Trauma Memories and Disturbances in Autobiographical Memory

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Declaration

This thesis is submitted for the degree of Doctorate in Clinical Psychology at the University of Sheffield. It has not been submitted for any other qualification or to any other academic institution.

Word count

Section 1- Literature Review: 7780

Section 2 - Empirical project: 8736

Section 3 - Appendix: 3366

Whole thesis: 165150

(Without references and appendix)

Whole thesis: 23175

(Including references and appendix)

Thesis Abstract

This thesis consists of a literature review and a research project. The review considers whether trauma memories are distinctive when compared to non-traumatic memories in the autobiographical memory base. Electronic databases were searched to identify published quantitative studies on the organisation and integration of memories in posttraumatic stress disorder (PTSD) in the adult population. Nine studies met the inclusion criteria and were included in the review. Studies were split into four groups: (i) PTSD versus non-PTSD, (ii) brain injury, (iii) PTSD only and (iii) high versus low PTSD. Results across the four groups were consistent, indicating that trauma memories are recalled in the present tense, have higher emotional intensity and greater rehearsal. Trauma memories did not appear to be disconnected, but are central to autobiographical memory.

The research report investigates whether disruptions in peri-traumatic processing (i.e., data driven processing, dissociation and self-referent processing) and trauma memory (i.e., disorganisation and intrusion) can explain PTSD symptom severity post-stroke. Stroke survivors ($N = 80$) were recruited from six-week follow-up clinics and completed questionnaires assessing PTSD symptoms, trauma memory and cognitive processing during stroke. The results showed that PTSD symptom severity was significantly correlated with age, time since stroke and all memory variables. Regression analyses revealed that self-referent processes and intrusion were the only predictors of PTSD severity. Further analysis highlighted that the effect of self-referent

processing on PTSD symptom severity was mediated by intrusion. The study provides support for Ehlers and Clark's cognitive model of PTSD.

Acknowledgments

This doctoral thesis would not have been possible without the encouragement and support of the wonderful people around me. First and foremost, I would like to offer my sincere gratitude to my supervisor Professor Paul Norman, who has supported me throughout my whole thesis offering me constructive feedback, immense knowledge and encouragement. I could not have wished for a better adviser and supervisor.

I would also like to express my greatest appreciation to my clinical supervisor Dr Jane Barton, who from the onset has provided me with her expertise in the area and provided valuable contacts, insights and support.

I am particularly grateful for the assistance given to me by Dr Hazel Reynders and Dr David Griffith; without their direction and advice, this thesis would not have materialised. In addition, I would also like to thank Dr Rosario, Dr Pratt and Amanda Jones for giving me permission to conduct my recruitment from their clinics. I would like to extend my gratitude to the nurses for all their help in the recruitment phase.

I would like to acknowledge the financial support provided by Sheffield Health and Social Care NHS Trust as well as the academic and technical support provided by the Clinical Psychology Unit at the University of Sheffield.

Thank you to my fellow course friends who offered advice and helped me retain a sense of humour along the way.

I also wish to express my love and gratitude to my parents, my family and friends for their understanding and compassion throughout this process.

Finally, my deepest heartfelt appreciation goes to my husband Saqib Nawaz, who has been my rock throughout this process and has kept me going through the highs and lows.

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Are trauma memories incorporated and retrieved differently to non-trauma memories in autobiographical memory?

Situations that are perceived as traumatic can happen to anyone at any time. There has been considerable research conducted into the onset of Post-Traumatic Stress Disorder (PTSD) following traumatic events. The Diagnostic and Statistical Manual of Mental Disorders (4th ed.; DSM-IV; American Psychiatric Association, 1994) classifies PTSD as an anxiety disorder. When patients who have a diagnosis of PTSD discuss their trauma, they refer to flashbacks, miss parts of the event, feel disconnected and recollect the most traumatic parts in a disorganised and jumbled way. Thus, research has focused on how trauma memories are perceived by the individual and stored in autobiographical memory, and how autobiographical memories may differ for trauma and non-trauma events.

