR TBI), but the difference was not significant (p = 0.308). Five NBR TBI patients and three BR TBI patients had hemianopsia, the most common VF defect. Analyses of combined data from the two patient groups showed that eight of the 13 patients with non–eye injury–associated VF defects had hemianopsia, and six of these eight had homonymous hemianopsia. One patient with hemianopsia was monocular, and one had a VF defect recorded as hemianopsia in one eye only. There were two patients with homonymous quadrantanopsia and three with VF constriction. Three NBR TBI patients and two BR TBI patients had VF defects secondary to an eye injury sustained in the TBI-inducing event. Of these, one patient had an orbital fracture and optic neuropathy with an associated hemianopsia in that eye. This patient's other eye did not have a VF defect. Another patient had an orbital floor fracture with globe displacement and VF constriction. The other three patients had nonspecific ocular trauma noted in their records and a VF defect only in the injured eye.

Significantly more BR TBI patients than NBR TBI patients had TBI classified as mild in this study (p = 0.0001). To determine whether this difference in TBI severity between the NBR TBI and BR TBI groups affected the results, the data were re-examined after removal of all mild TBI patients. This yielded 48 NBR TBI patients and 23 BR TBI patients who had TBIs

TABLE 2.

Acquired strabismus types and amounts

	Strabismus total		Strabismus type				
	% (n/N)	p	XT	ET	VT	Mixed XT VT	Mixed ET VT
NBR	46 (21/46)	0.125	10	1	3	6	1
BR	29 (12/42)		11	0	1	0	0

The p value is from Fisher exact test.
XT, exotropia; ET, esotropia; VT, vertical tropia.

TABLE 3.

Injury characteristics and vision deficits after removal of patients with mild TBI

Injury/anomaly	NBR TBI (n = 48), %	BR TBI (n = 23), %	p
Penetrating TBI	4	36	0.001
Ocular injury	28	52	0.063
Monocular	0	22	0.003
VA > 20/40 in worst eye	19	52	0.006
Vision complaints	67	70	1.0
Light sensitivity	33	55	0.162
Diplopia	38	31	0.766
Reading complaints	46	52	0.790
Reading deficit	53	67	0.390
Strabismus	43	31	0.554
Accommodation defect	62	61	1.0
Convergence defect	48	67	0.654
Pursuit defect	46	26	0.128
Saccade defect	84	48	0.004
Fixation defect	30	15	0.238
Visual field defect	18	14	1.0

p values from Fisher exact test.

rated as moderate/severe. Table 3 shows these results. With the mild TBI patients removed, the significantly higher rate of penetrating TBI in the BR TBI group remained ($p = 0.001$). In addition, the higher frequency of monocular status in the BR TBI patients increased and reached significance ($p = 0.001$). The percentage of worst eyes with logMAR VA poorer than 0.30 (20/40) was significantly greater in the BR TBI group ($p = 0.006$), mirroring the higher monocular status and ocular injury rates. The frequency of light sensitivity in the BR TBI group remained greater than that in the NBR TBI group, but the difference was no longer found to be statistically significant ($p = 0.162$). With respect to oculomotor measurements, the higher rate of NBR TBI patients with saccadic dysfunction compared with that in the BR TBI group remained significant ($p = 0.004$). The other measures of vision including subjective vision complaints, reading dysfunction, and oculomotor deficits did not change significantly with the mild TBI cases removed.

Table 4 shows that the time between the TBI-inducing event and eye examination varied significantly between the two patient groups ($p < 0.001$). The average time from injury to examination for the BR TBI group (1.01 ± 1.18 years) was more than three times longer than that for the NBR TBI group (0.32 ± 0.52 years). The reasons for this difference are unknown, but all patients received eye care in the PRC as soon after admission as their conditions allowed.

