MASTER

CHAPTER 5

Effects of Traumatic Brain Injury–Associated Neurocognitive Alterations on Posttraumatic Stress Disorder

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The high comorbidity of mild traumatic brain injury (mTBI) and posttraumatic stress disorder (PTSD) in those serving in Operation Enduring Freedom (OEF) and Operation Iraqi Freedom (OIF) (Hoge et al., 2008; Tanielian & Jaycox, 2008) has focused renewed attention on the nature of the relationship between the two disorders. In light of the high overlap in symptoms between mTBI and PTSD and their possible neural substrates (Vasterling, Verfaellie, & Sullivan, 2009), much of the current interest has focused on diagnostic questions, such as whether symptoms can best be understood as the result of physical or psychological trauma (Hoge, Goldberg, & Castro, 2009). Much less explored is the question as to how biomechanical injury to the brain may impact adjustment to psychological trauma.

This chapter examines the neurocognitive mechanisms by which mTBI may affect the development and course of PTSD. We make use of what is known about how sources of normal neurocognitive variance influence PTSD, on the assumption that the sequelae of mTBI represent another source of neurocognitive variance. To elucidate potential mechanisms of action, we draw on a cognitive model of PTSD (Ehlers & Clark, 2000) that

postulates that PTSD is maintained by (1) an autobiographical memory disturbance, whereby memory cues trigger the traumatic memory in such a way that it is experienced as reliving of the trauma; and (2) excessive negative appraisals that lead to exaggerated estimates of harm and negative outcome. We consider possible neurocognitive mechanisms in light of how mTBI may (1) alter the creation and correction of the traumatic memory and (2) influence the appraisal of the event and its consequences.

Although the physical trauma that leads to TBI can be temporally distinct from the life-threatening trauma that results in PTSD (e.g., a veteran who sustains mTBI after deployment), more commonly the physical trauma occurs as part of the same discrete event (e.g., a motor vehicle accident in a civilian setting) or an ongoing series of events (e.g., blast explosion in the context of ongoing combat operations) that are perceived as life-threatening. Thus, mTBI may have the greatest potential to affect the expression of PTSD symptoms in the immediate aftermath of the injury, when cognitive alterations are most pronounced. Nonetheless, among at least a subset of mTBI victims, chronic neurocognitive abnormalities may also influence subsequent PTSD symptom expression. Regarding treatment, what little preliminary evidence we have suggests that evidence-based interventions for acute stress-related symptoms following psychological trauma can be applied successfully to patients with mTBI (Bryant, Moulds, Guthrie, & Nixon, 2003). However, as suggested by Bryant and Litz (Chapter 11, this volume), many questions remain regarding how the wide range of variance in mTBI-related cognitive deficits may moderate responses to various evidence-based PTSD interventions.

In keeping with the theme of this volume, we limit our discussion to mTBI. We first review the empirical evidence that suggests that mTBI may affect the development of PTSD. To set the stage for a consideration of cognitive mechanisms that may be responsible for the association between mTBI and PTSD, we next review neurocognitive alterations commonly observed in mTBI. Subsequent sections address ways in which these neurocognitive factors may influence the development and course of PTSD, and may have implications for treatment. Our considerations are restricted to the possible impact of mTBI in adulthood, as it is unknown how disruption of ongoing neurodevelopment in children or adolescents who sustain mTBI influences subsequent maturation (for discussion, see Yeates, 2010). Finally, although we acknowledge that other comorbidities, such as alcohol use or pain, may modulate the effects of mTBI on PTSD, consideration of these factors is beyond the scope of this chapter (see Najavits, Highley, Dolan, & Fee, Chapter 7, this volume; and Otis, Fortier, & Keane, Chapter 6, this volume, for discussion of comorbid substance abuse and pain, respectively).

