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Traumatic Brain Injury

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In addition to being associated with psychological stress, war-zone participation increases risk of physical injury, including traumatic brain injury (TBI). TBI has long been a concern to military healthcare providers, as reflected in such terms as *shell shock* in World War I (Jones, Fear, & Wessely, 2007). More recently, TBI has emerged as a particular concern for those serving in Operation Enduring Freedom (OEF) and Operation Iraqi Freedom (OIF), given the nature of the warfare, which includes exposure to blasts associated with improvised explosive devices (IEDs). Mild TBI (mTBI) and posttraumatic stress disorder (PTSD) are often comorbid (Hoge et al., 2008; Tanielian & Jaycox, 2008) in war veterans, sharing common risk factors (e.g., combat intensity), symptoms (e.g., irritability, sleep disturbance, mild cognitive impairment), and, in some cases, underlying neural substrates (e.g., prefrontal and hippocampal involvement; Stein & McAllister, 2009). Yet mTBI remains poorly understood in terms of how it may complicate psychiatric outcomes, including PTSD and its treatment.

Reflecting the theme of this volume, this chapter addresses TBI from the perspective of PTSD, limiting discussion to milder cases of TBI, which are thought to be the most frequent among returning veterans (Hoge et al., 2008). Subsequent sections address the scope and impact of mTBI in returning veterans, review treatment approaches for mTBI, and discuss potential implications for the treatment of PTSD when associated with mTBI.

mTBI in Military Personnel and Veterans

Definition and Mechanisms of TBI

TBI refers to a physiological disruption of brain function caused by a traumatic mechanical (e.g., direct blow to the head) or biomechanical (e.g., blast injury) force. TBI diagnosis is historical and based on symptoms at the time of injury, such as alteration in mental status (e.g., confusion, amnesia, or loss of consciousness) or focal neurological signs. Most mTBI definitions specify that loss of consciousness cannot exceed 30 minutes and that posttraumatic amnesia (i.e., impaired encoding of new information after the injury) cannot exceed 24 hours. The biomechanics of TBI resulting from direct impact on the brain, as may occur in motor vehicle accidents, point to acceleration-deceleration of the head as the primary cause of injury. However, the biomechanics of blast-induced TBI, which are thought to be the most common source of mTBI among OEF/OIF veterans (Owens et al., 2008), are not as well understood. Animal studies suggest that blast injury is possibly due to direct pressure changes as the blast wave passes through the head and/or there is transfer of kinetic energy to the central nervous system through blood vessels in the chest and abdomen (Cernak & Noble-Haesslein, 2010). In addition to primary blast effects, brain damage (similar to that seen in nonblast TBI) can result from penetrating injuries due to shrapnel and debris and from direct impact of the head.

Epidemiology of TBI in OIF/OEF Veterans

Because milder deployment-related TBIs may have been undocumented at the time of injury, the prevalence of TBI in nonclinical samples has been difficult to ascertain; estimates range from 12% (Schneiderman, Braver, & Kang, 2008) to 23% (Terrio et al., 2009) in U.S. samples. Differences in prevalence rates across studies may also reflect sample-specific deployment exposures that are not necessarily representative of the deployed forces as a whole, especially as war-zone conditions evolve over time. In a large and possibly more representative sample, the prevalence of TBI was estimated at 19% (Tanielian & Jaycox, 2008).

The comorbidity of TBI and mental health disorders, including PTSD, is likewise high among OEF/OIF veterans. Tanielian and Jaycox (2008) estimated that one-third of combatants with TBI also suffered PTSD or depression. Hoge et al. (2008) similarly found that 44% of service members who reported TBI with loss of consciousness met screening criteria for PTSD, compared with 27% of those who suffered TBI with altered consciousness and 16% of those with non-TBI injuries. Whereas high comorbidity would not be unexpected given common risk factors such

as combat intensity, the degree to which reports of TBI are influenced by psychiatric symptoms remains controversial. The results of Hoge et al. (2008), which found that the association between TBI and PTSD remained even after controlling for combat experiences, can alternately be interpreted as mTBI creating additional risk of PTSD or PTSD influencing the reports of mTBI.