DISCUSSION

Abnormalities in visual and oculomotor functions have been previously documented in patients with acquired brain injury, including TBI.[10,11,14,27] In the current study, 97% of the 100 TBI patients had a subjective complaint about their vision, a vision function deficit, or both. Approximately 67% had one or more subjective vision complaints documented in their examination record (Fig. 2). Specific symptoms of light sensitivity, reading problems, and diplopia were common. Other researchers have also found subjective vision complaints to be common in TBI patients. Two previous studies from the Palo Alto PRC reported that approximately 75% of TBI patients had vision complaints.[14,27] Stelmack et al.,[8] in a study of 88 TBI patients from a Veterans Affairs hospital, also found that 75% had self-reported visual symptoms after their injury. Lew et al.[9] found that 66% of 62 TBI patients from a polytrauma network site reported visual disturbances. The causes of these visual symptoms in TBI patients are not well understood, although reading problems, diplopia, and other subjective vision complaints could be related to changes in oculomotor, binocular, and accommodative functions.

In this study, both NBR TBIs and BR TBIs were associated with similar frequencies and types of vision function deficits. Damage to the central nervous system in both TBI types might help explain these findings. If similar areas of the brain are damaged in NBR TBI and BR TBI, similar visual system function alterations might be expected. Thus far, it has been difficult to distinguish differences in brain morphology attributable to blast compared with nonblast causes, even with modern scanning techniques.[20] However, some studies have indicated that primary blast exposures may induce biochemical changes in the brain that differ from those seen in other TBI types, indicating that discernible injury mechanisms may exist between NBR TBI and primary BR TBI.[21] On the other hand, Stuhmiller[28] and Champion et al.[4] have argued that TBI after a blast event causes brain injury through secondary and tertiary mechanisms and not through primary blast exposure. Therefore, blast exposure may not produce uniquely different injuries compared with other traumas. Our findings largely support this position in that there were few visual dysfunction differences between NBR TBI and BR TBI groups.

One of the goals of this study was to determine whether vision symptoms and problems occurred more frequently in patients with NBR TBI or those with BR TBI. We found significant differences for two measures: light sensitivity and saccadic dysfunction. Subjective complaints of light sensitivity were significantly greater in the BR TBI group than those in the NBR TBI group (Fig. 1). When the patients with mild TBIs were removed from the analysis, the frequency of light sensitivity remained greater in the BR TBI group, but the difference was no longer significant (Table 3). In a related finding, Lew et al.[29] reported significantly greater sensitivity to light and/or noise in TBI patients who were wounded in combat (74% of whom were exposed to blasts) compared with patients with TBIs sustained in noncombat situations. Comparison with the results of the Lew et al.[29] report is confounded, however, because it did not distinguish light sensitivity from noise sensitivity. In general, light sensitivity was a common vision complaint of the TBI patients in the current study because more than 50% of the 100 TBI patients complained of increased sensitivity to light since their injury. Others have reported even higher percentages of light sensitivity in TBI patients. In a study of 88 TBI patients, Stelmack et al.[8] reported that 52 (59%) had complaints of light sensitivity. Similarly, Heitger et al.[30] found light sensitivity as a visual symptom in 20 (54%) of 37 patients with mild closed head injury during the first week after their injury.

Oculomotor dysfunction was also very common in this study, and one or more oculomotor deficits were measured in 88% of TBI patients whose records were reviewed. The numbers were similar for each patient group, with 90% of NBR TBI patients and 86% of BR TBI patients having at least one oculomotor defect. These results are comparable to the 90% frequency reported by Ciuffreda et al.[10] in a retrospective study of 160 TBI patient records. A much lower rate of visual dysfunction of 10.9% was reported by Dougherty et al.[13] in a retrospective study of 837 service members who sustained TBI while serving in

TABLE 4.

Years between injury date and examination date for the 50 NBR TBI patients and the 50 BR TBI patients

	NBR TBI	BR TBI
Mean*	0.32	1.01
SD	0.52	1.18
Median	0.15	0.48
Maximum	3.13	4.79
Minimum	0.02	0.03

*t test, $p < 0.001$.
SD, standard deviation

Operation Iraqi Freedom. The record selection process used by Dougherty et al.[13] may help explain this low frequency of ocular and vision problems identified compared with those in other studies. They reviewed each service member's medical record for *ICD-9-CM* (*International Classification of Diseases, Ninth Revision, Clinical Modification*) codes relating to ocular/visual dysfunctions. There is no indication of how many, if any, of the service members had eye examinations or whether oculomotor function was measured. Conversely, the current study as well as the study conducted by Ciuffreda et al.[10] included only TBI patients who were seen by referral for an eye examination, and these patients might be expected to exhibit more vision problems based on their referral.

