

Comparison of Concussive Symptoms, Cognitive Performance, and Psychological Symptoms Between Acute Blast-Versus Nonblast-Induced Mild Traumatic Brain Injury

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Abstract

Blast-related head injuries are one of the most prevalent injuries among military personnel deployed in service of Operation Iraqi Freedom. Although several studies have evaluated symptoms after blast injury in military personnel, few studies compared them to nonblast injuries or measured symptoms within the acute stage after traumatic brain injury (TBI). Knowledge of acute symptoms will help deployed clinicians make important decisions regarding recommendations for treatment and return to duty. Furthermore, differences more apparent during the acute stage might suggest important predictors of the long-term trajectory of recovery. This study evaluated concussive, psychological, and cognitive symptoms in military personnel and civilian contractors ($N = 82$) diagnosed with mild TBI (mTBI) at a combat support hospital in Iraq. Participants completed a clinical interview, the Automated Neuropsychological Assessment Metric (ANAM), PTSD Checklist-Military Version (PCL-M), Behavioral Health Measure (BHM), and Insomnia Severity Index (ISI) within 72 hr of injury. Results suggest that there are few differences in concussive symptoms, psychological symptoms, and neurocognitive performance between blast and nonblast mTBIs, although clinically significant impairment in cognitive reaction time for both blast and nonblast groups is observed. Reductions in ANAM accuracy were related to duration of loss of consciousness, not injury mechanism. (*JINS*, 2011, 17, 36–45)

Keywords: Blast injuries, Brain concussion, Brain injuries/psychology, Cognition disorder, Neuropsychological tests, Military personnel

INTRODUCTION

The military operations of Operation Iraqi Freedom and Operation Enduring Freedom (OIF/OEF) have sustained the highest ratio of wounded to killed-in-action in U.S. military history (Tanielian & Jaycox, 2008), with an estimated 1 of every 10 injuries being fatal, as opposed to approximately 1 of every 4 in the Persian Gulf, Vietnam, and Korean Wars (Leland & Oboroceanu, 2009). More service members are surviving their injuries due largely to sophisticated armor and medical technology (Tanielian & Jaycox, 2008); however, wounded survivors are often left coping with injuries that can have long-term and severely debilitating consequences, such

as traumatic brain injury (TBI). Estimates suggest that between 15% and 23% of service members experience a TBI during deployment to Iraq, ranging from mild to severe injuries (Hoge et al., 2008; MacGregor et al., 2010; Terrio et al., 2009). Though mild TBI (mTBI) is, by definition, a less serious injury than moderate or severe TBI, mTBI can be associated with substantial and occasionally long-term symptoms, especially when in combination with comorbid conditions such as posttraumatic stress disorder or depression, and is by far the most prevalent TBI diagnosis in veterans returning from OIF (MacGregor et al., 2010).

Although concussive symptoms from mTBI typically resolve within 1 to 3 months (Defense and Veterans Brain Injury Center [DVBIC], 2009), symptoms sometimes persist for longer. Persisting symptoms from mTBI are associated with long-term impairment in areas such as occupational and cognitive functioning. For example, almost half of the military personnel diagnosed with mTBI in one report (Drake, Gray, Yoder, Pramuka, & Llewellyn, 2000) were assigned to

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limited duty between 3 and 15 months post-injury, with younger age and poorer performance in memory and executive functioning being associated with occupational status. In contrast, other studies have suggested that mTBI is not associated with long-lasting cognitive impairment (Dikmen et al., 2009; Ivins, Kane, Schwab, 2009).

Though the findings concerning cognitive impairment are mixed, there is stronger support for the relationship between TBI and increased frequency and severity of psychological symptoms such as depression and anxiety, especially symptoms of posttraumatic stress disorder (PTSD), among military samples (Elder & Cristian, 2009; Hoge et al., 2008; Warden, 2006; Whelan-Goodinson, Ponsford, Johnston, & Grant, 2009). Furthermore, Hoge et al. found that the relationship between mTBI, somatic, and postconcussive symptoms and outcomes in patients with loss of consciousness (LOC) is largely explained by PTSD and depression, and by PTSD alone in mTBI patients without LOC, suggesting that psychological symptoms may play a key role in exacerbating physiological symptoms, or vice versa (Bryant, 2008).

