The Role of Memory for Trauma in the Development of Post-Traumatic Stress Disorder following Traumatic Brain Injury

and Research Portfolio

Vol I

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Chapter 1

Small Scale Research Project

A comparison of referrals made to a pilot EMDR service with referrals made to a Clinical Psychology Department for Post Traumatic Stress Disorder

Prepared in accordance with requirements for submission to
Clinical Psychology
(See Appendix 1.1)

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A comparison of referrals made to a pilot EMDR service with referrals made to a Clinical Psychology Department for Post Traumatic Stress Disorder.

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Comparison of referrals found no significant differences in age, gender, trauma type, time from trauma to referral, or attendance rates between services. Significantly more EMDR patients received additional professional support during their treatment.

1 Introduction

Post Traumatic Stress Disorder (PTSD) can present in survivors of a traumatic event and is defined in DSM-IV by the occurrence of 3 clusters of symptoms together – re-experiencing, avoidance and hyperarousal – persisting for a least one month (American Psychiatric Association, 1994). Lifetime prevalence rates vary considerably and have been reported as from 1% to 12.3% (Breslau et al, 1991). Co-morbidity is common, most often with depression (48.5% females, 47.9% males), anxiety (33% males and females), drug and alcohol abuse (33% females, 50% males) (Fairbank, Ebert and Costello, 2000).

A number of treatment approaches have been trialed with PTSD. Cognitive Behavioural techniques (eg Cognitive Restructuring, Anxiety Management, Exposure Therapy) have been found to have some of the largest treatment effects in meta-analysis (Van Etten & Taylor, 1998) with Exposure Therapy having particularly strong evidence for its effectiveness (Foa, Rothbaum, Riggs & Murdock, 1991).

Eye Movement Desensitisation and Reprocessing (EMDR) was first developed by Shapiro (1989). Her aim was to reduce the anxiety patients felt when recalling a traumatic event and to reduce to intrusiveness of related disturbing images. The technique involves the patient bringing to mind a particular memory from a traumatic event, along with associated sensations and cognitions, whilst focusing on the therapist's fingers moving back and forth in front of their eyes. The therapist

then gives instructions to 'let go' of the memory and to ask for feedback on any feelings and visual images experienced. The cycle is repeated until the distress associated with the target image is reduced. The same image is then paired with a positive thought using the same process until the patient rates the thought as feeling valid.

A number of studies into the effectiveness of EMDR have demonstrated its usefulness in alleviating the symptoms of PTSD (eg Boudewyns, Stewtka, Albrecht & Sperr, 1993, and Jensen, 1994). There remains some scepticism regarding its effectiveness when compared to other treatment approaches, for example CBT (Devilly & Spence, 1999) which in this study was found to have a more significant treatment effect. Discussion has also been raised regarding the mechanism of EMDR, with suggestions that the eye movements are not important to the treatment (Lohr, Tolin & Lilienfield, 1998). Further, imaginal focus on a traumatic image and the connection with positive cognitions are similar to techniques used in a trauma-focused CBT approach. However, the proposed NICE guidelines for the treatment of PTSD (www.nice.org.uk) consider that EMDR is an independent treatment from CBT, as particular training is needed to practice it.

1.2 National Standards

The current draft guidelines from NICE (2004) on the treatment of PTSD, states that 'all PTSD sufferers should be offered a course of trauma-focused psychological treatment (trauma-focused CBT or EMDR)'. Specifically, patients seen within 3 months of a traumatic event should be offered trauma-focused CBT, while patients who have had difficulties for more than 3 months should be offered either trauma-focused CBT or EMDR. The draft guideline further states that an important factor in deciding which treatment to provide should be patient preference and that enough information should be given for the patient to be able to make an informed choice.

1.3 Local Context

An EMDR pilot service was launched in March 2004 and consists of one trained EMDR therapist who provides around 1 ½ days per week to the service. Referrals are accepted for patients in the local area (Motherwell/Bellshill) who are suitable for this approach (Shaprio, 2001). Specifically, patients need not fulfil DSM-IV criteria for PTSD but should have experienced an event (real or imagined) that has led to distress on recollection and intrusions. Although the pilot service has only been operational for 1 year it is a new service in the area and therefore of interest. Agreement has been given by the practitioner for this audit to be carried out and she is aware of the purpose of the audit.

1.4 Aims

This audit seeks to:

1) Compare referrals made to the EMDR service with a sample of referrals seen by the local CP Department to discover how similar or different these two groups are in terms of gender, age, type of trauma, time from trauma to referral & ongoing support.

2) Investigate who is referring patients to each service to indicate whether there is a need for further information for referrers or wider publicity for the EMDR service.

3) Compare the attendance records of these patients as an indication of acceptability of treatment approach.

2 Method

2.1 Sample

All patients referred and accepted for treatment with the EMDR service from its launch in March 2004 to the end of April 2005 (n=18) are included in the audit. All new patients categorised as having PTSD symptomatology seen by the local CP

Department at Hartwood Hospital between March 2003 and March 2005 were also included (n=20). This time period was chosen to allow for a similar sample size. The CP department has a system of assigning each patient a number of codes that describes their symptoms. Codes are Broad (describing the main difficulty) and Fine (describing secondary problems). This sample represents those coded both Broad PTSD and Fine PTSD as both sets of patients would be considered suitable for EMDR.

2.2 Procedure

1) Ethical Approval

The audit proposal was presented to the local NHS Ethics Board and considered to be audit, therefore not requiring consent from patients as no identifying information would be included and the data included in the audit was routinely collected as part of clinical practice.

2) Data Collection

CP data was collected from patient files held in the department. If a patient was being seen by a psychologist, the psychologist's permission was sought to access their file. EMDR data was collected from the patient record file kept by the clinician providing the service, with her permission.

Data collected was; patient gender and age, referring agent, whether the trauma experienced was a single event (an example would be a road traffic accident) or multiple events (an example would be childhood sexual abuse), the length of time from the incident to referral, whether any other agency or service was also involved in their care, and finally attendance rates for the service as a whole, as individual attendance rates were not available.

3 Results

3.1 Age

Table 1 shows the mean age for patients in each group, the age range for each group and the statistical analysis carried out on these data. No significant difference was found between groups.

*****Insert Table 1*****

3.2 Gender

Table 2 shows the total number of male and female patients in each group and the statistical analysis carried out on these data. No significant difference was found between groups.

*****Insert Table 2*****

3.3 Trauma Type

Table 3 shows the total number of patients who had experienced a single trauma and the total number who had experienced multiple traumas for each group and the statistical analysis carried out on these data. No significant difference was found between groups.

*****Insert Table 3*****

3.4 Time from Trauma to Referral (years)

Histograms of the time from trauma to referral (see Figures 1 & 2) suggested that these data might be skewed. A Kolmogorov-Smirnov Test indicated that this was not the case therefore parametric statistics were considered suitable. Table 4 shows the mean time in years from the traumatic event to referral and shortest and longest

waiting time for each group and the statistical analysis carries out on these data. No significant difference was found between groups.

*****Insert Figures 1 and 2*****

*****Insert Table 4*****

3.5 Referring Agents

Referrals to Clinical Psychology

GP	12
Community Addictions Team (CAT)	4
CPN, Focused Intervention Team	2
Airbles Road Day Hospital	1

Referrals to EMDR

Psychiatry	8
Clinical Psychology	3
Airbles Road Day Hospital	3
Psychiatric Daycare Ward (Wishaw General)	3
Psychiatric Inpatient Ward (Wishaw General)	1

3.6 Additional Support Received During Treatment

Clinical Psychology

Seven of the 20 patients in the CP group received additional support during the time they were seen by this service. Those supports were;

Community Addictions Team -	4 patients
Psychiatry -	2 patients
Community Psychiatric Nurse -	1 patient

EMDR

Seventeen of the 18 patients in the EMDR group received additional support during the time they were seen by this service. Four of the 17 received two extra supports – 1) Mental Health support worker and Psychiatry, 2) CPN and Psychiatry, 3) Psychiatric Daycare Ward and Psychiatry, 4) Clinical Psychology and Psychiatry. The additional support therefore breaks down as follows;

Psychiatry -	12 patients
Clinical Psychology -	3 patients
Psychiatric Daycare Ward -	3 patients
CPN -	2 patients
Mental Health Support Worker -	1 patient

A chi-square comparing the number of patients receiving ongoing support in each group was significant, $X2=14.387$, $df=1$, $p<0.001$, with many more in the EMDR group having additional input.

3.7 Attendance

Table 5 shows the total number of hours given by each service to the treatment of patients with PTSD symptomatology, the number and percentage of those hours attended and not attended by patients in each group. These data are also presented in Figure 3.

*****Insert Table 5*****

*****Insert Figure 3*****

4 Discussion

The audit initially set out three aims. Findings relevant to each aim are discussed, followed by recommendations for service development and further research, and finally overall conclusions.

4.1 Comparison of referrals made to each service

No significant differences were found between the two groups' age ranges, gender distribution, or trauma type (single or multiple), suggesting a similar sample on these factors. Two areas of further interest were the length of time to referral from the traumatic event to referral, and the additional supports received during treatment. These will be discussed in greater detail.

Length of time from trauma to referral

A larger number of referrals were made up to 5 years from the trauma the CP Department (13) than to EMDR (8); however the time from the traumatic event occurring and referral to each service did not differ significantly between groups. Referrals met with NICE guidelines, with all EMDR patients having experienced the traumatic event 3 months or more previously. The length of time to referral was on average around 9 years, which raises some interesting issues for both services.

DSM-IV criteria state that symptoms occurring within the first three months of the event should be considered 'Acute PTSD', while symptoms persisting for 3 months or more are considered 'Chronic PTSD'. 'Chronic PTSD' therefore described all but one of the patients included in this sample, with a range of time lapse from 3 months to 36 years. Marshall et al (1999) criticises the definition of Acute PTSD as lacking utility as it infers no changes in terms of treatment and questions the ethical position of defining a problem chronic after only 3 months. Kessler, Sonnega, Bromet et al (1995) state that around 60% of people initially fulfilling diagnostic criteria will recover without treatment and that most cases of spontaneous recovery will take place in the first year following the event, with no further recovery having been found after symptoms have persisted for 6 years. If this is the case, perhaps it would be of more clinical use to differentiate between the time period where some recovery might occur naturally, and that when none is likely. A 6-year cut off for a more chronic PTSD would encompass 9/18 EMDR patients and 6/20 CP patients and be more meaningful for both services.

A further issue for both services is that of treatment efficacy with patients who have experienced symptoms for longer time periods. Some studies have carried out investigations with samples of Vietnam Veterans, who will most likely have experienced symptoms for many years. Jensen (1994) found that subjective units of distress reduced significantly more in a group of veterans treated with EMDR than standard services, but found no difference in PTSD measures. Silver et al (1995) reported a greater reduction of symptoms with Milieu treatment than EMDR than Milieu treatment alone, biofeedback or group relaxation, however the study was uncontrolled so limiting the strength of findings. Boudewyns and Hyer (1990) looked at the effects of Exposure Therapy with veterans and found some evidence but small effect sizes. There does therefore appear to be some difference in outcome with this population. It is difficult to generalise findings from such a specific sample and such specific trauma however services may find longer standing PTSD harder to treat.

Additional Supports

Significantly more EMDR patients received additional support (17 out of 18) than CP patients (7 out of 20). This may reflect a view of EMDR as a discrete piece of therapy which can be provided alongside other treatments and supports or that this service is not viewed as an independent treatment option. Or alternatively, perhaps the cases passed to EMDR were more complex than those seen by CP, with higher levels of co-morbidity. In this instance, EMDR may have been sought to help with PTSD while the clinician continued to provide help with any other difficulties.

4.2 Referring Agents

The majority of referrals to CP were made by GPs. Four referrals came from the Community Addiction Team (CAT) suggesting these 4 patients (who continued to be supported by CAT) had co-morbid alcohol or drug use issues.

The referrals made to the EMDR service were quite different. Any GP or clinician in the local area can make referrals, however the data suggest that only a small number of clinicians are aware of the service. Eight referrals came from Psychiatry, but this actually represents one Psychiatrist. Similarly, 3 referrals were made from

CP but this also only represents one Psychologist. The clinician providing the service made two referrals. In total, referrals came from only 8 clinicians.

It seems that clinicians who are aware of the EMDR service have made use of it, but perhaps it is not widely known about, suggesting greater publicity of the service should be undertaken.

4.3 Attendance

Attendance rates were very similar across both groups with attendance of 76.6% of sessions by CP patients and 74.8% of sessions by EMDR patients. Both services may therefore be considered as equally acceptable and valid to patients. The total hours given to treatment of PTSD demonstrates how valuable the EMDR service is to this locality, with 147 hours given over one year compared to 223 from the CP department over two years.

5 Recommendations

5.1 Suggestions for EMDR Service Development

The data indicate that the EMDR service is operating as a specialist service providing one part of a patient's care. As the service is time restricted this may be the most appropriate service delivery option at present. However, information about the EMDR service should be more widely disseminated to allow more clinicians (and therefore more patients) the option of accessing the service. NICE guidelines emphasise the importance of patient choice when deciding treatment approach and this should be made aware to referring agents.

5.2 Suggestions for Future Research

It appears from these data that a naturally occurring sample of PTSD sufferers will have experienced symptoms for an average of 9 years. The current differential of Acute and Chronic PTSD does not aid the clinician and further research into whether longer standing PTSD should be treated differently would be of mush use.

6 Conclusions

Patients referred to both the EMDR and CP services are very similar on measures of age, gender, trauma type, and time from trauma to referral. Fewer individual clinicians referred to the EMDR service than CP, which received most referrals from GPs. This suggests a need for wider publicity of the EMDR service. The average time from trauma to referral was 9 years, which raises questions regarding the clinical relevance of diagnosing chronic PTSD after 3 months. EMDR patients received many more additional supports than CP patients, which may reflect a view of the service as a specialist, additional treatment or a high rate of co-morbidity in this group. Similar attendance rates for both groups suggest both approaches were equally acceptable.

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Tables and Figures

Table 1 Age of Patients (years)

	Mean (SD)	Range	Statistics/Results
Clinical Psychology	38.45 (11.70)	26-58	Independent t-test t=1.097, df=36, p=0.280, n.s.
EMDR	42.17 (8.80)	23-68	

Table 2 Gender (total number)

	Male	Female	Statistics/Results
Clinical Psychology	12	8	Chi-square X2=0.920, df=1, p=0.338, n.s.
EMDR	8	10	

Table 3 Trauma Type (total number)

	Single	Multiple	Statistics/Results
Clinical Psychology	15	5	Chi-square X2=0.320, df=1, p=0.572, n.s.
EMDR	12	6	

Figure 1

Figure 1 - Histogram, CP Group, Time from Trauma to Referral

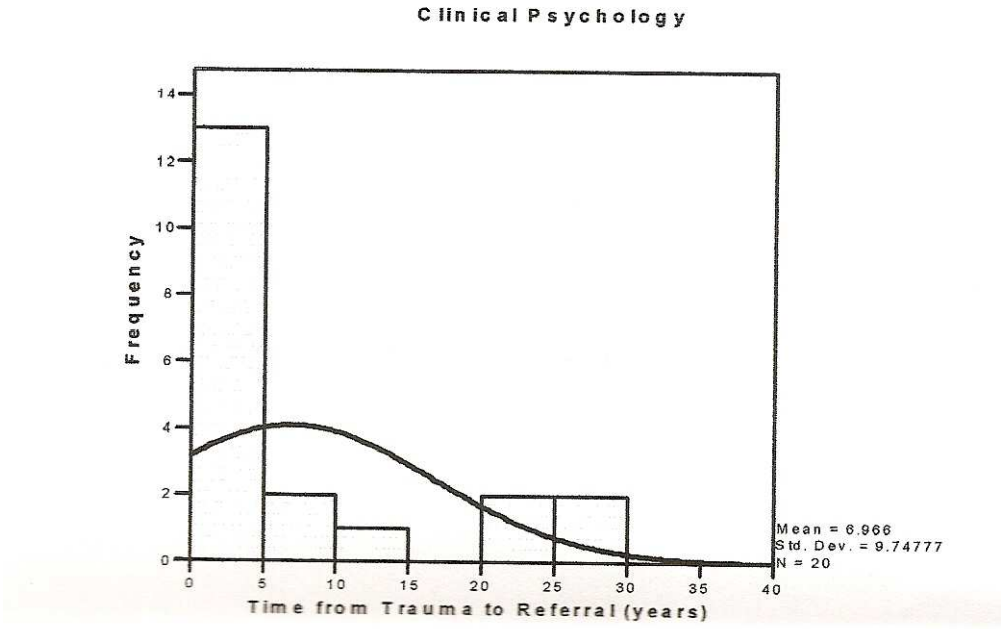


Figure 2

Figure 2 - Histogram, EMDR Group, Time from Trauma to Referral

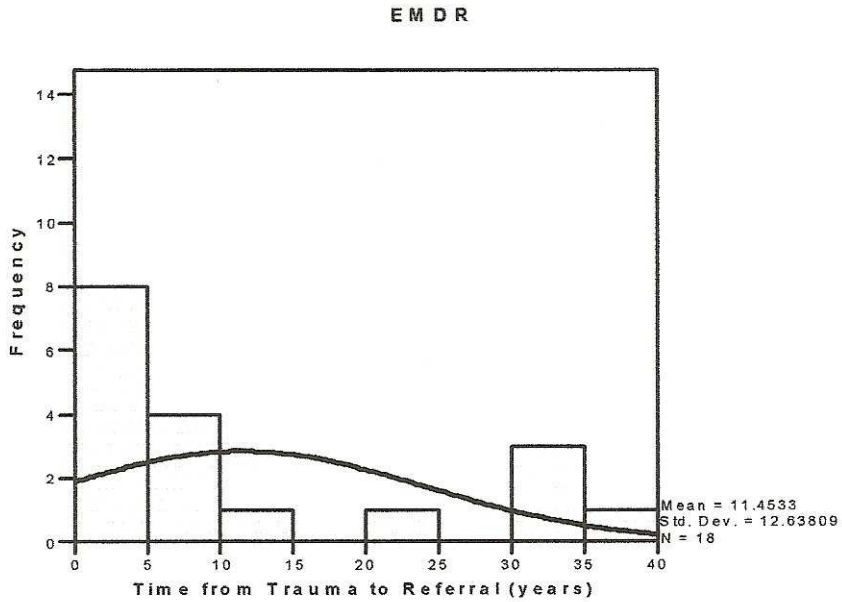


Table 4 Time from Trauma to Referrals (years)

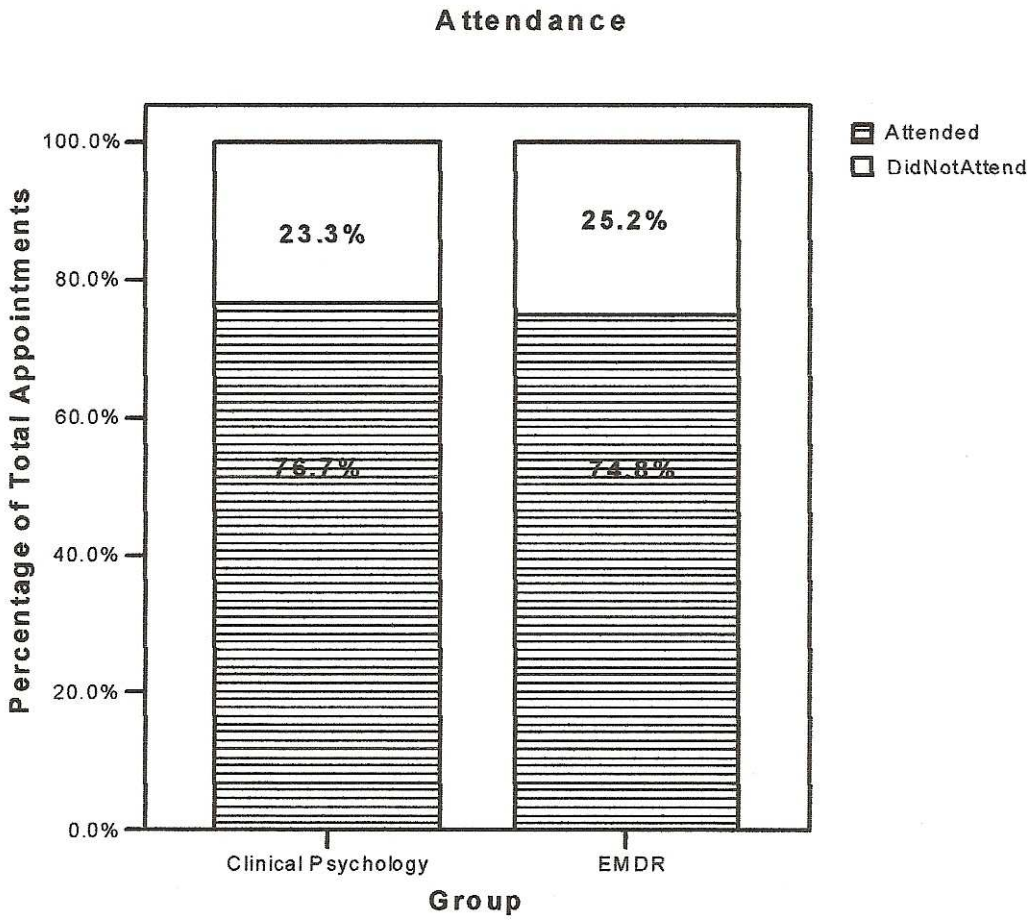
	Mean (SD)	**Range**	**Statistics/Results**
Clinical Psychology	6.96 (9.74)	1 month-30 years	Independent t-test t=1.233, df=36, p=0.226, n.s.
EMDR	11.45 (12.63)	3 months-36 years	

Table 5 Attendance Rates (total hours, percentages)

	Total Hours	**Patient Attended**	**Patient Did Not Attend**
Clinical Psychology	223	162 (76.7%)	49 (23.3%)
EMDR	147	110 (74.8%)	37 (25.2%)

Figure 3

Figure 3 - Percentage of Appointments Attended/Not attended by Patients.



Chapter 2

Major Research Project Systematic Review

Posttraumatic Stress Disorder and Traumatic Brain Injury: A Systematic Review of Causal Mechanisms

Prepared in accordance with requirements for submission to
Journal of the International Neuropsychology Society
(See Appendix 2.1)

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Abstract

This review describes the current evidence for causal mechanisms for the development of Posttraumatic Stress Disorder (PTSD) following traumatic brain injury (TBI) in an adult population. A systematic search strategy identified 9 studies published in 12 articles, which achieved the lowest SIGN grade of recommendation (D). The evidence suggests that fear conditioning may mediate PTSD after TBI. Symptoms of emotional and physiological reactivity are reported more often than intrusive memories and higher levels of arousal post-trauma are associated with PTSD symptom reporting. However methodological limitations in assessment of PTSD and measures of arousal confuse outcomes. Vulnerability factors predictive of PTSD in non-TBI populations (such as external attribution of causality) may also be relevant to TBI populations, and factors related to TBI outcome (such as reduction in executive functioning) may impact on PTSD symptom reporting. However the current evidence cannot confidently support these hypotheses. 'Recovery' of memory of the trauma over time may be due to confabulation, which could lead to an increase in reporting of re-experiencing symptoms. Differences reported between the trauma narratives of TBI and non-TBI groups indicate potential differences in the presence of intrusive symptoms. Lack of memory for the event may protect against PTSD, however limitations in assessment of recall exist. Limitations of the current evidence base are largely due to methods of assessment of PTSD. It has been demonstrated that assessment methods which do not allow for clinical judgement to be applied to symptoms reported can lead to over-diagnosis of PTSD (Sumpter and McMillan, 2005). It is recommended that further research into possible causal mechanisms is conducted, employing more valid methods of PTSD assessment.

