Post-traumatic stress disorder vs traumatic brain injury

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Post-traumatic stress disorder (PTSD) and traumatic brain injury (TBI) often coexist because brain injuries are often sustained in traumatic experiences. This review outlines the significant overlap between PTSD and TBI by commencing with a critical outline of the overlapping symptoms and problems of differential diagnosis. The impact of TBI on PTSD is then described, with increasing evidence suggesting that mild TBI can increase risk for PTSD. Several explanations are offered for this enhanced risk. Recent evidence suggests that impairment secondary to mild TBI is largely attributable to stress reactions after TBI, which challenges the long-held belief that postconcussive symptoms are a function of neurological insult. This recent evidence is pointing to new directions for treatment of postconcussive symptoms that acknowledge that treating stress factors following TBI may be the optimal means to manage the effects of many TBIs.

Dialogues Clin Neurosci. 2011;13:251-262.

Keywords: *post-traumatic stress disorder; traumatic brain injury; trauma; postconcussive syndrome*

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Overview

The intersection between traumatic brain injury (TBI) and post-traumatic stress disorder (PTSD) has become a major focus of attention in recent years. Stimulated largely by injuries sustained in the Iraq and Afghanistan wars, this issue has been debated widely because these conditions, both independently and additively, are regarded as being responsible for much impairment following deployments. This review will commence with defining these conditions, explain potential overlaps between them, and discuss the differential diagnosis challenges of determining the extent to which presenting symptoms can be attributed to organic or psychological factors. The review then discusses evidence of PTSD following TBI, and possible mechanisms that may impact on the nature of PTSD following TBI. The respective roles of PTSD and TBI in impairment after TBI are then addressed, with specific focus on the understanding of postconcussive symptoms. Finally, the implications for managing the effects of TBI and PTSD are discussed in terms of recent developments in how each condition can affect the other.

Definitional issues

TBI

TBI involves damage to the brain from an external force. Brain injuries can involve contusion, brain laceration, intracranial hematoma, contrecoup injury, shearing of nerve fibers, intracranial hypertension, hypoxia, anemia, metabolic anomalies, hydrocephalus, and subarachnoid

hemorrhage. Severity of TBI is typically described in terms of mild or moderate/severe; however, the exact definitions vary. Mild traumatic brain injury (MTBI) is usually defined as: (i) an external injury to the brain; (ii) confusion, disorientation, or loss of consciousness for 30 minutes or less; (iii) Glasgow Coma Scale score of 13 to 15; and (iv) post-traumatic amnesia for less than 24 hours.[1-3] Moderate TBI often involves loss of consciousness between 30 minutes and 24 hours, Glasgow Coma Scale score of 9 to 12, and post-traumatic amnesia between 1 and 7 days. Severe TBI involves more extended loss of consciousness and post-traumatic amnesia, which typically results in more severe cognitive impairment. These differences in TBI severity are important because they appear to interact differentially with PTSD.

PTSD

It is important to distinguish between immediate and longer-term PTSD reactions. Most diagnostic systems have distinguished between these two types of trauma response because acute stress reactions are frequent, but often transient, and they need to be distinguished from the less common persistent PTSD responses. In terms of the persistent responses, PTSD is described in the American Psychiatric Association's *DSM-IV* as an anxiety disorder that comprises five major criteria.[4] First, one must have been exposed to or witness an event that is threatening to safety, and one must respond to this event with fear, horror, or helplessness. Second, one must report a re-experiencing symptom, which may include intrusive memories, nightmares, a sense of reliving the trauma, or psychological or physiological distress when reminded of the trauma. Third, there need to be at least three avoidance symptoms, which can include active avoidance of thoughts, feelings, or reminders of the trauma, inability to recall some aspect of the trauma, withdrawal from others, or emotional numbing. Fourth, one must suffer marked arousal, which can include insomnia, irritability, difficulty concentrating, hypervigilence, or heightened startle response. These symptoms must cause marked impairment to one's functioning, and can only be diagnosed when they are present at least 1 month after the trauma.

