Review Paper

The Relation Between Memory of the Traumatic Event and PTSD: Evidence From Studies of Traumatic Brain Injury

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Objective: This paper focuses on the relation between memory and posttraumatic stress disorder (PTSD). More specifically, it addresses the debate regarding the role of memory of the traumatic event in the development of PTSD. Traumatic brain injury (TBI) is used as a naturally occuring model for traumatic exposure that is often associated with memory impairment.

Method: We present a critical review of the literature on studies assessing the relation between TBI and PTSD, with a focus on memory of the traumatic event as a critical factor. We also discuss results from recent studies conducted by our group.

Results: The literature review offers an inconclusive picture wherein a significant proportion of the studies indicate that PTSD and TBI are mutually exclusive, especially in individuals who exhibit lack of memory for the traumatic event. This finding supports the possibility that lack of memory may protect against the development of PTSD. However, some studies show that PTSD does occur in patients with head injury, suggesting that PTSD may develop in TBI survivors—even in those who cannot remember the traumatic event. Generally speaking, though, the overall balance of the findings (including our own findings) seems to support the possibility that, in subjects with TBI, impaired memory of the traumatic event is associated with reduced prevalence of PTSD.

Conclusions: The suggestion that amnesia regarding the traumatic event may protect against the development of PTSD has both theoretical and practical importance. This review focused on the case of trauamtic brain injury as a model for impaired memory for the traumatic event. However, it still remains to be proven that the conclusions based on these findings are generalizable beyond the case of TBI. While some patients with posttraumatic amnesia do develop PTSD despite lack of memory for the traumatic event, the majority of those who lack memory for the event seem to be protected from developing the disorder. Nevertheless, based on this assumption, we suggest that pharmacologic disruption of newly acquired—or even old—traumatic memories, which has been shown to be possible in animals, might therapeutically benefit trauma survivors.

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Highlights

- Impaired memory for the traumatic events seems to reduce the risk for developing posttraumatic stress disorder (PTSD).
- Pharmacologic disruption of traumatic memories might be therapeutically beneficial in people with PTSD.

Key Words: *posttraumatic stress disorder, traumatic brain injury, memory*

Posttraumatic stress disorder (PTSD) is a pervasive and highly debilitating condition that affects approximately 8% of the general population in the US and 15% to 30% in specific high-risk groups, such as survivors of war, torture, or rape (1,2). It is defined in the DSM-IV-TR (3) as an anxiety disorder comprising 4 major criteria: 1) exposure to or witnessing of an event that is threatening to one's well-being; 2) symptoms of reexperiencing, such as intrusive memories, nightmares, a sense of reliving the trauma, or psychological and physiological distress when reminded of the trauma; 3) avoidance of thoughts, feelings, or reminders of the trauma, and inability to recall parts of the trauma, withdrawal, and emotional numbing; and 4) arousal, as manifested in sleep disturbance, irritability, difficulty concentrating, hypervigilence, or heightened startle response. These symptoms

must cause marked impairment in functioning and persist for at least 1 month posttrauma.

PTSD is unique in having a specific etiology and time of onset subsequent to exposure to a traumatic event. Nevertheless, it is well known that the experience of a traumatic event in itself is not sufficient to evoke PTSD. While most trauma survivors develop a range of PTSD reactions in the initial weeks after a traumatic event, most also adapt effectively within approximately 3 months. This appears to be a critical phase during which, for most individuals, the stress response subsides (4). Those who fail to recover after approximately 3 months are at risk for developing a long-lasting chronic disorder (5). A smaller subpopulation fails to recover, even after many years of psychiatric treatment (2).

It is evident from both the phenomenology and the etiology of PTSD that the abnormal nature of the traumatic memory is a central feature of the disorder manifested by symptoms of re-experiencing, such as intrusive thoughts, nightmares, flashbacks, and physiological or psychological reactivity (6). At the same time, memory impairment in the form of amnesia and delayed recall is also a known phenomenon in traumatized individuals (7); it has been documented in response to such diverse traumatic events as natural disasters, war, torture, and physical or sexual abuse.