The Impact of mTBI on PTSD

Systematic reviews of the literature suggest that mTBI increases risk of psychiatric disorders (Jorge, 2005; Kim et al., 2007). Further, compared to traumatic injury not associated with TBI, mTBI confers additional risk for common trauma-related psychopathologies such as PTSD and depression. For instance, studying Cambodian survivors of mass violence, Molica, Henderson, and Tor (2002) found that psychologically traumatic events involving brain injury were more strongly associated with depression and PTSD than psychologically traumatic events not involving brain injury. In a prospective study of traumatically injured patients admitted to the hospital for at least 24 hours, Bryant et al. (2010) reported that patients were at higher risk for subsequent development of PTSD and several other anxiety disorders if they had sustained mTBI. Similar findings hold in military veterans exposed to war-zone trauma, even when potential differences in combat exposure between veterans with and without TBI are taken into account. For instance, Chemtob and colleagues (1998) reported that patients who sustained TBI had higher levels of PTSD symptoms than their non-TBI counterparts, and Vasterling, Constans, and Hanna-Pladdy (2000) reported an increased severity of depression associated with TBI. Similar findings have been reported in OEF/OIF veterans (Hoge et al., 2008; Schneiderman, Brawer, & Kang, 2008).

The association between mTBI and PTSD presents somewhat of a paradox, as the alterations in consciousness associated with TBI are thought to interfere with the formation of traumatic memories. According to one perspective (Sbordone & Liter, 1995), amnesia for the traumatic event might be incompatible with several core features of PTSD, including affective responses associated with the event, reexperiencing of trauma memories, and avoidance of trauma reminders. Consistent with this view, poorer memory for a traumatic event after mTBI is protective against select reexperiencing symptoms (Bryant, Creamer, O'Donnell, Silove, Clark, & McFarlane, 2009), and the likelihood of PTSD decreases as the severity of TBI increases (Glaesser, Neuner, Lutgehetmann, Schmidt, & Elbert, 2004). However, in patients with mTBI, amnesia for the event is not always complete, and some aspects of the trauma may be consciously accessible. Further, even when conscious retrieval fails completely, other mechanisms may be responsible for the development of symptoms: (1) affective and sensory–perceptual experiences associated with the trauma event may be encoded at an implicit, unconscious level, and subsequently impact on physiological, affective, and behavioral responses (Layton & Wardi-Zonna, 1995); (2) later reconstruction of memory from secondary sources such as family or observers may lead to the development of PTSD symptoms (Harvey & Bryant, 2001); and (3) the context of the trauma (e.g., sights and sounds

upon regaining consciousness) and peritraumatic events (e.g., medical procedures) can be traumatic in their own right, and therefore lead to PTSD (McMillan, 1996).

In military combat and other contexts characterized by ongoing psychological stress, unique factors may further permit the coexistence of TBI and PTSD. Both war-zone stress exposures and domestic violence, for example, are rarely limited to a single discrete event, but more commonly involve a series of repeated or ongoing threatening events. Thus, even if a specific traumatic event is not remembered, it may be embedded in a larger context of psychological trauma. Moreover, less severe levels of mTBI, such as commonly reported in military personnel, may entail only momentary alteration of consciousness. As such, partial encoding of the trauma event may allow some aspects of the trauma to be accessed and reexperienced at a conscious level.

Recent findings in OEF and OIF veterans have further highlighted the association between mTBI and PTSD. Hoge and colleagues (2008) reported that of those service members who reported sustaining TBI with loss of consciousness, 44% met screening criteria for PTSD as compared to 27% of those who suffered TBI with altered consciousness and 16% of those with other injuries not including TBI. Similar findings have been reported by Schneiderman et al. (2008). On the one hand, given the considerable overlap in symptoms of mTBI and PTSD, it has been argued that the observed association may reflect inflated reporting of mTBI on the basis of psychiatric symptoms. On the other hand, such findings give added weight to the notion that mTBI may serve as a risk factor for PTSD. Consistent with the latter view, a recent analysis of data from a large sample of Vietnam-era veterans enrolled in the Vietnam Experience Study indicated that remote history of TBI increased the risk of current PTSD, on average 16 years after deployment (Vanderploeg, Belanger, & Curtiss, 2009).