DSM-IV also introduced a new diagnosis, acute stress disorder (ASD), to describe acute trauma reactions that occur in the initial month following a trauma. As PTSD is only diagnosed 1 month after trauma, it was decided that there was a need to fill the nosological gap between the traumatic event and PTSD, in part to facilitate diagnosis and access to health care. A second major goal of the ASD diagnosis was to describe acute stress responses that precede longer-term PTSD, and therefore could be used to identify people who were at high risk for subsequent disorder and could benefit from early intervention. ASD is conceptually similar to PTSD and shares many of the same symptoms.[5] A key difference between ASD and PTSD is the former's emphasis on dissociative symptoms. Specifically, ASD requires the individual to experience at least three of the following: emotional numbing, reduced awareness of one's surroundings, derealization, depersonalization, and dissociative amnesia. These symptoms may occur either at the time of the trauma or during the subsequent month. The dissociative symptoms were included in ASD on the premise that dissociative responses following trauma are predictive of subsequent PTSD, presumably because they limit emotional processing of the traumatic experience.[5] Support for the inclusion of dissociative symptoms in the ASD diagnosis to predict subsequent PTSD came from evidence demonstrating an association between peritraumatic dissociation and subsequent levels of PTSD, a finding that has been replicated across several longitudinal studies.[6-10] Across many longitudinal studies, the ASD diagnosis has been shown to be a flawed predictor of subsequent PTSD.[11] Nonetheless, ASD is being retained in *DSM-5* as a descriptor of acute stress reactions.[12]

Differential diagnosis

A key issue in this discussion is the overlap between symptoms accompanying each condition. In terms of the dissociative symptoms often observed in PTSD, and especially in the acute phase in ASD, there is much evidence that TBI can result in emotional numbing, derealization, reduced awareness of surroundings, depersonalization, and amnesia.[13-15] The issue of amnesia is particularly important in cases of TBI and PTSD because of the difficulty in differentiating between organic and psychogenic amnesia.[16] Some commentators have adopted the approach of excluding dissociative amnesia as a possible symptom of ASD and PTSD following TBI to reduce the likelihood of falsely increasing

diagnostic rates.[17,18] In diagnosing PTSD, it is probably safer to not include dissociative amnesia as a potential symptom.

Relevant to the interplay with TBI is the proposed revision of PTSD in the upcoming revision of *DSM-5*, which suggests several changes to the PTSD criteria.[19] The subjective response to the trauma at the time of the event (Criterion A2) is to be deleted because it does not enhance accuracy of identifying people with PTSD. This is important for patients with TBI because many patients, especially those with more severe TBI, do not initially respond with a sense of fear or helplessness because of their impaired consciousness. Avoidance is being redefined to only include active avoidance of thoughts and situations, in recognition of the fact that numerous factor analytic studies have identified four factors of PTSD: re-experiencing, active avoidance, numbing/passive avoidance, and arousal.[20-24] Most of these studies have found that emotional numbing and social withdrawal are distinct from more active avoidance strategies. This is relevant because numbing and withdrawal can often be observed in more severe TBI; by separating these passive responses into a separate requisite cluster, it raises the possibility of differential diagnosis problems for more severe TBI patients, many of whom will display these symptoms. This cluster also includes alterations in mood and cognition, and comprises a range of symptoms that may include a range of emotional responses beyond fear and anxiety.[25] This may also be problematic in terms of differential diagnosis because of the frequent depressive and generalized anxiety seen in more severe TBI patients. Although the arousal cluster is retained, there is the expansion of several symptoms, including aggressive behavior and self-destructive/reckless behavior. These latter symptoms can be observed in the context of reduced inhibition in more severe TBI patients, thereby raising further differential diagnosis problems in distinguishing between symptoms of severe TBI and PTSD.

In contrast to ASD, the International Classification of Diseases[26] conceptualizes acute stress reaction as a transient reaction that can be evident immediately after the traumatic event and usually resolves within 2 to 3 days after trauma exposure. The ICD description of acute stress reaction includes dissociative (daze, stupor, amnesia), anxiety (tachycardia, sweating, flushing), anger, or depressive reactions, which may have more utility for clinicians than the more focused ASD criteria.[27] This position presumes that the initial period after trauma exposure may result in a rather general state of distress that can include many emotional responses that cannot be readily classified into different responses.[28] This construct has particular relevance for the acute phase of TBI, especially more severe TBI, when many of the symptoms described as acute stress reactions may be a function of impaired consciousness.