Though the typical resolution of TBI symptoms is fairly well documented, few studies have compared mechanisms of TBI to determine whether the mechanism of injury influences outcomes. TBIs can be caused by a wide variety of mechanisms such as motor vehicle accidents, blunt force trauma, falls, and blast injuries, the latter of which are particularly unique. Not only are blast injuries the most prevalent mechanism of TBI among OIF/OEF service members (DVBIC, 2009; Warden, 2006), but they are also unique because the air pressure wave caused by the explosion can cause barotrauma. Barotrauma (also called primary blast injury) results from over- or under-pressurization in organs filled with air or with air-fluid interfaces, such as the lungs, ears (tympanic membrane), eyes, brain, and spinal cord (Finkel, 2006). Blast injuries can additionally result from being struck in the head by flying objects set into motion by the blast wave (i.e., secondary injury), and/or as a result of the body striking an object (e.g., a wall or a vehicle) after being propelled outward from the blast source (i.e., tertiary injury). Other miscellaneous causes of injury associated with blasts, such as falling buildings, burns, inhalation of toxic fumes, or exposure to radiation, are classified as quaternary injury (Pennardt & Lavonas, 2009). Although nonblast TBIs commonly result from physical trauma equivalent to secondary, tertiary, and quaternary blast injuries, the uniqueness of barotraumata (i.e., primary blast injury) associated with blast-related TBIs might have distinct clinical implications.

For example, headaches seem to be particularly prominent among blast-injured patients. Wilk et al. (2010) found that among service members with mTBI and loss of consciousness (LOC), headaches and tinnitus were more common in blast injuries than in nonblast injuries, although this relationship did not hold true among service members with mTBI without LOC. Theeler and Erickson (2009) also found that among service members returning from Iraq with chronic headaches, 41% had experienced mild head or neck trauma, the majority (67%) of which were due to blasts. Results from

Belanger, Kretzmer, Yoash-Gantz, Pickett, and Tupler (2009) additionally suggest that there may be differences in psychological symptoms between blast and nonblast patients. They found a nonsignificant trend for service members with blast injuries to report more PTSD symptoms than service members with nonblast injuries.

Due to the increasing prevalence of blast injuries among deployed service members and the potential severity of the symptoms resulting from mTBI, it is critical that more research is conducted comparing blast to nonblast mTBIs. To the best of our knowledge, Belanger et al. (2009) conducted the only previous study with a primary purpose of comparing blast to nonblast injuries. However, interpretation of their results is limited because they evaluated participants an average of two years after TBI. Previous studies of mTBI among military personnel are similarly limited because measures are commonly administered after return from deployment, usually several months after injury (Hoge et al., 2008; Ivins et al., 2009; Sayer et al., 2008; Terrio et al., 2009; Wilk et al., 2010). Though measuring symptoms several months or years after the blast injury may provide significant insight into postconcussional disorder and long-term impairment, there is a lack of research measuring the physical, cognitive, and psychological sequelae immediately following the injury. Understanding the similarities and differences of blast and nonblast injuries in this acute phase (i.e., within 72 hr) of injury could provide critical information about the trajectory of recovery following mTBI of varying causes and could also provide valuable information to military clinicians in charge of recommending treatment and return to duty. The current study's primary aim is therefore to explore differences in concussive symptoms, cognitive performance, and psychological symptoms within the first 72 hr of an index mTBI associated with either a blast or nonblast mechanism of injury among deployed service members.

METHODS

Participants

Participants included military personnel ($n = 101$) and civilian contractors ($n = 3$) referred to an outpatient TBI Clinic located at a forward-deployed combat support hospital (CSH) in Iraq, assessed within 72 hr of the index injury. Participants were referred to the TBI Clinic via one of two primary routes, depending on where the personnel were stationed. The first route (45.2%) entailed personnel from outlying bases being medically evacuated directly from the battle field to the CSH for screening, CT imaging, and treatment. Personnel who did not meet criteria for a moderate or severe TBI (i.e., mild TBI or no TBI) were then discharged from the CSH to the outpatient TBI Clinic for further assessment and treatment. In the second route, personnel who were stationed at the same base as the TBI Clinic (54.8%) were referred by a primary medical provider following the index head injury. Only head injuries resulting in a diagnosis of mTBI or no TBI were evaluated and treated at the outpatient clinic; all cases

meeting criteria for moderate or severe TBI were immediately evacuated from Iraq for more advanced evaluation and treatment. Study approval was obtained from the Brook Army Medical Center Institutional Review Board, the U.S. Army Medical Research and Materiel Command's Office of Research Protection, and the Multi-National Force-Iraq Institutional Official.

Of the 104 patients seen within 72 hr of the index injury, only 78.8% ($n = 82$) met the Department of Defense and Department of Veterans Affairs Traumatic Brain Injury Task Force's (2008) criteria for mild TBI. The Task Force defined TBI as a traumatically-induced structural injury and/or physiological disruption of brain function as a result of an external force that is indicated by new onset or worsening of at least one of the following clinical signs immediately following the event: (1) any period of loss of or decreased level of consciousness; (2) any loss of memory for events immediately before or after the injury; (3) any alteration in mental state at the time of the injury; (4) neurological deficits that may or may not be transient; (5) intracranial lesion. Mild TBI (mTBI) is defined as any head injury meeting the following criteria: normal structural imaging, loss of consciousness less than 30 min, alteration of consciousness up to 24 hr, and posttraumatic amnesia of less than 24 hr.