Saccadic dysfunction was found at a significantly higher frequency in the NBR TBI group. This difference remained significant even when the mild TBI patients were removed from the analyses. With this information, the inference can be made that saccadic function was affected more by TBI mechanism (NBR TBI vs. BR TBI) than by TBI severity (mild vs. moderate/severe) in these patients. Few other papers have compared vision or oculomotor functions in patients with NBR TBI with those in patients with BR TBI. In a report comparing psychological symptoms between patients with primary BR TBI versus those with TBI from other causes (secondary/tertiary BR TBI and NBR TBI), Luethcke et al.[31] found that the prevalence of self-reported vision problems did not differ significantly between the groups. In a 2009 study of 192 TBI patients, Brahm et al.[14] found several oculomotor measures that differed between patients with NBR TBIs and those with BR TBIs. Summarizing the results of Brahm et al.,[14] higher occurrences of accommodative insufficiency and pursuit/saccadic dysfunction were found in the BR TBI group, and slightly higher frequencies of reading difficulties were present in the NBR TBI group of PRC patients. As reported by the authors, the higher number of patients with BR TBI compared with those with NBR TBI included in the study might have affected the validity of the results.[14] The current study included equal numbers of NBR TBI and BR TBI patients of similar ages, which yielded data suitable for statistical comparison.

The overall results of how both forms of TBI affected vision function in these patients are worth noting. Surprisingly, none of the TBI patients in this study met the criteria for legal blindness. The best eye VAs from the TBI patients were relatively good. Other researchers have reported more severe VA losses in TBI patients. A previous study of 68 PRC patients with moderate to severe TBI found that 32.3% had VAs of 0.48 (20/60) or worse.[14] Other studies have found legal blindness to be present in 3% to 14% of TBI patients.[8,27] Conversely, high rates of vision function defects were found in the current study. Overall, convergence insufficiency and accommodative insufficiency were found in more than 60% of the TBI patients, and this is greater than what has been reported by several researchers.[10,14,27] Acquired strabismus was recorded in about 38% of the TBI patients. Both Brahm et al.[14] and Ciuffreda et al.[10] have previously reported that about 25% of TBI patients had strabismus. Regardless of the absolute numbers, these results and others demonstrate that TBI is associated with high rates of vision disorders.

Finally, the elapsed time between sustaining a TBI and receiving an optometric vision function examination varied for the two patient groups in this study. Although the exact reasons for this difference are unknown, it might be related to the geographic location where each patient's TBI event occurred. All of the BR TBI patients were injured in Iraq or Afghanistan, whereas only two of the NBR TBI patients were injured in Iraq. In addition, the pathway to Veterans Affairs medical care and admission to the Palo Alto Veterans Affairs PRC may be longer for those injured in a combat zone. It is known that TBI-induced neurologic and vision symptoms can change over time, but the changes are not always consistent and can be influenced by a number of factors.[30] If vision function deficits improve over time, the BR TBI patients in this study might have been expected to exhibit fewer vision problems compared with the NBR TBI patients. If vision function worsens with time, more problems might have been expected in the NBR TBI group. For the most part, neither of these expectations was confirmed because both groups displayed similarly high rates of vision problems.