A further complicating issue in the differential diagnosis between PTSD and TBI is the range of other comorbid problems that commonly coexist with both TBI and PTSD. For example, depression is highly prevalent with both conditions. Numerous studies have suggested that TBI increases the risk for developing depression,[29,30] eg, refs 31,32,33. Some of the core symptoms noted across TBI and PTSD are also seen in depression, especially the more severe forms of TBI, including concentration problems, memory problems, irritability, reduced motivation, and fatigue. Highlighting this problem in one study was a finding that more than 50% of depressed patients met symptom criteria for moderate/severe postconcussive syndrome.[34] This contributes to the conclusion that some of the symptoms attributed to TBI may in fact be generic symptoms of psychological malaise, which are observed across anxiety and depressive responses. Complicating the issue of comorbidity is compounded by the fact that TBI, PTSD, and depression commonly occur in the context of chronic pain, which also results in symptoms that overlap with each of these conditions.[35-41]

Prevalence

PTSD and TBI are not uncommon. Epidemiological studies indicate that most people in the community have been exposed to traumatic stressors,[42,43] although anly a minority develop PTSD. For example, the National Comorbidity Survey found that 21% of the women and 8% of the men had developed PTSD.[42] Similarly, a Detroit study found that 13% of the women and 6% of the men had developed PTSD.[43] That is, although men are more likely to be exposed to trauma than women, women have at least a twofold risk of developing PTSD compared with men.[44] More severe traumas tend to result in more severe PTSD. Interpersonal violence leads to more PTSD than impersonal trauma; for example, whereas 55% of rape victims develop PTSD, only 7.5% of accident victims develop PTSD.[42,45]

In terms of TBI, there are between 1.5 and 2 million people in the USA alone who sustain a TBI, with approxi-

mately 70 000 to 90 000 experiencing persistent functional difficulties.[46] The Centers for Disease Control and Prevention estimates that approximately 5.3 million people in the USA are living with a disability due to TBI.[47] Certain populations appear to be more at risk of sustaining TBIs. For example, military estimates of mild TBI of deployed (non-medically evacuated) personnel indicate that between 10% and 20% may have suffered a mild TBI during deployment.[48] One study reported a rate as high as 23% in personnel assessed after returning to the USA.[49]

Can PTSD develop following TBI?

Some earlier commentators argued that PTSD could not develop following TBI because the impaired consciousness at the time of trauma precluded encoding of the traumatic experience, and this prevented trauma memories that are necessary for PTSD development.[50,51] In contrast, evidence has accumulated that PTSD can develop following mild TBI.[52-60] Intriguingly, both case studies[61-66] and cohort studies[17] have noted the existence of PTSD developing following severe TBI. In many of the latter cases, these individuals suffer very significant periods of retrograde and anterograde amnesia, such that they do not recall any episodes of the traumatic experience.

Fear conditioning

Several mechanisms have been put forward to explain how PTSD can develop following TBI. Fear conditioning models posit that the fear elicited during a traumatic event results in conditioning in which subsequent reminders of the trauma elicit anxiety in response to trauma reminders (conditioned stimuli).[67] This model proposes that extreme sympathetic arousal at the time of a traumatic event may result in the release of stress neurochemicals (including norepinephrine and epinephrine), mediating an overconsolidation of trauma memories. This proposal is consistent with animal studies that indicate that epinephrine administration after an aversive experience enhances fear conditioning.[68] Fear conditioning models are also supported by considerable evidence that people with chronic PTSD are hyperresponsive to trauma reminders.[69-71] The adrenergic increase occurring after trauma exposure that may contribute to fear conditioning may be reflected in increased sympathetic nervous system activation, including resting heart rate. Indirect support for this hypothesis comes from multiple longitudinal studies that indicate that elevated heart rate in the acute post-trauma phase is associated with subsequent development of PTSD[72]; elevated heart rate in the initial days after trauma may reflect stronger conditioning, which can then translate into longer-term PTSD. Although conditioning occurs optimally when one is aware of the contingency between the unconditioned and conditioned stimuli,[73] conditioning may occur with varying levels of awareness of the contingency between the trauma and the consequences, which may allow for some fear conditioning following TBI. Consistent with this proposal, there is evidence that people can develop PTSD following severe TBI, even though these patients do not recall the trauma and do not suffer intrusive memories of the event.[17] These patients display reactivity to reminders of the trauma in the absence of recall of the event; this observation is consistent with fear conditioning explanations of TBI-related PTSD. Further support for the possibility of fear conditioning leading to PTSD after severe TBI patients is evidence of higher heart rates immediately after the trauma in severe TBI patients who develop PTSD (even during dense post-traumatic amnesia) than those who do not develop PTSD.[74]