The autobiographical memory system is described as a combination of episodic memories consisting of personal events (such as people and time) and semantic memories (such as facts and general knowledge) about the world (Williams, Conway, & Cohen, 2008). Conway and Pleydell Pearce (2000) propose a model of how autobiographical memory is constructed and retrieved. They emphasise the role of the 'self' in relation to an underlying knowledge base which stores information in a hierarchy comprising of life time events, general events and event specific knowledge (ESK). When new information is received, it is processed through the self-memory system, which contains information about personal goals, cognition, behaviours and beliefs, providing a framework for the individual to interact with the world.

Memory is retrieved in two ways: generatively and directly. When information is retrieved generatively, it is recalled intentionally using a model of retrieval set up by the self-memory system. Elaboration cues are used to search records in the knowledge

base (i.e., lifetime, general and event specific records) and verification criteria interacts with central control processes (i.e., self-goals, cognitions, behaviours) to generate a memory. However, in direct retrieval the self-memory system only operates when memory construction has taken place. ESK is activated by patterns and cues without reference to the retrieval model causing spontaneous memory.

Furthermore, the model highlights that traumatic situations pose a threat to the self, as there are no current goals and plans in the self-memory systems to regulate the trauma experience. As the self-memory system cannot normalise or process the trauma experience it is integrated into long term memory with the goals of the self rather than in the knowledge base. Thus, when goals are activated, trauma related ESK is also triggered resulting in intrusive memories.

More recently, William et al. (2008) suggest that autobiographical memory processes use pre-existing schemas in order to reconstruct and remember past events and there are four ways in which autobiographical memories may be processed. First, memories can be processed using specific details about an event that has occurred, such as the colour of a dress worn to a party, or they can be processed more generally, for example, going to a party. Second, memories can either be processed as copies, where memories are based on vivid visual and sensory details, or as reconstructions where new information is stored as hindsight. Third, memories can be stored as biographies, which are important to who you are, for example, your graduation day or getting married. Fourth, memories can be stored as first person or third person accounts. Rubin (1995) suggests that during a highly emotive event, such as war, memories are likely to be encoded in the third person perspective. This is a protective factor, and happens to defend the individual from difficult feelings that occur during the event. Using the third person perspective in response to trauma means that the individual can distance

themselves. However, whilst this may be beneficial in the short term, it can cause difficulties with processing difficult memories, and may lead to PTSD.

Two main psychological theories have been developed to explain how traumatic experiences and the disruption of encoding, storage and retrieval of information into the autobiographical memory store can lead to, and maintain, PTSD. One model is proposed by Ehlers and Clark (2000). This cognitive model states that PTSD symptoms become persistent when the individual keeps processing the traumatic event in a way where there is always a current sense of threat. Ehlers and Clark (2000) propose that this happens in two ways. In the first, the individual has excessively negative appraisals of the trauma and/or its sequelae. This occurs because the individual overgeneralises from the traumatic experience and, as a consequence, perceives everyday activities as risky and threatening. Negative appraisals of one's behaviour during the event can also create unwarrantable implications for the individual, which maintain a sense of threat for the individual. In addition, negative appraisals about the trauma sequelae, such as consequences of the trauma in current life, or others' reactions to the trauma (e.g., others not talking about it), can sustain a current sense of threat. Furthermore, negative appraisals of emotional responses during traumatic situations such as guilt ('it's my fault'), loss ('my life has changed forever') and anger ('I have been treated unfairly') can also contribute to persistent PTSD. In the second way, Ehlers and Clark (2000) propose that the nature of trauma memory may also contribute to persistent PTSD. The model proposes that normal autobiographical memories are processed through a higher-order system which is meaning based. This route ensures that memories are not intentionally retrieved during everyday tasks. Furthermore, this ensures that memories are stored in context and time. On the other hand, trauma memories are poorly elaborated into autobiographical memory, they are not stored in context or time and are triggered by stimuli which are associated with the trauma. In addition, traumatic

memories have strong association memories which lead to re-experiencing. Furthermore, the model also highlights the relationship between trauma appraisals and trauma memories, and states that one process is dependent on the other. Thus, if one has disrupted memory of the trauma, only negative information may be retrieved about the trauma maintaining the cycle.