A chi-square analysis revealed a statistically significant difference in the frequency of mTBI diagnosis between groups, with 100% ($n = 42$) of nonblast injuries meeting the Task Force's criteria for mTBI, and only 65.2% ($n = 40$) of blast injuries meeting mTBI criteria. To make blast and nonblast groups more comparable, only the participants meeting mTBI criteria were included for analysis. The average participant was a young (26.86 ± 6.50 years), male (93.9%), junior enlisted (62.0% E1-E4 rank) Soldier (74.4% Army), and had been in Iraq for an average of 4.76 ± 2.72 months at the time of injury. Additional demographics are reported in Table 1. Chi-square and independent samples t-test analyses uncovered no demographic differences between those with blast and nonblast mechanisms of injury.

Procedures and Measures

Upon arrival at the outpatient TBI Clinic, personnel underwent a standardized intake evaluation which included computerized neurocognitive testing, psychological and physical health questionnaires, a clinical interview conducted by a clinical psychologist (the second author) or clinical social worker under the supervision of the psychologist, and a physical examination by a physician. Where indicated, personnel were referred to specialists (e.g., ear, nose, and throat, physical therapy, ophthalmology) for additional evaluation and treatment recommendations.

Injury mechanism

Cause of injury was determined via clinical interview and collateral reports, when available. Collateral report of injury

Table 1. Demographics for patients meeting criteria for mTBI according to injury mechanism

	Nonblast		Blast		χ^2	<i>p</i>
	<i>n</i>	%	<i>n</i>	%		
Male	38	90.5	39	97.5	1.765	.184
Race					3.795	.284
White	26	61.9	27	69.2		
Black	12	28.6	5	12.8		
Hispanic	3	7.1	6	15.4		
Asian/Pacific	1	2.4	1	2.6		
Status					4.673	.197
Active duty	28	73.7	19	52.8		
National Guard	7	18.4	14	38.9		
Reserve	2	5.3	1	2.8		
Civilian	1	2.6	2	5.6		
Branch					2.573	.462
Army	33	78.6	28	70.0		
Air Force	7	16.7	6	15.0		
Marines	1	2.4	4	10.0		
Civilian	1	2.4	2	5.0		
Rank					2.310	.067
E1-E4	25	61.0	24	63.2		
E5-E6	9	22.0	11	28.9		
E7-E9	3	7.3	2	5.3		
Warrant officer	1	2.4	0	0		
Officer	3	7.3	1	2.6		
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>t</i>	<i>p</i>
Age	26.62	6.68	27.13	6.38	0.351	.727
Days since injury	1.60	0.73	1.53	0.91	0.385	.702
Months in Iraq	4.63	2.96	4.90	2.48	0.432	.667
Treatment days	4.29	3.04	3.30	3.27	1.414	.161
Years of service	2.25	5.42	6.04	4.52	0.176	.859
Prior deployments	0.69	0.95	0.56	0.88	0.621	.537
Tour length (months)	10.63	2.37	10.85	2.94	0.354	.725
No. of past head injuries	2.00	1.78	2.23	2.15	0.514	.609

Note. mTBI = mild traumatic brain injury.

was usually obtained via medical records or notes accompanying injured service members, or via written or verbal report of witnesses (e.g., other members of the unit involved in the incident). Collateral reports were infrequently available since patients typically arrived via the medical aerovac system unaccompanied and without medical documentation.

Because the difference in mechanisms of blast and nonblast injuries is exposure to a blast wave (i.e., primary blast injury), participants were divided into groups based on whether their injury was most proximally related to primary blast injury ("blast injury" group) or another form of injury ("nonblast injury" group; including secondary, tertiary, or quaternary blast injuries and injuries not involving blasts). In cases in which participants were exposed to primary and other mechanisms of blast injury, participants were categorized based on whether primary blast injury was the most proximal cause of injury. For example, if a blast overturned a service member's vehicle, two potential head injury

mechanisms could exist (i.e., blast and MVA). If no evidence for a nonblast mechanism was reported or could be identified, the injury was coded as a blast injury. If, however, a nonblast mechanism was clearly related to the injury (e.g., the service member remained conscious up until the point of striking his head against the door frame), the mechanism of injury was coded as a nonblast injury. Nonblast mechanisms of injury included blunt object ($n = 14$; 32.6%), sport/recreation ($n = 11$; 25.6%), falls ($n = 10$; 23.3%), and motor vehicle accident ($n = 8$; 18.6%).

