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Amnesia, traumatic brain injury, and posttraumatic stress disorder: a methodological inquiry

Mark Creamer[a,b,*], Meaghan L. O'Donnell[a,b], Phillipa Pattison[c]

[a]*Australian Centre for Posttraumatic Mental Health, ARMC Repat Campus, PO Box 5444, Heidelberg, Victoria 3081, Australia*
[b]*Department of Psychiatry, University of Melbourne, Victoria 3052, Australia*
[c]*Department of Psychology, The University of Melbourne, Victoria 3052, Australia*

Abstract

This study explored the relationship between mild traumatic brain injury (MTBI), amnesia, and posttraumatic stress disorder (PTSD). MTBI status and amnesia for the event were assessed in 307 consecutive admissions to a Level 1 Trauma Center. Amnesia did not always occur concurrently with MTBI: 18% of those with MTBI had full recall and over half had partial recall of the event. Just over 10% of participants developed PTSD by 12 months post-injury, with prevalence comparable across MTBI and non-MTBI groups. Non-significant differences in incidence of PTSD were apparent between those with full recall (9%), partial recall (14%) and no recall (7%). These data highlight the fact that PTSD may develop following trauma despite amnesia for the event, and illustrate the importance in both clinical and research settings of carefully examining the extent of amnesia.
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*Corresponding author. Australian Centre for Posttraumatic Mental Health, ARMC Repat Campus, PO Box 5444, Heidelberg, Victoria 3081, Australia. Tel.: +61 3 9496 2922; Fax: +61 3 9496 2830.
E-mail address: markcc@unimelb.edu.au (M. Creamer).

Introduction

Posttraumatic stress disorder (PTSD) is known to develop in a significant proportion of individuals following exposure to a traumatic event (Kessler, Sonnega, Hughes, & Nelson, 1995). Such events are likely to involve physical harm, with the possibility of mild traumatic brain injury (MTBI). This is especially the case following motor vehicle accidents (MVAs), other accidental injury, and assaults where injury is serious enough to warrant hospitalization. Considerable debate has focused on the question of whether it is possible to develop PTSD following MTBI. It has been suggested (e.g., O'Brien & Nutt, 1998; Sbordone & Liter, 1995) that limited awareness at the time of the trauma prevents encoding and consolidation of the experience. It is argued that trauma survivors with MTBI have no painful traumatic memories and, therefore, cannot develop core PTSD symptoms characterized by intrusive re-experiencing of the event. This reasoning assumes that MTBI is always associated with amnesia.

Although early studies suggested that, indeed, PTSD did not occur following MTBI, (Mayou, Bryant, & Duthie, 1993; Sbordone & Liter, 1995) those studies have been criticized on methodological grounds (Bryant, 2001; McMillan, 1997). Recent research suggests that PTSD can, and does, occur following MTBI. Bryant and Harvey (1999) for example, reported that the prevalence of PTSD among MVA survivors was comparable across MTBI and non-MTBI groups. Hickling, Gillen, Blanchard, Buckley, and Taylor (1998) found that 40% of those who lost consciousness in their sample went on to develop PTSD. Similarly, Mayou, Black, and Bryant (2000) reported that PTSD was as common in MVA survivors who were briefly unconscious as in those who were not.

In light of these findings, several authors have proposed explanations for the mechanisms that may mediate the re-experiencing symptoms of PTSD in the wake of impaired consciousness at the time of the trauma (e.g., Bryant, 2001; McMillan, 1997). The simplest explanation is that the impaired consciousness may not last throughout the traumatic experience, thereby allowing the acquisition of traumatic memories. In such cases, aspects of the traumatic experience are presumably being encoded and may subsequently form the basis of PTSD. This highlights a major omission from much of the research on MTBI and PTSD. Although some studies define criteria against which MTBI was diagnosed, rarely is amnesia defined. The proposition that PTSD could not follow MTBI was based on the assumption that head injured survivors would be amnesic for the event; this is clearly not always the case. Thus, in addressing the question of whether PTSD can develop in trauma survivors with MTBI, it becomes crucial to examine the extent of any amnesia for the event.