1 Introduction

This paper systematically reviews studies investigating the development of Posttraumatic Stress Disorder (PTSD) in adults who have sustained a Traumatic Brain Injury (TBI). Studies into the incidence of PTSD following TBI demonstrate errors in overdiagnosis (Sumpter and McMillan, 2005, 2006). Symptoms of PTSD and TBI overlap and appear incongruous. For example, PTSD includes re-experiencing symptoms (such as intrusive memories of the event) however TBI can involve an extended period of unconsciousness and amnesia before and after the event. This review considers the current evidence base for mechanisms of symptom development following TBI.

1.1 Definitions of PTSD and TBI

Posttraumatic Stress Disorder (PTSD) involves key symptoms of intrusion, avoidance, and hyperarousal following a traumatic event which involves a threat to an individual's life or physical integrity and which is perceived as frightening. According to DSM-IV, symptoms must be present for at least one month to meet criteria for acute PTSD or three months to meet criteria for chronic PTSD and must have an impact on the individual's level of functioning (American Psychiatric Association, 1994; see Appendix 2.2).

Traumatic brain injury (TBI) severity is defined by the length of post-traumatic amnesia (PTA), duration of loss of consciousness (LOC), or Glasgow Coma Scale score (GCS). PTA is considered to be the most reliable predictor of adjustment following TBI (Bryant, 2001) and is defined as the period of time between the injury and return of continuous memory (Russell and Smith, 1961). Mild TBI involves PTA of less than one hour; moderate TBI involves PTA of one to 24 hours and severe TBI PTA of more than 24 hours (American Congress of Rehabilitation Medicine, 1993; Russell and Smith, 1961).

1.2 Prevalence of PTSD after TBI

PTSD is reported following mild TBI at rates of 14%-33% (Bryant and Harvey, 1999; Mayou et al., 2000) and following severe TBI at 3%-59% (Hibbard et al., 1998; Sumpter and McMillan, 2005). Sumpter and McMillan investigated diagnosis rates using three assessment tools – the Clinician Administered PTSD Scale (CAPS), Posttraumatic Diagnostic Scale (PDS) and Impact of Events Scale (IES). The PDS and IES, both self-report measures, led to reporting of symptoms related to TBI rather than PTSD and therefore over-diagnosis. The CAPS requires clinical judgement and allows for further investigation of each symptom, leading to the much lower incidence rate. The diagnosis of PTSD after TBI therefore requires careful interpretation of reported symptoms.

1.3 Development of PTSD after TBI

A number of possible mechanisms for the development of PTSD after TBI have been put forward. A brief overview of proposed mechanisms is outlined below.

Sub-conscious or neurological processes

Brewin et al. (1996) describe the 'dual representation theory' of PTSD which suggests that traumatic memories are stored as verbally accessible memories (VAMs) or situationally accessible memories (SAMs). VAMs can be intentionally retrieved and hold verbal and visual memories, whereas SAMs are generated subconsciously, possibly mediated by the amygdala, and may present as flashbacks or physiological symptoms. Therefore conscious processing of the trauma may not be necessary for PTSD to develop.

Theories of fear conditioning emphasise the increase in physiological arousal experienced during the traumatic event and suggest an association between cues to the trauma and further arousal can develop. van der Kolk (1996) hypothesises that this process occurs within limbic structures and outwith higher cortical processes. This hypothesis could indicate that people who do not have a conscious memory of

the traumatic event may still develop anxiety related to trauma cues as a result of fear conditioning.

It has been suggested that brain damage may itself play a part in the development of PTSD (Bryant, 2001). Biological theories of PTSD indicate a role for neurobiological factors in its development in non-TBI populations. These theories focus on the role of noradrenergic dysregulation which is hypothesised to create an inability to alter arousal levels, therefore creating the hyperarousal symptoms of PTSD (van der Kolk, 1996). TBI may interact with this process if brain damage impacts on areas of the brain involved in these functions (Bremner et al., 1995).

Pre and post-trauma vulnerability factors

A number of risk factors for PTSD have been identified in non-TBI populations, such as a previous psychiatric history, previous trauma, severity of threat during trauma, risk to life, dissociation during trauma, and an avoidant coping style (Davidson and Fairbank, 1993; Harvey and Bryant, 1998). One further area of interest is whether similar risk factors predict PTSD following TBI. TBI often results in disruption to cognitive abilities such as attention and memory along with physical disability and therefore alteration in lifestyle. It is possible that these difficulties may impact on an individual's coping abilities, which in turn could increase their likelihood of developing PTSD.

Memory for the event

The nature of the amnesia caused by TBI and the severity of memory loss is clearly an important area of research. Some studies have considered whether people with amnesia for trauma build their own 'memories' retrospectively, perhaps by incorporating third party reports, or through confabulation (Bryant, 1996, McMillan, 1996). Mild TBI involves a relatively short PTA and therefore some memories of the trauma may be retained. Recovery from PTA can be characterised by 'islands of memory' (King, 1997) which may include periods of memory during the trauma (McMillan, 1996). It has been suggested that there may be procedural memory for the event in absence of declarative memory (Layton and Wardi-Zonna,

1995). Therefore the quantity and quality of memory retained of the event may impact on the likelihood that PTSD will develop.

1.4 Objectives

This review aims to systematically identify the evidence for mechanisms of PTSD development following TBI. The review will establish the quality of evidence; the areas of mechanism investigated to date, and will make recommendations based on the findings for future research in this area.

2 Method

A systematic literature search was carried out using the OVID online interface to access the Psychinfo, Medline and Embase databases (See Figure 1). The search was conducted from 1980 (when DSM-III first included PTSD) to 2007 and included English language journal articles only. Search terms were '*traumatic brain injury'* or *'TBI'* or *'head injury'* combined with '*PTSD'* or *'post traumatic stress disorder'* or '*posttraumatic stress disorder'*. Additionally a hand search was conducted of the two most frequently identified journal titles (Brain Injury and the Journal of Traumatic Stress); the reference section of review articles identified from the search; and the reference sections of articles included in the review. The search identified 134 journal articles potentially suitable for inclusion.

[INSERT FIGURE 1]

2.1 Inclusion and Exclusion Criteria

Articles identified from the search strategy were assessed using structured inclusion and exclusion criteria. Articles were included in the systematic review if they met all of the following criteria:

1) The sample consisted of an adult population (aged over 16 years).
2) At least one group of participants had sustained a TBI.

3) TBI was classified through post traumatic amnesia (PTA), duration of loss of consciousness (LOC), or Glasgow Coma Scale (GCS) score.
4) PTSD was formally assessed by standardised questionnaire or structured interview.
5) The study aimed (as part or total objective) to investigate the development of PTSD following TBI.

Articles were excluded from the review if they met any of the following criteria:

1) The sample consisted of participants younger than 16 years of age.
2) None of the sample had sustained a TBI.
3) TBI severity was not classified by PTA, LOC or GCS.
4) PTSD was not formally assessed by standardised questionnaire or structured interview.
5) The study did not aim to investigate the development of PTSD after TBI.
6) The article was a review article.

Twelve articles describing 9 studies were included in the review.

2.2 Data Extraction

The characteristics of the sample investigated were extracted from each study. The methodology of each study was extracted and the methodological quality of each paper was rated. Quality criteria (outlined in Appendix 2.3) were established based on SIGN 50 guidelines (www.sign.ac.uk) and provided weighting for aspects of methodology considered particularly relevant to the current review. An independent rater graded the quality of the papers, leading to a 93% agreement rate with the writer. Disagreements were resolved through discussion. Each paper could achieve a maximum of 21 points and was graded from A (high quality, ≥ 75%) to D (poor quality, ≤ 49%). Each paper was also rated for the level of evidence according to SIGN 50 guidelines (see Appendix 2.4).

[INSERT TABLE 1]

3 Results

Articles are grouped according to the area of investigation – sub-conscious or neurological processes, predictive factors or memory for the event. Quality criteria points awarded, quality criteria grading and level of evidence for each article are given in brackets.

3.1 Sub-conscious or neurological processes

Bryant, Marosszeky, Crooks, and Gurka (2004) (Article 1 of 3 reporting from the same study) (*16 points, Grading A, level 2+*) investigated levels of arousal (measured by resting heart rate (HR)) in the two weeks following severe TBI and subsequent development of PTSD. Sixty-eight participants were recruited from a population of patients admitted to a brain injury rehabilitation unit over a 3 year period. PTSD was assessed by the Posttraumatic Stress Disorder Interview (PTSD-I). TBI was assessed through duration of PTA, assessed using the Westmead PTA Scale, and GCS. Sixteen patients (23%) met diagnostic criteria for PTSD 6 months post-injury and were found to have had higher resting HR ~9 days post-injury than those without PTSD ($t(66)=2.03$, $p<0.05$). However, when GCS score was controlled for, there was no significant difference between the groups. Those participants who had experienced more severe coma were less likely to have had a higher initial resting heart rate, suggesting severe coma reduced the impact of fear conditioning during the trauma. There was no significant difference in HR recorded 1 month after the injury. The authors propose that the association between initial HR and PTSD may provide evidence that fear conditioning can be experienced outwith conscious awareness and can contribute to PTSD development after severe TBI.

Bryant, Marosszeky, Crooks and Gurka (2000a) (Article 2 of 3 reporting from the same study) *(14 points, Grading B, level 2+)* report the symptom profile of their sample (n=96, the additional 28 excluded from the 2004 study due to a lack of HR data). Twenty-six participants (27.1%) were found to meet diagnostic criteria for PTSD. The predictive power of each of the 16 sections of the PTSD-I were calculated to determine which made diagnosis more or less likely. The symptoms

with the highest positive predictive power were intrusive memories (n=5), nightmares (n=6), and emotional reactivity (n=25). The authors claim that the low number of participants with PTSD reporting intrusive memories (n=5), along with higher numbers reporting emotional (n=25) and physiological (n=13) reactivity gives further weight to the hypothesis that PTSD is mediated by subconscious processes following severe TBI. The predictive power of re-experiencing symptoms was stronger in this sample than non-TBI samples.

Sojka, Stalnacke, Bjornstig and Karlsson (2006) (*10 points, Grading D, level 2-)* report on serum levels of cortisol as a measure of arousal and on the levels of two proteins (S-100B and neuron-specific enolase) as a measure of brain tissue injury, in a sample of patients admitted to hospital with mild TBI. Blood samples were taken on admission, 7 hours later, and at 1-year follow up. PTSD was assessed at 1-year follow up using the Impact of Events Scale (IES). Eighty-eight participants completed the first stage of assessment and 69 completed the follow up. Individual questions from the IES were compared with biological markers in a stepwise forward logistic regression analysis. This analysis found levels of S-100B at the 7 hour assessment to be significantly associated with three avoidance questions ('I tried to remove it from memory'; 'I felt as if it hadn't happened or it wasn't real'; 'My feelings about it were kind of numb'). The authors hypothesise that this association may indicate either the role of organic brain damage in altering neurological functions involved in posttraumatic stress, or that high levels of catecholamine (which they suggest may have been present in this sample) results in over-consolidation of memory for the trauma.

Limitations

Bryant et al., 2004, compared resting HR across participants as an indication of arousal levels. Resting HR in average adult populations can vary widely (eg Algra et al., 1993) and the differences found cannot be confidently ascribed to fear conditioning responses. The lack of significant difference at 1 month does add weight to this claim however it remains an uncontrolled variable. Sojka et al. employed cortisol as a measurement of stress but recognise that this is only reliable over a short time period. Additionally, *reduced* cortisol levels have been found to

correlate with PTSD development in non-TBI populations (e.g. Delahanty et al., 2000) whereas raised cortisol has been associated with brain injury (Woolf et al., 1990). Caution must be taken interpreting the conclusions of these studies due to the method of assessment of PTSD symptoms. Bryant et al. employ the PTSD-I as a diagnostic tool, which relies on participant ratings to establish the presence of symptoms. Sojka et al. rely on the IES, which assesses only intrusion and avoidance criteria and has been criticised as a diagnostic tool (Lees-Hayley et al., 2001; Sumpter and McMillan 2005). In both studies individual symptoms are analysed, however Sumpter and McMillan (2006) demonstrated that symptoms relating to TBI are often reported as PTSD. The three symptoms found to be associated with levels of S100-B may be symptoms of TBI rather than PTSD.

3.2 Predictive Factors

Bryant, Marosszeky, Crooks, Baguley and Gurka (2000b) (Article 3 of 3 reporting from one study) (*16 points, Grading A, level 2+)* investigated predictors of PTSD, established from research with non-TBI populations. It was hypothesised that pre-trauma functioning, trauma-related factors and response related factors, along with unemployment, shorter PTA and an avoidant coping style would predict PTSD severity. Assessment interviews were held between 5 and 7 months post injury. Participants also completed the Coping Style Questionnaire (CSQ) and the Functional Assessment Measure (FAM).

Comparison of the PTSD and no-PTSD groups found PTSD patients exhibited higher avoidance and emotion-focused scores on the CSQ. No difference was found in PTA between groups. Forward stepwise multiple regression with PTSD-I total score as dependent variable indicated avoidant coping style ($p<0.001$), behavioural coping style ($p<0.05$) and previous employment ($p<0.05$) were significant predictors of PTSD severity and together accounted for 40% of the variance. The finding that avoidant coping style was most strongly predictive of PTSD severity is in accordance with previous research on PTSD in non-TBI populations and in predicting ASD after mild TBI. The authors suggest severe TBI may compromise coping and problem-solving ability, increasing vulnerability to PTSD.

Williams, Evans, Needham, and Wilson (2002) *(12 points, Grading C, level 2-)* aimed to investigate the relationship between PTSD and severity of injury, level of insight into symptoms, severity of memory impairment, external attribution of causality and attribution of whether the event could have been avoided. Previous research has indicated increased risk for PTSD is associated with these factors.

Sixty-six participants were recruited from brain injury services and all had experienced a severe TBI between 1 and 26 years previously. The IES, dysexecutive questionnaire, Rivermead Behavioural Memory Test and questions relating to causality and avoidability of the traumatic event were administered. Memory impairment was not related to PTSD severity. Attribution for external causality positively correlated with PTSD severity. Rating of whether the event could have been avoided did not correlate significantly with PTSD severity. Insight was negatively correlated with PTSD severity. The authors conclude that lack of insight, indicated as moderate to severe dysexecutive disorder, might protect from PTSD or lead to inability to report symptoms. Holding attributions of external causality was associated with more severe PTSD symptoms and was hypothesised to relate to threat appraisal during the event.

Limitations

Williams et al. assess PTSD using the IES which as described has been demonstrated to have limited validity. The sample in this study was recruited from specialist services and may not be representative of the wider TBI population. Williams et al. note that their questions on event causality could not discriminate blame from causality, reducing the clarity of their results.

Bryant et al. hypothesise that reduced cognitive ability might increase vulnerability to PTSD, however do not include an assessment of cognitive functioning to explore this further. As noted previously, their assessment tool, the PTSD-I, may have led to reporting of TBI symptoms therefore their results cannot be reliably indicative of predictive factors in PTSD diagnosis in a TBI population.

3.3 Memory for the event

3.3.1 Symptom Profile

Glaesser, Neuner, Lutgehetmann, Schmidt and Elbert (2004) (*12 points, Grading C, level 2-)* investigated rates of PTSD and re-experiencing symptoms in a sample (n=46) of patients recruited from a neurological rehabilitation unit. Participants had experienced either a TBI or a traumatic injury to the cervical spine. They were divided into those who had or had not experienced loss of consciousness, established through patient self-report and collaborated by medical records. One group (n=31) had duration of LOC of at least 12 hours, and the other had either no LOC (n=9) or duration of LOC up to one hour (n=6). PTSD was assessed using the Posttraumatic Diagnostic Scale (PDS) and the PTSD section of the Structured Clinical Interview for DSM-IV (SCID). Those participants who reported intrusion symptoms were asked additional questions about the detail of their intrusive memories. Five participants (10.9%) were diagnosed as fulfilling PTSD criteria, only one of whom was in the 'unconscious' group. Ten participants from the 'conscious' group (66.7%) and 8 from the 'unconscious' group (25.7%) reported intrusions. The authors state that loss of consciousness may affect the form and frequency of intrusive symptoms, given that fewer participants from the 'unconscious' group reported intrusive memories and that they were less likely to re-experience physiological or emotional sensations or flashbacks.

Jones, Harvey and Brewin (2005) (Article 1 of 2 from one study) *(16 points, Grading A, level 2+)* report the symptom profiles of acute stress disorder (ASD) and PTSD in a consecutive sample of participants with (n=66) and without (n=65) mild or moderate TBI. ASD was assessed by the Acute Stress Disorder Interview (ASDI) as soon as possible following the event (mean time 5.98 days, SD 1.88). Additional questions were added to the ASDI to assess more thoroughly symptoms of dissociation during and since the trauma. PTSD was assessed by the interview version of the PTSD Symptom Scale (PSS) at around 6 weeks and 3 months following the event. The second assessment was completed with 118 participants (TBI n= 56, non-TBI n= 62), and the third with 119 (TBI n=58, non-TBI n= 65).

There was no significant difference in ASD rates between the TBI group (21.2%) and the non-TBI (20%) group, or in PTSD rates at 6 weeks (TBI 30.4%, non-TBI 27.4%) or 3 months (TBI 17.2%, non-TBI 18%). There was no significant difference between TBI and non-TBI participants with ASD in the number of dissociative symptoms reported. However amongst the participants without ASD, those with TBI reported more dissociative symptoms. There was no significant difference in the severity of reported symptoms at any time point. Participants in the TBI group reported significantly fewer re-experiencing symptoms at time 2 than those in the non-TBI group however there were no between group differences at time 3. Significantly fewer TBI participants reported feeling intense fear or helplessness during the trauma, or experiencing intrusive thoughts or images, but reported more emotional numbing, at time 1. At time 2 the TBI group reported fewer feelings of helplessness, reliving, physiological reactivity, hypervigilance, but more feelings of a foreshortened future. At time 3 there were no significant differences between the groups. The authors state that the TBI groups' lack of fear and helplessness, and fewer re-experiencing symptoms were likely due to lack of recollection of the event.

Limitations

The groups described in Glaesser et al. are not clearly defined. The 'no loss of consciousness' group also includes participants who had a TBI and loss of consciousness for up to an hour. The authors suggest that these participants were able to recall enough from the event to have 'sufficient islands of memory' of the event to have fully experienced it, however this was not assessed and assumes that brain injury itself does not alter the likelihood of developing PTSD. Additionally, the sample included participants who had experienced a traumatic injury to the cervical spine rather than TBI. It is not clear whether these participants also experienced loss of consciousness, further confusing group membership. Jones et al employed the PSS to assess for PTSD; a structured interview based on DSM-IV criteria. This assessment measure is comparable in validity to the CAPS in a non-TBI population (Foa and Tolin, 2001). It has not, however, been established as a valid tool with individuals with TBI therefore some confusion of TBI and PTSD symptoms may have occurred (Sumpter and McMillan, 2005, 2006). It is possible

that reported ‘dissociative’ symptoms (for example, feeling numb or distant from their emotions, or feeling distant from their normal selves) in those participants with TBI were symptoms relating to the outcome of their injury, which would explain the increased reporting of these symptoms in this group.

3.3.2 Content of Memories

Bryant and Harvey (1998) *(15 points, Grading A, level 2+)* investigated the nature of intrusive imagery in participants with PTSD and confirmed accurate recall of a traumatic event (motor vehicle accident) (n=12) compared with participants with PTSD and amnesia for the event due to TBI (n=6; 4 mild, 2 severe TBI), participants without PTSD (controls) (n=12), and participants who had not experienced trauma and did not have PTSD (simulators) (n=12).

The PTSD-I, IES and State Trait Anxiety Inventory were administered, along with the Vividness of Visual Imagery Questionnaire which established participants’ ability to imagine scenes. Participants were played an audio recording of a MVA and were asked to describe their experience of doing so. Results indicated that the PTSD, PTSD-TBI and simulator groups were rated higher than controls on vividness of imagery intrusiveness, poor control of imagery, affect and re-experiencing. Fewer participants in the PTSD-TBI group concentrated on intrusions than the PTSD group, and fewer of the PTSD group concentrated on intrusions than simulators or controls. Fewer PTSD-TBI participants stated their intrusive images included movement than PTSD participants, simulators or controls. Fewer simulators and controls saw the intrusions from their own perspective than the PTSD-TBI group or the PTSD group. The authors suggest that the PTSD-TBI group was able to experience intrusions due to non-verbal memories of the event being developed by the affective reactions they provide.

Harvey and Bryant (2001) *(14 points, Grading B, level 2+)* investigated memory for MVA 1 month and 2 years following the event in order to establish any alterations in recall. Seventy-nine participants were recruited consecutively following hospital admission due to MVA and mild TBI. Fifty were re-assessed 2 years post-injury. At first assessment participants were administered the ASDI and

asked to describe their recollections of the accident. At the second assessment the PTSD component of the Composite International Diagnostic Interview was administered with additional questions to establish severity. Memory for the accident was again assessed.

At first assessment, 14% (n=11) of participants were found to meet diagnostic criteria for ASD. PTSD was diagnosed in 22% (n=11) of the sample at the second assessment (8 of whom had met criteria for ASD). During the first assessment, all participants stated they had no recall of the accident. During the second assessment 30 participants stated they could not recall the accident, and 20 stated they could recall the accident fully. Of these 20, 4 met ASD criteria and 6 PTSD criteria. Participants who had recovered their memory had significantly shorter PTA, shorter duration of admission and lower injury severity score than those who continued to be amnesic. The authors considered that those participants who appeared to 'recover' memory of the accident may have retained islands of memory which developed over time, evidenced by the finding that they had shorter periods of PTA, or through information obtained through third party reports. Implicit encoding at the time of the accident may also have led to later development of explicit memory.