Concussive symptoms

Concussion-related symptoms were measured via a self-report questionnaire and clinical interview; they were differentiated between symptoms experienced immediately post-injury (“Which symptoms did you experience right after the injury?”) and symptoms experienced at the time of the evaluation (“Which symptoms are you currently experiencing?”). Symptoms assessed included those contained within the Military Acute Concussion Evaluation (DVBIC, 2007) symptom checklist: loss of consciousness, alteration in consciousness, headache, dizziness, memory problems, balance problems, nausea, vomiting, concentration impairment, irritability, visual disturbances, hearing problems, and sleep disruption.

Cognitive performance

The Automated Neuropsychological Assessment Metrics (ANAM, 2007) was used to measure neurocognitive performance. The ANAM is a computerized test that measures six cognitive domains suspected to be most highly impacted by concussion injuries: simple reaction time (SRT), procedural reaction time (PRT), learning (LRN), working memory (WM), delayed memory (DM), and spatial memory (SM). The ANAM records performance in two dimensions: speed (S) and accuracy (A). Scaled scores were used as dependent variables to account for age and gender effects.

Psychological symptoms

Psychological symptoms were measured using three self-report methods. Symptoms of PTSD were measured with the PTSD Checklist-Military Version (PCL-M; Weathers, Litz, Herman, Huska, & Keane, 1993), which is a 17-item self-report inventory that assesses the severity of each DSM-IV-defined PTSD symptoms. The PCL-M is widely used in the Department of Defense and the Veterans Administration, and it has excellent reliability and validity (e.g., Blanchard, Jones-Alexander, Buckley, & Forneris, 1996; Weathers et al., 1993). Overall mental health symptoms and functioning were measured with the Behavioral Health Measure (BHM; Kopta & Lowry, 2002), which is a 20-item self-report questionnaire that uses Likert rating scales to assess overall global mental health functioning (GMH). The BHM has good internal consistency ($\alpha = .89$), test-retest reliability ($r = .80$), adequate construct and concurrent validity as compared to other

widely-used measures of psychological functioning (Kopta & Lowry, 2002), and can be used to distinguish several levels of mental health impairment (e.g., normal, mild, moderate, severe) based on the clinical significance criteria recommended by Jacobson and Truax (1991). Current mood was assessed using the ANAM’s mood state scales (happiness, vigor, fatigue, restlessness, anxiety, depression, anger), which have demonstrated convergent and divergent construct validity as compared to other mood and symptom measures (Johnson, Vincent, Johnson, Gilliland, & Schlegel, 2008).

Insomnia and alertness

Insomnia was measured using the Insomnia Severity Index (ISI; Bastien, Vallières, & Morin, 2001), which is a 7-item index of insomnia severity that measures perceived severity of problems, dissatisfaction with current sleep patterns, and functional impairment due to sleep problems. The scale can be used to determine clinically significant levels of insomnia. It has demonstrated acceptable internal consistency and good concurrent validity with physiological measures of insomnia. In addition, the ANAM’s Sleep Scale is a single-item measure of subjective alertness included as a standard part of the ANAM evaluation.

RESULTS

Differences in Clinical Features and Concussive Symptoms by Injury Mechanism

Chi-square analyses were conducted to compare clinical and concussive symptom features of blast versus nonblast injury groups, and are summarized in Table 2. No significant difference was found in return to duty (RTD) disposition following treatment at the in-theater TBI clinic, with a greater than 90% RTD rate for both groups. Blast injuries were less frequently associated with loss of consciousness (LOC), with just over one-third of blast injuries reporting LOC as compared to around half of nonblast injuries (54.8%). When LOC occurred, blast injuries were marked by shorter duration LOC. Alteration in consciousness (i.e., feeling “dazed and confused”) was equally common in both injury types, as was amnesia for the index event (i.e., not remembering what happened). Not surprisingly, visibly observable bodily damage such as bruising, laceration, or swelling was more common in nonblast injuries. No differences were found between injury types in the reported frequencies of headache, dizziness, memory problems, concentration impairment, irritability, vision problems, or sleep disturbance. Balance problems, nausea, and vomiting were significantly more common in nonblast injuries, and hearing problems were much less common in nonblast injuries. At the time of intake, no differences in current concussive symptoms were found between blast and nonblast injuries with the exception of headaches. Around 83% of patients with nonblast injuries reported a current headache, whereas only half (52%) of blast injuries reported a current headache.