Memory reconstruction

An alternate mechanism is that TBI patients reconstruct trauma memories in ways that result in a traumatic representation of what occurred during impaired consciousness. A prospective study of mild TBI patients assessed motor vehicle accident survivors immediately after the accident, and subsequently reassessed for their memory of the event 2 years later; this study found that although all patients initially reported amnesia of some aspect of the accident, 40% reported 2 years later that they had subsequently achieved full recall of the experience.[75] Supporting this view, case reports describe severe TBI patients developing images of the traumatic event based on police reports, dreams, and other secondary sources.[61,65] For example, Bryant[76] reported a man who developed PTSD 12 months after his injury, which involved an extended period of anterograde and retrograde amnesia. When this man was directed to resume driving he developed distressing and intrusive images of his accident that were based on a newspaper photograph of his wrecked

car. Although he was densely amnesic of the accident, he developed a series of images that were founded on his memory of the photograph. Interestingly, these images changed with time. For example, when he became concerned that his children may be harmed when he was driving, his intrusive images changed to include his children lying dead in the car. Bryant and Harvey[62] compared the intrusive imagery of motor vehicle survivors who either (i) had PTSD and no TBI; (ii) had PTSD following severe TBI and reported intrusive memories that were inconsistent with objective reports of the accident; or (iii) had no PTSD. All participants were asked to listen to an audio tape of a car crash sound effect, and were then interviewed about their cognitive and emotional responses. When these responses were independently rated on a range of constructs, it was found that those PTSD participants with and without TBI reported comparable levels of vivid imagery, emotional response, involuntariness, and sense of reality. The only difference was that those with a TBI tended to report stationary images rather than moving sequential imagery. This finding highlights that the reconstructed memories that develop in TBI patients can be subjectively compelling and share may of the attributes of imagery experienced by people who have continuous recall of their trauma.

Postamnesia resolution

A third possible mechanism is that many people who sustain a TBI, and frequently those with MTBI, suffer traumatic experiences following resolution of their post-traumatic amnesia. One may be knocked unconscious in a motor vehicle accident victim but be fully aware of the experience of being cut out of the car by paramedics, experiencing severe pain, being treated in an emergency room, and fearing for their safety. These experiences function similarly to any traumatic scenario observed by people who develop PTSD in the absence of any TBI. Many MTBI patients will report distressing memories of their experience, despite islands of amnesia in which they cannot recall the point of impact in which they sustained their MTBI.

The impact of TBI on PTSD

One of the intriguing findings in recent years is that MTBI appears to *increase* the risk for PTSD. For example, Fann and colleagues reported from a large-scale study of 939 health plan members that patients with a history of mild TBI were 2.8 times more likely to develop a psychiatric disorder than patients with no TBI history.[77] In a large military survey, whereas 16% of troops who sustained a bodily injury indicated PTSD, 44% of those with MTBI screened positive for PTSD.[59] Further, a large civilian study that employed rigorous clinical interviews found that sustaining a MTBI significantly increased the risk for PTSD.[78] This development is in stark contrast to previously held views that TBI was protective of PTSD development.

This observation may have several possible explanations. The prevailing neurobiological model posits that PTSD involves exaggerated amygdala response associated with impaired regulation by the medial prefrontal cortex.[79] The amygdala appears to be pivotal to development and expression of conditioned fear reactions in human and animal studies, and that learning to inhibit these fear reactions (extinction learning) involves inhibition by the ventral medial prefrontal cortex.[80] Consistent with this model, numerous studies have reported that patients with PTSD have diminished medial prefrontal cortex during processing of fear.[81] It is possible that MTBI enhances risk for PTSD because neural damage sustained in the injury compromises the critical neural circuitry required to regulate fear following the traumatic experience.[82]

Alternately, the management of post-traumatic stress, as well as problems caused by ongoing stressors in one's environment, requires adequate working memory and cognitive resources[83]; it is possible that TBI depletes these resources to some extent, and this may contribute to increased PTSD risk. There is much evidence that PTSD is influenced by the compounding effects of stressors that occur following the precipitating trauma.[84,85] Pain, medical procedures, loss of employment, legal issues, and interpersonal conflict are commonplace following MTBI, and it is possible that the marginal deficits that may be attributed to MTBI could limit optimal management of these stressors.