Pathophysiological Sequelae

Mild TBI can be associated with persistent changes in brain structure and function. Diffusion tensor imaging studies measure the functional integrity of white matter and have demonstrated abnormalities in long white matter tracts that can be linked to aspects of cognitive dysfunction in individuals with persistent symptoms following mTBI (e.g., Niogi et al., 2008). Alterations in brain metabolism during task performance (McAllister, Flashman, McDonald, & Saykin, 2006) have also been observed in mTBI. A growing body of evidence in animals suggests that primary blast injury can affect the brain (Cernak & Noble-Haesslein, 2010), but data on the pathophysiology of blast-induced TBI in humans are limited.

Clinical Sequelae

Natural Recovery from mTBI

In civilian samples, the sequelae of mTBI are transient in an overwhelming majority of patients (Carroll et al., 2004). The acute symptoms include physical (e.g., headaches, dizziness, nausea), cognitive (e.g., difficulty concentrating, slowed thinking, memory dysfunction), and emotional (e.g., irritability, anxiety, depression) symptoms. Symptoms typically improve within days or weeks to full recovery, but in a minority of patients they persist at 3 months postinjury. Similar symptoms are also common among individuals with other medical or psychological (e.g., PTSD, depression) conditions. Although postconcussion symptoms are associated with the acute, transient neurological effects of mTBI, it is now generally accepted that psychological, social, and motivational factors may be associated with the maintenance of at least a subset of these symptoms (McCrae, 2008).

Within the first week of injury, measurable impairments in cognitive function are evident on tests that emphasize speed of processing, verbal fluency, and delayed memory (McCrae, 2008). Performance typically returns to baseline by 1–3 months postinjury (Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005). Despite this generally favorable recovery, patients may continue to show mildly reduced performance on measures of complex attention and working memory years after injury (Vanderploeg, Curtiss, &

Belanger, 2005). Clinically significant neuropsychological impairment may also persist in a small subset of individuals (Pertab, James, & Bigler, 2009). Of potential relevance to war-zone deployments during which service members may experience multiple TBI events, a history of multiple concussions is associated with poorer long-term outcomes in executive functioning and memory (Belanger, Spiegel, & Vanderploeg, 2010).

Comparison with Cognitive and Behavioral PTSD Sequelae

The neurocognitive sequelae of mTBI and PTSD overlap significantly, but the course of recovery differs notably in the two disorders. In contrast to the typically transient symptoms associated with mTBI, PTSD-related emotional and neuropsychological deficits frequently endure years after trauma exposure and may reflect pretrauma vulnerabilities in some cases. As in mTBI, PTSD is associated with relative weaknesses in attention, executive functioning, and memory (Vasterling, Verfaellie, & Sullivan, 2009). Aside from overlap in cognitive symptoms, somatic and emotional symptoms associated with TBI, such as sensitivity to noise, sleep problems, irritability, anxiety, and depression, are also commonly associated with PTSD (Benge, Pastorek, & Thornton, 2009). Moreover, comorbid psychiatric difficulties moderate postconcussion symptoms. In a study of Vietnam-era veterans, the effects of mTBI and PTSD on postconcussion symptoms were additive, but the effects of PTSD were consistently larger than those of mTBI (Vanderploeg, Belanger, & Curtiss, 2009).

Findings in OEF/OIF Military Personnel

The limited extant evidence suggests that the specific mechanism of injury (blast vs. nonblast) does not notably affect neurobehavioral symptoms (Belanger, Kretzmer, Yoash-Gantz, Pickett, & Tupler, 2009) or neuropsychological performance (Belanger, Kretzmer, Vanderploeg, & French, 2009). Several studies have focused on isolating the contribution of mTBI and PTSD to chronic neuropsychological sequelae, but results have been inconsistent, with some studies showing no impact of mTBI (Ivins, Kane, & Schwab, 2009) or PTSD (Brenner et al., 2010; Levin et al., 2010) and others showing poorer performance associated with TBI (Levin et al., 2010) or PTSD (Nelson, Yoash-Gantz, Pickett, & Campbell, 2009). With regard to neurobehavioral symptoms, Hoge et al. (2008) found that all observed variance in physical health and postconcussion symptoms (with the exception of headache) could be accounted for by PTSD and depression. In the same vein, Schneiderman et al. (2008) noted that the strongest factor associated with postconcussion symptoms was PTSD, even when overlapping symptoms between TBI and PTSD were parceled out.