Table 2. Differences in frequency of clinical features and concussive symptoms by injury type

	Nonblast		Blast		χ^2	<i>p</i>
	<i>n</i>	%	<i>n</i>	%		
Disposition RTD	40	95.2	37	92.5	0.268	.604
LOC Duration					8.603	.035
None	19	45.2	25	62.5		
<1 min	9	21.4	12	30.0		
1–20 min	12	28.6	3	7.5		
20+ min	2	4.8	0	0		
Dazed & confused	37	88.1	33	84.6	0.209	.648
Amnesia for index event	21	51.2	15	38.5	1.314	.252
Bruising/laceration/swelling	33	78.6	11	29.7	19.017	.000
Immediate symptoms						
Headache	34	81.0	28	70.0	1.333	.248
Dizziness	28	66.7	22	55.0	1.172	.279
Memory	19	45.2	11	27.5	2.779	.096
Balance	19	45.2	10	25.0	3.671	.055
Nausea	22	52.4	8	20.0	9.259	.002
Vomiting	11	26.2	3	7.5	5.055	.025
Concentration	19	45.2	12	30.0	2.023	.155
Irritability	8	19.0	8	20.0	0.012	.913
Vision	12	28.6	7	17.5	1.411	.235
Hearing	7	16.7	21	52.5	11.699	.001
Sleep	14	33.3	15	37.5	0.156	.693
Current symptoms						
Headache	35	83.3	21	52.5	8.995	.003
Dizziness	9	21.4	7	17.5	0.201	.654
Memory	13	31.0	8	20.0	1.290	.256
Balance	5	11.9	3	7.5	0.451	.502
Nausea	3	7.1	2	5.0	0.164	.685
Vomiting	1	2.4	1	2.5	0.001	.972
Concentration	15	35.7	8	20.0	2.507	.113
Irritability	6	14.3	9	22.5	0.925	.336
Vision	4	9.5	5	12.5	0.186	.666
Hearing	4	9.5	9	22.5	2.586	.108
Sleep	9	21.4	7	17.5	0.201	.654

Note. RTD = return to duty; TBI = traumatic brain injury; LOC = loss of consciousness.

Differences in Psychological Symptoms by Injury Mechanism

Independent t-test analyses with Bonferroni correction for multiple comparisons were conducted to explore differences in self-reported psychological symptoms between injury groups (see Table 3). No differences were found between blast and nonblast injuries in PTSD symptoms, global mental health functioning, global insomnia, or mood, although nonsignificant trends toward greater vigor and lower depression were observed on the ANAM mood scales in the blast injury group.

Differences in Postinjury Cognitive Performance by Injury Mechanism

Mean postinjury ANAM standard scores are reported in Table 4. A few notable observations were made based on initial visual inspection. First, standard scores for speed

generally tended to fall over one standard deviation below the mean, suggesting clinically meaningful reductions in speed. Standard scores for accuracy, in contrast, remained close to the normed mean, suggesting minimal change in accuracy relative to the reference group. One notable exception, however, was the procedural reaction time subtest, which had a mean accuracy standard score approximately two-thirds of a standard deviation below the mean. To investigate potential differences in postinjury performance on the ANAM subtests by injury type, separate multivariate analyses of covariance were performed for speed and accuracy standard scores, with injury type as the source of variation, and LOC duration entered as a covariate. LOC duration was entered as a covariate due to the previous finding that LOC duration was significantly longer among the nonblast injury group, suggesting that injuries could be more severe among the nonblast injuries. Missing ANAM scores reduced the total sample size to $N = 77$ (nonblast $n = 41$, blast $n = 36$) for speed scores,

Table 3. Differences in psychological symptoms by injury type

	Nonblast		Blast		<i>t</i>	<i>p</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>		
PCL-M ^a	26.65	13.22	27.77	8.91	-0.980	.330
GMH ^a	3.39	0.58	3.48	0.38	0.167	.868
ISI ^a	8.20	6.60	7.71	5.97	0.166	.869
ANAM Mood Scales						
Sleep ^a	2.95	1.36	2.91	1.27	0.184	.854
Happiness ^b	54.73	24.70	61.97	22.60	-1.409	.163
Vigor ^b	45.03	22.64	53.34	22.28	-1.803	.075
Fatigue ^b	37.28	24.26	32.17	24.33	1.144	.256
Restlessness ^b	20.20	18.76	22.60	20.38	-0.564	.574
Anxiety ^b	16.68	17.70	15.69	16.69	0.131	.896
Depression ^b	16.83	20.86	9.34	17.28	1.681	.097
Anger ^b	18.03	20.86	21.29	20.71	-0.773	.442

Note. PCL-M = Posttraumatic Stress Disorder Checklist, Military Version; GMH = Global Mental Health; BHM = Behavioral Health Measure; ISI = Insomnia Severity Index.