Aside from its impact on the prevalence of PTSD, mTBI may also be associated with qualitative differences in the development of specific stress symptoms and the course of adjustment. Focusing specifically on acute stress disorder, Broomhall and colleagues (2009) found several differences in the nature of acute stress symptoms reported by patients with and without mTBI. Although some of these symptom differences were likely due to overlap in symptoms between mTBI and acute stress disorder, of note was a higher level of behavioral avoidance in patients with mTBI, a symptom that is not in itself associated with mTBI. In another study (Bryant & Harvey, 1999), patients with mTBI reported less fear and fewer intrusive memories than patients without TBI acutely posttrauma, but this difference was not apparent 6 months later. The lack of group differences at 6 months was attributable to the rate of intrusions increasing over the first 6 months in those with mTBI, but decreasing in those without mTBI.

There is inconclusive evidence as to whether posttraumatic stress symptoms in the context of mTBI may be more persistent. Harvey and Bryant (2000) followed individuals with mTBI due to motor vehicle accidents and found that of those who had acute stress disorder, 82% had a diagnosis of PTSD 6 months postinjury, and 80% still suffered from PTSD at 2-year follow-up. This outcome is comparable to that of survivors of motor vehicle accidents without TBI (Harvey & Bryant, 1999a). However, these findings are specific to acute stress disorder. Many individuals develop acute posttraumatic stress symptoms without meeting diagnostic criteria for acute stress disorder. Prospective studies of civilian trauma survivors without TBI show that more than half of individuals who show stress-related symptoms in the initial weeks after trauma exposure remit within 3 months (Blanchard et al., 1996; Riggs, Rothbaum, & Foa, 1995). It is unknown whether this trajectory is different for patients with mTBI.

Neurocognitive Alterations in mTBI

Insight into the neurocognitive effects of mTBI comes primarily from studies in a civilian context, including motor vehicle accidents and sports-related injury. As summarized by Bigler and Maxwell (Chapter 2, this volume), in civilian samples, the sequelae of mTBI are mostly transient and rapidly or gradually resolve in an overwhelming majority of patients (Caroll et al., 2004; Iverson, 2005), but in a minority of patients cognitive deficits and symptoms persist at 3 months postinjury (Pertab, James, & Bigler, 2009; Ponsford et al., 2000). In sports-related concussion, cognitive impairments typically resolve within 7–10 days (Belanger & Vanderploeg, 2005; McCrae et al., 2003), whereas in other clinical samples deficits on neuropsychological measures can persist for several months (Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005). The temporal variability in the resolution of neurocognitive deficits may reflect individual differences in rate of recovery (McCrea et al., 2005), history of previous head injury (Belanger & Vanderploeg, 2005), and/or psychosocial factors including litigation (Belanger et al., 2005).

In the acute phase of mTBI, impairments in memory and executive function are most common. With regard to memory, deficient encoding of new information and delayed recall of this material frequently occur (Belanger et al., 2005; Belanger & Vanderploeg, 2005). Executive functions encompass a range of cognitive processes that are important for controlling or guiding behavior in a top–down manner. Specific executive control deficits that can follow mTBI include impaired ability to inhibit contextually inappropriate responses (McCrae et al., 2003), sustain and shift attention (Belanger et al., 2005; Belanger & Vanderploeg, 2005), manipulate

information in working memory (Stuss, Stetham, & Poirier, 1989), and organize information into meaningful units (Bruce & Echemendia, 2003).

Despite a generally favorable recovery, considerable heterogeneity in outcome is observed across both neuropsychological measures and patients. Although not in the range of clinical impairment, patients with mTBI may continue to show reduced performance on measures of working memory and enhanced sensitivity to proactive interference years after injury (Vanderploeg, Curtiss, & Belanger, 2005). These residual deficits may reflect in part underlying deficits in speed of processing and a reduction in the amount of information that can be processed accurately (Crawford, Knight, & Alsop, 2007; Van Zomeren, Brouwer, & Deelman, 1984)—impairments that become apparent under conditions of high cognitive load or other sources of stress. Furthermore, symptoms of depression and anxiety disorders can follow a TBI (Bryant, O'Donnell, et al., 2010) and, as discussed by Iverson (Chapter 3, this volume), may exacerbate these cognitive impairments.