The complexity of traumatic memories attracted the attention of early scholars, including Pierre Janet (during the 1880s, in his scientific examination of the memory process), Charcot, Breuer, and Freud (in 1893). It has since been accepted that memory is a dynamic organization of past experiences: it is often distorted and influenced by the person's emotional state at the time of recall and by the significance of the experience itself. Emotional memories, especially of traumatic events, seem to be fragmented and incomplete (8), inflexible and unchanged by other life experiences (9), and primarily sensory, emotional, and nonverbal (10). As a result, traumatic memories tend to intrude upon the individual's consciousness at any given time. Indeed, laboratory studies of individuals with PTSD have shown that they process trauma-relevant material selectively, demonstrate enhanced memory for trauma-related material, and exhibit difficulty forgetting trauma words during directed forgetting (11). They also experience problems retrieving specific autobiographical memories in response to cue words, recalling "overgeneral" memories instead (8).

Heightened emotion and arousal are considered to be key features of the trauma response. Studies examining the effect of heightened emotion and arousal on the accuracy of memory have found that, for nonviolent content, participants' confidence in their own testimony and accuracy of memory are related (12). However, this relation between confidence and accuracy was not found in response to violent content, implying that memory encoded under emotionally charged situations is potentially distorted. Other studies suggest that attention to details immediately relevant to the arousing situation is actually heightened, possibly at the expense of attention to general details. These findings may support the observation that memories of trauma are fragmented, displaying hyperawareness of some details and an apparent disregard of others (13).

These recurring observations have given rise to the perception that memories of traumatic events are inherently unique and probably encoded and processed differently than are nontraumatic events (14,15).

The intricate system of memory is commonly thought of as comprising 2 primary pathways. The first is regular memory, called explicit or declarative memory. This refers to conscious awareness of facts (17) and requires focal attention for processing; it is probably mediated by the medial temporal lobe system that includes the hippocampal formation and related structures that enable verbal representation (18,19). Conversely, the second pathway, called implicit or nondeclarative memory, refers to memories acquired during skill learning, habit formation, and simple, classic conditioning. It also refers to other knowledge expressed through performance rather than recollection (18). These memories are believed to be not easily accessible to consciousness (14). Traumatic memories that subjects can recall are thus part of explicit memory.

The notion that memory of the traumatic event is essential for the development and definition of PTSD raises the question whether it is possible for individuals who have no recollection of trauma to develop a posttraumatic response. It has been illustrated in case reports that individuals with no conscious memory of the traumatic event are still able to reenact their experiences; however, the commonly held view is that lack of memory precludes the development of PTSD (20).

The controversy regarding trauma and memory cannot be empirically resolved by experimental studies in humans, owing to practical and ethical limitations in the simulation of naturally occurring trauma and in manipulation of memory (7). As a compromise, many studies have focused on the occurrence of traumatic brain injury (TBI) (21–23). This traumatic event is often associated with loss of consciousness and impaired memory, and TBI can therefore serve as a naturalistic model for the study of memory and its role in the development of PTSD. Findings, however, are inconclusive. Studies have provided differing and sometimes conflicting results.

The following sections review these findings and discuss future directions for the study of PTSD and memory, together with implications for treatment.

Evidence Supporting Reduced Prevalence of PTSD After TBI

In the US alone, the estimated annual rate for TBI is 220 cases per 100 000 people (24). It is often accompanied by posttraumatic amnesia regarding events that occurred both after the injury (anterograde amnesia) and immediately prior to it (retrograde amnesia). Some researchers have argued that limited awareness at the time of the trauma makes it less likely

that traumatic memories can be encoded and that, as a result, these memories remain unavailable for the mediation of reexperiencing symptoms (20,25). Without the latter, PTSD or acute stress disorder (ASD), introduced in the DSM-IV to describe acute trauma reactions within the first month posttrauma, cannot be diagnosed. For example, in a study of 47 patients with moderately severe TBI, none fully met the criteria for PTSD: despite reporting partial PTSD symptoms—particularly, symptoms of avoidance and arousal—none endorsed symptoms of reexperiencing (26). Similarly, in a study comparing the acute stress reactions of road-accident victims both with and without head injury, the two groups reported high rates of anxiety, but the group with head injury reported fewer intrusive symptoms (27). Based on these findings, it was suggested that amnesia regarding the traumatic event minimizes the possibility that any cognitive representations of the trauma will be established (28). In another study of accidental head injury, PTSD was diagnosed in only 1 out of 107 injured patients, although other psychiatric problems were found in 22% of this group (29). Further, although some have suggested that TBI is a risk factor for PTSD (30), other research indicates that this is not the case and that PTSD is in fact rare among road-accident survivors with TBI, compared with road-accident survivors without TBI (31).