Jones, Harvey and Brewin (2007) (Article 2 of 2 from one study) *(17 points, Grading A, level 2+).* The transcribed scripts from the narratives described in the previous article were rated for disorganisation and dissociation according to a coding scheme. Sensory and emotional content was analysed with a computer package called 'Linguistic Inquiry and Word Count'. Analyses indicated that the narratives of those participants diagnosed with ASD at first assessment (mean 5.98 days, SD 1.88 days, post-trauma) were significantly less coherent ($p<0.001$) and showed more dissociation ($p<0.001$) in the narratives obtained at the first (~5.98 days post-trauma) and second assessments (6 weeks post-trauma) than participants without ASD at first assessment. Participants who had experienced a TBI presented with more confusion ($p<0.01$). At the third assessment the narratives of participants with PTSD were more repetitive ($p<0.01$), had more non-consecutive narratives ($p<0.001$), less coherence ($p<0.001$), more dissociation ($p<0.01$), and more sensory content ($p<0.001$) than those without PTSD. Participants with TBI presented with

more confusion ($p<0.01$). At all three time points a global coherence score was positively correlated with repetition and non-consecutive narrative. The finding that the TBI group exhibited more confusion in their narratives was considered to be consistent with the hypothesis that PTA results in disorientation and interrupted memory. The authors further suggest that confusion may be a particular type of disorganised memory, associated with TBI. They note that TBI was not associated with the content of narratives and suggest that this may be due to participants developing their memory by adding acquired information.

Limitations

As previously described, the assessment of PTSD may have been clouded by the use of tools which could allow for reporting of both TBI and PTSD symptoms, leading to higher rates of diagnosis and invalid severity scores. The samples reported in Harvey and Bryant, and in Jones et al., are described as having experienced a mild TBI however PTA is defined as less than 24 hours (mean 9.4 hours, SD 9.1 hours), which includes moderate TBI. The coded narratives in Jones et al. were compared across groups, which were unevenly sized, having many more participants without ASD or PTSD. Harvey and Bryant do not describe the method they used to assess recall for the event however it appears they did not establish whether participants had gained information about the event post-trauma, which could have been incorporated into their narrative. They acknowledge that asking an individual repeatedly to try to recall an event can lead to a belief in false memories (Roediger et al., 1997) which could have led to an apparent 'recovery' of memory.

3.3.3 Quantity of memory

Gil, Caspi, Ben-Ari, Koren and Klein (2005) *(15 points, Grading A, level 2+)* sought to establish the prevalence of PTSD in a sample of participants with 'good' and 'no' memory of the trauma which led to TBI and the pattern of symptoms associated with each group. Participants (n=120) were recruited from a medical centre following admission for mild TBI. They were assessed at four time points. Firstly within 24 hours of admission, then between 7-10 days, 4 weeks and 6

months post-injury. In the first assessment demographic information was gathered along with an injury severity rating, and the peri-traumatic dissociation questionnaire. In the next three assessments, PTSD was assessed using the Clinician Administered PTSD Scale (CAPS) and PSS, the Beck Depression and Anxiety Inventories were also administered, along with assessment of their memory of the event using a questionnaire designed for the study giving a rating of 1 (no memory) to 4 (good memory).

Results indicate that PTSD was significantly more prevalent in those participants categorised as having 'good' memory of the event (23%) than those with 'no' memory (6%) and that this finding was due to differences in reporting of re-experiencing symptoms. Acute posttraumatic symptoms, depression, and anxiety reported in the second assessment were associated with increased risk of PTSD in the fourth assessment. The authors concluded that these results provide evidence that having a memory for trauma increases the risk of developing PTSD and having no recall may be protective. They state that memory assessed at 24 hours post-injury may be a predictive factor.

Turnbull, Campbell and Swann (2001) *(15 points, Grading A, level 2-)* investigated whether amnesia for trauma following mild to severe TBI related to PTSD development or symptom profile. Fifty-three participants were recruited following admission to a hospital A&E department. They were posted the following questionnaires to complete; IES-R, HADS, a questionnaire designed for the study about their memory of the event and a questionnaire designed to establish the impact of physical injuries. Participants who scored over 20 on either subscales of the IES-R were administered the CAPS by telephone interview.

Memory for the event was categorised as 'no memory', 'untraumatic memory' or 'traumatic memory'. The 'no memory' and 'traumatic memory' groups had significantly higher avoidance and intrusion scores on the IES-R ($p<0.001$) than the 'untraumatic memory' group. These 2 groups also had higher levels of anxiety and depression. Higher levels of physical injury were related to higher levels of avoidance ($p<0.01$), intrusions ($p<0.01$), anxiety ($p<0.01$) and depression ($p<0.01$). PTSD was diagnosed in 17% of the sample using stringent criteria and 27% using

lenient criteria for CAPS rating. Severity scores were lower in the 'no memory' group, however by applying 'lenient' criteria (lower frequency and intensity scores considered symptomatic) 6 participants with traumatic memory and 5 with no memory reached PTSD caseness. Those with no memory reported fewer intrusions, but reported psychological and physiological distress to cues more often. The authors note the presence of 'pseudomemories' in 2 participants with no memory and suggest this as a possible mechanism for intrusions. They conclude that while amnesia for trauma does not prevent the development of PTSD, it was associated with lower severity in this sample and fewer intrusions.

Limitations

Gil et al. rely on GCS as a measure of TBI severity and do not take account of PTA. The categorisation of memory into 'good' and 'no' did not account for all participants and a continuous scale may have been more informative. Additionally they note that their assessment of memory established participants' confidence in their memory rather than the quantity of recall. Therefore results indicate participants who were unsure that their memory of the event was correct were less likely to develop PTSD – this does not necessarily equate to having 'no' memory for the event. Turnbull et al. grouped participants according to both quantity of memory and emotional reaction to memory. Therefore the impact of quantity of recall alone on severity scores was not established. There was a low response rate (15%) from potential participants to Turnbull et al., suggesting the sample may not represent the wider population. The authors indicate the low response rate could be due to the trauma sustained by the majority of participants (assault), as similar response rates have previously been reported with such a population. Symptoms reported using the IES-R and included in the between groups analyses cannot be assumed to relate to PTSD (Sumpter and McMillan, 2005). Additionally not all participants were assessed by interviewers for PTSD using the CAPS, the more robust measure, and those that were assessed using the CAPS were interviewed by telephone, which has not been proven to be a reliable assessment method (Blake et al., 1995).

4 Discussion

The studies reviewed consistently report the presence of PTSD symptoms following TBI, although some limitations in assessment have been identified. While it has been argued that PTSD cannot develop following TBI (Sbordone and Liter, 1995) it is clear that some individuals do present with symptoms of posttraumatic stress, even without memory for the event. The focus of this review was to establish current evidence for possible mechanisms of PTSD symptom development after TBI. All papers reviewed received SIGN level of evidence 2+ or 2- and are considered to fall under SIGN grade of recommendation D, which is the lowest grade of recommendation. Therefore the current quality of evidence is low and results must be interpreted as preliminary and investigative at this stage. The systematic literature search identified studies in three broad areas. The evidence within each area will be discussed separately. The limitations of the evidence base will be described. Finally, recommendations for future research will be made.

4.1 Evidence for Causal Mechanisms

4.1.1 Subconscious or neurological processes

The current evidence suggests a possible role for fear conditioning in the development of PTSD symptoms. Emotional and physiological reactivity symptoms were reported more often than intrusive memories, a pattern which could be interpreted as evidence of fear conditioning. However, methods of assessment of PTSD rely on participant reporting of symptoms, which may reflect the impact of TBI rather than trauma. Symptom profile cannot be considered as evidence for particular mechanisms of PTSD development unless valid assessment tools are employed. High levels of stress during trauma could result in over-consolidation of trauma memories and the development of PTSD symptoms. However the biological marker of stress reported (Sojka et al., 2006) may represent a measure of brain damage associated with TBI rather than hyper-arousal during trauma. Comparison of heart rate across the sample post-trauma as an indicator of arousal (Bryant et al., 2004) could reflect individual differences rather than fear conditioning. Higher arousal level after trauma has been hypothesised to indicate

risk of subsequent PTSD in non-TBI samples (van der Kolk, 1994) and is an important area of investigation with TBI samples. The presence of high arousal levels would indicate a role for fear conditioning however the current evidence cannot support this hypothesis. A further complication in the investigation of neurological processes is the interaction between brain damage from TBI and the brain damage which is thought to occur as a result of prolonged increases in stress hormones during traumatic events (Markowitsch, 1998). Participants with more severe brain damage, as assessed by GCS, were found to be less likely to have increased heart rate following trauma. This finding could indicate a negative relationship between fear conditioning and severe TBI which might explain the lower TBI prevalence rates reported following severe TBI (Sumpter and McMillan, 2005).

4.1.2 Predictive Factors

Factors which predict the development of PTSD in non-TBI samples could be vital in understanding the presentation of posttraumatic stress after TBI. Holding an external attribution of causality for the event has been linked with increased risk for PTSD without TBI (Delahanty et al., 1997). An important aspect in PTSD is the alteration of the individual's world view to one in which they feel unsafe and believe that events cannot be predicted, a view which could be developed from holding an external attribution of causality. Whether such an attribution and alteration of world view could exist if the trauma is not consciously experienced or recalled is an interesting area of investigation. Williams et al. (2002) reported that attribution for external causality was found to predict PTSD symptom severity. This finding would support a similar presentation of posttraumatic stress after TBI as after other trauma types however the results relied on an assessment measure which could have resulted in invalid reporting of symptoms. Therefore the current evidence cannot confidently support this hypothesis. Both studies reporting on predictive factors (Bryant et al., 2000b; Williams et al., 2002,) found an avoidant coping style to be associated with higher severity scores, suggesting individual risk factors to be relevant. Moderate to severe dysexecutive disorder resulting from TBI may reduce the ability to report symptoms or reduce insight into the impact of symptoms. However, methods of assessment of PTSD could have lead to reporting

of TBI symptoms. The association found between lower PTSD severity scores and dysexecutive disorder could indicate a lack of insight into the impact of TBI on functioning.

4.1.3 Memory for Event

Fewer or less severe re-experiencing symptoms were reported by participants with TBI than by those without TBI. Jones et al. (2005) reported that this pattern changed over time and by 3 months post-trauma the TBI group were reporting similar levels of re-experiencing symptoms as the non-TBI group. This is an interesting finding and suggests these symptoms had developed over time. It is possible that memories were confabulated by participants or that implicit memories of the trauma were expanded upon, and although lack of memory for the event may lead in the short term to a particular pattern of symptom presentation this may not remain the case. This would be an important area for future research to investigate more thoroughly. The trauma narratives of participants with TBI include confusion, which may be due to PTA. Narratives included less movement than non-TBI participants which could indicate a less dynamic memory for the trauma, perhaps resulting from a lack of conscious memory of the event. Intrusive images developed after TBI may therefore be qualitatively different from those described by non-TBI populations and these differences could indicate the role of confabulation in the development of re-experiencing symptoms. It would be important for future research to establish whether such differences in intrusions alter their emotional impact as this issue appears key to the development of PTSD after TBI. Some participants who were initially amnesic for the event reported full memory 2 years post-injury however this ‘recovery’ could be due to repeated questioning or to confabulation of memory incorporating information gained post-injury. This finding could provide further support for the hypothesis that re-experiencing symptoms can develop over time and that confabulation may contribute. Participants with non-traumatic memory were reported to be less likely to develop PTSD than those with no memory or traumatic memory (Turnbull et al., 2001) and those with no memory reported less severe symptoms. Gil et al. (2005) reported that participants with good memory were more likely to develop PTSD than those with no memory. These results suggest amnesia may be protective

against PTSD or severity of symptoms experienced. However assessment of memory of the event in one study (Gil et al.) established confidence in recall rather than quantity of recall and in the second study (Turnbull et al.) described the affect ascribed to memory as well as quantity. Results are therefore confused by the inclusion of a number of factors and the impact of quantity of memory alone cannot be concluded.

4.2 Recommendations for Future Research

It is clear that more research is needed in this field to more firmly and reliably establish the causal mechanisms underlying PTSD after TBI. The assessment of PTSD after TBI has been shown to be complicated by overlapping symptoms and thorough assessment is required to reliably obtain measures of PTSD symptom presentation and severity. Future research should rely on measures of PTSD which allow for the judgement of an experienced clinician (such as the CAPS) to ensure that reported symptoms pertain to trauma rather than TBI. The quality of future research would be improved by controlled group designs and larger sample sizes.

5 Conclusions

- The evidence indicates fear conditioning may be involved in the development of PTSD following TBI, however methodological limitations confuse outcomes.

- Vulnerability factors which are known to predict PTSD in non-TBI populations (such as external attribution for event causality) may also be relevant to TBI populations however research is in its early stages. Factors particularly related to TBI outcome, such as reduced executive functioning, may also be relevant.

- Individuals who have experienced TBI seem to initially report re-experiencing symptoms less often however symptoms may emerge over time. Apparent recovery of memory over time may be due to confabulation.

The trauma narratives of individuals with memory for the event and TBI may include more confusion and fewer dynamic images than non-TBI participants. Lack of memory for the event may protect against PTSD however methodological limitations in assessment of recall exist.

- Current research into the causal mechanisms of PTSD after TBI is limited by the use of assessment measures of PTSD which lead to mis-diagnosis, therefore results must be interpreted cautiously.

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Figure 1: Article Selection Process

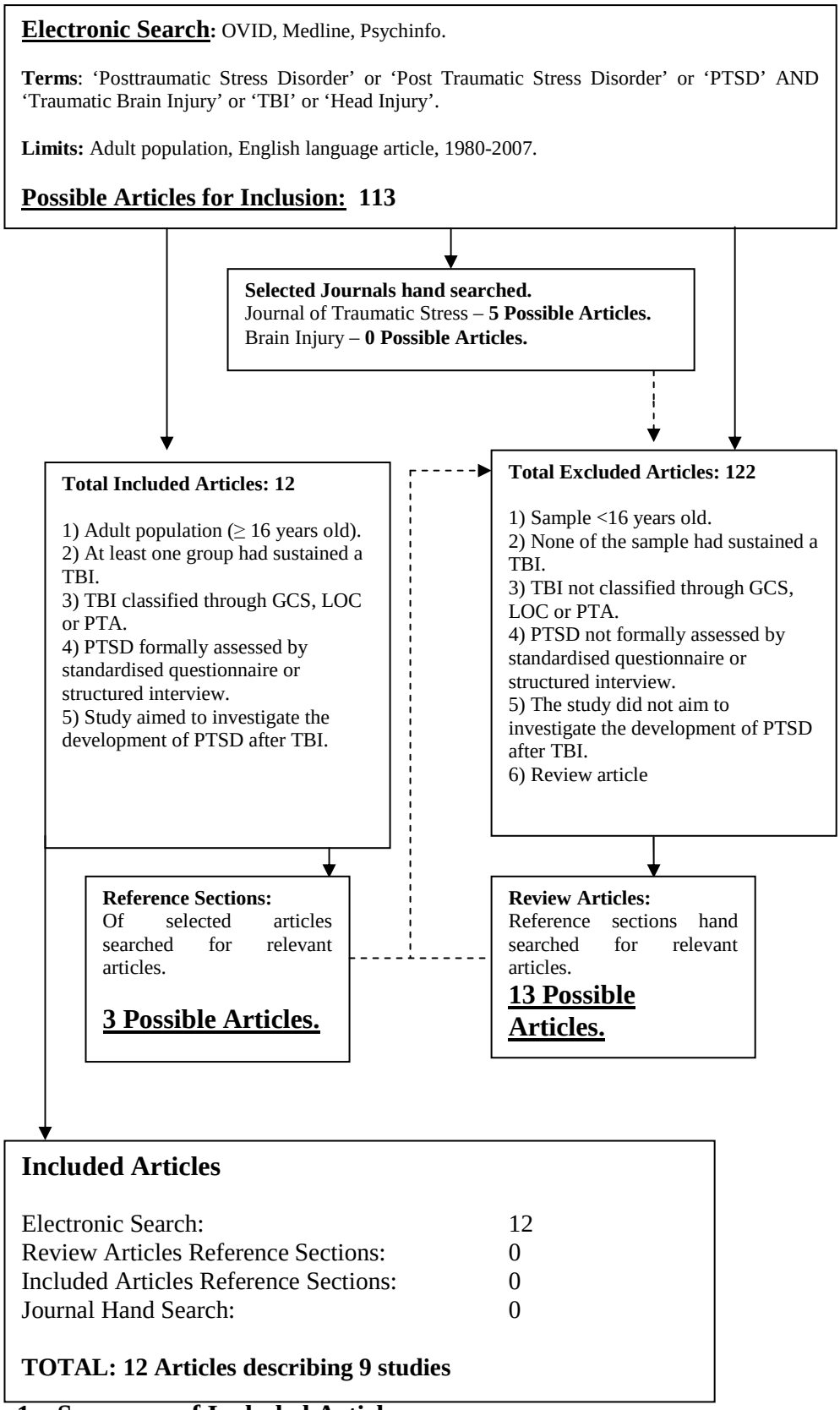

Table 1 – Summary of Included Articles

	Quality Rating (points/grade)	SIGN level of evidence	PTSD Assessment Method	TBI Assessment Method	Sample Size	Summary of findings
1)	**Bryant, Marosszeky et al**					
2004	16/A	2+	PTSD-I	Westmead PTA Scale	n=68	PTSD linked with higher resting heart rate ~9 days post trauma. Fear conditioning may mediate.
2000a	14/B	2+	PTSD-I	Westmead PTA Scale	n=96	Intrusive memories, nightmares and emotional reactivity strongest predictor of PTSD. High numbers reporting emotional and physiological reactivity indicates fear conditioning.
2000b	16/A	2+	PTSD-I	Westmead PTA Scale	n=96	Higher PTSD severity associated with avoidant and emotion focused coping style. Severe TBI may lead to reduced coping ability.
2)	**Sojka et al**					
	10/D	2-	IES	LOC/GCS	n=88	S-100B (biological marker of brain damage) ~7 hours post-injury associated with 3 avoidance symptoms. May indicate role of brain injury in PTSD.
3)	**Williams et al**					
	12/C	2-	IES	PTA	n=66	PTSD severity positively correlated with attribution for external causality for event. Insight negatively correlated with PTSD severity. Lack of insight may protect against PTSD.
4)	**Glaesser et al**					
	12/C	2-	SCID IES	LOC	n=46	LOC may affect form/frequency of intrusive symptoms. No LOC associated with fewer intrusions or re-experiencing symptoms.
5)	**Jones et al**					
2005	16/A	2+	PSS	PTA	n=131	TBI (no ASD) led to more reporting of dissociative symptoms. TBI associated with fewer re-experiencing symptoms.

	Quality Rating (points/grade)	SIGN level of evidence	PTSD Assessment Method	TBI Assessment Method	Sample Size	Summary of findings
5) Jones et al cntd.						
2007	17/A	2+	PSS	PTA	n=131	Those with ASD showed more dissociation and less coherence in trauma narrative. TBI associated with more confusion.
6)	**Bryant and Harvey**					
	15/A	2+	PTSD-I IES	LOC	n=42	Fewer TBI+PTSD focused on intrusions in narrative trauma than PTSD simulators or controls. Fewer PTSD+TBI included movement.
7)	**Harvey and Bryant**					
	14/B	2+	CIDI	PTA	n=79	t1 – no TBI recalled trauma. t2 – 20 recalled trauma; had shorter PTA than amnesics. Possible retention of some memory.
8)	**Gil et al**					
	15/A	2+	PSS CAPS	GCS	n=120	PTSD more prevalent in those with 'good' memory than 'no' memory. Memory for trauma thought to increase risk of PTSD.
9)	**Turnbull et al**					
	15/A	2-	IES-R CAPS	PTA	n=53	No memory/untraumatic memory associated with higher avoidance and intrusion scores. No memory associated with more frequent psychological and physiological distress. Amnesia led to less severe PTSD scores.

Chapter 3

Major Research Project Proposal

The Role of Memory for Trauma in the Development of Post-Traumatic Stress Disorder following Traumatic Brain Injury

Written according to course handbook guidelines for submission
(See Appendix 3.1)

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Summary of Project

It is becoming more accepted that post traumatic stress disorder (PTSD) can occur following traumatic brain injury (TBI). However, the mechanism through which PTSD can develop is not yet fully understood. TBI often involves a period of amnesia for the traumatic event, which has been thought to protect against the necessary re-experiencing symptoms of PTSD. If this were the case it would be assumed that those people with less memory for the event would not develop PTSD. To date two studies have investigated this link. Turnbull et al (2001) reported PTSD to be significantly more common in people with traumatic memories and no memories (of the event that led to their TBI) than those with non-traumatic memory. Gil et al (2005) found that people with 'good' memory were more likely to develop PTSD than those with no memory. This study expands on their findings by utilising a more detailed, structured measure of traumatic memory. This study will also measure physiological reactivity during recall of the traumatic event. It has been suggested that implicit memories of a traumatic event contain more sensory and physiological detail (Brewin et al, 1996). Changes in physiological reactivity may indicate that implicit memory for the event is present even when consciousness is impaired during TBI. It is hoped the results of this study will provide further evidence for the mechanism through which PTSD can occur following TBI.

1 Introduction

The occurrence of brain injury has been estimated from 92 per 100,000 (Thurman and Guerrero, 1999) to 618 per 100,000 (Sosin, Sniezek and Thurman, 1996) with differences in definitions and populations contributing to the variance. Using the Glasgow Coma Scale (Jennett and Teasdale, 1981) to classify severity of traumatic brain injury (TBI) Thornhill, Teasdale, Murray et al (2000) found, of a cohort admitted to hospitals in Glasgow, 90% of admissions were for mild TBI, 5% for moderate TBI and 3% for severe TBI. Common effects of TBI include fatigue, irritability, dizziness, poor concentration, and headaches (Bohnen and Jolles, 1992). Posttraumatic amnesia (PTA) frequently occurs. Mild TBI is associated with PTA for less than 24 hours, moderate to severe TBI with PTA for more than 24 hours (American Congress of Rehabilitation Medicine, 1993). PTA can include retrograde amnesia for events prior to the injury and recovery from PTA is often characterised by islands of memory for events after the injury before anterograde memory is completely restored.

Psychiatric disorders such as depression and anxiety have also been reported in TBI patients (Hibbard et al, 1998). However, the question of how commonly post-traumatic stress disorder (PTSD) occurs after TBI has yet to be conclusively answered. PTSD is categorised in DSM-IV (American Psychiatric Association, 1994) as an anxiety disorder. Criteria for diagnosis of PTSD are categorised in 4 areas. 1) Exposure to an event that is a threat to one's life or integrity. 2) Re-experiencing symptoms such as intrusive memories, re-living the trauma (flashbacks) and distress when reminded of the trauma. 3) Avoidance of thoughts, feelings or reminders of the trauma, inability to recall an important part of the trauma, social withdrawal, or emotional numbing. 4) Increased arousal as evidenced by insomnia, irritability, poor concentration, hypervigilance, or heightened startle response. It has been suggested that if memory for the traumatic event was not encoded due to loss of consciousness and subsequent amnesia during TBI then re-experiencing symptoms relying on recall of the event cannot be present. Therefore PTSD cannot be diagnosed (Sbordone, 1992). Indeed, some studies have found no PTSD following a traumatic event involving TBI (eg Sbordone and Liter, 1995).