With respect to subject heterogeneity, Pertab et al. (2009) concluded from a reanalysis of meta-analytic studies that residual mild neuropsychological impairment may occur more reliably than previously acknowledged in a small subset of individuals. In this regard, it is of note that a history of multiple concussions is associated with worse long-term neuropsychological functioning, particularly in the domains of executive control (e.g., inhibition, set shifting, verbal fluency) and memory (Belanger, Spiegel, & Vanderploeg, 2010). Further, even in individuals who perform within normal limits on standard neuropsychological tests, deficits on more taxing executive control tasks may remain (Ellemberg, Leclerc, Couture, & Daigle, 2007; Pontifex, O'Connor, Broglio, & Hillman, 2009). For instance, in patients with chronic mTBI, residual impairments in inhibitory control have been documented on an experimental task in which responding to a central stimulus required inhibition of flanking stimuli (Pontifex et al., 2009). Impairments were observed even in the absence of deficient performance on standard clinical neuropsychological tests, and these became more pronounced with repetitive mTBI.

The constellation of cognitive deficits that may accompany mTBI suggests a disruption of functions mediated by the frontal and medial temporal regions of the brain. Evidence for such disruption comes from diffusion tensor imaging studies that measure the integrity of white matter tracts, which conduct neural impulses between brain regions. Several studies have demonstrated abnormalities in long white matter tracts that connect anterior and posterior regions of the brain in patients evaluated in the semiacute phase (Mayer et al., 2010; Messé et al., 2010), as well as more chronically in individuals with persistent symptoms (e.g., Huang et al., 2009; Lo, Shifteh, Gold, Bello, & Lipton, 2009). Such measures have also been linked

to aspects of cognitive dysfunction (e.g., Kraus et al., 2007; Mayer et al., 2010). Studies using functional magnetic resonance imaging (fMRI) demonstrate that mTBI patients with normal structural imaging show alterations in neural activation relative to a control group. For instance, bilateral frontal and parietal lobe hypoactivation has been found on challenging tasks of working memory and spatial attention (Mayer et al., 2009; McAllister, Flashman, McDonald, & Saykin, 2006).

As shown by Bigler and Maxwell (Chapter 2, this volume), neuroimaging studies suggest that despite normal findings using the types of conventional MRI or computerized tomography (CT) commonly used in clinical settings, mTBI can alter brain microstructure and function in frontal and limbic regions. Given the significance of the limbic system to emotion, these regions are unsurprisingly also key in neural models of PTSD that postulate inadequate frontal lobe inhibition of the limbic system, and in particular a structure known as the amygdala, which is implicated in fear processing and learning (Rauch, Shin, & Phelps, 2006). In support of these models, a body of neuroimaging findings suggests that PTSD involves (1) exaggerated responsivity of the amygdala and (2) reduced activity in the frontal cortex (especially its medial, ventromedial, and orbital sections) and hippocampus (see Hayes & Gilbertson, Chapter 4, this volume). If mTBI affects the very same brain regions that account in part for the clinical presentation of PTSD, it stands to reason that mTBI may exacerbate symptoms associated with PTSD.

Cognitive Domains That May Influence the Development, Course, and Treatment of PTSD

Possibly the earliest evidence that sources of neurocognitive variance affect posttrauma psychological adjustment comes from studies examining the link between intellectual functioning and PTSD. Pitman, Orr, Lowenhagen, Macklin, and Altman (1991) reported that a lower score on the Arithmetic Reasoning subtest of the Armed Forces Qualification Test at enlistment predicted subsequent chronic PTSD, but their study remained inconclusive because it did not control for extent of combat exposure. Subsequent studies that adjusted for extent of stress exposure, however, have confirmed these findings (Macklin et al., 1998; Vasterling et al., 2002), although one study demonstrated that intellectual resources may be influential only at relatively lower levels of stress with less impact at higher stress levels (Thompson & Gottesman, 2008).