Although MTBI does appear to increase the risk of PTSD, it needs to be remembered that the association between TBI and PTSD is complex, and much is not understood. There is evidence of an inverse relationship between extent of one's memory of the traumatic experience and the occurrence of re-experiencing memories. One study of 228 motor vehicle accident survivors indexed the extent to which patients with MTBI recalled

details of the traumatic accident,[87] and found that the less patients recalled of their traumatic event, the less likely they were to develop PTSD. Another study assessed 1167 traumatic injury patients in hospital (459 with mild TBI and 708 with no TBI) for post-traumatic amnesia and PTSD in hospital immediately, and subsequently reassessed them for PTSD 3 months later.[87] Although this study found an inverse relationship between the length of post-traumatic amnesia and intrusive memories in the initial phase after trauma, MTBI patients were more likely to develop PTSD than no-TBI patients, after controlling for injury severity (adjusted odds ratio: 1.86, 95% confidence interval, 1.78-2.94). This finding suggests that although duration of amnesia appears protective of development of intrusive traumatic memories, MTBI nonetheless confers risk for developing PTSD. It is for this reason that whereas mild TBI appears to increase risk for PTSD, presence of PTSD after more severe TBI (in which there is limited encoding of trauma memories) is less common.

Delayed-onset PTSD

Post-traumatic stress symptoms typically occur in the initial days and weeks after trauma exposure, and then gradually abate in most people; a minority of trauma survivors can suffer persistent PTSD.[88] Delayed-onset PTSD refers to cases of PTSD in which the condition develops at least 6 months after the trauma. Most studies indicate that delayed-onset PTSD is rare. Although uncommon following civilian trauma, it has been reported to occur more frequently in troops returning home from deployment.[89,90] A review of delayed-onset PTSD studies found that it was rare for PTSD to develop outside military samples, with up to one third of military cases presenting as delayed-onset[91]; specifically, it is reported in 38% of military cases compared with 15% in civilian cases.

To date, there has not been any systematic study of delayed-onset PTSD following MTBI. It is possible that sustaining an mild TBI may contribute to delayed-onset PTSD in the military, and this may be one factor in the increased rates of delayed-onset PTSD in the military. It is possible that following MTBI sustained in combat, one feels the need to fill the gap of knowledge of the events that affected them. Consistent with reports of TBI patients confabulating events in order to make sense of what occurred to them during the loss of consciousness,[61] it is possible that one explanation of delayed-onset PTSD, especially in the military, is the pattern of subsequently reconstructing the traumatic events in the wake of impaired consciousness. The possibility that trauma memory reconstruction in the post-deployment period contributes to PTSD needs to be studied in military populations, and points to the potential importance of ensuring that adaptive, rather than maladaptive, reconstructions of events occur in the months after injury.

Impairment following MTBI

There is enormous concern in the wake of the Iraq/Afghanistan wars over the impairment caused by MTBI. Many millions of dollars are being devoted to rehabilitation procedures to minimize the potential adverse effects of MTBI on soldiers affected by it. Recent studies are indicating, however, that MTBI itself is responsible for minimal impairment. One large-scale survey of US troops reported that health impairment was markedly higher in deployed personnel who sustained a MTBI, including reported poor general health, days off work, and medical visits. Importantly, the influence of MTBI on these measures of impairment was not significant after controlling for the effects of PTSD and depression.[59] This conclusion was supported in a second large-scale military study.[92] Similarly, a large-scale study of civilians found that impaired functioning was not increased by the presence of MTBI; however, there were very significant functioning deficits if a patient sustained a psychological disorder in conjunction with the MTBI.[78] This convergent evidence points to physical, social, and occupational impairment being strongly related to psychological factors occurring after trauma exposure, such as PTSD and depression, rather than the presence of MTBI.