Implications for Assessment

As indicated before, the nonspecific nature of postconcussion symptoms lessens their diagnostic utility. More broadly, however, the failure to observe consistent links between neuropsychological performance and mTBI and PTSD in OEF/OIF veterans challenges the usefulness of attributing complex neuropsychological sequelae to one of two potential diagnoses that share not only significant core features but also underlying neural substrates, including the hippocampus and prefrontal regions (Stein & McAllister, 2009). Rather than attempt to ascribe specific and definitive etiologies to symptoms that may be determined by multiple conditions, a more promising approach may be to focus on better characterization of cognitive, behavioral, and functional problems that manifest postdeployment, regardless of underlying etiology.

Impact of TBI on the Development and Persistence of PTSD

There is now strong evidence that PTSD can develop in the context of mTBI, even following a single incident in which the individual lost consciousness and had no opportunity to encode the event into memory (Harvey, Kopelman, & Brewin, 2005). In the context of military combat, war-zone stress exposures are rarely limited to a single discrete event but more commonly involve a series of repeated or ongoing life-threatening events. Thus, if a specific traumatic event is not remembered, it is still likely embedded in a larger context of psychological trauma. Even when conscious retrieval for a single incident fails completely, implicit encoding of certain aspects of the TBI event, and memory for associated events occurring before or after the TBI event itself, may be responsible for the development of symptoms (Harvey et al., 2005).

Psychologically traumatic events that occur in the context of physical trauma to the brain are more likely to result in PTSD than those not involving brain injury (Bryant et al., 2009; Hoge et al., 2008), although this association is typically limited to the mild end of the TBI spectrum. PTSD can be associated with moderate or severe TBI, but its likelihood is reduced with increased injury severity (Glaesser, Neuner, Lütgehetmann, Schmidt, & Elbert, 2004).

The mechanisms by which mTBI increases the risk of PTSD are likely multifaceted, involving not only the broader context of multiple disabilities and associated psychosocial stressors but also the integrity of neurocognitive processes both during and after the injury (Vasterling et al., 2009). For example, if altered consciousness at the time of the injury leads to a poorly integrated memory of the event or persistent executive dysfunction

interferes with memory reconstruction, retrieval of the trauma memory may be difficult to regulate. Such poorly controlled recall of trauma memories may be particularly problematic if intrusive memories lead new contextual elements to be incorporated into the memory, thus creating additional triggers for emotional distress. Brain dysfunction at the time of the injury may also alter affective processing of the trauma event. Diminished cognitive resources can interfere with engagement in adaptive appraisal of the trauma event and active coping strategies that help reduce stress. Reduced cognitive efficiency can also indirectly affect how individuals cope with PTSD through their adverse effects on psychosocial and occupational functioning, leading to additional stress.

Treatment Approaches for mTBI

Treatment interventions for mTBI differ qualitatively from those used with more severe brain injuries and have primarily focused on early education to prevent the development of persistent postconcussion syndrome. However, the smaller group of patients experiencing persisting symptoms may require additional treatment approaches, particularly when there are comorbid conditions that have an impact on recovery and when early intervention is not possible (Howe, 2009). In these cases, a comprehensive neuropsychological assessment can provide critical information about cognitive, emotional, and other noninjury-related factors that may be compromising functioning and can assist in guiding treatment for these patients.

Interventions

A number of evidence-based psychoeducational interventions for mTBI have been developed (Helmick et al., 2010). The Department of Veterans Affairs (DVA)/Department of Defense (DoD) *Clinical Practice Guidelines* (Department of Veterans Affairs, 2009) recommend early education of patients and families with an emphasis on expectation of full recovery within weeks to a few months; symptom management (e.g., headache, pain, anxiety/depression); and a gradual return to normal daily activities as soon as possible postinjury to minimize the risk of developing persisting symptoms. In deployment contexts, early intervention would ideally occur in theater during the acute or subacute period of recovery by providing clear information that accurately describes the expected recovery after mTBI. For veterans and military personnel who have returned from deployment but who experience barriers to in-person treatment, effective psychoeducation and symptom management may also be provided by telephone. Bell et al. (2008)

found that patients who received four to five telephone counseling sessions directed at education and symptom management during the first 3 months after mTBI reported fewer symptoms and reduced impact of symptoms on everyday functioning at 6 months after injury relative to a control group.