This shift in focus has the advantage of reducing the importance of defining MTBI. While operational definitions are provided for the more severe end of the TBI range (i.e., to differentiate mild from moderate or severe TBI; see, for example, American Congress of Rehabilitation Medicine, ACRM, 1993), the decision about whether a person has any kind of TBI is more complex. This has particular relevance in the context of PTSD and the acquisition of traumatic memories. In the absence of a head injury, it would be assumed that the event was encoded at the time; the memory exists even if it is not accessible. In the case of head injury, it is assumed that the memory was not encoded (because of lack of consciousness); thus, no memory of the event exists. This confusion regarding the diagnostic limits of MTBI makes previous research on the relationship between head injury and PTSD difficult to interpret.

In order to further explore the relationships among MTBI, amnesia, loss of consciousness, and PTSD, and to focus on amnesia as the primary area of interest, we compared the prevalence of PTSD in a population of severely injured trauma survivors with and without amnesia for the event.

Method

Participants

Participants were consecutive admissions to a Level 1 Trauma Service. Individuals were included if they: (a) experienced a physical injury that required an admission of at least 24 h, (b) experienced either no brain injury or mild traumatic brain injury (as defined by the ACRM, 1993), (c) were aged between 18–70 years, and (d) had a reasonable comprehension of English. Participants were excluded if the injury was a result of deliberate self-harm, or if they were currently using intravenous drugs or had a current psychotic disorder.

Over an 18-month period, 412 individuals met entry criteria and were approached to participate in the study. A total of 363 agreed, representing an 88% participation rate. Following detailed explanation of the study, written informed consent was obtained from all participants. A total of 307 individuals completed the 12-month assessment, representing an 85% retention rate.

The majority (76%, $N = 232$) of participants were male and the average age was 37 years ($SD = 13.66$). Participants spent an average of 10.52 days in the Trauma Centre ($SD = 10.13$) with 32% ($N = 97$) requiring an Intensive Care Unit (ICU) admission. The mean Injury Severity Score (ISS; Baker, O'Neil, Haddon, & Long, 1974) was 13.67, $SD = 9.34$.

Assessment issues

Initial assessments were conducted in hospital, a mean of 8 days post-injury and 2.5 days prior to discharge. This assessment was timed to minimize the potential confounding effects of issues such as hypovolumaemia, pain, and analgesia use. At the time of assessment, patients were haemodynamically stable, were relatively pain free, and were a minimum of 24 h post opioid analgesia. At this point, MTBI status was determined and participants were asked about their memory for the event. Regrettably, accurate data regarding substance use intoxication at the time of the event, which may have affected memory, were not available.

MTBI

Individuals were assumed to have suffered a head injury if they experienced one of: (a) a period of unconsciousness, (b) a period of PTA and/or any loss of memory for the event, or (c) an altered mental state, defined as a Glasgow Coma Scale (GCS: Teasdale & Jennett, 1974) of less than 15. Individuals were diagnosed with mild, rather than moderate or severe, TBI if they had: (a) LOC of 30 min or less, (b) a GCS of 13 or more after 30 min, and (c) PTA not greater than 24 h (ACRM, 1993). Information regarding GCS and LOC were obtained from the medical notes. While the Westmead PTA Scale (Marosszeky et al., 1998) was effective at identifying PTA greater than 24 h in order to exclude patients with moderate or severe TBI, assessing PTA for MTBI inclusion was

more difficult. Following Gronwall and Wrightson (1980), participants were asked to describe the event, starting from just before when the injury occurred. They were then asked "And what happened then?" until the account reached the arrival of the ambulance or arrival in hospital. Witnesses and ambulance records were consulted to set the times of the accident and subsequent events. The duration of PTA was defined as the elapsed time between the accident and the return of continuous memory. While recognizing that retrospective reports of amnesia may not be entirely accurate, these criteria conform to internationally accepted guidelines and were the only means available to assess short periods of PTA. Finally, in line with the ACRM guidelines, any participant reporting less than 100% recall in response to the amnesia question below was included in the MTBI group. Since this may include some participants with psychogenic amnesia, this definition of MTBI is relatively inclusive.