Regarding response to treatment, Rizvi, Vogt, and Resick (2009) found that lower general intelligence and education were associated with higher treatment dropout in a sample of 145 women being treated with a

cognitive-behavioral therapy (CBT) for PTSD from sexual assault. Notably, however, general intelligence and education were not significantly associated with level-of-symptom reduction among treatment completers. These findings raise the possibility that cognitive deficits in patients with mTBI could potentially influence early treatment response (leading some patients to opt out early), but would not necessarily alter the eventual outcome of treatment for patients who became engaged for the full span of treatment.

Intellectual functioning is not a unitary construct, and as such, there are a number of possible mechanisms that may explain the observed association between intelligence and PTSD. Intellectual resources may broadly impact the ability to mobilize coping resources and problem-solving skills important for posttrauma adjustment, but may also have an impact through more specific neurocognitive processes that are potentially affected by mTBI independently of intellectual functioning. Possible candidates include (1) autobiographical memory; (2) executive control; and (3) verbal skills. We elaborate on each of these cognitive factors in turn, with special consideration of how they may be affected by mTBI.

Autobiographical Memory

Most cognitive theories of PTSD hold that autobiographical memory disturbance is a central element of the disorder, with enhanced involuntary recall of the trauma as well as decreased voluntary access to trauma memories (Brewin, 2007). Both in acute stress disorder (Harvey & Bryant, 1999b) and PTSD (Amir, Stafford, Freshman, & Foa, 1998), trauma memories have been described as being fragmented, disorganized, and lacking internal coherence. Such fragmentation is thought to result from disorganized initial encoding of the traumatic event, which leads to inconsistent consolidation and poorly regulated retrieval. Further, the failure to properly contextualize a memory by placing it in its appropriate spatial and temporal context may be responsible for the occurrence of involuntary intrusive memories, consisting of vivid, detailed images accompanied by a sense of reliving in the present. Evidence suggests that these characteristics of intrusions strongly predict PTSD severity (Michael, Ehlers, Halligan, & Clark, 2005) and that resolution of PTSD symptoms is associated with reduced frequency of intrusions (Hackmann, Ehlers, Speckens, & Clark, 2004) and establishment of a cohesive organization of the trauma memory (Foa, Molnar, & Cashman, 1995).

mTBI may exacerbate the development of PTSD by weakening the encoding and integration of a traumatic event into a temporally and conceptually organized autobiographical memory (Conway & Pleydell-Pearce, 2000). Disruption of consciousness during the mTBI is likely to interfere with the formation of a coherent memory of the trauma and altered mental

status may have a blunting effect on memory abilities (Stuss et al., 1999). Even after consciousness returns to normal, however, difficulties with strategic learning and organization in the immediate aftermath of mTBI may continue to disrupt encoding of trauma-related information received from medical personnel or witnesses of the event. Thus, although degraded encoding of the trauma may be associated with protection against development of select reexperiencing symptoms (Bryant et al., 2009), reduced control over retrieval resulting from fragmented encoding could arguably be detrimental to recovery from psychological trauma.

Extending this line of reasoning, it is possible that accessibility to, and the coherence of, the trauma narrative would likewise be relevant to those PTSD interventions dependent on retrieval of the trauma memory. Evidence-based CBTs for PTSD typically include exposure-based components, which require retrieval of trauma memories so that new affective associations may be formed. Thus, even if an mTBI is fully resolved at the time of the intervention, it is plausible that acute neurocognitive impairment at the time of the trauma could influence the encoding of the trauma event, how associated emotions are processed, and therefore the degree to which trauma-related memories and affect can be retrieved in a controlled, verbally accessible manner during therapy (for fuller discussion of treating PTSD in the context of mTBI, see Bryant & Litz, Chapter 11, this volume).