^aRaw scores. ^bPercentile scores.

and $N = 73$ (nonblast $n = 38$, blast $n = 35$) for accuracy scores. Results indicated that speed did not differ by injury type ($F_S(6, 65) = 0.582$; $p = .744$; *partial* $\eta^2 = .051$) or LOC duration ($F_S(18, 201) = 1.405$; $p = .132$; *partial* $\eta^2 = .112$). Accuracy scores likewise did not significantly differ according to injury type ($F_A(6, 61) = 0.699$; $p = .651$, *partial* $\eta^2 = .067$), but longer periods of LOC demonstrated a nonsignificant trend toward lower accuracy scores ($F_A(18, 189) = 1.611$; $p = .061$; *partial* $\eta^2 = .133$). Although nonsignificant, this trend nonetheless demonstrated a reasonable effect size. An injury

Table 4. Differences in post-injury mean ANAM standard scores by injury type

	Nonblast		Blast	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Simple reaction time				
Speed	81.12	32.72	81.72	45.96
Accuracy	101.05	0.23	101.09	0.28
Procedural reaction time				
Speed	74.61	66.14	79.42	46.97
Accuracy	90.34	36.30	92.17	31.11
Learning				
Speed	86.10	25.68	93.61	19.78
Accuracy	103.08	12.70	105.20	8.42
Delayed memory				
Speed	78.61	25.51	76.28	36.09
Accuracy	97.00	16.85	98.71	16.67
Working memory				
Speed	73.02	27.34	80.39	27.60
Accuracy	106.32	8.67	107.97	9.60
Spatial memory				
Speed	80.00	26.73	90.53	22.95
Accuracy	97.66	18.08	105.11	7.73

Note. Speed: $F_S(6, 70) = 1.919$, $p = .090$, *partial* $\eta^2 = .141$; Accuracy: $F_A(6, 66) = 0.861$, $p = .528$, *partial* $\eta^2 = .073$.

type-by-LOC duration interaction (i.e., 2×4) could not be tested, however, due to small cell counts (i.e., $n = 2$ in two cells, $n = 0$ in one cell).

To identify potential differences in the *magnitude* of postinjury declines in cognitive performance as compared to preinjury baseline performance, we used repeated-measures analysis of covariance to test the main effects of time (i.e., change in ANAM score from baseline to postinjury) and injury type (i.e., blast versus nonblast), and the interaction of time with injury type (i.e., change in ANAM scores by injury type). LOC duration was again entered as a covariate to account for injury severity. Separate analyses were conducted for speed and accuracy scores. Only mTBI patients who had completed the ANAM before their deployment as a part of routine baseline assessment ($N = 53$; nonblast $n = 26$, blast $n = 27$) were included in these analyses. Means and standard deviations are displayed in Table 5. In terms of speed, a significant reduction in speed was found when comparing postinjury performance to baseline performance ($F_S(1, 50) = 10.638$, $p = .002$, *partial* $\eta^2 = .175$), but no significant difference by injury type ($F_S(1, 50) = 0.009$, $p = .923$, *partial* $\eta^2 = .000$) or LOC duration ($F_S(1, 50) = 0.179$, $p = .674$; *partial* $\eta^2 = .004$) was observed. Analysis of the time-by-injury interaction likewise was not significant ($F_S(1, 50) = 0.057$, $p = .812$, *partial* $\eta^2 = .001$). Results suggest that speed significantly slows following TBI relative to predeployment baseline levels, with no differences in magnitude according to injury type, even when accounting for differences in LOC duration.

In terms of accuracy, no significant differences were found from baseline to postinjury ($F_A(1, 46) = 0.086$, $p = .770$, *partial* $\eta^2 = .002$), or between blast and nonblast injury type ($F_A(1, 46) = 0.209$, $p = .650$, *partial* $\eta^2 = .005$). The main effect of LOC duration was significant, however ($F_A(1, 46) = 8.182$, $p = .006$, *partial* $\eta^2 = .151$), with greater duration LOC being associated with lower accuracy scores. Analysis of the time-by-injury interaction likewise was not significant

Table 5. Mean ANAM baseline, post-injury, and pre-/post standard score changes by injury type

Subtest		Speed				Accuracy			
		Nonblast		Blast		Nonblast		Blast	
		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
SRT	Baseline	100.88	16.26	101.78	7.81	101.22	0.85	101.31	1.01
	Postinjury	74.42	38.06	75.44	51.65	101.04	0.21	101.12	0.33
	ΔM	-26.46	43.18	-26.33	50.25	-0.17	0.83	-0.19	0.98
PRT	Baseline	99.50	17.47	96.74	15.01	99.83	11.96	101.92	8.74
	Postinjury	68.88	78.33	72.89	52.09	102.57	10.49	88.73	35.29
	ΔM	-30.62	79.75	-23.85	51.29	2.52	15.55	-13.19	34.48
LRN	Baseline	98.42	14.45	96.56	13.69	104.13	9.96	105.62	6.26
	Postinjury	88.65	27.20	91.04	21.76	104.00	9.77	105.77	8.25
	ΔM	-9.77	24.82	-5.12	22.17	-0.13	7.36	0.15	7.58
DM	Baseline	92.38	16.85	88.52	21.91	105.70	10.84	104.19	9.44
	Postinjury	79.65	27.51	70.78	39.56	96.17	18.52	98.96	17.11
	ΔM	-12.73	24.70	-17.74	30.85	-5.92	20.30	-3.56	19.01
WM	Baseline	85.77	27.99	80.59	27.28	108.00	6.85	112.04	4.12
	Postinjury	73.15	30.43	75.89	30.17	104.78	9.10	108.58	9.18
	ΔM	-12.62	22.05	-4.70	17.52	-2.69	9.41	-4.67	10.92
SM	Baseline	94.15	16.67	96.89	18.57	106.96	6.43	105.65	5.53
	Postinjury	79.38	30.15	86.56	24.37	95.35	19.07	104.23	8.34
	ΔM	-14.77	26.74	-10.33	19.53	-12.46	20.45	-1.37	10.63