A less extreme view suggests that, although PTSD is unlikely to occur following TBI, it can develop following mild TBI (32). However, 1 study reports that, while patients with PTSD provided emotionally charged accounts of their traumatic experience (including nightmares, flashbacks, and intrusive imagery), such symptomatology was absent in a comparison group with mild TBI, who did not even report symptoms of emotional arousal when describing their traumatic event (20). An additional study involving 188 road-accident victims who sustained loss of consciousness reports that none exhibited symptoms of PTSD (30).

Posttraumatic amnesia (PTA) is considered a marker for the degree of TBI severity and a sensitive predictor of outcome (33). To assess the extent to which PTA influences PTSD symptom development and prevalence, particularly reexperiencing of the traumatic event, 282 TBI patients were examined within a few months after injury. The sample was stratified into 4 groups according to their degree of PTA and severity of brain injury (34). All 4 groups reported intrusive and avoidant symptoms relating to the trauma, but these symptoms diminished significantly when PTA exceeded 1 hour. In some subjects, however, PTSD symptoms were present even when PTA exceeded 1 week. The nature of the intrusive phenomenology did not vary by length of PTA. This is one of few studies that tried to account for the degree of memory disruption following TBI and its relation to subsequent development of PTSD.

Evidence Supporting PTSD After TBI

Alongside the studies indicating that subjects who sustain TBI develop PTSD less frequently are several studies suggesting that TBI in trauma survivors is not associated with a lower risk for PTSD. A study that followed victims of motor vehicle accidents during the acute phase of recovery found that symptoms such as fear and intrusive recollections of the accident were present both in those with and in those without head injury, although more commonly in the latter (27). Of those who sustained mild brain injury, 24% met criteria for PTSD in the initial month. Subsequent studies reported that 14% of subjects with mild TBI developed ASD, and an additional 4% to 5% were diagnosed with subsyndromal ASD (35). A 6-month follow-up of the same cohort revealed that 24% had proceeded to develop PTSD, suggesting that risk factors other than ASD were involved (36). A prospective study of 97 people admitted to hospital with mild TBI following motor vehicle accidents found that, after 2 years, 80% of those initially diagnosed with ASD still suffered from PTSD (37). In 2 additional studies, it was reported that 33% of the patients with brain injury met criteria for PTSD (22,38). Another study selected a random sample of 100 subjects from 400 patients with mild-to-severe TBI, and using the Structured Clinical Interview for DSM-IV, diagnosed PTSD in 17% of this sample (39). In a prospective study of 107 road-accident survivors who sought medical attention within 2 days, 36% were diagnosed with PTSD, including 9 out of 16 who had lost consciousness (40). Indeed, there are several case studies of patients with severe head injury who met criteria for PTSD despite extended periods of amnesia and a self-reported inability to recall any aspect of the trauma (41,42).

A few studies examined whether PTSD symptoms with TBI are similar or dissimilar to PTSD symptoms without TBI. For example, ASD and PTSD symptoms in accident survivors with and without TBI were compared after 1 and 6 months. At 1 month, traumatic memories of the accident were less common in the mild TBI group, but by 6 months, this difference was no longer apparent (43). Similarly, patients without brain injury have reported less intrusive memories with time, while those with TBI have displayed increased intrusive memories, suggesting a unique course of posttraumatic adjustment after TBI (27,44).

In a retrospective study of 312 patients with severe TBI referred for neuropsychological assessment (45), 10 had symptoms of PTSD, suggesting that at least 3% of survivors may experience the disorder. In a representative sample of 66 survivors of severe TBI, 18.2% had clinically significant PTSD symptomatology; of these, 6.1% had severe symptoms (46). These rates are lower than rates reported in subjects with mild brain injuries, yet comparisons with other studies are problematic, given varying definitions and methodological differences.

Taken together, these studies support the notion that TBI does not reduce the prevalence of PTSD following exposure to traumatic events. Further, they call into question whether TBI protects trauma survivors from subsequently developing PTSD. However, it is noteworthy that none of these studies carefully addressed memory for the traumatic event as an important variable that differentiates people with TBI. As we discuss in the following section, this factor seems to be of major importance.