In addition to the coherence of the trauma memory, more general aspects of autobiographical memory (extending to nontrauma memories) may also contribute to PTSD outcomes following trauma exposure. Specifically, trauma survivors with acute stress disorder (Harvey, Bryant, & Dang, 1998) or PTSD (McNally, Lasko, Macklin, & Pitman, 1995) tend to provide fewer specific details on autobiographical memory tasks, instead producing overgeneral memories, particularly in response to positive cues. Longitudinal studies have clarified that this tendency to produce overgeneral memories is a risk factor for the development of PTSD. For instance, Bryant, Sutherland, and Guthrie (2007) reported that a failure to retrieve specific memories in response to positive cues prior to trauma exposure in a group of firefighter trainees predicted symptom severity after trauma exposure.

To our knowledge, no studies have examined autobiographical memory in TBI samples limited to mTBI, although two studies have addressed autobiographical memory in samples with more severe TBI or in mixed severity groups. In the first study, more severe TBI was associated with significant difficulty in the retrieval of specific memories (Williams, Williams, & Ghadiali, 1998). Furthermore, the extent of overgeneral retrieval was related to impaired memory function on an immediate story recall task (Williams et al., 1998), suggesting a link between disruption of general

memory formation and disturbance of autobiographical memory following TBI. In the second study, Bessel, Watkins, and Williams (2008) found that overgeneral memory was linked to depression in TBI. In that study, patients with mild and moderate–severe TBI showed reduced specificity of autobiographic memory retrieval following induction of depression-like ruminative self-focus. Moreover, baseline depression severity was inversely correlated with autobiographical memory retrieval. Thus, both general memory inefficiency and negative mood are associated with reduced ability to retrieve specific autobiographical memories in mTBI. One mechanism that may mediate these associations is reduced executive resources (Williams, 2006). Specifically, limited executive capacity may lead to a truncated search through the hierarchy of memory representations, resulting in the retrieval of memories at a general level of description rather than memories corresponding to specific events.

Executive Control

In addition to its role in guiding memory search, executive control may affect the development and course of PTSD through its impact on the control of memory retrieval, regulation of affective processing, and cognitive appraisal of the trauma and its sequelae. We discuss each of these possible mechanisms in turn.

Studies in normal cognition point to inefficient cognitive control as a vulnerability factor for reexperiencing symptoms. Specifically, the ability to inhibit interference from irrelevant emotionally neutral information in working memory has been linked to the presence of spontaneous intrusive memories (Verwoerd, Wessel, & de Jong, 2009) and experimentally induced undesirable intrusive memories (Wessel, Overwijk, Verwoerd, & de Vrieze, 2008). Likewise, in patients with PTSD, Vasterling, Brailey, Constans, and Sutker (1998) found that disinhibition and intrusion errors on emotionally neutral neuropsychological tasks were positively correlated with reexperiencing symptoms, suggesting that a more general deficit gating information contributes to difficulties with the control of emotional information. If extrapolated to mTBI, it is possible that TBI-related impairments in working memory and inhibitory control lead to difficulty suppressing intrusive information, thereby contributing to the development and maintenance of reexperiencing symptoms. Moreover, a reduction in executive resources may lead not only to an inability to control intrusive thoughts, but may additionally enhance precisely those thoughts that are intended to be controlled (Wegner, 1994).

The same executive impairments that potentially render mTBI patients more vulnerable to uncontrolled, intrusive memories may also compromise

the controlled, voluntary retrieval of the trauma memory. This is so because controlled retrieval requires specification of appropriate retrieval cues and inhibition of inappropriate ones, as well as monitoring of the suitability of retrieved memories, all of which depend on the integrity of executive function. As discussed above, difficulty retrieving the trauma memory in a controlled manner may impede natural recovery from PTSD and hamper interventions with exposure components.

Executive control plays a similarly important role in affective regulation. Several studies have concluded that PTSD reflects an inability of dorsolateral prefrontal cortex to inhibit a hyperresponsive emotional system mediated by the limbic system (e.g., Bremner et al., 1999; Shin et al., 2005). Extending a framework on the normal interaction between cognition and emotion, Morey, Petty, Cooper, LaBar, and McCarthy (2008) examined how PTSD symptoms relate to the reciprocal relationship between executive functioning and processing of emotional information. They found that in a sample of OEF/OIF veterans recently returned from deployment, severity of PTSD symptoms was positively associated with activation in ventral frontal-limbic regions during processing of combat-related pictures and negatively associated with activation in dorsal-frontal regions during executive processing. Because these findings are correlational, they do not indicate whether alterations in brain function were caused by PTSD or were a precipitating factor. However, it stands to reason that reductions in executive control, as seen in mTBI, might exacerbate any imbalance between affective and executive processing in PTSD.