Postconcussive syndrome and PTSD

The issue of postconcussive syndrome is a vexed one, both in terms of its definition and its purported causes. It is also an issue that intersects with symptoms of PTSD. PCS is generally defined as a syndrome that involves headache, dizziness, fatigue, sensitivity to light or sound, sleep disturbance, and concentration difficulties.[93] The definitions of PCS vary, and generally overlap somewhat with symptoms of PTSD. For example, the *International Classification of Diseases (ICD-10)*[26] stipulates that PCS

is defined by headaches, dizziness, general malaise, fatigue, noise intolerance, irritability, emotional lability, depression, or anxiety, concentration or memory difficulty, sleep disturbance, reduced tolerance to alcohol, and a preoccupation with these symptoms and fear of permanent brain damage. The Appendix of the *DSM-IV*[4] describes PCS as fatigue, sleep disturbance, headaches, dizziness, irritability, anxiety or depression, changes in personality, and apathy. These descriptions clearly overlap with common symptoms of post-traumatic stress, and represent differential diagnosis problems insofar as how one attributes these symptoms to PCS or PTSD.

Recent evidence is highlighting that symptoms described as PCS are common in many populations, and actually reflect a diffuse collection of frequently experienced sensations. In healthy individuals, headaches, sleep difficulty, irritability, and memory failures are relatively common in daily life.[97-98] One study found that 72% to 79% of healthy adults reported at least three or more PCS symptoms; further, a significant minority of subjects met *DSM-IV* (14.6%) or *ICD-10* (12.5%) criteria for PCS.[99] Interestingly, these observed rates of PCS in non-MTBI are comparable to the rates noted in TBI populations, highlighting the fact that PCS are not unique to TBI.

There has been much debate over the extent to which persistent PCS develops as a result of neurological damage,[100] psychological distress,[101] or a combination of both.[102] One recent study that assessed PCS in both MTBI and non-MTBI injured patients found that comparable proportions of patients reported PCS (MTBI: 40%; no-TBI: 50%).[103] A subsequent follow-up at 3 months post-injury found that a similar pattern (mild TBI: 46.8%; control: 48.3%).[104] Interestingly, across these studies, PCS was predicted by pain levels and PTSD symptoms. These data indicate that PCS is not unique to MTBI, and that these symptoms that are commonly attributed to MTBI are more parsimoniously explained by the effects of high arousal associated with the stress of surviving a traumatic injury.

The problem of confusing MTBI and PTSD

Military agencies have implemented programs for troops in Iraq and Afghanistan targeted towards treating the effects of MTBI. Much attention has been given to the "problem" of mild TBI, communicating to troops that MTBI is a syndrome that causes marked problems. Given the evidence that so-called postconcussion-like symptoms and general health problems are largely related to psychological factors, there are likely risks in suggesting to troops that the problems experienced following MTBI should be attributed to neurological damage. Communicating to personnel who sustained a MTBI that a range of nonspecific symptoms are caused by brain damage communicates a cause with a poor prognosis. This expectation that common sensations are signs of permanent dysfunction can result in hypervigilance to every sensation, followed by catastrophic attributions about the adverse consequences of the sensations. This pattern has been well-documented across a range of disorders, including panic disorder, health anxiety, and hypochondriasis.[105-107] In these disorders, people tend to be hypervigilant to somatic cues because they believe they represent a threat to their physical well-being. For example, the patient with panic disorder may believe that an alteration in his or her respiration is a sign of imminent choking or that a slight pain in the chest is indicative of an approaching cardiac arrest. Similarly, someone with health anxiety may constantly search their body for any alterations in appearance of function to determine if there are signs of malignancy. Once the sensation or sign is detected, the person can catastrophize the sign in an extremely negative manner, such that the slightest somatic cue is perceived as indicative of dire outcomes. This is a common pattern in people with PTSD. Fear network models of PTSD propose that these individuals preferentially allocate attention to stimuli of concern because of their fear of threat.[108] Consistent with this proposal, people with PTSD are hypervigilent to threat on a range of paradigms.[109-111] Further, people with PTSD not only catastrophize about external threats,[112] they also catastrophize about somatic and physical sensations.[113] Therefore, people who are suffering the effects of PTSD will be attentive to any information that is perceived as threatening, and will likely attribute a range of physical, cognitive, and emotional responses to brain injury if this is provided as a salient explanation. This response may exacerbate the PTSD reaction, as well as promote continued hypervigilence to sensations and subsequent maladaptive appraisals that these reactions are indicative of permanent brain injury.