However, there is increasing evidence that PTSD is present in TBI populations. Mayou, Black and Bryant (2000) compared the prevalence of PTSD (assessed using the PSS) in road traffic accident victims in a group with mild TBI and definite loss of consciousness, a second group with mild TBI and probable loss of consciousness, and a third group with no TBI. Three months after the trauma 48% of the definite group, 23% of the probable group and 23% of the non-TBI had PTSD. One year after the trauma 33% of the definite, 14% of the probable and 17% of the non-TBI group had PTSD. These results suggest loss of consciousness may increase vulnerability for subsequent PTSD, contrary to earlier views. Bryant and Harvey (1999) report an incidence of 25% after mild TBI (assessed with the CIDI); Hickling et al (1998) found 36% of a group with mild TBI to have PTSD using the SCID (Structured Clinical Interview for DSM-IV Diagnosis); Hibbard et al (1998) report 19% of a group with severe TBI to have PTSD on the CAPS.

Reported incidence rates are varied and it may be that methodology for diagnosing PTSD influences outcome. Sumpter and McMillan (2005) and Sumpter and McMillan (2006) found that using the Clinician Administered PTSD Scale (CAPS: Blake, Weathers, Nagy et al, 1995), an interview based measure requiring clinical judgement, only 3% of TBI patients fulfilled DSM-IV symptom criteria for PTSD whereas 59% were identified using the Post-traumatic Diagnostic Scale (PDS: Foa, Cashman, Laycox et al, 1997), a self-report measure. It was hypothesised that part of this large discrepancy could be due to participants identifying symptoms relevant to their brain injury rather than to PTSD. For example, reported intrusive thoughts about the trauma were often found to be due to a desire to recall more information because of amnesia for the event, rather than a desire to avoid thinking about the memory. However, other studies also rating PTSD caseness using the CAPS have reported a higher percentage of patients with TBI to have PTSD, including Bryant and Harvey and Hickling et al. Additionally, Gil et al (2005) reported 14% of patients with TBI to have PTSD, as diagnosed by the CAPS. It is as yet unclear why such discrepancies are found. A possibility is that people with TBI experience PTSD symptoms that do not reach diagnostic criteria for PTSD, but may reflect PTSD in a less severe form. Hence there may be variability in the incidence found between studies due to relatively small numbers of cases falling

one side or the other of ‘caseness’ criteria for symptoms. This is an area that deserves further investigation.

Some people who experience a mild TBI lose consciousness for only a few minutes and the duration of post traumatic amnesia is also short, hence they will have a relatively intact memory for the trauma. Those who recall only brief snatches of memory for the trauma may still have encoded the memory along with associated horror or fear responses. This memory can become intrusive (McMillan, 1996). Additionally, amnesic gaps in memory for the trauma can be filled in by information imagined or obtained after the event and this ‘confabulated’ memory can also form the basis for intrusions (Bryant, 1996). It seems important to investigate the link between memory for the traumatic event and subsequent PTSD to establish whether amnesia for the event is protective. Gil et al (2005) investigated whether the quality of memory reported for the traumatic event was related to diagnosis of PTSD. PTSD was present in 23% of participants with ‘good’ memory for the event compared to 6% with no memory for the event, suggesting that having no memory is somewhat protective. However, memory was assessed with a simple self-report scale devised for the study with participants being asked to rate on a 4-point scale how good their memory was for 9 separate items. They were then categorised as either having ‘no memory’ or ‘good memory’. It is unclear how valid a representation this is of their narrative memory for the event and also whether their memory had been influenced by information acquired after the event, which could have led to a mixture of memory for the traumatic event, knowledge from the report of others and confabulation.

Turnbull, Campbell and Swann (2001) reported that of 55 participants who had experienced TBI and loss of consciousness, those with no memories or traumatic memories of the event were significantly more likely to be diagnosed with PTSD on the Impact of Events Scale – Revised (IES) than those with non-traumatic memories. Memory was assessed with a questionnaire developed for the study to categorise participants into these three groups. This finding is in contrast to Gil et al and further suggests that having less memory for the event is not protective against the development of PTSD. Findings did suggest having no actual memory for the event was related to less severe intrusion symptoms. This is an area of

research that must be further investigated before the association between memory for trauma and PTSD caseness can be fully understood.

It has been theorised that trauma memories can be encoded and retrieved implicitly. Dual Representation Theory (Brewin, Dalgleish and Joseph, 1996) describes how traumatic memories may be stored as verbally accessible memories (VAMs) or situationally accessible memories (SAMs). VAMs are described as verbal or visual and can be intentionally recalled. SAMs comprise of subconscious memory that includes sensory information and are recalled unintentionally in the form of flashbacks. In this way, while impaired consciousness during TBI may lead to poor declarative memory for the traumatic event, implicit encoding may still occur.

Brewin additionally proposes that SAMs are mediated via the amygdala (while VAMs are processed by the hippocampus). This theory links cognitive processes to biological theories that suggest fear conditioning to a traumatic event occurs in limbic structures. It is suggested that heightened physiological arousal to a traumatic event leads to an association between an anxiety response and reminders of the trauma (Kolb, 1987). There is support for this theory from studies that have found increased physiological arousal in people with PTSD (Bryant et al, 2003) and in response to trauma related cues in people with PTSD (Orr and Kaloupek, 1997). Bryant, Harvey, Guthrie and Moulds (2003) found that re-experiencing symptoms reported following TBI consisted of physiological distress or physiological reactivity when reminded of the trauma.

Hellawell and Brewin (2002) have also found autonomic and motor behaviour to increase during recall of flashback memory compared to non-flashback memory of the trauma in a sample of participants with PTSD. They propose that dynamic movement or stasis during recall may indicate a specific response to a particular part of the memory. Such a response is more likely to occur during flashback memory as it holds more sensory information. They further propose that physiological changes are only likely to occur during particular points of recall, experienced as flashbacks. There has been no research as yet into changes in physiological responsivity in patients with TBI and PTSD during recall of their trauma. Such research would provide additional evidence as to whether the

processes indicated during flashback memory in other populations with PTSD are also present after TBI. In turn the amount of responsivity and its relation to degree of PTSD caseness will provide information about the mechanism through which people without full memory for their trauma could develop PTSD and particularly re-experiencing symptoms. Sumpter and McMillan (2006) report that, on the PDS, 42% of participants experienced intrusive memories, and 30% experienced nightmares. It will be interesting to establish whether physiological change occurs during recall of both flashbacks and nightmares. Holmes et al (Holmes, Brewin and Hennesyt, 2004) described 'hot spots' in trauma memories which are suggested as likely to be intrusive and to be accompanied by intense emotion. Holmes found that parts of a trauma film later rated as intrusive were associated with brief episodes of decreased heart rate. This finding was related to the occurrence of 'hotspots' and it is possible that brief decreases in heart rate are found in this sample.

2 Aims and Hypotheses

Aims

1) To investigate the relationship between memory of the traumatic event and PTSD caseness in people with TBI.
2) To establish whether any changes in physiological arousal (motor or heart rate) are associated with recall of the traumatic event and PTSD symptom severity or caseness in people with TBI.
3) To repeat the study by Sumpter and McMillan comparing PTSD caseness using questionnaire and structured interview methods.

Hypotheses

1) PTSD caseness or symptom severity, as assessed by the PDS and the CAPS, will be associated with higher scores on the TMI.
2) Physiological arousal, indicated by changes in heart rate and motor activity, will occur whilst participants are recalling memories previously experienced as flashbacks or nightmares during administration of the TMI.
3) PTSD caseness or symptom severity will be associated with greater physiological arousal.

4) About 60% of cases will fulfil DSM-IV criteria for criteria using the PDS and less than 10% will fulfil PTSD criteria using the CAPS.

3 Plan of Investigation

3.1 Participants

All participants will be aged over 16 years and will have experienced a TBI at least 3 months previously (DSM-IV criteria for PTSD define this time scale from the trauma for diagnostic purposes). A range of severity of TBI from mild to severe will be included in the study to ensure a range of post traumatic amnesia and therefore memory for the trauma, which will provide a more informative picture of association between memory and PTSD. Severity will be informed from records at the recruitment centres (see *Recruitment*) and using the following criteria. Mild TBI will be considered if loss of consciousness was for less than 30 minutes, post-traumatic amnesia less than 24 hours (American Congress of Rehabilitation Medicine, 1993), and Glasgow Coma Score between 13-15. Severe TBI will be considered if loss of consciousness was for at least 6 hours, post-traumatic amnesia was for at least 24 hours (McMillan and Greenwood, 2003) and Glasgow Coma Score of 8 or less. A moderate TBI will be assumed if loss of consciousness is between 30minutes and 6 hours and Glasgow Coma Score is between 8 and 13. Participants who are receiving treatment for a psychiatric disorder will be considered for inclusion on a case by case basis. If it is considered that their treatment will impact on their responses they will not be included.

3.2 Recruitment

Participants will be recruited from several sources – the Community Treatment Centre, Glasgow; Headway, Glasgow; the Glasgow Royal Infirmary; and Professor McMillan's outpatient clinic at the Southern General. Clinicians at the above centres will be asked to identify potential participants from their caseloads using the recruitment criteria previously outlined. Clinicians will be asked to hand over to identified participants an envelope which will contain an information sheet, letter to participants and consent form. Clinicians will indicate that the envelope contains details about a research study being carried out at the centre, about which they are informing a number of patients. Clinicians will otherwise offer no encouragement to patients to take part and will only give further information about the study if asked. The letter and information sheet outline the recruitment process to participants, who will sign and return the consent form in a pre-paid envelope to the researcher if they decide to take part. They will indicate a telephone number on the consent form by which the researcher can contact them. The researcher will contact those participants who return the form to arrange an appointment to complete the assessment measures.

3.3 Measures

PTSD Caseness

The Post-traumatic Diagnostic Scale (PDS: Foa, Cashman, Laycox et al, 1995). A self-report questionnaire based on DSM-IV criteria.

The Clinician Administered PTSD Scale (CAPS: Blake, Weathers, Nagy et al, 1995) will also be administered due to the discrepancy found in previous studies between self-report and clinician administered measurements. This is a structured clinical interview also assessing DSM-IV symptoms.

Depression and Anxiety

The Hospital Anxiety and Depression Scale (HADS: Zigmond and Snaith, 1983) is a self-report questionnaire that assesses symptoms of anxiety and depression.

Disability following TBI

The Glasgow Outcome Scale-Extended (GOS-E: Wilson, Pettigrew and Teasdale 1998) is a clinician rated scale and assesses social and functional disability after TBI.

Pre-morbid IQ

The Weschler Test of Adult Reading (WTAR: Weschler, 2001) will assess estimated IQ prior to TBI.

Learning and memory

The Rey Auditory-Verbal Learning Test (AVLT: Vakil and Blachstein, 1997) will assess memory and learning so that any variability in ability can be included in data analysis.

Memory for Traumatic Event

The Traumatic Memory Inventory (TMI: van der Kolk and Fisler, 1995) is a structured interview that assesses sensory, affective and narrative memory for the event. It covers 6 main areas. 1) Background of the event and contextual information, such as nature and duration of the trauma, whether the person had always remembered all of it and if not when they became aware. 2) Sensory memories of the trauma such as images, sounds, emotions, tactile or bodily sensations, smells. 3) Whether the memory is recalled as a coherent narrative. 4) The nature of nightmares. 5) The nature of flashbacks. 6) Precipitants of flashbacks and nightmares. 7) The way intrusive recollections are dealt with. It is expected that the range of questions described above will cover the issue of confabulation (as it specifically asks whether total recall was always present) and also will tap into flashback type memories.

Physiological Reactivity

Heart rate will be monitored using a Polar heart rate monitor (S610i, www.heartratemonitor.co.uk/polar_s610i_uk.html). This will be purchased for the study. The watch records heart rate at 5 second intervals and stores these data for later use. Data can be downloaded to a PC using the software provided. Motor movement will be measured using an Actiwatch Plus (Cambridge

Neurotechnology, www.camtech.com) placed on the participant's non-dominant hand. This will also store information about movement across a particular time period for later use.

Demographic Information

Each participant's age and gender will be recorded. They will also be asked whether they have previously experienced a TBI or any psychiatric condition for which they received treatment. Previous trauma will not be assessed, as it is important participants only concentrate on the event in which their most recent TBI occurred.

3.4 Design and Procedures

Procedure

Participants will be seen individually in a private room. Initially the researcher will talk through the procedure of the study with them and collect demographic details. The WTAR and AVLT will be administered first so that fatigue factors do not affect their performance. The HADS and GOS-E will then be administered, followed by the PDS and the CAPS. Participants will be offered breaks between these measures. The last measure will be the TMI. It will be necessary for participants to put on a chest strap for the Polar watch. They will have the opportunity to do this either in private or with assistance from the researcher. They will also put on the Actiwatch. The watches will be started in synchrony and then a 5 minute rest period given to achieve a baseline measure of heart rate and activity levels. After 5 minutes the TMI will be started, along with a digital watch with which the researcher will note the time alongside each question. This will allow data from the watches to be linked to particular points in the TMI. After removing the watches, the participant will receive a short de-briefing to assess their mood and conclude the appointment.

It is estimated that administration will take around 2 hours. Data will be stored securely in a locked filing cabinet at the Section of Psychological Medicine, Gartnavel Royal Hospital. Participants will be allocated a randomised number and

this number will identify their data. The list of participants and allocated numbers will be kept separately in a secure location.

3.5 Data Analysis

1) PTSD caseness or symptom severity, as assessed by the PDS and the CAPS, will be associated with higher scores on the TMI. A correlational analysis will establish the relationship between symptom severity and degree of traumatic memory. Additionally a between groups comparison will be carried out using a t-test to compare TMI scores with participants who reach caseness on the PDS and those who do not.

2) Physiological arousal, indicated by changes in heart rate and motor activity, will occur whilst participants are recalling memories previously experienced as flashbacks. Independent t-tests will assess mean heart rate change during 're-experiencing' memory (flashbacks or nightmares) compared to 'non re-experiencing' memory and also mean activity level change during flashback memory compared to non flashback memory.

3) PTSD caseness will be associated with greater physiological arousal. Analysis of variance will compare PTSD severity scores with mean heart rate change from baseline and mean activity level change from baseline.

4) About 60% of cases will fulfil PTSD criteria using the PDS and less than 10% will fulfil PTSD criteria using the CAPS. This data will be categorical and will be analysed by chi-square.

3.6 Settings and Equipment

As previously stated, a base will be established for data collection at the Sackler Centre, Southern General Hospital, Glasgow. It will be necessary to purchase the Polar heart rate monitor which will cost £169.00, including software. The Actiwatch required for the study can be borrowed from the Section of Psychological Medicine, University of Glasgow.

3.7 Power Calculation

Power was calculated for the primary hypothesis – that PTSD symptom severity, as measured by the PDS, will correlate with scores on the TMI, with higher severity scores being associated with higher TMI scores.

To establish the necessary sample to size to test this hypothesis a power calculation was performed using data from Turnbull et al (2001). This study was chosen rather than Gil et al (2005) because the latter study does not report the means and standard deviations of their participant's scores. The data used from Turnbull were the scores of avoidance and intrusion severity measured by the IES, from the non-traumatic memory group and the traumatic memory group. The third group from the study, no memory, was not included in the calculation. It was considered that the comparison between the degree of trauma in memory was more relevant to the current hypothesis. The calculation, with alpha=0.05 and power=0.8, indicated sample sizes from the avoidance data of 5 (non-traumatic memory) and 7 (traumatic memory). From the intrusion data indicated sample sizes were 4 and 6 respectively. However, the current study will use a correlational design to test this hypothesis. Cohen's sample size tables (Cohen, 1988) indicate that with alpha=0.05, power=0.80 and r=.50, the sample needed to detect an effect will be 22. The study will therefore aim to obtain a sample size of at least 22 which would give an expected 13 PTSD 'cases' assessed by the PDS and 9 non-cases.

3.8 Pilot Study

The measures and procedure will be piloted on a control participant, without TBI, recruited from the University of Glasgow.

4 Ethics

The study will seek ethical approval from Greater Glasgow NHS Mental Health Division Ethics Committee. The following issues are noted.

It is possible that some participants will have difficulty with the Polar watch chest strap due to physical disability. This will be assessed case by case and if it will cause any discomfort or pain, the participant will be excluded from the study. However it is anticipated that most people will not have continuing physical disability as persisting physical disability is rare after even severe TBI (McMillan and Greenwood, 2002).

There is the potential for participants to become distressed from re-visiting the trauma that led to their TBI, either during assessment or after leaving. The de-brief at the end of testing will allow opportunity for the researcher to assess their mood. If a participant indicates distress they will be advised to contact either their clinician at the relevant centre or their GP. It is considered unlikely that participants will become acutely distressed as it is not anticipated many will meet diagnostic criteria for PTSD (Sumpter and McMillan, 2005) and so any symptoms experienced will be relatively mild. Additionally, previous studies have included participants who do meet criteria for PTSD and expose them to traumatic memories with no persisting difficulties (eg Clohessy and Ehlers, 1999).

It will be necessary to ensure that participants can understand the informed consent form, as cognitive ability can be compromised following TBI. This will be done by asking participants prior to starting testing to explain their understanding of the consent form to the researcher. Any participant who does not understand the form will be excluded. Most of the TBI population live independently (McMillan and Greenwood, 2002) and so it is anticipated the majority will be able to give informed consent.

There are no major safety concerns with this study. Participants will be seen in a staffed facility and the researcher will ensure a member of staff is aware of their appointment times with participants.

5 Practical Applications

The study will provide further evidence for the incidence of PTSD following TBI. It will expand existing knowledge regarding the link between memory for a traumatic event and subsequent PTSD and particularly the importance of this link following TBI. The study will also provide evidence of physiological reactivity in people with PTSD following TBI and whether this follows a similar pattern to people with PTSD after non-TBI events. The above will help to inform triage regarding appropriate treatment for people who have experienced a TBI.

6 Timescale

2006

March-April	Finalise Proposal
May	Ethical Approval sought
June	Amendments to proposal in accordance with Ethics Committee recommendations.
July	Records at the identified centres will be examined to identify potential participants according to inclusion criteria.
August	Initial letters will be sent to participants inviting them to take part in the study.
September	Contact will be established with participants and appointments arranged for data collection. As the study will depend on participants agreeing to take part and attending appointments it is assumed that participant identification and contact will be ongoing.
October-March 2007	Data Collection.

2007

March-April	Data Analysis.
May-June	Drafts written and amended.
July	Finalise report and submit.

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Chapter 4

Major Research Project

The Role of Memory for Trauma in the Development of Post-Traumatic Stress Disorder following Traumatic Brain Injury

Prepared in accordance with requirements for submission to
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Abstract

Posttraumatic stress disorder (PTSD) has been reported following traumatic brain injury (TBI), even when TBI leads to amnesia for the traumatic event. This study aimed to investigate the relationship between memory for the event (as assessed by the Traumatic Memory Inventory) and reporting of PTSD symptoms in a sample of adults with mild-severe TBI (n=21). Physiological reactivity (heart rate and activity level) was recorded in order to investigate the possible role of sub-conscious processes (such as implicit memory or fear conditioning) in the development of PTSD after TBI. PTSD symptoms were assessed by a self-report questionnaire (Post-traumatic Diagnostic Scale; PDS) and the Clinician Administered PTSD Scale so as to compare previously reported diagnostic rates established with these measures (Sumpter and McMillan, 2005). Higher PTSD severity scores were not, as predicted, associated with recall of the event. Amnesia for the traumatic event may not protect against PTSD development and other factors associated with recall (such as emotional response and confidence in accurateness) may be relevant. Predicted increases in heart rate and activity level during trauma recall were not found and results do not support the role of sub-conscious processing as a causal mechanism for PTSD development after TBI. Rates of diagnosis established using self-report and interview measures support previous evidence that the assessment of PTSD after TBI is confused by overlapping symptoms and that valid diagnosis can only be established with clinician judgement.

1 Introduction

Psychiatric disorders such as depression and anxiety have frequently been reported following traumatic brain injury (TBI) (Hibbard et al., 1998), however conclusive incidence rates of post-traumatic stress disorder (PTSD) following TBI have yet to be established. Studies investigating the incidence of PTSD following TBI have established a range of occurrence rates. PTSD has been reported following mild TBI at rates of 14%-33% (Mayou, Black and Bryant, 2000, Bryant and Harvey, 1999) and following severe TBI at 3%-59% (Sumpter and McMillan, 2005, Hibbard, Uyssal, Bogdany and Silver, 1998). Sumpter and McMillan demonstrated that due to an overlap in symptoms experienced after TBI and after trauma (such as problems with concentration, sleep and irritability), the method of assessment can impact on diagnosis rates. They suggest that the most reliable tool with which to assess PTSD following TBI is the Clinician Administered PTSD Scale (Blake, Kaloupek, Gusman, Charney and Keane, 1995) which allows clinical judgement to be applied to reported symptoms, therefore distinguishing between symptoms related to TBI and trauma-related symptoms.

PTSD is categorised in DSM-IV (American Psychiatric Association, 1994) as an anxiety disorder. Criteria for the diagnosis of PTSD include the following (Full DSM-IV criteria are outlined in Appendix 4.2): A) Exposure to an event that is a threat to one's life or integrity during which helplessness, horror or intense fear is experienced. B) Re-experiencing symptoms such as intrusive memories, re-living the trauma (flashbacks) and distress when reminded of the trauma. C) Avoidance of thoughts, feelings or reminders of the trauma, inability to recall an important part of the trauma, social withdrawal, or emotional numbing. D) Increased arousal as evidenced by insomnia, irritability, poor concentration, hypervigilance, or heightened startle response. It has been suggested that if memory for the traumatic event is not encoded due to loss of consciousness (as can occur during TBI) then the event is not truly 'experienced'. Re-experiencing symptoms relying on recall of the event cannot therefore be present and PTSD cannot be diagnosed (Sbordone, 1992). Indeed, some studies have found no incidences of PTSD following TBI (eg Sbordone and Liter, 1995). However there is increasing evidence that PTSD can occur after TBI and case studies of PTSD after severe TBI provide evidence that

re-experiencing symptoms can develop even with loss of memory of more than 24 hours (eg King, 2001). It has therefore been suggested that it may not be necessary to fulfil criterion A) - direct experience of the event including intense fear or helplessness, for symptoms of PTSD to develop (McMillan, 2001). The protective value of not recalling a traumatic event has been investigated by Gil, Caspi, Ben-Ari, Koren and Klein (2005) who found that 6% of participants with 'no memory' of their TBI had developed PTSD compared to 23% of participants with 'good memory', indicating amnesia may reduce the likelihood of PTSD. Turnbull, Campbell and Swann (2001) found that participants with either no memory or traumatic memory of the event were significantly more likely to develop PTSD than participants with non-traumatic memory. Therefore it appears that individuals who have experienced complete loss of memory can still develop the symptoms associated with PTSD.

A number of possible mechanisms through which PTSD may develop after TBI have been proposed. Some individuals who experience a mild TBI lose consciousness for only a few minutes and the duration of post traumatic amnesia is also short, hence they will have a relatively intact memory for the trauma. Those who recall only brief snatches of memory (termed 'islands' of memory, King, 1997) for the trauma may still have some recall along with associated horror or fear responses. This brief memory can become intrusive (McMillan, 1996). Additionally, amnesic gaps in memory for the trauma can be filled in by information imagined or obtained after the event and this 'confabulated' memory can also form the basis for intrusions (Bryant, 1996).