Aside from its effect on emotional experience, executive impairment may also impact the modulation of emotional expression. TBI-related difficulty monitoring and inhibiting behavioral expressions of anger, frustration, and other negative emotions may result in behaviors that are contextually inappropriate in non-life-threatening situations. It is possible that dysregulated emotional responses, such as irritability, anger, and aggression (Alderman, 2003; Johansson, Jamora, Ruff, & Pack, 2009), decrease feelings of control and lead to a negative self-perception. The emotional consequences of poor affect regulation, such as anger, shame, and guilt, have also been found to predict PTSD status (Andrews, Brewin, Rose, & Kirk, 2000; Marx et al., 2010). Emotional lability similarly may have deleterious effects on interpersonal relationships, reducing access to sources of social and economic support, thus further hindering recovery from psychological trauma. Dysfunctional emotional regulation could also interfere with relationships within a treatment context. For example, poor emotional control could potentially interfere with group treatment interventions, in which the focus of the group is vulnerable to inappropriate disruptions posed by individual group members.

Finally, executive dysfunction possibly adversely affects cognitive appraisal of the trauma and its consequences, a factor known to affect the development and maintenance of PTSD (Ehlers & Clark, 2000). More specifically, reduced cognitive flexibility in mTBI, due to compromised attentional switching and inhibitory processes, may interfere with the ability to reappraise stimuli in the environment as nonthreatening. This also has implications for the therapeutic context. Because cognitive therapy requires consideration of alternate appraisals of negative or distorted thoughts, with the goal of generating more realistic and constructive explanations (see Bryant & Litz, Chapter 11, this volume), cognitive flexibility may be particularly important for PTSD interventions with a cognitive component. Presumably, successful reappraisal would require both intact inhibition (of maladaptive thoughts) and cognitive flexibility (to reappraise thoughts), which may be particularly challenging for the subset of patients with mTBI who show lingering cognitive deficits. On the other hand, CBT offers a structured therapeutic context, which could arguably benefit patients with mild executive deficits. The success of CBT with patients with mTBI and acute stress disorder (Bryant et al., 2003) supports this perspective and argues for continued use of cognitive therapy with patients with mTBI and PTSD. The question remains, however, whether PTSD interventions with cognitive components would benefit from strategies that enhance the potential for efficient and adaptive reappraisal processes.

Verbal Skills

As described above, patients with mTBI may exhibit impairments in memory and executive functioning that affect both verbal and nonverbal information processing. Impairments in verbal processing may be particularly relevant to the development and course of PTSD. The association between impaired verbal memory and PTSD is well established (Brewin, Kleiner, Vasterling, & Field, 2006), and memory impairment appears to affect in particular the initial acquisition of verbal information into memory. Both prospective (Bustamante, Mellman, David, & Fins, 2001) and twin (Gilbertson et al., 2006) studies suggest that impaired verbal function, and especially verbal learning and memory, may be a risk factor for PTSD. Further, Bustamante and colleagues (2001) found that verbal learning and recall as well as verbal fluency measured in the first few weeks after trauma predicted the development of PTSD 6 weeks later.

There are a number of ways in which TBI-related deficits in verbal processing, including the ability to flexibly and effectively encode and manipulate verbal information, may impact on the development and maintenance

of PTSD. Brewin (2005) has emphasized the relationship between verbal processing and the ability to suppress intrusive thoughts. In normal individuals, low verbal working memory has been associated with more intrusions of arbitrary (Brewin & Beaton, 2002) as well as personally relevant obsessive thoughts (Brewin & Smart, 2005). Further, in participants who watched a traumatic film, limiting spontaneous verbal processing by means of a concurrent verbal task enhanced the likelihood of intrusions, a finding that was interpreted as suggesting that intrusive memories are normally suppressed by verbal processes (Holmes, Brewin, & Hennessy, 2004). We highlighted above how deficiencies in cognitive control associated with mTBI may be a vulnerability factor for reexperiencing symptoms. Although not limited to the verbal domain, mTBI-related working memory impairments provide a possible mechanism by which top-down control of intrusive memories may be compromised.