There are also potential functional benefits of cognitive rehabilitation, which refers to a range of interventions that retrain prior skills and/or teach compensatory strategies that exploit cognitive strengths to compensate for relative cognitive weaknesses. Cognitive rehabilitation may be domain (e.g., attention) specific or may target multiple types of cognitive and functional impairments simultaneously. However, much of the evidence base for cognitive rehabilitation concerns moderate to severe TBI or nontraumatic brain injury (Rohling, Faust, Beverly, & Demakis, 2009).

Examples of cognitive rehabilitation interventions include attention process training (APT; Sohlberg & Mateer, 1987), an intervention that presents increasingly challenging tasks as patients master each level of difficulty, and goal management training (GMT; Levine et al., 2000). GMT teaches strategies to improve the ability to plan, organize, and achieve goals by following a series of structured steps, including orienting to the current context, selecting appropriate goals, breaking tasks into simpler subtasks, learning and remembering the goals and subgoals, and assessing the usefulness of selected strategies and solutions. Sohlberg, McLaughlin, Pavese, Heidrich, and Posner (2001) found significant improvement in complex attention in postacute patients with mild to severe brain injuries following APT. Likewise, GMT has demonstrated effectiveness for TBI patients with executive dysfunction (Kennedy et al., 2008).

The "burden of adversity" hypothesis (Brenner, Vanderploeg, & Terrio, 2009) suggests that mTBI is just one of a complex constellation of problems affecting recovery of returning veterans and that treating symptoms regardless of etiology reduces the cumulative effects of many contributing problems. Consistent with this perspective, manualized rehabilitation interventions targeting multiple cognitive and functional domains have been developed and/or adapted for use with OEF/OIF veterans who sustained mTBI during deployment and who experience cognitive and functional impairments months or years postinjury due to a variety of possible etiologies. For example, cognitive symptom management and rehabilitation therapy (CogSMART), a 12-week intervention adapted for OEF/OIF veterans from a treatment program initially developed for patients with schizophrenia (Twamley, Savla, Zurhellen, Heaton, & Jeste, 2008), includes psychoeducation, symptom management, and compensatory cognitive components. Preliminary data suggest that, among veterans who were enrolled in supportive employment and had sustained mild to moderate TBI an average of 4 years earlier, those who participated in CogSMART were more likely to find jobs

and report fewer postconcussive symptoms than those receiving supportive employment only (Thomas, Williams, Bondi, Dellis, & Twamley, 2010).

Healthcare Delivery Models

Patients with mTBI often have multiple physical and mental health concerns, referred to as "polytrauma" by the VA. Providers reported coordinating the care of patients with polytrauma to be among their most daunting challenges (Sayer et al., 2009), and many questions remain about how to best structure care for these patients. For example, are returning veterans with histories of mTBI and PTSD best treated in specialty clinics (e.g., mental health, neurology), multidisciplinary rehabilitation settings, or primary care? Are treatments best integrated, delivered sequentially, or delivered concurrently? Prototypes of two possible approaches (biopsychosocial and step-care) to healthcare delivery for mTBI follow.

The biopsychosocial approach takes into account the influence of somatic, psychological, social, and motivational factors contributing to persisting symptoms and complaints following mTBI (McCrea et al., 2009). In the military population, chronic pain, sleep disturbance, PTSD, depression, substance abuse, and other psychological disorders all may contribute to the maintenance of symptoms. McCrea (2008) implemented an early multidisciplinary treatment model for civilian TBI that relies on liaison between the emergency department and a team of specialists (neuropsychologist, psychiatrist, nurse coordinator) in a TBI clinic during the acute postinjury phase (1–5 days). Based on neuropsychological and medical evaluations, patients are given immediate feedback and recommendations that are often largely psychoeducational, with a follow-up plan if needed. However, most providers caring for OEF/OIF veterans will not see these patients until long after their injuries, when early prevention is no longer an option. Like McCrea's model, the VA polytrauma system of care includes settings that are typically multidisciplinary and consider both physical and psychological issues. Unfortunately, little outcome data are yet available on either model.