CONCLUSIONS

Vision dysfunction occurred after all severities of TBI in this study. Visual acuity remained normal in most of these patients and was not a reliable predictor of visual outcome after brain injury. The mechanism of injury, blast or nonblast, did not seem to result in different frequencies or types of visual dysfunction. The detrimental impacts of TBI on vision function found here are in general agreement with those of previous studies.[10,14,27] With the exception of two areas, light sensitivity and saccadic dysfunction, no significant differences in visual dysfunction were found between the study groups. Deficits in oculomotor, vergence, and accommodative function were common in both groups. Reading problems, both reading complaints and observed reading deficits, were also found in about one-half of the patients and may be related to the oculomotor dysfunctions identified. Visual acuity and VF deficits, although present and sometimes severe, were found less frequently than most visual symptoms and oculomotor defects. In many cases, the VA or VF deficit could be traced to an ocular injury. The increased rate of light sensitivity in the BR TBI patients might be consistent with the increased sensitivity to light and noise in TBI patients with blast injuries reported by Lew and colleagues.[29] Further research is needed to evaluate this hypothesis. In addition, more research is needed to understand the pathophysiological and neurologic changes that occur in all types of acquired brain injury. This, in turn, will aid us in understanding the subsequent changes that take place in the visual system and in making specific recommendations to improve the eye and vision care of individuals with TBI. Because of the high prevalence of subjective visual complaints and oculomotor dysfunctions in the TBI patients in the current study, as well as in previous studies, a comprehensive vision examination should be conducted after brain injury, regardless of injury type or severity.

ACKNOWLEDGMENTS

This project was supported in part by Veterans Affairs Quality Enhancement Research Initiative (QUERI) grant RRP 11-008.

Received June 6, 2012; accepted November 13, 2012.