The extent to which the trauma memory itself can be verbally accessed and processed also plays an important role in the course of PTSD. Brewin, Gregory, Lipton, and Burgess (2010) postulated that reexperiencing symptoms in PTSD occur when low-level sensory representations of the trauma are insufficiently integrated with corresponding contextual representations that allow an event to be placed in its appropriate spatial–temporal context. Sensory representations are triggered involuntarily by reinstatement of situational and affective cues, and it is by virtue of their association with contextual representations that they can be intentionally controlled and experienced as a memory from the past rather than relived in the present. Although contextual representations are not inherently verbal, they provide the basis for narrative memories, in that they allow information to be deliberately retrieved and manipulated and integrated into a person's individual history and knowledge base. Assimilation of trauma memories and their associated emotions into an ongoing life narrative may be critical for symptom resolution (Conway, 2005).

Contextual representations depend on functioning of the medial temporal lobe memory system, which may be compromised under conditions of extreme stress (Metcalfe & Jacobs, 1998). Because the hippocampus is uniquely vulnerable to the effects of brain injury (Lowenstein, Thomas, Smith, & McIntosh, 1992), even mTBI (and even more so repeated insult [Slemmer, Matser, De Zeeuw, & Weber, 2002]) may further exacerbate hippocampal dysfunction. Thus, mTBI may further interfere with the ability to establish contextual memory representations, leaving trauma memories largely dependent on sensory representations that are not verbally accessible. As discussed earlier in regard to autobiographical memory more generally, access to the trauma memory is key to successful implementation of exposure-based PTSD interventions. Given that these interventions

rely specifically on the ability to form a verbal trauma narrative, verbal processing (both at the time of the trauma and subsequently during treatment) may be particularly important in the treatment context.

Conclusions

Although mTBI is associated with good short-term recovery, both acute neurocognitive deficits and subtle postacute deficits in a subgroup of patients potentially influence the development and course of PTSD. This possibility is borne out by empirical findings indicating that mTBI is associated with enhanced risk for PTSD. Little is currently known about the ways in which mTBI may affect how individuals cope with psychological trauma. It is apparent that a range of factors will need to be taken into account, including not only the neurocognitive sequelae of mTBI, but also its emotional and psychosocial consequences. Similarly, conditions that complicate recovery from mTBI, such as repeated head injury or alcohol abuse, will require consideration. In this chapter, we focused on the neurocognitive impairments associated with mTBI that may mediate vulnerability to PTSD, including impairments in autobiographical memory, executive function, and verbal processing. Given the lack of studies examining the effects of mTBI-related cognitive impairments upon the expression of PTSD, we drew on evidence regarding cognitive factors that have been postulated to mediate the variability in recovery from psychological trauma in the general population. Although theoretical considerations suggest multiple avenues by which mTBI-related neurocognitive impairment may influence the development, course, and recovery of PTSD, future empirical studies are clearly needed to evaluate these possibilities.

Treatment of course also potentially affects the course of PTSD, but limited data address whether PTSD treatments are as effective for patients with mTBI, and in particular for those with enduring deficits. What little evidence we have suggests that CBT is likely an effective treatment option for patients with comorbid mTBI and PTSD. Of less certainty is whether patients with mTBI would be more likely to terminate treatment prematurely, whether treatment response would be attenuated, and whether certain aspects of various CBT components could be enhanced via modifications that take into account cognitive deficits, or even via integrated treatments that address PTSD and mTBI sequelae concurrently, as described by Bryant and Litz (Chapter 11, this volume). Answers to these questions await carefully designed longitudinal studies and clinical trials that take into account the variability in outcome of mTBI and possible factors that may affect PTSD recovery.

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