The second psychological theory, proposed by Brewin (2001), is the Dual Representation Theory, which is based on the notion that there are multiple memory systems that work together and independently to process autobiographical memories. Brewin states that there are many features of traumatic events, such as sounds, smells, and sights and these are initially retained in a memory system called situationally accessible memory (SAM). This SAM system analogous the episodic memory store. Brewin (2001) explains that after a traumatic event the individual will consciously reflect and think about the event. This process ensures that the memory becomes integrated, and stored into a different system called the verbally accessible memory system (VAM), which equivalents the semantic memory store. However, peri-traumatic processes during trauma, such as distraction, negative mood and dissociation, can disrupt the process of storing the memories in the verbally accessible system, and the memories stay in the situationally accessible memory. This, in turn, increases the likelihood of PTSD.

A number of reviews have been conducted on research addressing different aspects of trauma memories. Bedard-Gilligan and Zoelinger (2012) conducted a recent review of the literature specifically examining the link between the role of dissociation and its effect on memory fragmentation (i.e., perception of one's own memory). In total, they included 16 studies, and the results indicated mixed evidence for an association between dissociation and memory fragmentation. However, some studies

found that those individuals who reported higher levels of peri-traumatic dissociation were more likely to have memory fragmentation. Furthermore, trait dissociation was reliably associated with meta-memory fragmentation. Bedard-Gillingan and Zoelinger concluded that further research on the role of dissociation and memory fragmentation is required that employs a more precise definition of key terms, such as dissociation, and stronger research designs (e.g., using multiple measures) that account for factors such as distress and cognitive ability, which may influence both dissociation and fragmentation.

There has also been a body of research that has utilised an experimental design to simulate peri-traumatic processes. This trauma film paradigm was devised in the 1960s by Lazarus and colleagues (Lazarus & Albert, 1964; Lazarus & Opton, 1964), initially to examine the physiological responses that occur while participants watch a traumatic film. This paradigm further developed to incorporate the monitoring of film-related intrusions (Butler, Wells, & Denwick, 1995). Participants are usually recruited from non-clinical backgrounds and are shown a film which depicts stressful or traumatic events including, industrial accidents (Butler et al., 1995) and a workplace film about fire safety (Stuart, Holmes, & Brewin, 2006). Holmes and Bourne (2008) recently reviewed the trauma film paradigm, specifically examining the development of intrusive emotional memories. They concluded that watching traumatic films can produce PTSD-like symptoms (such as intrusive memories of the film content, dissociation, fear and avoidance). However, there is debate in the literature about whether watching a film of individuals that have no connection with the audience can produce the exact same feelings as experiencing a traumatic event which happens to one self, or witnessing it in real life.

Brewin (2007) conducted a review examining four areas of autobiographical memory and trauma. First, the review examined forgetting trauma memories, and

recalling them unexpectedly later, by evaluating the literature on 'recovered memories'. Brewin concluded that research to date has been unable to identify the processes that highlight why traumatic memories can be forgotten, and then retrieved later on. Second, the review also examined if special memory processes were used to forget traumatic events (such as dissociation or repression). Brewin concluded that traumatic memory is likely to be forgotten through normal memory processes, such as decaying and blocking, as new information is learnt. Third, the review examined whether trauma memory is distinctive when compared with other memories in the autobiographical store. Any differences in traumatic memory were only found in clinical samples and contained features of reliving and sensory information. Fourth, Brewin reviewed whether high levels of emotion impair or enhance memory for traumatic events. The evidence from animal studies indicated that the same biological mechanisms are involved to either enhance or impair memory for traumatic events. However, real life traumatic events are complex, and depending on how recall is measured, traumatic memory can be seen as better (i.e., clear in recall) or worse (i.e., have missing details) than non-traumatic memory. Brewin concluded that future investigations should be carefully controlled, and individuals should be able to provide details about traumatic and non-traumatic memories over time.