This pattern was reflected in the aftermath of the 1991 Gulf War, when there were widespread concern of chemical weapons, which apparently contributed to medically unexplained symptoms that were linked to

concerns about somatic sensations purportedly linked to chemical agents.[106,114,115] It seems that a cohort of soldiers after the 1991 Gulf War misattributed somatic experiences to chemical agents, which led to persistent concerns about their health. There are potential similarities between Gulf War Syndrome and the manner in which MTBI is currently being understood; both comprise general sensations that are commonly reported in stress responses, and both mistakenly attributed to common stress reactions. This can be problematic because it can reduce people's optimism or expectancy for recovery.

Implications for treatment

This review has several implications for how symptoms following TBI are addressed in treatment. In terms of treating the symptoms of PCS, current evidence suggests that simple neuropsychological education is modestly useful in reducing symptoms of PCS.[116] The emerging evidence that PCS is predominantly influenced by post-traumatic stress reactions suggests that addressing these problems may be crucial in alleviating PCS. That is, by reducing the arousal-inducing symptoms of PTSD, it is possible that many of the symptoms associated with PCS will be alleviated. Similarly, by minimizing catastrophic appraisals that exaggerate the severity or adversity of PCS sensations it is probable that anxiety about these reactions would be eased. For example, patients who are overly concerned about the adverse outcomes of dizziness or sensitivity to light can be taught to normalize these reactions in ways that minimize distress about these sensations. Cognitively reframing the perception of these reactions is akin to established treatments for panic disorder or health anxiety, in which patients are taught to tolerate somatic experiences in ways that discourage inferences involving an adverse outcome. Although this approach has been proven to be very effective in treating panic disorder[117] and health anxiety,[118] it has yet to be tested with PCS.

In terms of treating symptoms of PTSD, prevailing cognitive models posit that recovery from a traumatic experience involves integrating the trauma memory into one's autobiographical memory base in a way that allows a coherent narrative of the experience in which the person can contextualize the experience and consequently currently feel safe.[112] This perspective proposes that a major reason trauma memories are difficult to integrate into memory is the manner in which they are encoded[119]; specifically, experiences are often fragmented because they are encoded under conditions of extreme arousal, and this purportedly disturbs the ability to form the required coherent narrative. Fragmented memories of the traumatic experience can also occur in the context of TBI because of the impaired consciousness secondary to the injury. As noted above, TBI patients can reconstruct aspects of the traumatic experience that were not adequately encoded during the period of impaired consciousness. This scenario raises the possibility that treating PTSD after TBI will require adaptive reconstruction of this narrative in a way that facilitates adaptation rather than retraumatization. For example, a patient who reconstructs their memory of a car accident in which they were excessively responsible for someone's death will have marked depressive responses relative to a patient who reconstructs the memory in a way that accepts a more reasonable level of responsibility. Alternately, a patient can be encouraged to tolerate a level of uncertainty insofar as there is permanent amnesia of some aspect of the event; inability to tolerate uncertainty is linked to enhanced anxiety and worry.[120] One of the challenges for treating PTSD after TBI is the patient's ability to either reconstruct events in a coherent and adaptive way or to accept the uncertainty of how events transpired when they suffered their TBI.

The extent to which a person with TBI needs to reconstruct the trauma narrative to recover from PTSD has yet to be empirically determined. As noted above, several large-scale studies have reported that MTBI is associated with increased risk for PTSD.[59,92,78] One possibility for this observation may be that people who sustain a MTBI do not have a coherent narrative of their traumatic experience because of the impaired consciousness secondary to the brain injury, and this may impede their capacity to contextualize the experience in their autobiographical memory base.

A second implication for PTSD treatment after TBI is that the treatment of choice for PTSD involves trauma-focused exposure therapy.[121] This treatment is based on extinction learning, which occurs when a conditioned stimulus is repeatedly presented in the absence of an aversive outcome, thereby facilitating new learning that the stimulus is no longer signaling threat. In the context of therapy, presenting memories or reminders of the trauma to the patient in the safety of therapy typically leads to symptom reduction. Exposure can either be *imaginal*, which involves focusing on one's memories of

the traumatic event, or *in vivo*, in which approaches and remains with reminders that usually trigger anxiety about the event. On the premise that fear conditioning and extinction still occurs in the context of TBI, it would seem that that exposure-based therapy is the indicated intervention for PTSD following TBI. Supporting this conclusion is evidence in one controlled trial of patients with acute stress disorder following MTBI that CBT effectively treated PTSD symptoms to a similar extent as when applied to non-TBI samples.[122]