Amnesia

Participants were asked about the extent of their memory for the traumatic event. However, opioid analgesic medication was administered to the majority of participants shortly after the trauma (upon arrival of the paramedics) and was often maintained for several days in hospital. Since opioids may impact upon awareness, information processing, and memory, a narrow definition of the traumatic event was adopted. In order to minimize the potential influence of opioids, questions regarding memory for the event were limited to recall of the period "from just prior to the event to when the ambulance arrived". Participants were asked to nominate a percentage figure to estimate their level of recall for that period. Those nominating 100% were classified as "full recall", those nominating 0% were classified as "no recall", and those nominating any percentage greater than 0 but less than 100 were classified as "partial recall".

PTSD

PTSD at 12 months post-injury was diagnosed using the Clinician Administered PTSD Scale for DSMIV (CAPS-IV: Blake et al., 1995) administered by trained mental health clinicians as part of a longer telephone interview. Thirty percent of all interviews were audiotaped, with one third of those randomly chosen for interrater reliability. Agreement on the absence or presence of a CAPS diagnosis was 100%.

Given the difficulty in differentiating between organic and psychogenic amnesia, a strategy to deal with the DSM-IV PTSD Criterion C3 (psychogenic amnesia) was developed. An a priori decision was made to exclude psychogenic amnesia in the assessment of PTSD for all participants. This is consistent with other similar research (Bryant, Marosszeky, Crooks, Baguley, & Gurka, 2001; Schnyder, Moergeli, Klaghofer, & Buddeberg, 2001).

Results

Over half the participants (62%; $N = 189$) met study criteria for MTBI. Of those, 69% ($N = 130$) experienced at least brief LOC, 82% ($N = 155$) had less than complete memories of the event and/or a period of PTA, and 50% ($N = 95$) had a GCS of 13 or 14 after 30 min.

As shown in Table 1, nearly 18% ($N = 54$) of the sample was amnesic for the event, 33% ($N = 101$) had partial recall, and 49% ($N = 152$) had full recall. Interestingly, of those who

Table 1
Comparisons between participants with full, partial, and no recall of the event

	Total ($n = 307$)		Full recall ($n = 152$)		Partial recall ($n = 101$)		No recall ($n = 54$)	
	N	%	N	%	N	%	N	%
Loss of consciousness	130	42	21	14	70	69	39	72
GCS after 30 min of 13–14	95	31	18	12	39	39	38	70
MTBI								
Yes	189	62	34	22	101	100	54	100
PTSD diagnosis at 12 months	32	10	14	9	14	14	4	7
Re-experiencing	78	25	37	24	29	29	12	22
Avoidance	39	13	18	12	15	15	6	11
Arousal	61	20	26	17	24	24	11	20

claimed to have full recall of the event, 14% ($N = 21$) had experienced a loss of consciousness at the time (albeit usually of less than 2 min) while 28% ($N = 15$) of those with no recall of the event had apparently not lost consciousness. These apparent contradictions highlight some of the difficulties in making a diagnosis of MTBI.

At 12 months post-injury, 10% ($N = 32$) of the sample met criteria for PTSD. Prevalence was higher in those with MTBI than in those without, although a χ^2-test of independence revealed that this difference was not significant (15%, $N = 24$ vs. 7%, $N = 8$), χ^2 (1, $N = 307$) = 2.73, $p = .10$, *ns*. As shown in Table 1, the prevalence of PTSD was highest in the partial recall group (14%, $N = 14$), followed by the full recall (9%, $N = 14$) and no recall (7%, $N = 4$) groups although, again, a χ^2-test revealed that these differences were not significant, χ^2 (2, $N = 307$) = 2.04, $p = .36$, *ns*. The number of participants meeting criteria for each PTSD symptom cluster is also shown. All groups were least likely to meet diagnostic criteria for the avoidance cluster and there was little to suggest differential patterns across the three recall groups.