To conclude, the literature highlights a link between trauma memories and the probability of developing PTSD. The aim of the current review is to examine whether traumatic memories are assimilated and recalled differently to non-traumatic memories within autobiographical memory. The review will also examine if traumatic memories are recalled differently in those individuals with PTSD, compared to those who do not develop PTSD following a traumatic event, as outlined in DSM IV. These questions have not been addressed in previous reviews (Brewin, 2007; Bedard-Gilligan & Zoelinger, 2012; Holmes & Bourne 2008).

Aims

The main aims of this review are:

1. To examine how trauma memories in individuals with and without PTSD are incorporated and retrieved from autobiographical memory.
2. To examine whether retrieval of trauma memories in individuals are different from the retrieval of non-trauma memories comparing individuals with and without PTSD.

Method

Identification of studies

Three electronic databases (Web of Knowledge, PsychINFO, and Medline) were searched for relevant articles to be included in the review. The searches spanned from 1980 (when PTSD became a diagnosis in DSM III) to 31 January 2013. To identify relevant studies, the terms *autobiographical memory/memories, post traumatic stress disorder/PTSD, trauma memory/memories, and acute stress disorder/ASD* were entered into the search engine for each database. Within the searches the Boolean operator "AND" was used with different combinations of the search terms. Furthermore, the citation histories and references of relevant articles were also scanned manually to identify any further studies to be included in the review.

Inclusion and exclusion criteria

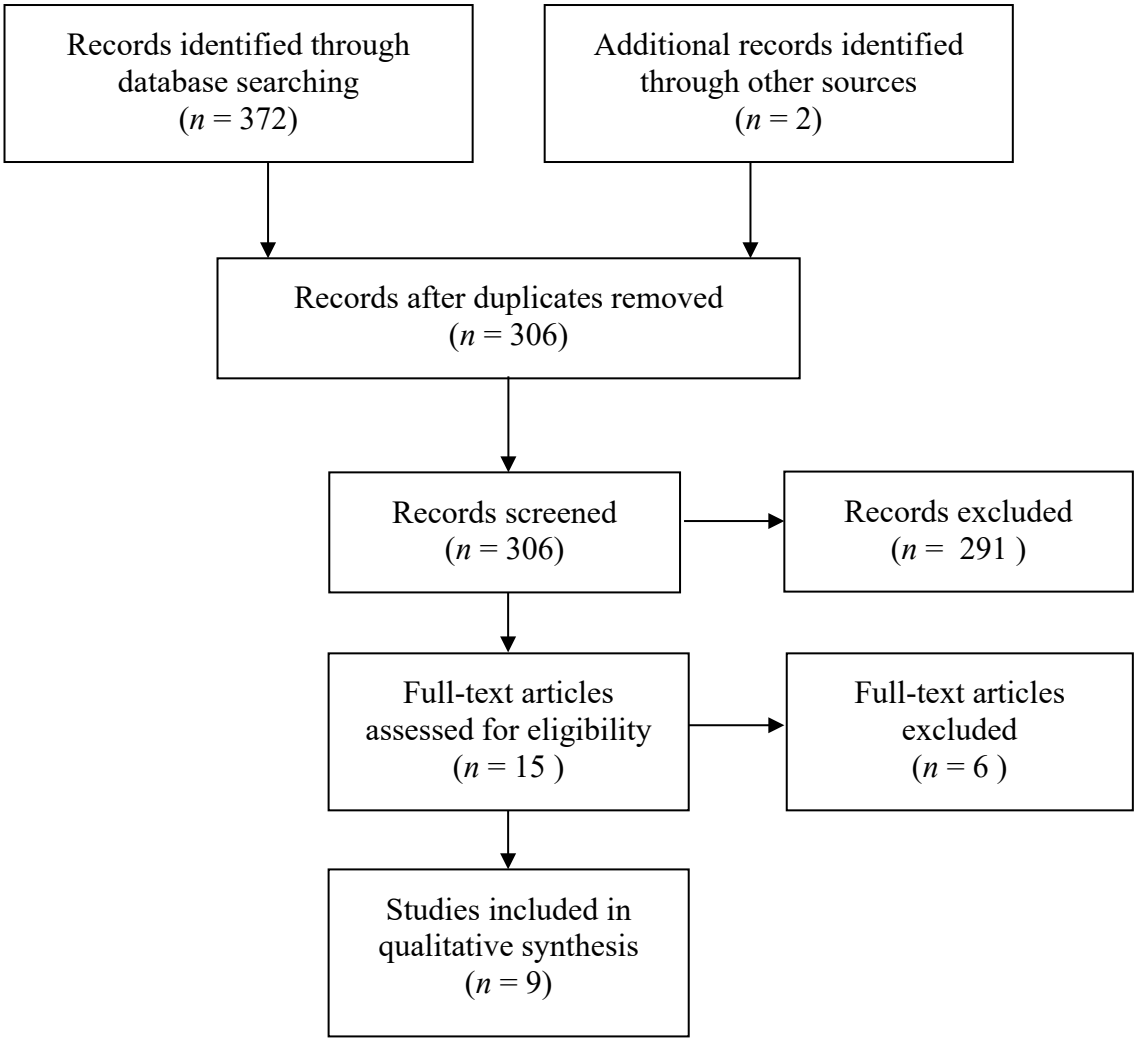
Studies examining trauma memory in relation to autobiographical memory were included in the review. Those studies looking at other types of memory (i.e. semantic) were excluded. Furthermore, only studies looking at a clinical sample of participants who had been diagnosed with PTSD, or those who had survived a traumatic event and were experiencing symptoms of PTSD as described by the DSM–IV, but did not have a formal diagnosis, were included in the review.