It has been theorised that trauma memories might be encoded and retrieved implicitly. Dual Representation Theory (Brewin, Dalgleish and Joseph, 1996) describes how traumatic memories may be stored as verbally accessible memories (VAMs) or situationally accessible memories (SAMs). VAMs are described as verbal or visual and can be intentionally recalled. SAMs comprise of subconscious memory that includes sensory information and is recalled unintentionally in the form of flashbacks. In this way, while impaired consciousness during TBI may lead to poor declarative memory for the traumatic event, implicit encoding may still occur.

Brewin et al. propose that SAMs are mediated via the amygdala (while VAMs are processed by the hippocampus). This theory links cognitive processes to biological theories that suggest fear conditioning to a traumatic event occurs in limbic structures. It is suggested that heightened physiological arousal to a traumatic event leads to an association between an anxiety response and reminders of the trauma (Kolb, 1987). There is support for this theory from studies that have found increased physiological arousal in individuals with PTSD (Bryant, Harvey, Guthrie and Moulds, 2003) and specifically in response to trauma related cues (Orr and Kaloupek, 1997). Bryant et al. (2003) found that re-experiencing symptoms reported following TBI consisted of physiological distress or physiological reactivity when reminded of the trauma.

Hellawell and Brewin (2002) have also found autonomic and motor behaviour to increase during recall of flashback memory compared to non-flashback memory of the trauma in a sample of non-TBI participants with PTSD. They propose that dynamic movement or stasis during recall may indicate a specific response to a particular part of the memory. Such a response is more likely to occur during flashback memory as it holds more sensory information. They further propose that physiological changes are only likely to occur during particular points of recall, experienced as flashbacks. Changes in physiological responsivity in patients with TBI and PTSD during recall of their trauma have not yet been investigated. Such research would provide additional evidence as to whether the processes indicated during trauma recall in other populations with PTSD are also present after TBI. In turn the amount of responsivity and its relation to PTSD symptom severity will provide information about the mechanism through which people without full memory for their trauma could develop PTSD and particularly re-experiencing symptoms.

1.1 Aims and Hypotheses

Aims

1) To investigate the relationship between memory of the traumatic event and PTSD caseness in people with TBI.
2) To establish whether any changes in physiological arousal (motor activity or heart rate) are associated with recall of the traumatic event and PTSD symptom severity or caseness in people with TBI.
3) To repeat the study by Sumpter and McMillan comparing PTSD caseness using questionnaire and structured interview methods.

Hypotheses

1) PTSD caseness or symptom severity, as assessed by the PDS and the CAPS, will be associated with higher scores on the TMI.
2) Physiological arousal, indicated by changes in heart rate and motor activity from baseline, will occur whilst participants are recalling the event that led to their head injury.
3) PTSD caseness or symptom severity will be associated with greater changes in physiological arousal.
4) About 60% of cases will fulfil DSM-IV criteria for PTSD using the PDS and less than 10% will fulfil PTSD criteria using the CAPS.

2 Method

2.1 Participants

Participants were recruited from a head injury outpatient clinic at a city centre hospital, which was attended by patients initially brought to the Accident and Emergency Department due to TBI (169 current and discharged patients were invited to take part; 31 consented to taking part), and from Headway, a voluntary support organisation (15 current service users were invited to take part; 9 consented to taking part). (See Appendix 4.3 for further details of recruitment.) Participants were considered for inclusion if they were aged over 18 years, had sustained a head injury at least three months previously (to fulfil DSM-IV criteria for chronic

PTSD) and not be receiving psychiatric treatment for PTSD. Informed consent was obtained from all participants who took part. Participants were considered unsuitable for inclusion if they were younger than 18 years or were currently receiving psychiatric treatment for PTSD. Participants receiving psychiatric treatment or counselling for reasons other than PTSD were considered on a case by case basis.

2.2 Power Calculation

Power was calculated for the primary hypothesis using data from Turnbull et al. (2001). Turnbull et al. compared avoidance and intrusion symptoms between groups categorised by their memory of the event. The power calculation, with alpha=0.05 and power=0.8 (indicated as a reasonable level of power, Cohen, 1988), indicated sample sizes from the avoidance data of 5 (non-traumatic memory) and 7 (traumatic memory). The intrusion data indicated sample sizes of 4 and 6 respectively. The data reported in Turnbull et al. indicated the relationship between PTSD symptoms and memory for the event produced a small to medium effect size (Cohen's d = 0.31 and 0.47).

However, the current study will employ a correlational design to test the relationship between memory for the event and PTSD symptom severity, anticipating a positive correlation between these two variables. Therefore, Cohen's sample size tables were consulted. For the purposes of the current study, power was set at 0.8 and the correlational effect size at r=0.50, which would give a large effect size (Cohen, 1988), considered appropriate given the small to medium effect size found by Turnbull et al. The calculation indicated that to reliably reject the null hypothesis using a correlational design a sample of n=22 should be recruited, matching the total number calculated from Turnbull et al. A sample of n=22 was therefore considered sufficient to detect a significant relationship.

2.3 Measures

TBI Severity

TBI severity was estimated by retrospective questioning of post-traumatic amnesia (PTA) with each participant. PTA is defined as the return of continuous memory (Russell and Nathan, 1946) and can be established by questioning the patient regarding their memory of events following return to consciousness (McMillan, Jongen and Greenwood, 1996). PTA was used as a measure of severity as other indicators, such as Glasgow Coma Scale score, or records of length of loss of consciousness, were not available for all participants. Additionally, PTA is considered to be the more reliable measure of severity of injury and one which better predicts outcome (Wilson, Pettigrew and Teasdale, 1998).

Attention

The Wechsler Adult Intelligence Scale – III (WAIS-III, Wechsler, 1997) subtest Digit-Symbol Coding gave an indication of sustained attention (Lezak, 1995). This test is known to be sensitive to brain damage and performance has been negatively correlated with coma duration (Correll, Brodginski and Rokosz, 1993). A key with 9 symbols is shown, each given a number from 1 to 9. Participants are given 2 minutes to copy as many symbols into boxes underneath this key, ensuring they place the right symbol with the right number. A score is calculated by totalling the number of correct symbols copied. Scaled scores according to age are given, from 0 to 19. Normative scaled scores indicate a mean of 10, and standard deviation of 3.

Pre-morbid IQ

The Wechsler Test of Adult Reading (WTAR: Wechsler, 2001) estimated IQ prior to TBI. The participant reads a list of words out loud and a point is given for each correctly pronounced word. Scaled scores were calculated from age categories. Normative scores indicate a mean of 100 and standard deviation of 15.

Learning and memory

The Adult Memory and Information Processing Battery (AMIPB) (Coughlan and Hollows, 1985), list-learning sub-test provided a measure of short-term memory retention and recall. A list of 15 words is read out to the participant who is then asked to recall as many as possible, in any order. This is repeated 5 times and a total score for list learning is calculated across the 5 trials. For the purposes of this study, this score is used as an indication of short-term memory, with a range from 0 to 75. Normative scores indicate a mean score of 50.3, standard deviation of 9.7 and a range from 20-68.

Executive Functioning

The Hayling (The Hayling and Brixton Tests; Burgess and Shallice, 1997) provides a brief assessment of three executive functions and therefore gives an indication of possible frontal lobe damage. Two sets of fifteen sentences, each with the last word missing, are read to the participant. In the first set they are asked to provide a word which completes the sentence and a scaled score (calculated from time taken to respond) gives a measure of their response initiation speed. In the second set they are asked to provide a word unconnected to the sentence, giving a scaled score (calculated from time taken to respond and also 'errors' made in giving words connected to the sentence) of their suppression ability and thinking time. A total score from 1 (impaired) to 10 (very superior) is given.

Depression and Anxiety

The Hospital Anxiety and Depression Scale (HADS: Zigmond and Snaith, 1983) is a self-report questionnaire that assesses symptoms of anxiety and depression and has been found to be reliable with medical outpatient populations (Zigmond and Snaith, 1983). Symptoms are scored for their presence over the past week on a scale from 0 to 3 and total scores for anxiety and depression are provided, from 0 to 21.

Disability following TBI

The Glasgow Outcome Scale-Extended (GOS-E: Wilson, Pettigrew and Teasdale 1998) is a clinician rated scale and assesses social and functional disability after TBI. A total score from 0 (Dead) to 8 (Upper Good Recovery) is given, based on

the participants ability to self-care, engage in leisure pursuits, return to work, and remaining symptoms of TBI.

PTSD Severity and Caseness

The Post-traumatic Diagnostic Scale (PDS: Foa, Cashman, Laycox & Perry, 1997) is a self-report questionnaire based on DSM-IV criteria for PTSD. Forty-nine items are rated for frequency of presence over the past month (0 = not at all/only one time, 1 = once a week or less/once in a while, 2 = 2 to 4 times a week/half the time, 3 = 5 or more times a week/almost always). Duration and onset of symptoms are rated along with impact on functioning. The frequency scores are summed to give an overall severity score, from 0 to 51. To achieve PTSD caseness, criterion B to F must be met. Criterion A, feeling helpless or terrified during the event, is not considered essential with a TBI population (McMillan, 2001).

The Clinician Administered PTSD Scale (CAPS: Blake et al., 1995) is a structured clinical interview also assessing DSM-IV symptoms, with and without clinician judgement. The clinician assesses the presence of each symptom over the past month with standard prompt questions and rates the frequency and intensity on a scale from 0 to 4. A symptom is considered present if frequency is rated at least 1 and intensity 2. Total score is calculated by summing the frequency and intensity scores across all 17 symptoms, giving a potential range of 0 – 136. The clinician also rates the impact of symptoms on functioning and distress. Caseness is initially met by fulfilling criteria B to F. Caseness by clinician judgement is further established by the clinician considering whether the presence of nine of the avoidance and hyperarousal symptoms are trauma-related. In the current study this allows the impact of TBI on symptoms such as irritability and poor concentration to be taken account of.

Memory for Traumatic Event

The Traumatic Memory Inventory (TMI: van der Kolk, 1990, unpublished paper – TMI obtained directly from the author) is a structured interview that assesses sensory, affective and narrative memory for the event (see Appendix 4.4). The TMI allows for separate assessment of initial post-trauma memory; memory at the time that symptoms of PTSD were most severe; and current memory. van der Hart, Bolt

and van der Kolk (2005) suggest retrospective recall of memory may not reliably distinguish between these three time periods, so for the purposes of this study only current memory is assessed. A score for the number of memories under each sensory modality is achieved. A participant will achieve a score of 0 if they are unable to reproduce any memory of the event. They will score one point for each separate memory recalled visually, as physical sensations, as smells, as sounds, and as emotions. Categorical data are given for whether memory is integrated (yes/no), narrative (yes/no), has been confirmed by others (yes/no), and whether this confirmation incorporates details of the event (as opposed to, for example, a police report based on the aftermath of the event, with no witnesses of the event itself) (yes/no). The TMI additionally assesses the nature of intrusions, however as intrusive symptoms are accounted for during assessment with the PDS and CAPS this information will not be detailed for the current study.

Physiological Reactivity

Heart rate was monitored using a Polar heart rate monitor (S610i, www.heartratemonitor.co.uk/polar_s610i_uk.html). Heart rate was recorded at 5 second intervals throughout the interview. Mean heart rate was calculated for each assessment measure separately. Additionally each section of the CAPS (re-experiencing, avoidance and hyperarousal) was calculated separately. Motor movement was measured using an Actiwatch Plus (Cambridge Neurotechnology, www.camtech.com) placed on the participant's dominant wrist. Activity was recorded in 2 second intervals and mean activity level was calculated for each interview section apart from when completing self-report measures. Baseline mean heart rate and activity rate was calculated using the data collected during administration of the GOS-E and WTAR. It was considered that this would give a measure of physiological reactivity during interview conditions but without discussion of the trauma (WTAR) and during discussion of the impact of injury, without discussion of the event itself (GOS-E).

2.4 Procedure

The procedure was piloted with one non-TBI individual (a colleague of the researcher) to ensure the timing and physiological measures were reliable. Measures were administered to participants in one individual interview. The heart rate monitor and actiwatch were worn throughout the interview. They were started together, along with a digital stopwatch, and times recorded at the beginning and end of each section. Demographic information was initially collected followed by assessment of PTA. The cognitive screen measures were then administered, followed by the HADS, GOS-E, PDS, CAPS (times were recorded at the beginning and end of each section to establish any changes in physiological reactivity relating to discussion of re-experiencing, avoidance and hyperarousal symptoms) and TMI in order. The trauma-related measures were administered last to ensure any physiological change did not carry over into other measures. Heart rate and activity were therefore recorded for twelve time periods. The interview took between one and two hours, depending on the time taken to describe the event and subsequent symptoms.

2.5 Data Analysis

Data were analysed using SPSS 14.0. Prior to formal analysis, data were checked to ensure they met the assumptions for parametric statistical analysis. A Kolmogorov-Smirnov analysis indicated all data were normally distributed. Therefore Pearson's correlations were calculated for all tests of association. Differences in physiological measures between separate sections of the interview were calculated with paired sample t-tests. Due to the unequal number of participants who met criteria for PTSD, comparison between PTSD cases and non-cases was conducted using non-parametric analysis in order to interpret the data more conservatively. Therefore between subjects analysis was conducted using Mann-Whitney U tests.

3 Results

3.1 Demographics

Twenty-one participants took part in the study; 14 (66.7%) male and 7 (33.3%) female. Thirteen (61.9%) were recruited from the hospital out-patient clinic and eight (38.1%) from Headway. Table 1 outlines the demographic profile of the sample.

*****Insert Table 1*****

Thirteen participants had suffered a severe TBI (62%), 4 participants a moderate TBI (19%) and 4 a mild TBI (19%). Cause of TBI were road traffic accident (driver/passenger n=6, 28.6%, pedestrian n=3, 14.3%), fall (n=6, 28.6%), assault (n=5, 23.8%), and work-related accident (n=1, 4.7%).

3.2 Assessment Measures

Table 2 outlines the descriptive data for additional assessment measures. Scaled scores calculated for the Hayling ranged from 1-6, with three participants scoring 6 (Average), four scoring 5 (Moderate Average), three scoring 4 (Low Average), three scoring 3 (Poor), one scoring 2 (Abnormal) and seven scoring 1 (Impaired). Clinician ratings given for the GOS-E ranged from 5-8, with seven participants rated 8 (Upper Good Recovery), ten participants rated 7 (Lower Good Recovery), two rated 6 (Upper Moderate Disability) and two rated 5 (Lower Moderate Disability). One participant declined to complete the HADS. For the remaining participants the mean anxiety score is rated as ‘mild’ (8.80, sd 4.77) and the mean depression score is rated as ‘minimal’ (5.85, sd 3.42). Mean severity score on the PDS was 13.66 (sd 13.02), range from 0-47. Seven participants met PDS diagnostic criteria for PTSD. Mean CAPS total score was 22.66 (sd 21.18), range from 0-72. Four participants met CAPS diagnostic criteria for PTSD and three met CAPS with clinician-judgment criteria. TMI scores for memory of the event ranged from 0-9 (mean 1.85, sd 3.10). Fourteen participants (66.7%) scored 0, indicating they had no recall of the event (12 of whom had sustained a severe TBI).

*****Insert Table 2*****

3.3 Hypothesis One

PTSD caseness or symptom severity, assessed by the PDS and CAPS, will be associated with higher scores on the TMI.

Severity scores achieved using the PDS did not correlate with TMI scores (r=0.156, n=21, p=0.250), nor did CAPS total scores (r=0.222, n=21, p=0.167). As the sample size is small caution should be taken when interpreting analyses between 'PTSD' and 'no PTSD' groups. Comparison of TMI scores achieved by those participants who reached PTSD caseness on the PDS indicated no relationship (U=30.50, N1=7, N2=14, p=0.172). Comparison of PTSD caseness as defined by the CAPS with clinical judgement confirmed this result (U=17.50, N1=3, N2=18, p=0.356). Of the seven participants who reached diagnostic criteria for PTSD on the PDS, 3 scored '0' on the TMI. Of those who reached caseness on the CAPS with clinical judgement, 1 scored '0'. Additional data from the TMI is presented in Appendix 4.5.

3.4 Hypothesis Two

Physiological arousal, indicated by changes in heart rate and motor activity from baseline, will occur whilst participants are recalling the event that led to their head injury.

Preliminary analyses indicated a significant decrease in heart rate from baseline to TMI administration (t=4.442, df=20, p=0.000) and from baseline to administration of CAPS section C (avoidance) (t=2.149, df=21, p=0.044) and section D (hyperarousal) (t=3.193, df=20, p=0.005). Activity rate did not differ significantly from baseline to TMI (t=0.120, df=20, p=0.906) or CAPS (section B: t=1.530, df=20, p=0.142, section C: t=1.184, df=20, p=0.250, section D: t=0.546, df=20, p=0.591).

Analysis of heart rate over the course of the interview was carried out to establish the overall trend. A boxplot (see Figure 1) of mean heart rate across all twelve time periods did not suggest significant lowering of heart rate during T10-12 (CAPS section C, section D and TMI). Due to a number of outliers identified by the boxplot, the data for mean heart rate across the entire interview was subjected to a Kolmogorov-Smirnov test which indicated the data were parametric. A Univariate Analysis of Variance (ANOVA) indicated a significant main effect of Time ($F_{1,228} = 61.920$, $p=0.000$). Parameter estimates of this analysis indicated that heart rate dropped across time (Beta = -0.447, $p=0.000$). Including T10, T11 and T12 as covariates indicated no significant change from the downward trend (T10: $F_{1,227} = 1.647$, $p=0.201$, Beta = 0.865); (T11: $F_{1,227} = 0.240$, $p=0.625$, Beta = 0.342); (T12: $F_{1,228} = 0.770$, $p=0.381$, Beta = 0.623). These results indicate that mean heart rate showed a general decline across the interview.

*****Insert Figure 1*****

3.5 Hypothesis Three

PTSD caseness or symptom severity will be associated with greater changes in physiological arousal.

Initial analyses were conducted to investigate the relationship between baseline heart rate and PTSD severity scores in order to establish whether participants with more severe PTSD scores had higher baseline mean heart rate (higher resting heart rate has been reported in non-TBI PTSD populations, eg Buckley and Kaloupek, 2001). A scatterplot of PDS severity and baseline heart rate, and a scatterplot of CAPS total score and baseline heart rate were developed (see Figures 2 and 3).

*****Insert Figure 2 and 3****

No relationship between baseline heart rate and PTSD severity score was observed from the scatterplots, confirmed by correlational analyses (baseline heart rate and PDS severity: $r=0.055$, $n=21$, $p=0.812$, baseline heart rate and CAPS total score:

r=0.071, n=21, p=0.758). Therefore analyses based on changes in physiological measures from baseline were conducted.

CAPS total scores did not correlate significantly with the difference in heart rate (r=0.138, n=21, p=0.080) nor with the difference in activity level (r=0.162, n=21, p=0.242) from baseline to administration of the CAPS.

PDS severity scores did not correlate significantly with the difference in heart rate (r=0.380, n=21, p=0.089) from baseline to administration of the PDS. Activity level data was not available during PDS as it involved writing.

PTSD severity scores were also correlated with the difference in physiological measures from baseline to TMI administration. Higher PDS severity scores correlated significantly with greater decrease in heart rate (r=0.456, n=21, p=0.016), however activity level did not correlate significantly with PDS severity score (r=0.072, n=21, p=0.378). Higher CAPS total scores correlated significantly with greater decrease in heart rate (r=0.447, n=21, p0.021), however activity level did not correlate significantly with CAPS total score (r=0.099, n=21, p=0.335).

3.6 Hypothesis Four

About 60% of cases will fulfil DSM-IV criteria for PTSD using the PDS and less than 10% will fulfil PTSD criteria using the CAPS.

Seven participants fulfilled criteria for PTSD caseness using the PDS (33.3%). Severity ratings of PTSD indicated one participant reported mild PTSD, two participants had moderate PTSD, two had moderate-severe PTSD and two had severe PTSD. Of these participants, three had sustained a moderate TBI and four a severe TBI. Four participants fulfilled CAPS criteria without clinical judgement (19%) (TBI severity – 3 moderate, 1 severe) and three with clinical judgement (14%) (TBI severity – 2 moderate, 1 severe). All participants identified as reaching caseness by the CAPS were also identified by the PDS as fulfilling PTSD criteria.

The three participants who met criteria for PTSD on the PDS but not the CAPS were found not to meet intensity criteria for re-experiencing symptoms and to have reported symptoms relating to their TBI in the avoidance and hyperarousal sections. The participant who met criteria for PTSD on the CAPS without clinical judgement was considered to have described one avoidance and 2 hyperarousal symptoms which related to their TBI rather than the impact of the trauma. Of the total scores recorded using the PDS; 16.47% were re-experiencing symptoms; 38.08% avoidance symptoms; and 45.45% hyperarousal symptoms. Of the total scores recorded using the CAPS; 23.29% were re-experiencing symptoms; 34.68% avoidance symptoms; and 42.03% hyperarousal symptoms.

3.7 Analysis of Association between Assessment Measures

Anxiety and depression scores from the HADS were positively correlated with PDS severity and CAPS total scores. Bonferroni correction was applied to this correlation table, giving an accepted significance value of p=0.0125. Both anxiety and depression scores correlated significantly with PDS severity scores (respectively, r=0.751, n=20, p=0.000, r=0.630, n=20, p=0.003). Anxiety and depression scores also correlate significantly with CAPS total scores (respectively, r=0.744, n=20, p=0.000, r=0.698, n=20, p=0.001).

Demographic and cognitive functioning measures were correlated with TMI score, PDS severity score and CAPS total score. Bonferroni correction gave an accepted significance value of p=0.0027. Age was not associated with TMI score (r=0.318, n=21, 0.160), PDS severity score (r=0.051, n=21, p=0.825) or CAPS score (r=0.039, n=21, p=0.865). Time since injury was not associated with TMI score (r=0.350, n=21, p=0.119), PDS severity (r=0.286, n=21, p=0.208) or CAPS score (r=0.337, n=21, p=0.136). Estimated pre-morbid IQ (WTAR) was not associated with TMI score (r= -0.083, n=21, p=0.719), PDS severity (r= -0.383, n=21, p=0.086) or CAPS score (r= -0.430, n=21, p=0.052). Digit-Symbol Coding score (sustained attention) was not associated with TMI score (r=0.479, n=21, p=0.028), PDS severity (r=0.227, n=21, p=0.323) or CAPS score (r=0.235, n=21, p=0.306). Short-term memory (AMIPB) was not associated with TMI score (r=0.272, n=21, p=0.233), PDS severity (r=0.147, n=21, p=0.524), or CAPS score (r=0.267, n=21,

p=0.243). Executive functioning (Hayling) was not associated with TMI score (r= -0.170, n=21, p=0.461), PDS severity (r= -0.356, n=21, p=0.113), or CAPS score (r= -0.409, n=21, p=0.065).