TBI-Induced Amnesia and the Risk for PTSD

The assumption underlying many of the studies examining the relation between TBI and PTSD is that impaired consciousness precludes memory of the trauma (47). However, as mentioned before, most studies did not adequately address the degree to which victims of head injury actually remember the traumatic event.

In an effort to fill this gap, we designed and recently completed 2 studies that employed a memory questionnaire requiring participants to assess the degree to which they remember the following 9 domains of the traumatic event: the nature of the event, where it took place, who was involved, when it took place, sounds, sights, odours, things said by the participant during the event, and things said by others. In the first study, we used a prospective design to examine the relation between participants' appraisal of their memory for details of the traumatic event and the later development of PTSD (48). The participants (n = 120) were admitted to the surgical ward following a trauma involving mild head injury, and we evaluated them at 4 different time points over 6 months (within 24 hours and after 1 week, 1 month, and 6 months). We used the Structured Clinical Interview for Axis I Disorders-Nonpatient Edition (SCID-NP) (49) to diagnose psychiatric disorders and the Clinician-Administered PTSD Scale (CAPS) (50) to assess current PTSD symptoms. Most participants (91%) were motor-vehicle accident survivors with mild physical injury. Responses on the memory questionnaire showed a bimodal distribution in most respondents, with reports of memory for all the domains or for none. Overall, 14% of the participants met full criteria for PTSD 6 months after the trauma. However, PTSD was nearly 5 times more prevalent among participants with memory of the trauma than among those without memory of the trauma. Appraised memory for the traumatic event as early as 24 hours after its occurrence was shown to strongly predict the presence of PTSD at 6 months. It is noteworthy that the positive relation between memory and PTSD was owing primarily to the difference in the reexperiencing cluster. When we accounted for other PTSD risk factors, memory of the traumatic event was associated with more than twice the risk of developing PTSD following a traumatic event involving TBI.

These findings were corroborated in a second study using a retrospective design. We recruited 120 patients with head injury (on average, 3 years posttrauma) from an outpatient neurocognitive clinic and evaluated them in a single, 3-hour interview (51). Overall, 22% of the participants in this study met full criteria for PTSD. The prevalence of PTSD among subjects with memory of the trauma was more than 5 times higher than among participants who did not remember the traumatic event. Moreover, when we accounted for anxiety and depressive symptoms, sex, and age, memory for the details of the traumatic event was associated with a threefold increase in the risk for a PTSD diagnosis. As in the previously mentioned study, the positive relation between memory and PTSD was primarily attributable to a difference in the reexperiencing cluster.

While these 2 studies clearly show the positive correlation between memory for the traumatic event and PTSD, they also show that, in a subset of patients, lack of memory does not fully protect against the development of PTSD. There are several mechanisms by which PTSD could develop in subjects lackng memory of the traumatic event, but a detailed discussion of this complex issue is beyond the scope of this review. Suffice it to say here that emotionally charged traumatic memories may be initially processed via brain circuits that bypass cortical structures and are mediated primarily through the amygdala, resulting in the formation of implicit (unconscious) memories (52). At the same time, stress-induced secretion of glucocorticosteroids, which have been shown to impair hippocampal functioning (53), may disrupt the formation of explicit (conscious) memory.

Conclusions and Implications

This review addresses the relation of traumatic memory to PTSD in the context of TBI, a naturally occurring model for studying the role of memory in trauma response. The literature review is not conclusive, but taken together with the results from our own studies, there seems to be support for the notion that PTSD is more prevalent in TBI victims with memory of the traumatic event and less prevalent when memory of the traumatic event is impaired. This clearly indicates that, at least in this patient population, amnesia regarding the event may protect against the development of PTSD.

While speculative, it is tempting to generalize from this to other traumatic conditions that do not involve head trauma but in which traumatic exposure is nevertheless associated with impaired memory of the event. Taking this even further, one might suggest that deliberately disrupting the memory of the traumatic event might prove therapeutically beneficial. This possibility has been addressed in a recent double-blind study that compared the severity of acute PTSD symptoms in 18 subjects who were given 40 mg of propranolol 6 hours after the trauma with symptoms in 23 participants who received placebo (54). In this study, subjects in the experimental group tended to have lower levels of PTSD symptoms 10 days posttrauma. If further corroborated, these findings may support the notion that not only does lack of memory protect against the development of PTSD but also that pharmacologically induced disruption of traumatic memories can therapeutically benefit trauma survivors.