Non-English studies were excluded from the review, as were any studies examining PTSD and autobiographical memory in children aged between 0 and 18 years. Furthermore, papers examining the trauma film paradigm were also excluded from the review.

Study selection

From the initial search, a total number of 374 studies were found, combining the results from the different databases. Screening was conducted on a number of levels. First, all articles were initially scanned for duplicates, and 68 were removed following this process, leaving 306 articles. Second, the titles and abstracts of these 306 articles were examined, and after applying the inclusion/exclusion criteria, 291 were excluded; 98 articles researched the trauma film paradigm, 42 focused on children and adolescents, 31 looked other aspects of memory (i.e., declarative memory), 28 looked at verbal memory and trauma, 21 examined voluntary and involuntary memories not in relation to PTSD, 19 examined the role of fragmentation, 17 examined dissociation, 12 examined responses to PTSD retrospectively, but not in relation to autobiographical memory, 11 focused on hormone, anxiety or emotional interference and the development of PTSD, 7 looked at medical drugs and PTSD, and 5 looked at eye movement desensitisation and reprocessing therapy. Thus, 15 articles remained to be included in the review. However, six of these articles were removed after reading the full papers, as they did not fully meet the inclusion criteria (i.e., three examined risk factors for PTSD, one examined cue words and memory, one examined disorganisation in memory and onset of PTSD, and one examined behavioural observations). Therefore, a total of nine studies were included in the final review.

Figure 1. PRISMA Diagram (Moher, Liberati, Tetzlaff, Altman, 2009).



Data organisation

For the initial analysis, the nine papers were split into four groups. The studies in group one had one sample of participants who were diagnosed with PTSD and compared trauma memories and non-traumatic autobiographical memories for those individuals. The studies in group two compared traumatic/unpleasant memories in those who had PTSD and those who did not have PTSD. The study in group three had one sample of participants with PTSD, and compared those with higher and lower scores on PTSD measures to ascertain whether severity of PTSD impacts on autobiographical memory processes. The study in group four focused on traumatic brain injury (TBI) as a result

of road traffic accidents, and examined whether patients who experienced TBI were more likely to show PTSD symptoms than those without.