Note. Speed: $F_S(1, 50) = 0.923$, $p = .923$, *partial* $\eta^2 = .000$; Accuracy: $F_A(1, 46) = 0.209$, $p = .650$, *partial* $\eta^2 = .005$. ANAM = Automated Neuropsychological Assessment Metric; SRT = simple reaction time; PRT = procedural reaction time; LRN = learning; DM = delayed memory; WM = working memory; SM = spatial memory.

($F_A(1, 46) = 0.316$, $p = .577$, *partial* $\eta^2 = .007$). Results suggest that accuracy does not significantly decrease following mTBI relative to predeployment baseline levels, with no differences in magnitude according to injury type. Closer inspection of pre- and postinjury accuracy scores (see Table 5) found that, in contrast to overall nonsignificant findings in accuracy scores over time, procedural reaction time (PRT) demonstrated a one standard deviation reduction in accuracy for blast injuries, and spatial memory (SM) demonstrated an almost one standard deviation reduction in accuracy for nonblast injuries. Follow-up analyses for each subtest were not conducted, however, to further test these findings due to limited power related to the small sample size.

DISCUSSION

The current study investigated the clinical presentations of deployed service members (and three civilians) presenting to an outpatient medical clinic located in central Iraq within 72 hr of a possible head injury, and aimed to describe any differences between individuals injured via blast or nonblast mechanisms. Results revealed that patients referred to the clinic for a nonblast injury were more likely to meet criteria for a diagnosis of mTBI than patients referred for a blast injury. Though this might suggest that nonblast injuries result in more frequent and severe concussion diagnoses, this conclusion cannot be drawn because participants with

moderate and severe TBI were evacuated and could not be included in the sample, resulting in potential sampling bias. Among those meeting criteria for a diagnosis of mTBI, results suggest that there are few differences in clinical presentation between mTBIs caused by blast versus nonblast mechanisms among deployed military personnel when assessed within 72 hr of the index injury.

Several differences in concussive symptoms were found, with nonblast mTBI being associated with greater frequency of LOC, longer duration LOC, and immediate experience of balance problems, nausea, and vomiting. Differences in the latter symptoms disappeared by the time of evaluation. In contrast, blast mTBIs were more frequently associated with hearing problems immediately following the injury, as might be expected due to barotrauma to the tympanic membrane and/or the intense volume of the explosion, but this difference did not persist between groups at the time of evaluation. Concussive symptoms by and large did not differ by the time participants arrived for a medical evaluation, although nonblast mTBI was more frequently associated with headaches. No differences were found between injury groups in terms of psychological symptoms, suggesting that within 72 hr of an index injury, psychological well-being and functioning do not differ between blast versus nonblast mTBIs. Overall, symptomatic differences between nonblast and blast injury were limited.

Analyses of cognitive performance within 72 hr of injury resulted in interesting patterns of findings. First, there were

no overall differences in speed or accuracy associated with injury type, even when accounting for duration of LOC. Second, LOC duration was associated with reduced accuracy, but not speed, on ANAM subtests. Unfortunately, our limited sample size restricted our ability to further test between-group differences and LOC duration by injury type interactions. Omnibus statistical analyses suggest, however, that there is little difference between blast and nonblast injuries in cognitive performance across most domains measured by the ANAM, and differences observed in terms of accuracy might be due to severity of injury. This pattern of results is consistent with the findings of Belanger et al. (2009), who reported no differences in cognitive performance between blast versus nonblast injuries among patients with TBI evaluated an average of 2 years postinjury, but found that severity of injury did predict cognitive sequelae. In combination with the current findings, the Belanger et al. study suggests that blast versus nonblast TBI might have similar presentations and courses over time, beginning from the acute phase and continuing over the course of years postinjury.