Terrio and colleagues (cited in Brenner et al., 2009) proposed a "step-care" treatment model that first provides education and expectation of recovery while simultaneously treating psychiatric symptoms and subsequently addresses somatic complaints and self-care routines. Because cognitive complaints can often be related to poor sleep, chronic pain, and psychological distress, treatment of cognitive symptoms is postponed until the former steps are adequately taken. Adopted in some active-duty military healthcare settings, the step-care approach may decrease unnecessary utilization of medical services. It is also purported to potentially reduce the impact of secondary gain and overuse of disability benefits, which could be framed as a roadblock to recovery during an earlier stage of treatment.

Summary of mTBI Treatment Approaches

Although evidence supports the utility of psychoeducational strategies and cognitive rehabilitation strategies for TBI, less is known about the application of psychoeducation in postacute phases of recovery or of cognitive rehabilitation for milder TBI (Snell, Surgenor, Hay-Smith, & Siegert, 2009). Evidence for the efficacy of post-acute treatment strategies is particularly relevant to military veterans, who may not be diagnosed or treated until months or years after their deployment, when symptoms may have been significantly exacerbated or maintained by a host of other conditions and psychosocial stressors. As important, as veterans return with multiple psychological and physical injuries, questions remain regarding the best models for healthcare delivery and coordination of care.

Treatment of PTSD in Patients with a History of mTBI

We know little about how mTBI may affect PTSD interventions, but the extent to which mTBI affects PTSD treatment outcomes will bear on the clinical management of comorbid PTSD and mTBI. Two core questions pertain to PTSD interventions in patients with history of mTBI and/or persistent cognitive compromise: (1) Are evidenced-based PTSD interventions contraindicated? (2) Is PTSD treatment response attenuated? Unfortunately, empirical evidence addressing these questions is scant. At the writing of this chapter, a VA–DoD Consensus Conference determined only that there was no evidence that evidence-based PTSD interventions would be contraindicated by mTBI (Department of Veterans Affairs PTSD/Mild TBI Consensus Panel, 2009). We focus here on psychosocial interventions for PTSD.

Potential Neurocognitive Mechanisms Affecting PTSD Treatment Response

In considering potential neurocognitive mechanisms affecting PTSD treatment response, we take into account three factors: (1) the nature of the cognitive deficit (e.g., memory, attention, executive); (2) the timeline of the cognitive deficit (i.e., resolved vs. enduring); and (3) the intervention component (e.g., exposure, cognitive reappraisal).

Nonspecific Effects of Neurocognitive Deficits

Neurocognitive compromise may hinder the treatment process in ways non-specific to particular treatment components. For example, forgetfulness, distractibility, slowed information processing, and difficulties with planning

and organization could arguably hamper more general aspects of treatment, such as reliable attendance, follow-through with homework, keeping pace with group discussions, and taking medications as prescribed. Interference of this type implies that deficits are current and, as reviewed earlier, likely affect only a small subset of patients with mTBI.

Deficits Potentially Interfering with Core Elements of Specific Interventions

Cognitive-behavioral therapies (CBTs) with cognitive reappraisal and/or exposure-based components are considered to be among the most empirically supported of any PTSD treatments. These interventions, however, also depend on the successful engagement of cognitive resources that may be impaired following mTBI. At its core, cognitive therapy requires consideration of alternate appraisals of negative or distorted thoughts attached to trauma experiences, with the goal of generating more realistic explanations and thoughts. Such modifications presumably require both sufficiently intact inhibition (of maladaptive thoughts) and cognitive flexibility (to reappraise thoughts and memories) for their success. Exposure-based treatments require controlled retrieval of the trauma memory and the subsequent modification of the memory and associated emotions. Much of the cognitive demand required by cognitive reappraisal, manipulation of trauma memories, and the formation of new associations is dependent on cognitive status at the time of the PTSD intervention and therefore compromised in only a subset of patients. However, even if neurocognitive deficits are resolved at the time of the intervention, neurocognitive impairment at the time of the trauma event potentially influences the encoding of the trauma event, associated affective processing, and the degree to which trauma-related memories and affect can be retrieved in an accessible, controlled manner during therapy.