Quality assessment

To assess the quality of the remaining nine papers, a standardised checklist by Downs and Black (2009) was used. This checklist was originally created to access the quality of both randomised and non-randomised studies in health care settings. The checklist was used because it has high internal consistency (KR-20 = 0.89), good external validity (KR-20 = 0.54), and high scores for test-retest reliability (r = .88). None of the papers included in the review were randomised control trials (RCTs) and, as a result, questions relating specifically to RCTs were scored as 'not applicable'. Therefore, the scale was modified for the final review (See appendix A). The final checklist consisted of 19 items with a maximum score of 19. All studies were prospective or cross-sectional in design and were therefore rated together. Quality scores ranged from 12 – 14 with a mean of 13. In order to check the reliability of the ratings, a second rater, a final year trainee clinical psychologist, selected five of the papers at random from each group. As group three and four only consisted of one article, another paper was selected at random from the remaining articles and was from group two (Jelinek et al., 2010; Jones, Harvey, & Brewin, 2007; Kleim, Wallott, & Ehlers, 2008; O'Kearney, Hunt, & Wallace, 2011; Rubin, Berntsen, & Boals, 2008).

To address the level of agreement between raters, differences between the positive and negative agreement scores were calculated and incorporated into kappa calculations. Interrater reliability was kappa = .67, p =.005. A value between .61 - .80 is considered to indicate substantial agreement between raters (Lardis & Koch, 1977). As all studies scored similarly in terms of quality, all studies were considered in the review.

Data extraction

Once all studies had been quality rated, further data was extracted from each study and tabulated. Details of date of publication, country of origin, type of design used, main aims, sample size, recruitment location, main exclusion criteria and main outcomes were recorded (see Table 1).

Table 1

Data Extraction

Authors, Year and Country	Study Design, Recruitment	Main Aim	Sample, Exclusion Criteria	Main Findings	Quality Rating Scores
Group 1					
Hellawell & Brewin (2004) UK	Prospective Recruitment: Variety of local hospitals and veteran services	To test the theory that periods of flashback will differ from periods of ordinary memory in specific aspects of content and grammatical form.	*N* = 62 Male *N* = 48 Inclusion criteria: Not adequately described	Flashbacks differed from ordinary memory; they contained primary emotions such as fear.	12
Rubin, Feldman, & Beckham (2004) USA	Prospective Recruitment: Durham veterans affairs medical center	Four types of autobiographical memories (i.e., two years before service, non-combat memory, a combat memory and an intrusive memory) were examined from war veterans to examine any differences in fragmentation, cognitive and visceral emotions and reliving.	*N* = 50 100% Male Exclusion criteria: Not described	Trauma related memories showed an increase in cognitions and visceral emotions.	12
Peace, Porter, & ten Brinke (2008) Canada	Within subjects Recruitment: Through media	To look at the characteristics of trauma memories for sexual violence, non-sexual trauma memories and a positive experience.	*N* = 44 Male: *N* = 0 Exclusion criteria: Not reported	Sexually traumatic events were no more fragmented or impaired than other trauma memories.	13

O'Kearney, Hunt, & Wallace (2011) Australia	Prevalence study Recruitment: Not reported in sufficient detail	To examine PTSD symptom severity and the integration of trauma memory, and to examine PTSD symptom severity and trauma memory organisation using participants' self-reports.	*N* = 47 Male: *N* = 9 Exclusion criteria: Not reported	Inhibition or enhancement of the integration of trauma memory within autobiographical memory was not associated with severity of PTSS.	14
Group 2					
Kleim, Wallott, & Ehlers (2008) UK	Cross-sectional study	To experimentally test the hypothesis that trauma memories are poorly integrated with other autobiographical information.	*N* = 74 Male: *N* = 45 Exclusion criteria: Not reported	Those with PTSD took longer to retrieve information while imagining the worst moment of their assault. PTSD participants also took longer to retrieve autobiographical memories during hot spots. Thus, it appears that trauma memories are disjointed from autobiographical memory.	13
Jelinek, Stockbauer, Randjbar, Kellner, Ehring, & Moritz (2010) Germany	Cross-sectional study Recruitment: Participants recruited through media advertising and through university medical center, and department of psychiatry and psychotherapy.	To compare worst moment of trauma memories of participants with and without PTSD.	*N* = 81 Male: *N* = 37 Exclusion: History of psychotic symptoms, neurological disorder, TBI or alcohol/substance dependence.	Non-PTSD group had longer narratives. Less cognitive processing in PTSD group. Affective words and alexithymia were negatively related and more present tense was	14