Imaginal exposure with people following TBI will usually be dependent on the amount of memory that the patient is reporting. It may not be as useful to patients with more severe TBI because they are largely amnesic of their trauma. As noted above, some severe TBI patients can have nightmares or intrusive memories on the basis of reconstructions of their trauma; in these cases, imaginable exposure to those mental representations that are causing anxiety. In most cases of moderate/severe TBI, however, it is more useful to employ in vivo exposure because reminders of the trauma can elicit stronger anxiety in the absence of actual memories or images. A survivor of a motor vehicle accident who sustained a severe TBI may experience marked fear when watching film footage of traffic; in such a case, the patient could complete exposure by repeatedly watching traffic footage. Through these techniques it would be hoped that extinction learning can be achieved, even though the patient may never retrieve direct memories of the traumatic event.

Conclusions

The coexistence of TBI and PTSD is frequent, and the extent to which the symptoms of TBI and PTSD are confused may be as frequent. Increasing evidence indicates that many previously termed PCS responses are a function of psychological responses, and it hampers a patient's recovery if they mistakenly perceive these reactions as indicators of a brain injury that may be permanent. In this sense, the field is recognizing the distinction between TBI as an event rather than a syndrome, whereas PTSD or PCS are symptoms that arise secondary to the event. The likelihood that the presumed sequelae of MTBI are actually attributed to psychological responses to the traumatic experience is becoming more apparent. Accurate identification of the true nature and cause of the symptoms experienced after TBI is important because if stress-related disturbances are mistakenly attributed to neurological factors, patients may be deprived of effective treatments that can, in most cases, alleviate the symptoms. As we learn more about the interaction of TBI and PTSD, it seems that we will be discovering much about how the brain responds to traumatic experiences, both in cases when there has and has not been a TBI. Understanding this interaction between neurological insult and psychological response has the potential to shed light on the key mechanisms underpinning trauma response generally, and how it is impacted by different levels of brain injury. ❑

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Trastorno por estrés postraumático versus daño cerebral traumático

Con frecuencia el trastorno por estrés postraumático (TEPT) y el daño cerebral traumático (DCT) coexisten, ya que las lesiones cerebrales a menudo son parte de las experiencias traumáticas. Esta revisión esquematiza la sobreposición significativa entre TEPT y DCT comenzando con un resumen crítico de los síntomas que se sobreponen y de los problemas en el diagnóstico diferencial. A continuación se describe el impacto del DCT en el TEPT y se señala que hay una evidencia creciente que sugiere que el DCT leve puede aumentar el riesgo de TEPT. Se ofrecen algunas explicaciones para este incremento del riesgo. Hay evidencia reciente que propone que el deterioro secundario al DCT leve se puede atribuir en forma importante a las reacciones de estrés después del DCT, lo que desafía la antigua creencia de que los síntomas post contusionales se producen a raíz del daño neurológico. Esta evidencia reciente está apuntando hacia nuevas orientaciones para el tratamiento de los síntomas post contusionales, reconociendo así que el manejo de los factores de estrés después de un DCT puede ser la forma óptima de abordar los efectos de muchos DCTs.

L'état de stress post-traumatique versus lésion cérébrale traumatique

L'état de stress post-traumatique (ESPT) et la lésion cérébrale traumatique (LCT) coexistent souvent car la survenue de lésions cérébrales est souvent retardée lors des événements traumatiques. Cet article souligne le chevauchement significatif entre l'ESPT et les LCT en débutant par une description indispensable des symptômes communs et des problèmes liés au diagnostic différentiel. L'influence de la LCT sur l'ESPT est ensuite décrite, de plus en plus d'arguments suggérant qu'une légère LCT augmenterait le risque d'ESPT, ce qui s'explique de différents façons : d'après des résultats récents, le déficit secondaire à une légère LCT est largement attribuable aux réactions de stress après la LCT, ce qui contredit une croyance ancienne selon laquelle les symptômes post-commotionnels sont fonction d'une lésion neurologique. Ceci ouvre de nouvelles voies de traitement des symptômes post-commotionnels, traiter les facteurs de stress après une LCT s'avérant peut-être le meilleur moyen de prendre en charge les effets des LCT.

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