REFERENCES

1. Faul M, Likang X, Wald MM, Coronado VG. Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations and Deaths 2002–2006. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Injury Prevention and Control; 2010. Available at: http://www.cdc.gov/traumaticbraininjury/pdf/blue_book.pdf. Accessed November 15, 2012.
2. Veterans Affairs Office of Research and Development, Health Services Research and Development Service. Polytrauma and Blast-Related Injuries. VA Quality Enhancement Research Incentive (QUERI) Fact Sheet. Minneapolis, MN: VA Office of Research and Development, Health Services Research and Development Service; 2011. Available at: http://www.queri.research.va.gov/about/factsheets/polytrauma_factsheet.pdf. Accessed November 15, 2012.
3. Defense and Veterans Brain Injury Center. DoD Worldwide Numbers for TBI; 2012. Available at: http://www.dvbic.org/dod-worldwide-numbers-tbi. Accessed: July 26, 2012.
4. Champion HR, Holcomb JB, Young LA. Injuries from explosions: physics, biophysics, pathology, and required research focus. J Trauma 2009;66:1468–77.
5. Hibbard MR, Uysal S, Sliwinski M, Gordon WA. Undiagnosed health issues in individuals with traumatic brain injury living in the community. J Head Trauma Rehabil 1998;13:47–57.
6. Doble JE, Feinberg DL, Rosner MS, Rosner AJ. Identification of binocular vision dysfunction (vertical heterophoria) in traumatic brain injury patients and effects of individualized prismatic spectacle lenses in the treatment of postconcussive symptoms: a retrospective analysis. PM R 2010;2:244–53.
7. Green W, Ciuffreda KJ, Thiagarajan P, Szymanowicz D, Ludlam DP, Kapoor N. Accommodation in mild traumatic brain injury. J Rehabil Res Dev 2010;47:183–99.
8. Stelmack JA, Frith T, Van Koevering D, Rinne S, Stelmack TR. Visual function in patients followed at a Veterans Affairs polytrauma network site: an electronic medical record review. Optometry 2009;80:419–24.
9. Lew HL, Poole JH, Vanderploeg RD, Goodrich GL, Dekelboum S, Guillory SB, Sigford B, Cifu DX. Program development and defining characteristics of returning military in a VA polytrauma network site. J Rehabil Res Dev 2007;44:1027–34.
10. Ciuffreda KJ, Kapoor N, Rutner D, Suchoff IB, Han ME, Craig S. Occurrence of oculomotor dysfunctions in acquired brain injury: a retrospective analysis. Optometry 2007;78:155–61.
11. Du T, Ciuffreda KJ, Kapoor N. Elevated dark adaptation thresholds in traumatic brain injury. Brain Inj 2005;19:1125–38.
12. Lew HL, Weihing J, Myers PJ, Pogoda TK, Goodrich GL. Dual sensory impairment (DSI) in traumatic brain injury (TBI)—An emerging interdisciplinary challenge. NeuroRehabilitation 2010;26:213–22.
13. Dougherty AL, MacGregor AJ, Han PP, Heltemes KJ, Galarneau MR. Visual dysfunction following blast-related traumatic brain injury from the battlefield. Brain Inj 2011;25:8–13.
14. Brahm KD, Wilgenburg HM, Kirby J, Ingalla S, Chang CY, Goodrich GL. Visual impairment and dysfunction in combat-injured service members with traumatic brain injury. Optom Vis Sci 2009;86:817–25.
15. Martin EM, Lu WC, Helmick K, French L, Warden DL. Traumatic brain injuries sustained in the Afghanistan and Iraq wars. J Trauma Nurs 2008;15:94–9.
16. Cernak I, Noble-Haeusslein LJ. Traumatic brain injury: an overview of pathobiology with emphasis on military populations. J Cereb Blood Flow Metab 2010;30:255–66.
17. Smith DH, Meaney DF, Shull WH. Diffuse axonal injury in head trauma. J Head Trauma Rehabil 2003;18:307–16.
18. Werner C, Engelhard K. Pathophysiology of traumatic brain injury. Br J Anaesth 2007;99:4–9.
19. Taber KH, Warden DL, Hurley RA. Blast-related traumatic brain injury: what is known? J Neuropsychiatry Clin Neurosci 2006;18:141–5.
20. Mac Donald CL, Johnson AM, Cooper D, Nelson EC, Werner NJ, Shimony JS, Snyder AZ, Raichle ME, Witherow JR, Fang R, Flaherty SF, Brody DL. Detection of blast-related traumatic brain injury in US military personnel. N Engl J Med 2011;364:2091–100.
21. Svetlov SI, Larner SF, Kirk DR, Atkinson J, Hayes RL, Wang KK. Biomarkers of blast-induced neurotrauma: profiling molecular and cellular mechanisms of blast brain injury. J Neurotrauma 2009;26:913–21.
22. Warden D. Military TBI during the Iraq and Afghanistan wars. J Head Trauma Rehabil 2006;21:398–402.
23. United States Department of Veterans Affairs. Polytrauma/TBI System of Care. Understanding Traumatic Brain Injury: Definition and Background. 2011. Available at: http://www.polytrauma.va.gov/understanding-tbi/definition-and-background.asp. Accessed: November 30, 2011.
24. American Congress of Rehabilitation Medicine. Definition of mild traumatic brain injury. J Head Trauma Rehabil 1993;8:86–7. Available at: http://www.acrm.org/pdf/TBIDef_English_Oct2010.pdf. Accessed November 15, 2012.
25. MacGregor AJ, Dougherty AL, Morrison RH, Quinn KH, Galarneau MR. Repeated concussion among US military personnel during Operation Iraqi Freedom. J Rehabil Res Dev 2011;48:1269–78.
26. Hofstetter HW. A comparison of Duane's and Donder's tables of the amplitude of accommodation. Am J Optom Arch Am Acad Optom 1944;21:345–63.
27. Goodrich GL, Kirby J, Cockerham G, Ingalla SP, Lew HL. Visual function in patients of a polytrauma rehabilitation center: a descriptive study. J Rehabil Res Dev 2007;44:929–36.
28. Stuhmiller JH. Blast Injury: Translating Research into Operational Medicine. Fort Detrick, MD: US Army Medical Research and Material Command; 2008.
29. Lew HL, Poole JH, Guillory SB, Salerno RM, Leskin G, Sigford B. Persistent problems after traumatic brain injury: the need for long-term follow-up and coordinated care. J Rehabil Res Dev 2006;43:vii–x.
30. Heitger MH, Jones RD, Frampton CM, Ardagh MW, Anderson TJ. Recovery in the first year after mild head injury: divergence of symptom status and self-perceived quality of life. J Rehabil Med 2007;39:612–21.
31. Luethcke CA, Bryan CJ, Morrow CE, Isler WC. Comparison of concussive symptoms, cognitive performance, and psychological symptoms between acute blast-versus nonblast-induced mild traumatic brain injury. J Int Neuropsychol Soc 2011;17:36–45.

Gregory L. Goodrich
Department of Veterans Affairs
Palo Alto Health Care System (124)
795 Willow Rd
Menlo Park, CA 94025
e-mail: gregory.goodrich@va.gov