4 Discussion

4.1 Memory for the event

It was hypothesised that PTSD severity scores achieved on the PDS and the CAPS would be associated with TMI scores, reflecting previous findings that the amount and quality of memory recalled of the traumatic event affected severity of PTSD symptoms (Gil et al., 2005, Turnbull et al., 2001). No such association was found. It is possible that this result was due to small sample size and the proportion of participants (66.6%) achieving a score of 0 (indicating no memory) therefore reducing the usefulness of a continuous scale. It is also possible that this result indicates that 'memory' as assessed by the TMI differed from 'memory' assessed in previous studies. Both Gil et al. and Turnbull et al. devised their own self-report questionnaires to measure the quality of participant's memory. Gil et al. reported that their questionnaire assessed participants' confidence in their memory (by their ratings from *no memory* to *good memory* on 9 items) of the event rather than measuring the detail recalled. Participants were divided into 'no memory' and 'good memory' groups from their ratings, therefore the quality of memory was not objectively measured. Turnbull et al. defined groups as having 'no memory', 'traumatic memory' and 'untraumatic memory' on the basis of self-report. Memory was therefore assessed for emotional content as well as quantity, which reflects an additional variable. Memory as assessed by the TMI comprised all details recalled of the event, therefore capturing islands of memory as well as elaborated narrative memories, while separating details later incorporated into the trauma narrative by third party information or confabulation. The lack of relationship between quantity of memory and PTSD severity may indicate that volume is not the most influential factor. Variables such as participants' confidence in the accurateness of their memory and the affect attached to recall might also have a role to play in predicting PTSD symptom severity.

The TMI allowed for investigation of information gained by participants after their injury (Appendix 4.5). Interestingly, all three participants who reached diagnostic criteria using the CAPS with clinical judgement had received information from witnesses to the event and had included this information in their account of the event (these details were not included in their TMI score). Bryant (1996) and McMillan (1996) report that information gained post-injury could provide a mechanism for the development of PTSD after TBI. These three participants described feelings of shock or horror in response to descriptions of their injury, which may be an important factor in the development of PTSD after TBI. While these individuals had no conscious recollection of the injury itself they were provided with information post-injury which allowed them to 'imagine' the event vividly. Rumination over the traumatic event which involves elaboration of events to include catastrophic outcome is implicated in poorer outcome following trauma (Ehlers, Mayou and Bryant, 1998). Individuals with TBI may therefore be more likely to develop PTSD if they are later provided with details of the event, particularly if their reaction to these details reflects the fear or horror as described in Criterion A of DSM-IV diagnostic criteria.

Turnbull et al. suggest that individuals without memory of the event may not experience intrusive memories. In the present study only one participant without memory reported intrusive memories and reliving. This participant had incorporated information learned from witnesses into her account of the event and described these details as appearing in intrusions and flashbacks. Therefore lack of memory for the event does appear to protect against intrusive memories.

4.2 Physiological Responses

Activity level did not change significantly between baseline and discussion of trauma and activity level was not significantly associated with PTSD severity scores. Hellawell et al. (2002) reported an increase in non-writing movements and vocalisations during recall of memory reported as 'flashback' by participants. The lack of effect in the current study could be due to differences in methodology. Hellawell observed and recorded movement (according to a coding scheme devised

for the study) at set time points whilst participants were writing a trauma narrative. Comparisons for observations were then made between sections of the narrative participants rated as 'flashback' and 'ordinary' memory. No inter-rater reliability checks were performed. The current study utilised the Actiwatch, which provided a continuous measurement of physical movement throughout the interview. The Actiwatch is arguably a more objective measure of activity level and results could demonstrate the effect found in Hellawell would be non-significant when measured in this way. Hellawell et al. reported on findings from a non-TBI population, while this study included participants without memory for the event. The lack of change in activity level in the current study could reflect a difference in presentation between the two samples. Bryant and Harvey (1998) investigated reactions to audio of a road traffic accident (RTA) in participants with PTSD following RTA, some of whom had experienced TBI. Participants with PTSD and TBI reported less movement in their intrusions than participants without TBI. Increase in 'mobilisation' behaviour was suggested by Hellawell to reflect retrieval of dynamic memories which are linked to perceptual processing of the traumatic event. It was hypothesised that a significant change in activity level in the current sample would suggest a similar process could underlie re-experiencing symptoms in the absence of conscious memory for the event. As no change in activity level was observed it must be considered that perceptual processing linked to non-conscious memory does not mediate re-experiencing symptoms following TBI.

While the data indicated a drop in heart rate during discussion of the trauma, this was found to represent a general decline in heart rate over time. Heart rate is known to increase in response to anxiety (eg Hofmann et al., 2005) and it is possible that participants demonstrated higher heart rate at the start of the interview due to anxiety about taking part. The decrease observed over time could indicate a general decrease in their anxiety levels as the interview progressed.

Higher PTSD severity scores were significantly associated with greater decrease in heart rate during recall of the trauma (TMI administration). Previous studies into PTSD without TBI have found an increase in heart rate during recall of trauma (Cohen et al., 1998) and in response to trauma related stimuli (e.g. Blanchard, Kolb, Pallmeyer, and Gerardi, 1982). An increase in arousal in response to feared

stimuli is considered to represent the 'fight or flight' reaction to threat (e.g. Pitman, Orr and Shalev, 1993) and has also been demonstrated in other anxiety disorders (e.g. Cohen et al., 2000). The decrease observed in association with higher severity scores is therefore interesting. Studies investigating physiological reactions to affective stimuli have demonstrated an association between motivated orienting and decreased heart rate. Viewing unpleasant pictures (Lang, Bradley and Cuthbert, 1997), pleasant pictures (Sanchez-Navarro, Martinez-Selva and Roman 2006), and novel or complex stimuli (Stekelenburg and van Boxtel, 2002) led to reduced heart rate. This reduction is considered to represent an orienting response as opposed to the threat reaction which results in increased arousal. It is possible that the decrease in heart rate observed when discussing recall of the trauma indicates attentional orientation. This association is stronger in participants reporting higher severity scores which might suggest discussion of the trauma to have greater personal salience, a factor which is thought to impact on attention orientation (Sanchez-Navarro et al., 2006). McMillan (2001) demonstrated that curiosity about the gap in memory left by PTA can exist and this curiosity can be reported as 'intrusive' due to a desire to recover memory. This finding was supported by Sumpter and McMillan (2006). Appraisal of the traumatic event as time-limited and without implications for future safety is protective against PTSD (Ehlers and Clark, 2000). Perhaps participants with higher severity scores seek to integrate their unknown experience in order to evaluate future threat. This would lead to motivated orientation to discussion of the trauma and attendance to salient stimuli rather than a threat response because of a lack of awareness of specific trauma cues. If this is the case there is little evidence for the encoding of implicit or sensory memory in the absence of conscious recall. Indeed, considering the role of the amygdala in the production of defence and startle responses (e.g Davis, 1996) implicit memories, mediated by the amygdala, do not appear to produce physiological reactivity in individuals without memory for trauma.

4.3 Diagnosis of PTSD

While proportions of participants reaching caseness on the PDS and CAPS differed from those hypothesised, the pattern followed that predicted from Sumpter and McMillan (2005). As expected, more participants met PTSD diagnostic criteria

when self-reporting symptoms on the PDS (33.3%) than met criteria as assessed by the CAPS with clinical judgement (14%). This incidence rate included participants with moderate and severe TBI and falls within previously reported incidence ranges of 3-33% (Sumpter and McMillan, 2005, Hibbard et al., 1998, Mayou, Black and Bryant, 2000).

4.4 Additional Results

Depression and anxiety symptoms are known to present after TBI (Hibbard et al., 1998) and it would be expected that reporting of low mood and anxiety would be associated with higher levels of PTSD symptom reporting. Depression and anxiety symptoms reported on the HADS correlated significantly with PDS and CAPS scores, although levels of depression and anxiety were sub-clinical. This association was also reported in Sumpter and McMillan (2005). Associations between cognitive screen measures and PTSD severity scores were treated conservatively due to the large number of comparisons made. CAPS total scores were negatively associated with Hayling scores. Seven of the current sample scored '1' (impaired) on the Hayling, suggesting reduction in executive functioning. A lack of insight, associated with dysexecutive disorder, could therefore have led to reduction in ability to report symptoms. Sustained attention (Digit-Symbol Coding) was positively associated with TMI scores. This would be expected. Participants with lower scores on the TMI were likely to have sustained more severe TBI and therefore achieve lower scores on this test.

4.5 Limitations and Recommendations for Future Research

A priori hypotheses regarding aspects of memory assessed by the TMI other than quantity of recall were not established due to the investigative nature of the study. Investigation of additional variables associated with recall is indicated, for example confidence in accuracy of recall, affect associated with memory and the emotional reaction to third party information about trauma. The decrease of heart rate over the course of the interview is a limitation in the interpretation of the heart rate data. Future studies should consider random administration of measures. It is

recommended that further research is conducted into motivated orienting in individuals with TBI.

5 Conclusions

The quantity of memory recalled of the traumatic event may not be the most influential factor in predicting PTSD symptom severity or caseness after TBI. Individuals' confidence in the accurateness of their memories and their emotional response to memory may also impact on likelihood of PTSD development. Gaining information post-trauma regarding details of the event may increase the likelihood of PTSD, particularly if emotional responses to that information reflect those required in Criterion A of diagnostic criteria. Lack of memory alone does not therefore protect against PTSD.

The lack of activity change could indicate that perceptual processing linked to sub-conscious memory does not mediate re-experiencing symptoms after TBI. The decrease in heart rate associated with higher PTSD severity scores may reflect a high level of personal salience and motivated orientation to attempted trauma recall. The lack of physiological arousal in response to trauma cues provides no evidence for the encoding of implicit or sensory memory mediated by the amygdala as a mechanism for PTSD development in individuals without memory for the trauma.

Proportions of PTSD diagnosis using the PDS and the CAPS support the use of assessment tools requiring clinician-judgement in order to reliably establish the presence of PTSD after TBI.

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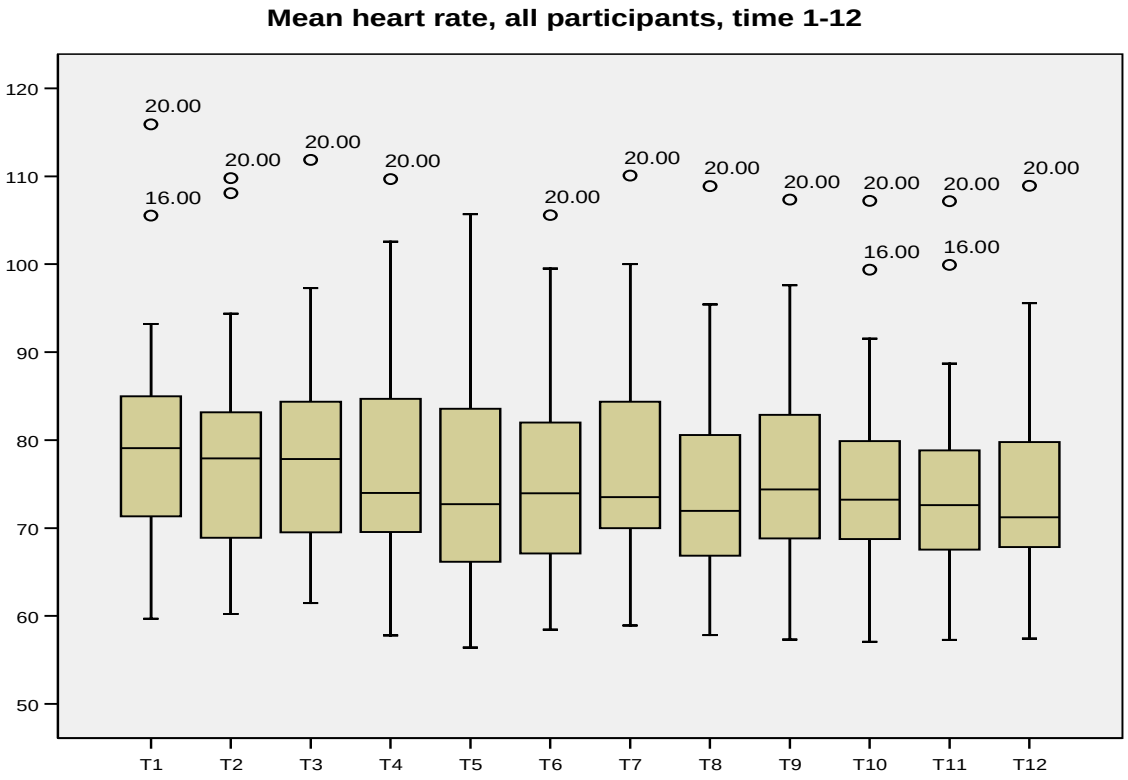
Table 1 – Demographic Information

	Mean (s.d)	**Range**
Age (Years)	42.90 (10.98)	20-65
Time since TBI	5 years 10 months (8 years 10 months)	3 months – 29 years 11 months
PTA	28 days (54 days)	0.50 hours – 6 months
Years of Education	12.61 (2.85)	10-20
	YES	**NO**
Previous TBI	4 participants (19%)	17 participants (62%)
Previous Trauma	6 participants (28.5%)	15 participants (71.5%)
Previous Psychiatric Treatment or Counselling	6 participants (28.5%) *(2 of these 6 reported previous trauma)*	15 participants (71.5%)
Currently Employed	8 participants (38%)	13 participants (62%)
Alcohol drunk prior to TBI	6 participants (28.5%)	15 participants (71.5%)

Table 2 – Assessment Measures

Measure	**Mean (s.d)**	**Range**
WTAR (estimated IQ)	98.38 (9.63)	78-114
Digit-Symbol Coding (scaled score)	6.95 (2.85)	3-14
AMIPB (total number of words recalled)	39.04 (10.62)	18-56
Hayling (total scaled score)	3.23 (1.92)	1-6
HADS – Anxiety (total)	8.80 (4.77)	2-20
HADS – Depression (total)	5.85 (3.42)	1-13
GOS-E (clinician rating)	7.04 (0.92)	5-8
PDS (severity score)	13.66 (13.02)	0-47
CAPS (total score)	22.66 (21.18)	0-72
TMI (total score)	1.85 (3.10)	0-9

Figure 1 – Boxplot Mean Heart Rate across Interview Time Periods



T1 – Demographic Information

T2 – Digit-Symbol Coding

T3 – WTAR

T4 – AMPIB

T5 – Hayling

T6 – HADS

T7 – GOS-E

T8 – PDS

T9 – CAPS B

T10 – CAPS C

T11 – CAPS D

T12 - TMI

Figure 2 – Scatterplot PDS Severity Scores and Baseline Mean Heart Rate

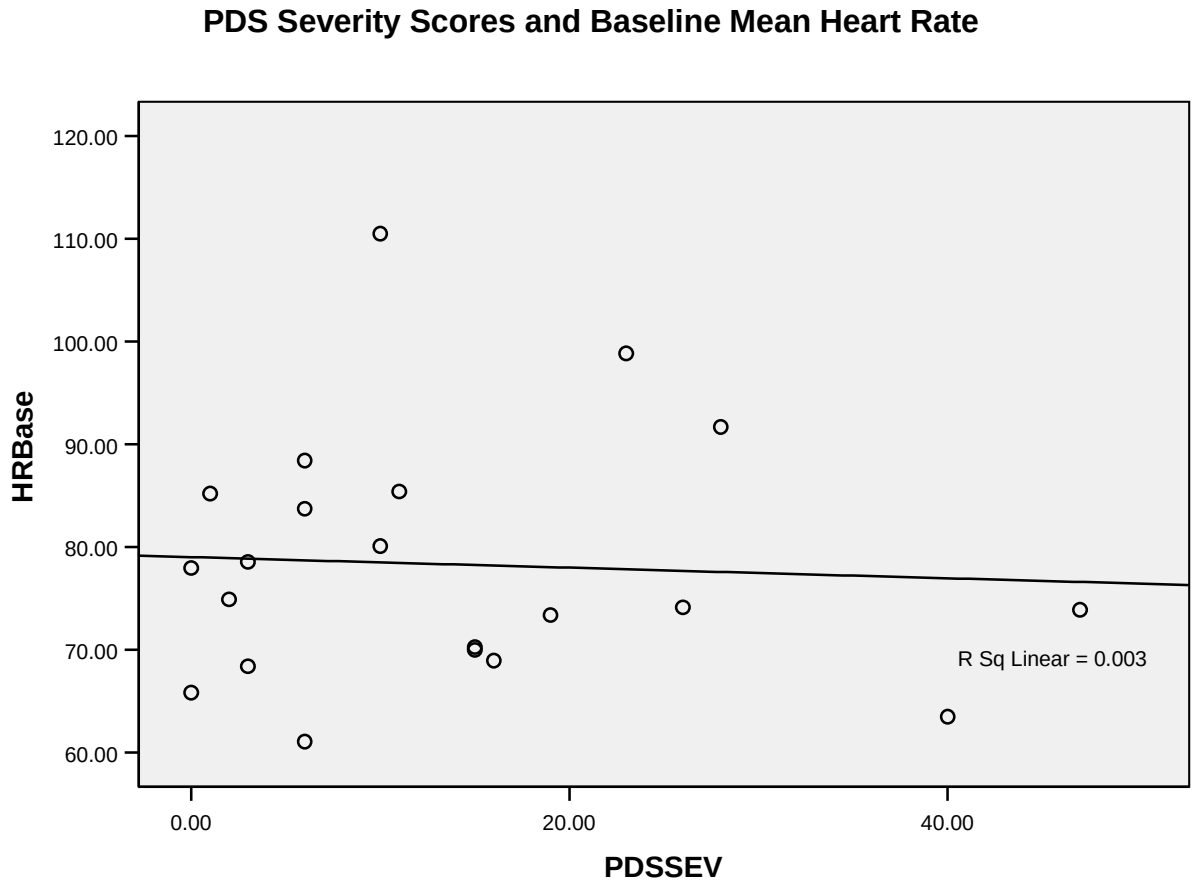
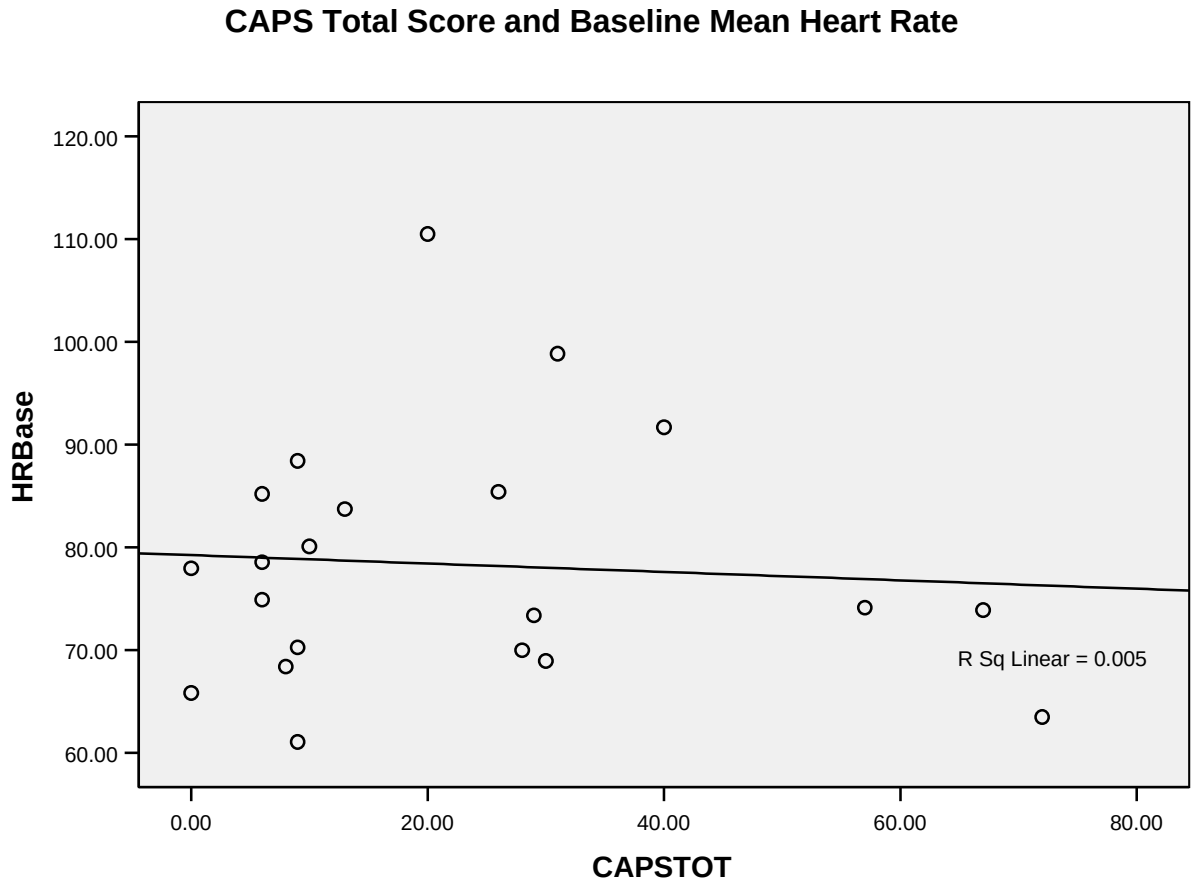


Figure 3 – Scatterplot CAPS Total Score and Baseline Mean Heart Rate



Chapter 5

Single N Proposal

Conversion Disorder in adolescence: a single case study investigating the additive effects of a four-stage treatment approach

Written according to course handbook guidelines for submission
(See Appendix 5.1)

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Submitted in partial requirement for the degree of Doctorate in Clinical Psychology (DClinPsy)

Abstract

Conversion Disorder is defined by DSM-IV as the presentation of uncontrolled physical symptoms which emerge due to underlying emotional distress. There is little research on the treatment of Conversion Disorder in adolescence. Research on the treatment of Conversion Disorder in adult populations has emphasised cognitive and behavioural interventions, however the active treatment components have not been identified. Approaches which include intervention with the whole family have generally been applied to somatoform disorders in childhood. A 16 year old girl presenting with unexplained collapse fulfilled diagnostic criteria for Conversion Disorder. Maintaining factors include behavioural, cognitive, psychosomatic and family systems factors. A four stage treatment approach is proposed to investigate the additive effects of behavioural, cognitive, psychosomatic, and family system interventions. It is hypothesised that behavioural intervention will decrease the frequency of collapse and the addition of cognitive, psychosomatic and family intervention will each further decrease the frequency of collapse. This case study will add to the evidence base for the treatment of Conversion Disorder in adolescence and will indicate the effectiveness of combining a number of approaches.

Research Portfolio Volume I Appendices **Page**

Appendix 1.1 – Submission guidelines to Clinical Psychology

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- Spell out all acronyms the first time they appear.
- Include the first names of all authors and give their employers, and remember to give a full postal address for correspondence.
- Give references in Clinical Psychology style, and if a reference is cited in the text make sure it is in the list at the end.
- Don't include tables and figures unless they save space or add to the article.
- Ask readers to request a copy of your questionnaire from you rather than include the whole of it in the article.