Effectiveness of Psychosocial PTSD Interventions for Patients with Comorbid mTBI

Virtually no evidence exists addressing the effectiveness of PTSD interventions in patients with history of mTBI, although two small randomized studies of CBT to treat emotional symptoms in patients with mTBI provide some information. In evaluating CBT for the treatment of acute stress disorder following mTBI, Bryant, Moulds, Guthrie, and Nixon (2003) found that CBT reduced the development of PTSD immediately posttreatment and 6 months later. Although CBT was applied to patients with mTBI in the context of acute stress symptoms, it is plausible that it would also successfully reduce more enduring PTSD symptoms in patients with mTBI. In a sample

of 20 mild to moderate TBI patients, Tiersky et al. (2005) found that CBT combined with neurorehabilitation reduced anxiety and depression symptoms compared with a wait-list control. Although far from definitive, these studies suggest that CBT can effectively improve emotional outcomes in the context of mTBI.

There is likewise no direct evidence addressing whether PTSD treatment response is attenuated or slowed in patients with mTBI. However, several studies examining normal variation in brain integrity in small non-brain-injured samples suggest that PTSD treatment response may be influenced by neural integrity and/or baseline neurocognitive functioning. Specifically, attenuated CBT response was associated with less proficient pretreatment verbal memory and narrative encoding (Wild & Gur, 2008), smaller rostral anterior cingulate cortex volumes (Bryant, Felmingham, Whitford, et al., 2008), and increased bilateral activation of the amygdala and ventral anterior cingulate (Bryant, Felmingham, Kemp, et al., 2008).

Summary of PTSD Treatment Implications

There is no evidence that current evidence-based PTSD interventions would be contraindicated in patients with history of mTBI, even in cases with persistent mild neurocognitive deficits. Instead, preliminary evidence from randomized trials using small samples suggest that CBT interventions targeting acute stress disorder and other emotional symptoms (anxiety and depression) were successful in preventing PTSD and/or reducing symptoms. As Soo and Tate (2007) suggest, the structure provided by CBT interventions may in itself benefit patients with cognitive deficits. The question remains whether PTSD treatment response is attenuated in mTBI patients, particularly when the mTBI was associated with transient or enduring neurocognitive compromise. If treatment response is attenuated, it may be that PTSD interventions will benefit from augmented strategies (e.g., cognitive rehabilitation) to address neurocognitive deficits or that PTSD interventions will need to be tailored to minimize the impact of the deficits (e.g., additional rehearsal of key treatment concepts, provision of written instructions, removal of distractions in the treatment setting). Until there is more evidence, we recommend using evidence-based PTSD interventions in patients with history of mTBI but with a watchful eye toward monitoring treatment response.

Conclusion

The nature of contemporary warfare has led to increased attention to mTBI among returning veterans. Although we know much about TBI at more severe levels, the factors that determine mTBI outcomes in a military con-

text and the best treatment approaches for mTBI are not as well understood. The introduction of explosive blasts as an etiology has contributed to the many unknowns regarding combat-related mTBI. Moreover, the often prolonged and repetitive psychological stress exposures inherent in combat and consequent development of PTSD and other mental disorders complicate the picture. These unique combat-related factors limit to some degree inferences from the more extensive civilian mTBI literature. Not surprisingly, such ambiguities have fueled controversy surrounding healthcare delivery policies for returning veterans with history of mTBI, particularly in the context of comorbid PTSD. Yet, because of the high rates of mTBI among returning veterans with PTSD, providers caring for OEF/OIF veterans with PTSD regularly confront clinical presentations that include mTBI.

Although there is still much to be learned about mTBI and PTSD, we are able to draw on several basic observations to guide the care of veterans with PTSD and mTBI. First, most people with history of mTBI function relatively well. Psychoeducation, including a clear statement of expectations regarding recovery, currently ranks as the most effective tool in preventing adverse mTBI outcomes. On the other hand, the course of recovery from mTBI is not uniform across individuals, and subtle problems may surface, particularly in contexts of stress (e.g., war-zone participation) or high demand (e.g., transitioning back to civilian life). These seemingly contradictory findings call for a delicate balance of trying not to convey undue alarm or pessimism while nonetheless taking seriously difficulties that veterans may experience. Second, because we know that certain factors may complicate recovery from TBI (e.g., substance abuse, additional TBI), an important component of treatment will be preventing such complications from arising and addressing them, if they do. Finally, because there is no evidence to suggest that current evidence-based PTSD interventions are contraindicated in patients with mTBI, it is critical to continue to use our very best tools to treat PTSD as we await further evidence that addresses whether augmentation or modifications to current treatments may optimize outcomes among patients with mTBI.

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