The above-mentioned study assumed that it is crucial to intervene in the initial process of memory consolidation, which is believed to take place within the first 12 hours after traumatic exposure. However, this may not be the case. Findings from recent studies indicate that memory is a dynamic process: in it, the interplay between retrieval of consolidated memory and its reconsolidation after further processing in the working memory constantly reshapes old memories (52). This ongoing process strengthens the memory, but at the same time it renders retrieved memories amenable to disruption (52). This could indeed be one of the mechanisms by which recurrent intrusive memories in PTSD operate as enhancers, constantly strengthening the memory of the traumatic event and

preventing its decay. From a neurobiological perspective, this process reinforces neural networks and involves protein synthesis (52,55). It has been further shown in animal studies that, during this process, retrieved memories are sensitive to pharmacologic disruption (for example, by the use of protein synthesis inhibitors), which may inhibit reconsolidation and result in the extinction of old memories (55,56). Thus, memory disruption can occur in already-consolidated memories, once they are retrieved and processed in the working memory. Taken together with the previously mentioned findings in patients with TBI, this may suggest that old traumatic memories in patients suffering from PTSD could be disrupted by pharmacologic means. If proven empirically, such a possibility might have far-reaching theoretical and practical implications, given that most psychotherapeutic treatments for PTSD emphasize the importance of exposure to and confrontation of the traumatic memories. Currently available treatments for PTSD are frustrating and unsatisfactory; thus, a search for new and different treatment approaches is timely and of great importance.

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Résumé : La relation entre le souvenir de l'événement traumatique et le syndrome de stress post-traumatique : preuves de lésion cérébrale traumatique, selon certaines études

Objectif : Cette étude se penche sur la relation entre la mémoire et le syndrome de stress post-traumatique (SSPT). Plus précisément, elle aborde le débat concernant le rôle du souvenir de l'événement traumatique dans le développement du SSPT, en se servant de la lésion cérébrale traumatique (LCT) comme modèle survenant naturellement dans une exposition traumatique et souvent associé à l'affaiblissement de la mémoire.

Méthode : Nous présentons une analyse critique des études de la documentation qui évaluent la relation entre la LCT et le SSPT, en mettant l'accent sur le souvenir de l'événement traumatique comme facteur déterminant. Nous discutons également des résultats d'études récentes menées par notre groupe.

Résultats : L'analyse de la documentation offre un tableau non concluant où une proportion significative d'études indiquent que le SSPT et la LCT sont mutuellement exclusifs, surtout chez les personnes qui ne se souviennent pas de l'événement traumatique. Ce résultat confirme la possibilité que l'absence de souvenir soit une protection contre le développement du SSPT. Cependant, certaines études indiquent que le SSPT survient chez des patients blessés à la tête, ce qui laisse croire que le SSPT peut se développer chez les survivants d'une LCT – même chez ceux qui ne se souviennent pas de l'événement traumatique. En général, toutefois, la majorité des résultats (y compris les nôtres) semblent appuyer la possibilité que chez les sujets ayant une LCT, la mémoire défaillante de l'événement traumatique soit associée à une prévalence réduite du SSPT.

Conclusions : L'hypothèse que l'amnésie concernant l'événement traumatique puisse protéger contre le développement du SSPT a une importance à la fois théorique et pratique. Cette analyse présentait le cas d'une lésion cérébrale traumatique comme modèle de mémoire entravée de l'événement traumatique. Cependant, il reste à prouver que les conclusions fondées sur ces résultats peuvent se généraliser au-delà de la LCT. Même si certains patients qui présentent une amnésie post-traumatique développent le SSPT malgré l'absence de souvenir de l'événement traumatique, la majorité de ceux qui ne se souviennent pas de l'événement semblent être protégés contre le développement du syndrome. Néanmoins, selon cette hypothèse, nous suggérons que la suppression pharmacologique des souvenirs traumatiques récemment acquis – ou même depuis longtemp – qui s'est révélée possible chez les animaux, puisse avoir un avantage thérapeutique chez les survivants de traumas.