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Appendix 2.1 – Requirements for Submission to Journal of the International Neuropsychological Society

JOURNAL OF THE INTERNATIONAL NEUROPSYCHOLOGICAL SOCIETY
Instructions for Contributors

Aims and Scope:

The Journal of the International Neuropsychological Society welcomes original, creative, high quality research papers covering all areas of neuropsychology. The focus of articles may be primarily experimental, more applied or clinical. Contributions will broadly reflect the interest of all areas of neuropsychology, including but not limited to: development of cognitive processes, brain-behavior relationships, adult and pediatric neuropsychology, neurobehavioral syndromes, such as aphasia or apraxia, and the interfaces of neuropsychology with related areas such as behavioral neurology, neuropsychiatry, and cognitive neuroscience. Papers that utilize behavioral, neuroimaging, and electrophysiological measures are appropriate. Book reviews will also be published.

To assure maximum flexibility and to promote diverse mechanisms of scholarly communication, the following formats are available in addition to Regular Research Articles: Brief Communications are shorter research articles; Rapid Communications are intended for "fast breaking" new work, that does not yet justify a full length article, and which are put on a fast review track; Neurobehavioral Grand Rounds are unique case studies, which are published in tandem with an introduction by an expert in the field to put the case into a more global perspective; Critical Reviews are thoughtful considerations of topics of importance to neuropsychology, including associated areas, such as functional brain imaging, neuroepidemiology, and ethical issues; Dialogues provide a forum for publishing two distinct positions on controversial issues in a point-counterpoint form; Symposia consist of several research articles that are thematically linked; Letters to the Editor respond to recent articles in the Journal of the International Neuropsychological Society; and Book Reviews.

Critical Reviews, Dialogues, and Symposia may be invited by the appropriate Department Editor or proposed by individual authors. Such proposals should be discussed with the Editor-in-Chief or the Department Editor before submission. Book Reviews are invited by the Book Review Editor.

Originality and Copyright

To be considered for publication in the Journal of the International Neuropsychological Society, a manuscript cannot have been published previously, nor can it be under review for publication elsewhere. Papers with multiple authors are reviewed with the assumption that all authors have approved the submitted manuscript and concur with its submission to the Journal of the International Neuropsychological Society. A Copyright TransferAgreement, with certain specified rights reserved by the author, must be signed and returned to the Editor by the corresponding author of accepted manuscripts, prior to publication. This

is necessary for the wide distribution of research findings, and the protection of both author and the society under copyright law.

Disclosure Form

An Author Disclosure Form must be signed by the corresponding author at the time the manuscript is submitted. This form includes an attestation that the manuscript is original and not under review in another journal, research was conducted in compliance with institutional guidelines, and any potential conflict of interest has been reported. Such disclosure will not preclude publication, but it is critical because of the potential of negative or positive bias. Potential conflicts of interest include funding sources for the reported study or financial interest in a test or product or with a company that publishes a test that is being investigated in the manuscript. In addition to signing this attestation, compliance with institutional research standards for animal or human research (including a statement that the research was completed in accordance with the Helsinki Declaration http:00www.wma.net0e0policy017-c_e.html) should be included in the methods section of the manuscript, and funding sources and other potential conflicts of interest should be included in the acknowledgements. See the Author Disclosure Form on website for specific details.

Manuscript Submission and Review

The Journal of the International Neuropsychological Society uses online submission and peer review. Paper submissions are not accepted. Authors who are not able to submit their manuscripts online are asked to contact the editorial office at: jins@unm.edu. The website address for submissions is: http:00mc.manuscriptcentral.com0cup0jins, and complete instructions are provided on the website. Prior to online submission, please consult http:00www.ncbi.nlm.nih.gov0entrez0query.fcgi?db5mesh for 6 keywords or mesh terms that are different from words in the title. Accurate mesh terms will increase the probability that your manuscript will be identified in online searches. Please follow the instructions carefully to avoid delays. The menu will prompt the author to provide all necessary information, including the manuscript category, the corresponding author including phone number, fax number and e-mail address, and suggested reviewers.

The website will automatically acknowledge receipt of the manuscript and provide a manuscript reference number. The Editor-in-Chief will assign the manuscript for review to an Associate or Department Editor and at least two other reviewers. Every effort will be made to provide the author with a review within 6 to10 weeks of manuscript assignment. Rapid Communications will be reviewed within 6 weeks. If the Editor requests that revisions be made to a manuscript before publication, a maximum of 3 months will be allowed for preparation of the revision, except in unusual circumstances.

Manuscript Length

In order to increase the number of manuscripts that can be published in the JINS, please adhere to the following length requirements. Please provide a word count on

the title page for abstract and for manuscript (not including abstract, tables, figures, or references). Manuscripts will be returned if they exceed length requirements.

Regular Research Articles: Maximum of 5,000 words (not including tables, figures, or references) and a 200 word abstract.

Brief Communications: Maximum of 2,500 words (not including abstract, tables, figures, or references) and a 150 word abstract, with a maximum of two tables or two figures, or one table and one figure, and 20 references.

Rapid Communications: Maximum of 1,000 words (not including abstract, tables, figures, or references) and a 150 word abstract, with a maximum of two tables or two figures, or one table and one figure, and 10 references.

Critical Reviews: Maximum of 5,000 words (not including abstract, tables, figures, or references) and a 200 word abstract. Critical Reviews must be pre-approved by the Department Editor. Please e-mail your abstract to jins@unm.edu in order to receive prior approval.

Dialogues: Maximum of 2,000 words for each segment (not including abstract, tables, figures, or references) and a 100 word abstract, with a maximum of two tables or two figures, or one table and one figure and 20 references.

Dialogues must be pre-approved by the Department Editor. Please e-mail your abstract to jins@unm.edu in order to receive prior approval.

Symposia: Maximum of 5,000 words (not including abstract, tables, figures, or references) and a 200 word abstract. Symposia must be pre-approved by the Department Editor. Please e-mail your abstract to jins@unm.edu in order to receive prior approval.

Neurobehavioral Grand Rounds: Maximum of 5,000 words with an informative literature review (not including abstract, tables, figures, or references) and a 200 word abstract.

Letters to the Editor: Maximum of 500 words (not including table, figure, or references) with up to five references, one table, or one figure.

Book Reviews: Approximately 1,000 words. Manuscript Preparation and Style

The entire manuscript should be typed double-spaced throughout using any word processing program. Unless otherwise specified, the guideline for preparation of manuscripts is the Publication Manual of the American Psychological Association (5th edition) except for references with 3 or more authors (see References section). This may be ordered from: APA Order Dept., 750 1st St. NE, Washington, DC 20002-4242, USA.

Pages should be numbered sequentially beginning with the Title Page. The Title Page should contain the full title of the manuscript, the full names and affiliations of all authors, a contact address with telephone and fax numbers and e-mail

address, and the word count for abstract and for manuscript (excluding title page, abstract, references, tables, and figures). At the top right provide a short title of up to 45 characters preceded by the lead author's last name. Example: Smith-Memory in Parkinson's Disease. This running headline should be repeated at the top right of every following page.

The Abstract and Mesh terms (Keywords) on page 2 should include a brief statement of the problem, the method, the key findings, and the conclusions. Six mesh or key words should be provided (see http:00www.ncbi.nlm.nih.gov0entrez0query.fcgi?db5mesh for list), and they should not duplicate words in the title. The full text of the manuscript should begin on page 3. For scientific articles, including Regular Research Articles, Brief Communications, Rapid Communications, and Symposia, the format should include an Abstract, Introduction, Method, Results, and Discussion. This should be followed by References, Appendixes, Acknowledgments, Tables, Figures, and Figure Legends.

The use of abbreviations, except those that are widely used, is strongly discouraged. They should be used only if they contribute to better comprehension of the manuscript. Acronyms should be spelled out at first mention. Metric system (SI) units should be used.

Figures

High quality digital images (600 dpi or higher) should be provided in PDF, EPS, or TIFF formats. If a digital image is not available, please scan in the image. Figures should be numbered consecutively as they appear in the text. Any indication of features of special interest should also be included. Figures should be drawn or composed on computer to about twice their intended final size and authors should do their best to construct figures with notation and data points of sufficient size to permit legible photo reduction to one column of a two-column format. As a guide, no character should be smaller than 1 mm wide following reduction.

Tables and figures should be numbered in Arabic numerals.
The approximate position of each table and figure should be provided in the manuscript: [INSERT TABLE 1 HERE]. Tables and figures should be on separate pages. Tables should have short titles and all figure legends should be on separate pages.

Color figures can be accepted. All color graphics must be formatted in CMYK and not in RGB, because 4-color separations cannot be done in RGB. However, the extra cost of printing these figures must be paid by the author, and the cost typically ranges from $700 to $1500 per figure.

References

References should be in American Psychological Association, 5th Edition, style (see the examples presented below). Text references should be cited as follows: ". . . Given the critical role of the prefrontal cortex (PFC) in working memory (Cohen et al., 1997; Goldman-Rakic, 1987; Perlstein et al., 2003a) . . ." with multiple

references in alphabetical order. Another example is: “For example, Cohen et al. (1994, 1997), Braver et al. (1997), and Jonides and Smith (1997) demonstrated . . .” If multiple works by Perlstein et al. (1977) are cited, use a, b, c, in the order these appear in the text, even if the subsequent authors are different. References cited in the text with three or more authors should state et al. (e.g., Smith et al.) even at first mention (this deviates from the APA 5th Edition style). However, in the reference section all authors should be listed. Reference entries should be alphabetically listed in the reference section with all authors being cited. Examples of the APA reference style are as follows:

Scientific Article:
Haaland, K.Y., Price, L., & LaRue, A. (2003). What does the WMS-III tell us about memory changes with normal aging? Journal of the International Neuropsychological Society, 9, 89–96.

Book:
Lezak, M.D., Howieson, D.B., & Loring, D.W. (2004). Neuropsychological Assessment. New York: Oxford University Press

Book Chapter:
Knopman, D. & Selnes, O. (2003). Neuropsychology of Dementia. In K.M. Heilman & E.E.Valenstein (Ed.), Clinical Neuropsychology. New York: Oxford University Press.

Report at a Scientific Meeting:
Rothi, L.J.G. (2003, February) Use-dependent learning and neural plasticity: A revision of the pessimism surrounding neurorehabilitation. International Neuropsychological Society, Honolulu, Hawaii.

Manual, Diagnostic Scheme, etc.:
American Psychiatric Association (1994). Diagnostic and Statistical Manual of Mental Disorders (4th ed.). Washington, DC: American Psychiatric Association Press.

Proofs
The publisher reserves the right to copyedit manuscripts.
The corresponding author will receive pdfs for final proofreading. These should be checked and corrections returned within 2 days of receipt. The publisher reserves the right to charge authors for excessive correction of nontypographical errors.

Offprints and PDF Files
The corresponding author will receive a free pdf. This pdf can also be mounted on the authors’ web pages. Offprints must be ordered when page proofs are returned. The offprint order form with the price list will be sent with page proofs.

Appendix 2.2 – DSM-IV Criteria for Posttraumatic Stress Disorder (309.81)

A) The person has been exposed to a traumatic event in which both of the following have been present:

(1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others (2) the person's response involved intense fear, helplessness, or horror. **Note:** In children, this may be expressed instead by disorganized or agitated behaviour.

B) The traumatic event is persistently re-experienced in one (or more) of the following ways:

(1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. **Note:** In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.

(2) recurrent distressing dreams of the event. **Note:** In children, there may be frightening dreams without recognizable content.

(3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur upon awakening or when intoxicated). **Note:** In young children, trauma-specific re-enactment may occur.

(4) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

(5) physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

C) Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:

(1) efforts to avoid thoughts, feelings, or conversations associated with the trauma

(2) efforts to avoid activities, places, or people that arouse recollections of the trauma

(3) inability to recall an important aspect of the trauma

(4) markedly diminished interest or participation in significant activities

(5) feeling of detachment or estrangement from others

(6) restricted range of affect (e.g., unable to have loving feelings)

(7) sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)

D) Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:

(1) difficulty falling or staying asleep
(2) irritability or outbursts of anger
(3) difficulty concentrating
(4) hypervigilance
(5) exaggerated startle response

D) Duration of the disturbance (symptoms in Criteria B, C, and D) is more than one month.

F) The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:
Acute: if duration of symptoms is less than 3 months.
Chronic: if duration of symptoms is 3 months or more.

Specify if:
With Delayed Onset: if onset of symptoms is at least 6 months after the stressor.

Appendix 2.3 - Quality Criteria Rating Scale

1) Assessment of PTSD:

Structured interview with clinical judgement (eg CAPS)	3 points
Structured interview without clinical judgement	2 points
Self-report questionnaire	1 point

2) Criteria for TBI severity:

PTA assessed formally (interview/questionnaire)	3 points
PTA assessment informal or unclear, or duration of LOC	2 points
GCS only	1 point

3) Outcome measures are clearly defined and relate to the aims of the study:

Measures clear and related to aims	2 points
Description of symptom profile only	1 point
Measures unclear or unrelated to aims	0 points

4) Sample size was justified:

Power calculation was conducted	2 points
Limitations of sample size acknowledged	1 point
No justification of sample size	0 points

5) Study type:

Matched control group	3 points
Unmatched groups	2 points
Groups defined after assessment	1 point
Uncontrolled group	1 point

6) Confounding variables accounted for (time since injury/type of trauma/previous head injury/current psychiatric treatment/previous trauma):

3 or more confounding variables accounted for	2 points
1- 2 confounding variables accounted for	1 point
No confounding variables accounted for	0 points

7) The study indicates how many of the people asked to take part did so:

Opt-in rates are clear	1 point
No opt-in rates reported	0 points

8) The percentage of individuals who dropped out before the study was completed is clear:

Drop-out rates clearly reported/not applicable	1 point
Drop-out rates not reported	0 points

9) The demographics of the sample population were reported (eg age/gender/employment):

Clear reporting of demographics	1 point
Demographics not reported	0 points

10) Appropriate analysis of the data was conducted:

Analysis appropriate for data and aims	1 point
Analysis unsuitable to address aims or for data	0 points

11) Interpretation of analysis based on data provided:

Interpretation based on reported data	1 point
Interpretation based on data other than that reported	0 points

12) Study aims were addressed in discussion:

Discussion or results related to study aims	1 point
Discussion of results not related to study aims	0 points

Maximum score: 21
Minimum score: 3

Grading system:

75%+	A = high quality
60-74%	B = moderate quality
50-59%	C = low quality
0-49%	D = poor quality

Appendix 2.4 – SIGN 50 Grading System (SIGN 50: A guideline developer's handbook, Section 6: Forming guideline recommendations)

Levels of evidence

1++ High quality meta analyses, systematic reviews of RCTs, or RCTs with a very low risk of bias

1+ Well conducted meta analyses, systematic reviews of RCTs, or RCTs with a low risk of bias

1 - Meta analyses, systematic reviews of RCTs, or RCTs with a high risk of bias

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2++ High quality systematic reviews of case-control or cohort studies
High quality case-control or cohort studies with a very low risk of confounding, bias, or chance and a high probability that the relationship is causal

2+ Well conducted case control or cohort studies with a low risk of confounding, bias, or chance and a moderate probability that the relationship is causal

2 - Case control or cohort studies with a high risk of confounding, bias, or chance and a significant risk that the relationship is not causal

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3 Non-analytic studies, e.g. case reports, case series

---

4 Expert opinion

Grades of recommendation

A	At least one meta analysis, systematic review, or RCT rated as 1++, and directly applicable to the target population; or A systematic review of RCTs or a body of evidence consisting principally of studies rated as 1+, directly applicable to the target population, and demonstrating overall consistency of results
B	A body of evidence including studies rated as 2++, directly applicable to the target population, and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 1++ or 1+
C	A body of evidence including studies rated as 2+, directly applicable to the target population and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 2++
D	Evidence level 3 or 4; or Extrapolated evidence from studies rated as 2+

Appendix 3.1 - Submission guidelines for Major Research Project Proposal: Doctorate in Clinical Psychology Handbook 2006-2007

7.8.1 Major Research Project
In accordance with research governance prior to carrying out their Major Research Project all trainees, as employees of the NHS (in Scotland) are required to submit their application for ethical approval to their relevant Research Ethics Committee. Details of how to apply for ethics approval are available at the following URL: http:// www.corec.org.uk/applicants/index.htm . In addition, Research cannot be carried out without Management approval. Advice of local processes on management approval can be sought through local Research and Development Departments. Trainees are unable to submit for ethical or management approval until the Major Research Proposal must have been formally examined and passed by the Research Director.

7.10 Major Research Proposal (including Draft)
The Major Research Project Proposal should include the following headings.

1. Full title of project
2. Trainee Name, Research Supervisor, Field Supervisor and / or Local Lead Investigator
3. Structured Abstract of Project (200 words max)
    - Background
    - Aims
    - Methods
    - Applications
3. Introduction
4. Aims and hypotheses
    - Aims
    - Hypotheses
5. Plan of Investigation
    - Participants
    - Inclusion and Exclusion Criteria
    - Recruitment Procedures
    - Measures
    - Design
    - Research Procedures
    - Justification of sample size
    - Settings and Equipment
    - Data Analysis
6. Health and Safety Issues
    - Researcher Safety Issues
    - Participant Safety Issues
7. Ethical Issues
8. Financial Issues
    - Equipment costs etc
9. Timetable
10. Practical Applications
11. Ethical and Management Approval Submissions
12. References

Appendix 4.1 – Requirements for Submission to Journal of Traumatic Stress

Instructions to Authors

Instructions to Contributors

1. The *Journal of Traumatic Stress* accepts submission of manuscripts online at:

http://mc.manuscriptcentral.com/jots

Information about how to create an account or submit a manuscript may be found online in the "Get Help Now" menu. Personal assistance also is available by calling 434-817-2040, x167.

Please note: This journal does not accept Microsoft WORD 2007 documents at this time. Please use WORD's "Save As" option to save your document as an older (.doc) file type.

2. Three paper formats are accepted. All word counts should include references, tables, and figures. *Regular articles* (no longer than 6,000 words) are theoretical articles, full research studies, and reviews. Purely descriptive articles are rarely accepted. In special circumstances, the editors will consider longer manuscripts (up to 7,500 words)that describe complex studies. Authors are requested to seek special consideration prior to submitting manuscripts longer than 6,000 words. *Brief reports* (2,500 words) are for pilot studies or uncontrolled trials of an intervention, case sudies that cover a new area, preliminary data on a new problem or population, condensed findings from a study that does not merit a full article, or methodologically oriented papers that replicate findings in new populations or report preliminary data on new instruments. *Commentaries* (1,000 words or less) cover responses to previously published articles or, occasionally, essays on a professional or scientific topic of general interest. Response commentaries, submitted no later than 8 weeks after the original article is published (12 weeks if outside the U.S.), must be content-directed and use tactful language. The original author is given the opportunity to respond to accepted commentaries.

3. The *Journal* follows the style recommendations of the 2001 *Publication Manual of the American Psychological Association* (APA; Fifth Edition), with exceptions indicated below. Contributors should refer to this publication when preparing a manuscript for submission. Manuscripts should use nonsexist language. Type double-spaced on one side of 8.5 X 11 inch or A4 white paper using 1-inch margins on all sides and a font no smaller than 12-point.

4. The *Journal* uses a policy of **unmasked review**. Author identities are known to reviewers; reviewer identities are not known to authors or other reviewers. During the submission process, authors may request that specific individuals not be selected as reviewers; the names of preferred reviewers also may be provided. Authors may request blind review by contacting jots@dartmouth.edu prior to submission in order to provide justification and obtain further instructions.

5. The title page should include the title of the article, author's name (no degrees), author's affiliation, acknowledgments, and suggested running head. The affiliation should comprise the department, institution (usually university or company), city and state (or nation) and should be typed as a footnote to the author's name. The suggested running head should be less than 80 characters (including spaces) and should comprise the article title or an abbreviated version thereof. Also include the *word count*, the complete mailing address, telephone and fax numbers, and e-mail address for the corresponding author during the review process, and, if different, a name and address to appear in the article footnotes for correspondence after publication.

6. An abstract is to be provided, no longer than 120 words.

7. Reports of randomized clinical trials should include a flow diagram and a completed CONSORT checklist (available at http://consort-statement.org/Downloads/download.htm). The checklist should be designated as a "Supplementary file not for review" during the online submission process. As of 2007, the *Journal of Traumatic Stress* now follows CONSORT Guidelines for the reporting of randomized clinical trials. Please visit http://consort-statement.org for information about the consort standards and to download necessary forms.

8. Format references in APA style and list them alphabetically at the end of the text. Refer to them in the text by name and year in parentheses. In the text, all authors' names must be given for the first citation (unless six or more authors), while the first author's name, followed by et al., should be used in subsequent citations.

Journal Article
Friedrich, W.N., Urquiza, A.J., & Beilke, R.L. (1986). Behavior problems in sexually abused young children. Journal of Pediatric Psychology, 11, 47-57.

Book
Kelly, J.A. (1983). Treating child-abusive families: Intervention based on skills-training principles. New York: Plenum Press.

Book Chapter
Feindler, E.L., & Fremouw, W.J. (1983). Stress inoculation training for adolescent anger problems. In D. Meichenbaum & M.W. Jaremko (Eds.), Stress reduction and prevention (pp. 451-485). New York: Plenum Press.

9. Tables and figures should be formatted in APA style. *Count each full-page table or figure as 200 words and each half-page table or figure as 100 words.* Tables should be numbered (with Arabic numerals) and referred to by number in the text. Each table should be typed on a separate page. Only black and white tables and figures will be accepted (no color). Figures should be in Word, TIFF, or EPS format.

10. Footnotes should be avoided. When their use is absolutely necessary, footnotes should be formatted in APA style.

11. Submission is a representation that the manuscript has not been published previously and is not currently under consideration for publication elsewhere. A statement transferring copyright from the authors (or their employers, if they hold the copyright) to the International Society for Traumatic Stress Studies will be required before the manuscript can be accepted for publication. The Editor will supply the necessary forms for this transfer. Such a written transfer of copyright, which previously was assumed to be implicit in the act of submitting a manuscript, is necessary under the U.S. Copyright Law in order for the publisher to carry through the dissemination of research results and reviews as widely and effectively as possible.

12. **The journal makes no page charges**. Reprints are available to authors, and order forms with the current price schedule are sent with proofs.

Appendix 4.2 – DSM-IV Criteria for Posttraumatic Stress Disorder (309.81)

A) The person has been exposed to a traumatic event in which both of the following have been present:

(1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others (2) the person's response involved intense fear, helplessness, or horror. **Note:** In children, this may be expressed instead by disorganized or agitated behaviour.

B) The traumatic event is persistently re-experienced in one (or more) of the following ways:

(1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. **Note:** In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.

(2) recurrent distressing dreams of the event. **Note:** In children, there may be frightening dreams without recognizable content.

(3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur upon awakening or when intoxicated). **Note:** In young children, trauma-specific re-enactment may occur.