Regarding PTSD symptoms, the present study contrasts with Belanger et al. (2009). Belanger et al. identified a trend for blast injuries to be associated with a greater endorsement of PTSD symptoms than nonblast injuries, whereas this study found no evidence of a difference in PTSD symptoms between groups. This discrepancy could be explained by the difference in the sample populations. While the current study focused on mTBI only, Belanger et al. included participants with mild, moderate, and severe TBI. Another potential explanation is that PTSD symptoms have a differential course over time following an injury. In their sample assessed approximately 2 years post-injury, Belanger et al. reported that endorsement of PTSD symptoms increased with the passage of time following injury. In the current study, mean PCL-M scores did not differ between groups and were lower than mean scores reported by Belanger et al., who also found a significant effect of time on PCL-M scores and a non-significant trend ($p < .07$) toward higher scores in their blast group relative to the nonblast group. Another possibility is that the Belanger et al. sample had greater exposure to trauma relative to the current sample, the latter of which had been in theater an average of only 4 to 5 months. Belanger et al. did not report the duration of combat tours for their sample, however, to determine the reasonableness of this possible explanation.

Analyses likewise revealed little evidence of differences in other types of psychological symptoms between injury groups. Though there was a trend for more depressive symptoms and less vigor after nonblast injuries in comparison with blast injuries, these trends did not reach significance, and the mean scores for both blast and nonblast injuries were still well within normative ranges. Furthermore, mean scores for all psychological symptom variables considered in this study were well below clinical levels. Although this finding might seem to contrast with previous research suggesting that mTBI is associated with increased psychological impairment (Elder & Cristian, 2009; Hoge et al., 2008; Warden, 2006), the current study differs

considerably from prior research in that assessment occurred within the first 72 hr of injury while still deployed in a combat zone, as opposed to measuring symptoms after return from deployment several months or years after the index injury (Elder & Cristian, 2009; Hoge et al., 2008). It is, therefore, possible that during the acute stage of mTBI, concussive symptoms and cognitive impairment are more prominent than psychological symptoms, whereas the reverse is true in the chronic stages of mTBI. Longitudinal studies should be conducted to determine whether clinical presentation, concussive symptoms, and cognitive impairment during the acute stage after an mTBI are predictive of later onset of psychological symptoms such as PTSD. For example, it is possible that cognitive impairment during the acute stage of mTBI may diminish cognitive resources needed to cope with the traumatic experiences often associated with TBI and other combat-related traumas (Bryant, 2008), increasing the risk of later onset of PTSD. Another possibility is that more psychological symptoms arise during the process of readjustment after return from deployment, and are therefore not evident in an acutely injured, deployed population.

In addition to providing information concerning psychological outcomes, this study also provides key information concerning the immediate cognitive repercussions of mTBIs in the deployed setting. Results suggest that mTBIs sustained in the deployed setting led to clinically significant impairment in overall speed of cognition regardless of injury mechanism. Declines in accuracy appear to be more directly related to injury severity (as measured by LOC duration) than injury mechanism, however. Though cognitive symptoms may resolve over time, evidence that mTBIs sustained in the deployed setting cause an acute slowing of response time has critical implications for military clinicians. Clinicians in the combat zone must make recommendations for rehabilitation and dispositional determinations to return to full or limited duty in a short period of time. Increases in reaction time immediately following an mTBI are particularly salient in combat zones, as this symptom may jeopardize the safety of the service member, the combat team, and the mission.

Several limitations warrant discussion. Results of the current study would be considerably strengthened via replication with a larger sample size to increase power to detect differences. Also, with a larger sample size, participants could be divided into more refined groups to better isolate various mechanisms of injury (i.e., primary, secondary, tertiary, quaternary, and any combinations of these). An attempt to isolate mechanisms of injury was made in this study by categorizing participants into blast and nonblast categories according to the mechanism deemed to be the most proximal cause of injury. However, to isolate the mechanism of injury more accurately, a larger sample size would allow researchers to break participants into more subgroups, such that exposure to a primary blast injury alone could be compared with, for example, a combined primary and secondary mechanism of injury. In addition to the small sample size, another limitation is the lack of effort testing. Effort testing was not routinely included in the assessment battery based primarily on the clinical experience of many deployed health care providers

(including the second author), which suggests that poor effort is exceedingly infrequent among deployed military personnel, who are generally very highly motivated to return to duty as quickly as possible. Furthermore, effort testing is typically not part of the routine assessment battery for acute TBI in the wider TBI literature of athletic injuries. Future studies, however, might include effort testing in the deployed setting so that empirical data can be gathered on the frequency of poor effort in this scenario. A final limitation of this study is the lack of follow-up data, which could be of critical importance for understanding the trajectory of symptom expression over time. Longitudinal studies that track recovery and disposition over time would be exceedingly valuable in determining the eventual health outcomes of service members injured during deployment. Despite this limitation, these results have important clinical implications and provide a solid foundation for future research, since this is the first study that we could identify to capture symptom expression among deployed military personnel within 72 hr of mTBI.

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