Discussion

This large, prospective study of injured trauma survivors supports the proposition that PTSD can develop following MTBI. As such, the findings are consistent with much recent research in the field (e.g., Bryant & Harvey, 1999; Hickling et al., 1998). In the current data set, PTSD prevalence was comparable across MTBI and non-MTBI groups, with a trend towards a higher rate among those with mild brain injury. Interestingly, differences across symptom cluster were small, with little to suggest that those with more complete recall were more prone to re-experiencing symptoms. Importantly, however, the current study highlighted a difficulty confronting all researchers in this field—that of defining viable inclusion and exclusion criteria for MTBI. The difficulty in obtaining accurate information regarding whether a head injury occurred, and the high degree of overlap between functional and organic symptoms in the acute aftermath of trauma, result in relatively arbitrary applications of MTBI inclusion criteria. In the

absence of more reliable criteria for MTBI, comparison across existing studies becomes difficult and the benefits of focusing on MTBI as a mediator of subsequent psychopathology are limited.

Findings regarding the nature and extent of amnesia provide an interesting focus within this data set. The fact that nearly one-fifth of those with MTBI had full recall, and over half had partial recall of the traumatic event, illustrates that current MTBI criteria leave ample opportunity for awareness and encoding of at least some of the traumatic experience. This may go some way to explaining the discrepancies in previous research on head injury and PTSD. Sbordone and colleagues, (Sbordone & Liter, 1995) while generally arguing against the possibility of PTSD developing after head injury, admit that PTSD may be an outcome if the traumatic event commences before, or continues beyond, a brief episode of LOC. The current data suggest that those with partial recall are certainly able to develop PTSD; in fact, prevalence in that group (14%) was higher than the full recall (9%) and no recall (7%) groups. Thus, the MTBI/PTSD debate should, perhaps, focus on the extent of amnesia rather than the presence of a head injury per se.

Even those with no recall, however, were not immune to PTSD, prompting the need to consider alternative explanations for the development of the condition in those with full amnesia. It has been suggested that traumatic experiences may be processed at an implicit level during periods of impaired consciousness (e.g., Bryant, 2001). This is consistent with the finding that psychological distress and physiological reactivity in response to reminders of the trauma can occur in patients who have no memory of the event itself (Bryant, Marosszeky, Crooks, & Gurka, 2000). There is also evidence that some people with MTBI reconstruct memories of experiences that occurred during periods of impaired consciousness. Harvey and Bryant (Harvey & Bryant, 2001), for example, assessed the memories of MVA survivors with MTBI immediately after the accident and again 2 years later. Despite high levels of initial amnesia, 40% of the sample reported that they had recovered full recall of the experience 2 years later.

The period of the traumatic event was defined very conservatively for this study. While this was important in differentiating amnesia resulting from head injury from that caused by opioid intoxication, it is recognised that this definition is narrow. Clearly, secondary stressors, such as the ambulance journey and time in the specialist trauma unit, may themselves be traumatic and may play an etiological role in the subsequent development of PTSD. The absence of reliable data regarding intoxication at the time of the trauma (which may, of course, have impaired encoding) is a further limitation.

This study, despite being one of the largest to focus exclusively on severely injured trauma survivors, is limited by subject numbers. With only 10% developing chronic PTSD, it is premature to make definitive statements about the predictive utility of amnesia for a PTSD diagnosis. Such statements would require larger numbers, probably only possible in the context of multi-site trials using identical methodologies. The absence of reliable data regarding intoxication at the time of the trauma (which may, of course, have impaired encoding) is a further limitation.

Nevertheless, the current data highlight the importance in both clinical and research settings of carefully examining the nature and extent of amnesia. It is not sufficient to report simply on the prevalence of MTBI, since not all persons with MTBI are amnesic to the event.

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