(4) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

(5) physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

C) Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:

(1) efforts to avoid thoughts, feelings, or conversations associated with the trauma

(2) efforts to avoid activities, places, or people that arouse recollections of the trauma

(3) inability to recall an important aspect of the trauma

(4) markedly diminished interest or participation in significant activities

(5) feeling of detachment or estrangement from others

(6) restricted range of affect (e.g., unable to have loving feelings)

(7) sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)

D) Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:

(1) difficulty falling or staying asleep
(2) irritability or outbursts of anger
(3) difficulty concentrating
(4) hypervigilance
(5) exaggerated startle response

D) Duration of the disturbance (symptoms in Criteria B, C, and D) is more than one month.

F) The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:
Acute: if duration of symptoms is less than 3 months.
Chronic: if duration of symptoms is 3 months or more.

Specify if:
With Delayed Onset: if onset of symptoms is at least 6 months after the stressor.

Appendix 4.3 – Details of Recruitment

As detailed in the Method section, a total of 184 individuals were invited to take part in the study. Fifty individuals consented to taking part, amounting to a 27% response rate. Of these 50 individuals only 21 were recruited to the study, giving an overall recruitment rate of 11.4%. The difficulties in recruiting from this population and the strategies employed to overcome these difficulties are outlined below.

Recruitment Difficulties

A number of individuals who consented to taking part were uncontactable by the telephone numbers they provided. On two occasions this was due to a sudden change of address. The majority of people who consented to take part agreed to arrange an interview time. However attendance rates were poor, with 55% of appointments being unattended. Only one individual who did not attend their first appointment subsequently attended a further appointment. Most participants contacted following a missed appointment requested a further appointment. Reasons for missing the appointment included forgetting, having to do something else suddenly (such as help out a relative or friend), and having a lot of stressful events occurring in their lives at present (eg medical appointments, organising benefits, legal proceedings). A few individuals were offered more than three appointments without attending any, but insisted that they wanted to take part in the study.

Strategies Employed

All participants were sent a letter detailing the time, date and location of their appointment along with directions. Additionally the researcher telephoned them either the day before or the day of their appointment to remind them of the appointment. Travel expenses were offered and participants who appeared to be struggling with many other stresses were offered a taxi to bring them to and from the appointment. Flexibility in appointment times and days was necessary to accommodate other commitments.

While these strategies were not always successful, offering travel expenses and taxis was fundamental in achieving the recruitment of those individuals who did participate as was reminding them of their appointment and providing clear details in writing. Future studies hoping to recruit individuals who have suffered a head injury would be advised to follow similar procedures to enable participants to contribute to research. Additionally, it should be expected that many participants will not attend their appointments and therefore recruiting should focus on establishing a large pool of potential participants. It would be advisable to avoid offering numerous appointments as it became apparent that individuals were unlikely to attend after missing the first appointment. Consideration should be given to a limit being set on the number of appointments offered to one individual to reduce time spent by the researcher arranging unattended appointments.

Appendix 4.5 – Traumatic Memory Inventory

TRAUMATIC MEMORY INVENTORY

Patient name_____ Patient ID# _____
Interviewer_____ Date of interview___/___/___
DES Score_____ PDEQ Score _____

PART I: TRAUMATIC MEMORY

I. INTRODUCTION

1) Age_____
2) Sex ___Male ___Female

Indicate age(s) of trauma(s) on the timeline below

BIRTH_____NOW

Type of trauma(s)

__Sexual abuse/assault
__Physical abuse/assault
__Accident
__Witness death
__Natural disaster
__Being injured (as the trauma)
__Injured/killed someone
__Combat
__Imprisonment/torture
__Emotional abuse
__Death of child
__Other (Specify)

3) Which trauma has had the greatest effect on your life?_____

☞ **Focus on the memories for this trauma for the entire interview.**

4) _____ Age of onset of trauma

5) _____ Total duration of trauma (put X for one-time event)

6) If interpersonal violence is involved, relationship to perpetrator

__1) father
__2) stepfather/mother's boyfriend
__3) grandfather
__4) brother
__5) other male relative
__6) mother
__7) other female relative
__8) family "friend"
__9) teacher or priest
__10) stranger
__11) spouse
__12) acquaintance
__13) other (Specify_____)

_____Total number of perpetrators

II. HISTORY OF MEMORY (write narrative of memory here and on the opposite blank page; be sure to include the information necessary to answer the following questions):

7) Have you always known that this trauma happened to you?
(Was there a time that you had no recollection that these things ever happened to you?)

0	1	2	3
no recollection at times	<---->		always known what had happened

☞ **If answer is 3, skip to question #10**

8) How have you remembered the event(s) over time ?

__1) always had memories, but did not think of events as trauma
__2) always had some memories, but details were filled in later
__3) had period of complete amnesia, now have clear memories
__4) had complete amnesia, filled in some blanks, but missing pieces remain
__5) have fragments of memories, but no coherent picture of what happened
__6) have no clear memories, but feelings, or other evidence makes me believe that I was traumatized

9) Under what circumstances did forgotten memories come up ?

__1) related to anniversary
__2) related to emotions having to do with the trauma (such as intimacy, trust, power, fear, anger)
__3) related to sensory reminders (eg sounds, sights, smells, etc)
__4) retrieved in talking therapy
__5) retrieved in altered state of consciousness (hypnosis, meditation, drugs)
__6) spontaneous (no awareness of precipitants)
__7) other (specify)_____

III. Awareness of Memories

10) How have you remembered the event(s)?

Initially

When you first became aware of what had happened, how was the memory registered in your mind?
(Listen for patient's report first, then probe for specific details, ie What did you see?)

___ (X) As visual images (What did you see?)_____

___ (X) As physical sensations (kinesthetic) (What did you feel?)_____

___ (X) As smells (Olfactory) (What did you smell?)_____

___ (X) As sounds (Auditory) (What did you hear?)_____

___ (X) As intense emotions (Affective) (How did you feel?)_____

___ (X) All of them together (Did you see, feel, smell, and hear at the same time?)_____

___ (X) As a story (Narrative) (Were you capable of telling other people what had happened?)_____

Peak

When you were most haunted by the memories, how was the memory registered in your mind? **(Listen for patient's report first, then probe for specific details, ie What did you see?)**

___ (X) As visual images (What did you see?)_____

___ (X) As physical sensations (kinesthetic) (What did you feel?)_____

___ (X) As smells (Olfactory) (What did you smell?)_____

___ (X) As sounds (Auditory) (What did you hear?)_____

___ (X) As intense emotions (Affective) (How did you feel?)_____

___ (X) All of them together (Did you see, feel, smell, and hear at the same time?)_____

___ (X) As a story (Narrative) (Were you capable of telling other people what had happened?)_____

Currently

When the event(s) come(s) to mind, how do you remember it? **(Listen for patient's report first, then probe for specific details, ie What do you see?)**

___ (X) As visual images (What do you see?)_____

___ (X) As physical sensations (kinesthetic) (What do you feel?)_____

___ (X) As smells (Olfactory) (What do you smell?)_____

___ (X) As sounds (Auditory) (What do you hear?)_____

___ (X) As intense emotions (Affective) (How do you feel?)_____

___ (X) All of them together (Do you see, feel, smell, and hear at the same time?)_____

___ (X) As a story (Narrative) (Are you capable of telling other people what had happened?)_____

How long did it take before you could talk to someone else about what had happened in a coherent fashion ?

___ immediately ___ les than a day
___ less than a week ___ less than a month
___ I still cannot tell the whole story of what happened

How long did it take before you could talk to someone else about what had happened without being interrupted by intense feelings or sensations related to the event ?

___ immediately ___ les than a day
___ less than a week ___ less than a month
___ I still cannot tell the whole story of what happened without getting intense feelings or sensations

11) FLASHBACKS

A. Do you have flashbacks in which the event(s) comes back as if it were happening all over again (while you are awake) ?

__1) yes, currently
__2) used to, no longer
__3) no

☞ **skip to 12 if no flashbacks at all**

B. If yes, does the entire event come back, or only parts of it (ie. just the smell, sound or the hand of the perpetrator)?

__1) entire trauma
__2) fragments
__3) both

☞ **Complete next question only if the flashbacks are fragments of the trauma**

C. If fragments, does the event come back as (check all that apply):

__1) Visual (as images)
__2) Tactile/kinesthetic (physical sensations)
__3) Olfactory (smells)
__4) Auditory (sounds)
__5) Affective (emotions)
__6) All of them together
__7) As a story (narrative)

Compare the modalities from Question #10 (Initially/Peak/Current) with the modalities of the flashback. If different, explain the discrepancies._____
_____
_____
_____
_____
_____

12) How often do memories (flashbacks, nightmares, unwanted memories, etc) of the trauma come to mind without your wanting them to?

A __0) never
__1) daily
__2) 2-4/wk
__3) weekly
__4) monthly
__5) less than once a month

B. Longest intrusion free period
__1) more than a week
__2) more than a month
__3) more than a year

13) CURRENT TRIGGERS
What sort of things trigger memories of the event ?

__1) anniversaries
__2) being upset with people
__3) people being upset with me
__4) other emotions
__5) sensory reminders (such as sounds, sights, smells)
__6) being touched in certain ways
__7) in talking therapy
__8) relived in altered state of consciousness (hypnosis, mediation, drugs)
__9) getting off alcohol or drugs
__10) spontaneous (no awareness of precipitants)
__11) other (specify) _____
__12) nothing triggers memories

14) NIGHTMARES

Do you have nightmares about the trauma ?

__1) yes , currently
__2) used to, but have not had them in 3 months
__3) no

If yes, are they :
__1) Dreamlike (bizarre, illogical)
2) Lifelike: exact representations of some aspect of the trauma- no admixture of other elements
__a) replay of entire trauma
__b) fragments (sights, smells, feelings, etc)
__3) Combination of dreamlike and lifelike

15) **If you have both nightmares and flashbacks, do they have the same content?**

__1) same
__2) different
__3) do not have both

If answer is 2, how are they different?_____
_____

IV. CONTROL AND MASTERY

16) What do you do to control the intrusive memories ?

	in past (X)	currently (X)
1) eating	—	—
2) talking with people	—	—
3) alcohol or drugs (which ones)_____	—	—
4) work, keeping busy	—	—
5) cleaning	—	—
6) religion	—	—
7) being with friends	—	—
8) music	—	—
9) therapy (what sort)_____	—	—
10) self harm (how)_____	—	—
11) sex	—	—
12) sleeping	—	—
13) television	—	—
14) other _____	—	—
15) nothing helps to control the memories	—	—

17) Interviewer

A On the basis of subject's narrative rate for:

__1) Significant functional impairment in effort to avoid re-exposure
__2) Avoids exposure, but no significant effects on occupational or interpersonal functioning
__3) Find self in situations reminiscent of trauma, but unaware of setting it up
__4) Attracted to trauma-related feelings, thoughts or actions.

B. On the basis of subject's narrative, rate cohesiveness of narrative:

0 1 2 3
Least cohesive <---> Most Cohesive

IV. ACCURACY AND CONFIRMATION

Use this scale for question #18 through #19

0 1 2 3
Not at all <---> Completely

18) Do you think that your perceptions of the event(s) have changed over time (ie the role in the trauma or the extent of the trauma)?

0 1 2 3

If yes, in what way ?_____
_____
_____

19) How sure are you that your memories are accurate in regards to:

a) time	0 1 2 3
b) place	0 1 2 3
c) person	0 1 2 3
d) events	0 1 2 3

20) Have you ever checked out what you remember with others ?

__1) Not tried to confirm
__2) Disconfirmed by others only
__3) No confirmation, but no alternative versions are offered by other potential witnesses
(what _____
_____)
__4) Others who knew subject at time of trauma support subject and BELIEVE it is true
__5) Clear confirmatory evidence
(what _____
_____)
__6) Adult trauma; No delayed memories, issue of confirmation not relevant
__7) Other _____
_____
_____

Interviewer's comments about reliability of information

_____
_____
_____
_____
_____
_____

Appendix 4.5 - Additional data collected from TMI

Six participants (28.5%) described integrated memories of the trauma, indicated by all details recalled occurring together rather than separately. Of these three met diagnostic criteria for PTSD on the PDS and one on the CAPS. Two had sustained a mild TBI, three had sustained a moderate TBI and one a severe TBI.

Seven participants (33.3%) were able to give a narrative account of the event, indicated by the ability to describe the events they could recall 'like a story'. Of these three met diagnostic criteria for PTSD on the PDS and one on the CAPS. Six had sustained a mild TBI, three a moderate TBI and one a severe TBI.

Thirteen participants (61.9%) had received information about their injury from a third party after the event. Of these five met diagnostic criteria for PTSD on the PDS and three on the CAPS. Of the thirteen, seven (33.3%) received details of the event itself from witnesses who had observed the event. Of these seven, three met diagnostic criteria for PTSD on the PDS and on the CAPS.

These data are presented in Table 1.

Table 1 – TMI additional data

Participant/Severity of TBI	PTSD PDS	PTSD CAPS	Integrated Memory	Narrative Account	3rd Party Confirmation	Details During Trauma
1 - Sev					●	
2 - Sev					●	
3 – Mild						
4 - Mild				●	●	●
5 – Mod	●		●	●		
6 – Mod			●	●	●	●
7 – Sev					●	
8 – Mod	●	●			●	●
9 – Mod	●	●	●	●	●	●
10 – Mild			●	●	●	
11 - Sev						
12 – Sev	●		●	●	●	
13 – Sev	●				●	
14 - Sev						
15 – Sev	●	●			●	●
16 – Mild			●	●		
17 – Sev	●				●	
18 - Sev						
19 - Sev						
20 – Sev					●	●
21 - Sev						

Appendix 4.6 – Participant Information Sheet and Consent Form

PARTICIPANT INFORMATION SHEET

Study on stress symptoms after head injury

Introduction
You are invited to take part in a research study that is being carried out by the University of Glasgow. Before you decide, it is important for you to understand why the research is being done and what is involved. Please take time to read the following carefully and discuss it with friends, relatives and any medical professionals you have contact with if you wish. If you would like more information or if there is anything that is not clear please ask us.

What is the purpose of the study?
We would like to know more about how people develop stress symptoms after a head injury (for example a knock to the head) so that we can better help people with these problems**.**

Why have I been chosen?
The people who have been invited to take part are all adults who experienced a head injury at least 3 months ago. We will not know whether the people invited to take part have any symptoms of stress until we have carried out the study with them.

Do I have to take part?
No. You can refuse to take part in this study now or at any time during the study. Your treatment will not be affected in any way.

How do I agree to take part?
If you want to take part you should fill in the enclosed consent form, sign it and return it in the envelope provided. I will telephone you or write to you within two weeks of receiving the consent form to set up a time for you to meet and take part.

What will happen to me if I take part?
You will have to travel to an agreed place, which is most likely to be a clinic you have attended already. I will be able to give you the money back for taxi fares to and from the clinic, if you keep the receipt. When you arrive, I will go over the information in this sheet and make sure you understand what will happen. You will complete some questionnaires about your mood, about stress symptoms, about your memory of the event in which you had your head injury, and also about your memory in general. I will go over all of the questionnaires with you.

When completing the questions about your memory of your head injury we will measure your heart rate and also how much you move around by placing a watch on each of your wrists. We realise that your memory of the head injury may be limited. You will also be asked to wear a strap around your chest to measure your heart rate. You will be given the opportunity to put this on in private if you prefer.

You can wear your clothes on top of the strap. The whole study will last for around an hour and a half. You can have regular breaks during this time.

Do you need any other information about me?
The researcher would also like to look at the medical records of your head injury. This is to get some information about how severe your injury was.

Will my taking part in the study be kept confidential?
Your identity and personal information will be completely confidential and known only to Lindsay Smith and Professor McMillan.

Who is organising and paying for the research?
Lindsay Smith from the University of Glasgow is organising the research, supervised by Professor TM McMillan, at the University of Glasgow. The study is funded by NHS Greater Glasgow.

Contact for Further Information
If you have any questions about the study please contact Lindsay Smith on 0141 211 0694.

Thank you for your time and co-operation.

23 August 2006 :Version 2

Lindsay Smith
0141-211-0694

Participant Consent Form

Study on Stress Symptoms after head injury

Please Tick:	YES	NO
Have you read the information sheet?	[ ]	[ ]
Have you received enough information about the study?	[ ]	[ ]
Have you had opportunity to ask questions and to discuss the study?	[ ]	[ ]
Do you have any unanswered questions about the study?	[ ]	[ ]
Do you understand that you are free to withdraw from the study…		
at any time?	[ ]	[ ]
without having to give a reason?	[ ]	[ ]
and without affecting future NHS care?	[ ]	[ ]
Do you agree to being contacted by telephone?	[ ]	[ ]
Do you agree to the researcher accessing medical notes about your head injury?	[ ]	[ ]
Do you agree to take part in the study?	[ ]	[ ]
If you are agreeing to take part, do you agree to the researcher writing to your GP and your clinician at the head injury clinic to let them know?	[ ]	[ ]

Continued on next page, please turn over…

Participant signature: ……. Date: ……

Name in Block Letters: ……..

Witness signature: …….. Date: ……..

Name in Block Letters: ……..

If you agree to take part in the study, I will contact you by telephone to arrange an appointment time and date. Please write down the telephone number you would like her to contact you on:

Preferred contact telephone number ..……

If you have no telephone, I will write to you with an appointment date. Please write down the address you would like me to write to:

Preferred contact address:

……..

……..

……..

……..

……..

Version2:
23/08/2006

Appendix 4.7 – Ethical Approval

Primary Care Division

Divisional Headquarters
Gartnavel Royal Hospital
1055 Great Western Road
GLASGOW G12 0XH
Telephone 0141 211 3600
www.nhsgg.org.uk

NHS Greater Glasgow

Miss Lindsay Smith
Trainee Clinical Psychologist
University of Glasgow
Psychological Medicine
Gartnavel Royal Hospital
1055 Great Western Road, Glasgow
G12 0XH

Date 24 November 2006
Your Ref
Our Ref

Direct line 0141 211 3824
Fax 0141 211 3814
E-mail Liz.Jamieson@gartnavel glacomen.scot.nhs.uk

Dear Miss Smith

Full title of study: **The role of memory for trauma in the development of post-traumatic stress disorder (PTSD) following traumatic brain injury.**

REC reference number: **06/S0701/81**

Thank you for your email of 14 November 2006, responding to the Committee's request for further information on the above research.

The further information has been considered on behalf of the Committee by the Chair.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised.

Ethical review of research sites

The favourable opinion applies to the research sites listed on the attached form.

Conditions of approval

The favourable opinion is given provided that you comply with the conditions set out in the attached document. You are advised to study the conditions carefully.

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document	*Version*	*Date*
Application		29 June 2006
Application	two	23 August 2006
Application		23 October 2006
Investigator CV		29 June 2006
Protocol	2	

Letter of invitation to participant	tw0	23 August 2006
Letter of invitation to participant		29 June 2006
GP/Consultant Information Sheets		29 June 2006
GP/Consultant Information Sheets	3	23 October 2006
Participant Information Sheet: Professional	two	23 August 2006
Participant Information Sheet	two	23 August 2006
Participant Information Sheet		29 June 2006
Participant Consent Form		29 June 2006
Participant Consent Form	two	23 August 2006
Response to Request for Further Information		23 October 2006
Response to Request for Further Information		14 November 2006
Response to Request for Further Information		23 August 2006
Supervisor CV		29 June 2006

Research governance approval

The study should not commence at any NHS site until the local Principal Investigator has obtained final research governance approval from the R&D Department for the relevant NHS care organisation.

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

06/S0701/81	**Please quote this number on all correspondence**

With the Committee's best wishes for the success of this project

Yours sincerely

Liz Jamieson

Liz Jamieson
Research Ethics Committee Co-ordinator on behalf of Dr Paul Fleming, Chair

Enclosures: Standard approval conditions
Site approval form

Copy to: NHS Greater Glasgow Primary Care Division Research and Development Department

Appendix 5.1 – Submission guidelines for Single N Proposal: Doctorate in Clinical Psychology Handbook 2006-2007

7.14 Single N Proposal (Evidence Based Practitioner)

The explicit purpose of the Single N Research Examination is to assess competency in the ability to design recognised single case methodology. In previous years, trainees selected a suitable case from any point in training, and implemented a single case methodology with that patient, which was then included in the final thesis submitted in July of the third year.

However, recent changes in NHS research governance and ethics have severely limited what trainees are able to do within this examination format. Any practice considered to deviate from routine clinical practice must now be submitted for ethical approval. This has made conducting single case design within the Doctorate Programme time scale largely unviable. Increasingly therefore, trainees have become restricted to monitoring treatment phases using tailored patient centred measures, which does not allow the key competencies of the single N experimental study to be properly assessed.

Therefore, a change to curriculum has been introduced. Trainees now are required to submit a single case experimental design research proposal. This must outline a single case based on a case seen during placement, although the intervention need not actually be implemented. Trainees are not therefore required to deliver the single N case design with that patient, merely to design it. This new format allows better assessment of single N competencies whilst being responsive to the changing requirements of NHS clinical governance, research governance and ethics.

As before, the Single Subject Research Study should be designed to address an area of conceptual and/or clinical importance. It must address a hypothesis or answer a question, and it must be presented in the context of the available published literature. Trainees are urged to consider opportunities for selection of the single N research proposal during any clinical placement. The range of appropriate methodologies includes single case designs incorporating procedures for experimental control (e.g. reversal phases, multiple baseline measurement), and time series analyses. Appropriate quantitative measurements must be detailed in the proposal and, where appropriate, statistical procedures and tests outlined. Qualitative proposals may also be acceptable providing recognised procedures are described, and recognised methods for analyses and presentation qualitative data outlined. Simple narrative case presentation and uncontrolled case study proposals will not be acceptable.

In order to protect the anonymity of the subject it is essential that all identifying information is removed from the proposal prior to presentation for examination purposes. Furthermore, only the abstract from the single Case Research Proposal should be bound into the Research Portfolio and this must be similarly anonymised.

The proposal should adhere to the following broad format:

1. Title
2. Abstract
3. Relevant theoretical and clinical literature
4. Case description
a. Brief background
b. Presenting Problems
c. Theoretical Formulation
d. Hypotheses
5. Methodology
a. Design
b. Measures
c. Procedures
d. Data Analysis
6. Ethical Issues
7. Practical Applications.

Appendix 5.2 – DSM-IV Diagnostic Criteria for Conversion Disorder (300.11)

A) One or more symptoms or deficits affecting voluntary motor or sensory function suggest a neurologic or other general medical condition.

B) Psychological factors are judged to be associated with symptom or deficit because initiation or exacerbation of symptom or deficit is preceded by conflicts or other stressors.

C) The symptom or deficit is not intentionally produced or feigned (as in factitious disorder or malingering).

D) The symptom or deficit cannot, after appropriate investigation, be fully explained by a general medical condition or by the direct effects of a substance or as a culturally sanctioned behaviour or experience.

E) The symptom or deficit causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or warrants medical evaluation.

F) The symptom or deficit is not limited to pain or sexual dysfunction, does not occur exclusively during the course of somatisation disorder, and is not better accounted for by another mental disorder.

G) The type of symptom or deficit should be specified as follows: (1) with motor symptom or deficit, (2) with sensory symptom or deficit, (3) with seizure or convulsions, or (4) with mixed presentation.