The Role of Posttraumatic Stress in Acute Postconcussive Symptoms following Blast Injury in Combat

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Mild traumatic brain injury (mTBI) has been called the 'signature' injury of recent wars in Iraq and Afghanistan. Estimates of mTBI in deployed personnel are as high as 20% [1]. Postconcussive symptoms (PCS) are regarded as the core problem following mTBI, comprising headaches, dizziness, sensitivity to light and sound, fatigue, and concentration deficits. Although PCS have traditionally been presumed to result from neurological insult, evidence points to a role of psychological factors in these symptoms [2]. Current military evidence is limited by the reliance on retrospective reports collected after deployment through surveys. Research on mTBI in combat settings is needed to more accurately determine the nature of blast-related mTBI. This study reports the first analysis of military personnel assessed in theatre shortly after exposure to a blast.

Participants were US military personnel serving in Iraq who were examined as part of routine assessments of blast exposure at a military combat theatre hospital between September 2006 and September 2007. The study was approved by the Institutional Review Board at Wilford Hall Medical Center, Lackland Air Force Base, San Antonio, Tex., USA.

There were 685 personnel (mean age 26.4 ± 6.8 years) who had been exposed to an explosive blast. mTBI was defined as documented occurrence of injury to the head, loss of consciousness for less than 30 min, posttraumatic amnesia of less than 24 h, and normal computerized tomography findings with no focal neurological deficit or intracranial complications [3]. A total of 567 (83%) participants suffered an mTBI, and 118 (17%) had no TBI. Six personnel were excluded because of moderate/severe TBI.

Assessed demographic information included gender, age, service branch, marital status, date of head injury, location and cause of head injury, and history of prior blast exposures. The Military Acute Concussion Evaluation (MACE), a clinician-administered assessment, was derived from the Standardized Assessment of Concussion [4] assessed for PCS. Posttraumatic stress disorder (PTSD) symptoms were derived from the PTSD Checklist - Military Version (PCL-M) [5]. Participants were examined an average of 7.4 days (±2.3) after blast exposure. The results from the completed assessments varied, including the MACE (n = 685), PCL-M (n = 475), and time since blast (n = 612). Bivariate analyses were done with the χ² test, the t test or non-parametric statistics. Multiple regression analysis was used to predict PCS severity from age, time since blast, number of combat deployments, number of blast-related TBIs, PTSD severity, and presence of recent mTBI. Multiple imputation with 100 imputations was done to accommodate missing data, and the analysis was conducted using SAS version 9.3 PROC MI and MIANALYZE from SAS/STAT[6].

The 567 (83%) participants with an mTBI were more likely to be males (p = 0.04) and Marines (p < 0.0001) than the 118 (17%) participants with no TBI. At least one prior blast-related TBI was reported by 13%. Personnel diagnosed with mTBI were more likely to report a prior blast-related TBI (14 vs. 5%; χ² = 5.8, d.f. = 1, p = 0.016), more likely to meet the criteria for PTSD without the minimum 1-month duration (25 vs. 11%; χ² = 10.8, d.f. = 1, p = 0.001), and more likely to meet the criteria for PCS using a diagnostic threshold of at least three PCS based on the ICD-10 criteria [6] (62 vs. 31%; χ² = 37.1, d.f. = 1, p = 0.001). In the multiple regression analysis, PCS severity was predicted by a diagnosis of mTBI, longer duration since blast, and PTSD severity (table 1).

This is the first study to show an effect of stress reactions on PCS in blast-exposed combat troops in the acute combat setting over and above a diagnosis of mTBI. The strong contribution of stress reactions to PCS may occur because (a) heightened arousal after combat may exacerbate sensations that are reported as PCS, (b) posttraumatic stress is characterized by attentional focus on threat, including somatic symptoms, which may lead to increased awareness of PCS, and/or (c) patients with posttraumatic stress may display cognitive biases leading to attributing sensations to medical conditions such as PCS [7]. The sensations reported as PCS are very common in the population, and there is evidence that they can occur comparably in TBI and non-TBI populations [8].

A longer duration since blast may have been associated with PCS because more urgent medical issues were the primary focus at the time of the initial injury. PCS were still predicted by the diagnosis of mTBI even after controlling for PTSD symptom severity, suggesting that the blast-related mTBI has an acute impact on these sensations. This has some support from evidence of mild neurological damage underpinning PCS [9]. Despite uncertainty about the actual aetiology, it does appear that both mTBI and PTSD symptom severity are factors that need to be considered in understanding PCS in the acute aftermath of a blast injury. We note that these conclusions are limited by the lack of a control group not exposed to blasts, and so we cannot disentangle the et-
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Effects of blast and other combat experiences. The absence of longitudinal data limits our ability to map the course of posttraumatic stress and PCS reactions, which is pivotal because both reactions generally abate over time, and it is critical to understand how one may influence the other.

These limitations notwithstanding, this study has implications for how military personnel are managed in the acute aftermath of a blast injury. Management of PCS needs to recognize the role of acute posttraumatic stress responses in the immediate aftermath of a blast injury. As most acute stress reactions abate in the following months, it is reasonable to encourage expectancy of recovery and resolution of PCS. Attributing the array of somatic sensations experienced after battle to mTBI may create the perception that the individual has suffered a disabling and permanent brain injury. Historians have documented the pattern of attributing symptoms similar to postconcussive syndrome to various causes over the years, ranging from ‘mustard gas hysteria’ in World War I to Gulf War syndrome in the 1991 Gulf War [10]. The current finding supports the proposal that PCS are impacted, at least in part, by psychological factors and should not be regarded solely as the result of neurological insult on the battlefield.

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Disclosure Statement
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References

Table 1. Summary of the regression model predicting PCS

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<thead>
<tr>
<th></th>
<th>Estimate ± SE</th>
<th>t</th>
<th>p</th>
</tr>
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<tbody>
<tr>
<td>Age (n = 683)</td>
<td>0.010 ± 0.012</td>
<td>0.81</td>
<td>0.420</td>
</tr>
<tr>
<td>Time since blast (n = 676)</td>
<td>0.020 ± 0.004</td>
<td>5.14</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Combat deployments (n = 597)</td>
<td>−0.001 ± 0.061</td>
<td>−0.02</td>
<td>0.981</td>
</tr>
<tr>
<td>Prior blast-related mTBIs (n = 570)</td>
<td>0.193 ± 0.168</td>
<td>1.15</td>
<td>0.251</td>
</tr>
<tr>
<td>PTSD symptom severity (n = 475)</td>
<td>0.054 ± 0.006</td>
<td>9.13</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Recent mTBI (n = 685)</td>
<td>1.250 ± 0.211</td>
<td>5.92</td>
<td>&lt;0.0001</td>
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Estimate = Unstandardized B-coefficients. The results are based on multiple imputation with 100 imputations (n = 685).