				used in worst moments for PTSD group.	
Rubin, Dennis, & Beckham (2011) USA	Prospective Study Recruitment: From community	To compare most stressful memories to other memories in both voluntary and involuntary recall.	*N* = 177 Male: *N* = Not reported Exclusion: Meeting the criteria for substance or alcohol use and psychotic disorders. Additionally, in the control group, meeting the PTSD criteria currently or in the past.	There were consistent differences between the control and PTSD group in rehearsal emotions and centrality of memories in the autobiographical base. Further, these differences were shown in both voluntary and involuntary memories. Trauma memories had less emotional regulation and are relived in the present tense.	12
Group 3					
Rubin, Berntsen, & Boals (2008) USA	Cross-sectional study Recruitment: Undergraduate students	To conduct three way comparisons of memories for stressful events, between voluntary and involuntary memories. To compare the differences in those with high PTSD symptom severity versus those with low PTSD symptom severity.	*N* = 115 Male: *N* = 44 Exclusion: Potential participants who scored below 25 on the PTSD checklist (PCL) and potential participants who scored above 40 on the PCL.	Results found that differences in personality traits (e.g., neuroticism) were found among those with high and low PTSD symptom severity. Those with high PTSD symptom severity reported more emotional intensity and more centrality of events. Further, involuntary memories had less emotional regulation, had more emotional reaction and were less specific.	14

Group 4					
Jones, Harvey, & Brewin (2007) UK	Cross-sectional and prospective Recruitment: A&E and trauma ward	To extend previous findings that narrative disorganisation predicts future PTSD and address the discrepant findings concerning the association between recovery from trauma and decrease in narrative disorganisation.	*N* = 131 Male: 60.3 % Inclusion criteria: Fluent English, age between 18 - 65, medically well enough to take part within 14 days of trauma, resident within 30 km of Oxford city centre.	Those with ASD had less coherent narratives and dissociative content 1 & 6 weeks post-trauma. Three months post-trauma PTSD narratives showed more repetition, more non-consecutive chunks, less coherence and were more dissociated. TBI participants showed more confusion, 1 and 6 weeks and 3 months post trauma. Disorganisation in trauma narrative shortly after traumatic event predicted PTSD severity at three month follow-up.	13

Results

Samples

The nine studies included in the review all had small samples ranging from 44 (Peace, Porter, & ten Brinke, 2008) to 131 (Jones, Harvey, & Brewin, 2007). Using smaller samples has limitations as it increases the chance of making type II errors (i.e., false negative). There is also a greater risk of bias in the results with smaller samples and this can affect the generalizability of the research. Peace et al. (2008) recruited only female participants who had experienced a sexually traumatic experience, and Rubin et al. (2004) recruited only male war veterans. As both studies recruited small samples from very specific groups it may be more difficult to generalise from their findings.

PTSD measures

The majority of studies only used self-report questionnaires, such as the Posttraumatic Diagnostic Scale (Foa, 1995), in order to assess for PTSD symptoms and severity. Although these measures follow a structured pattern in line with the DSM-IV criteria, there are many limitations in only using self-report assessments of PTSD. Relying solely on participants' self-reports means that the validity of the study may be impaired; for example, answers may be open to bias due to social desirability effects, or demand characteristics.

Two studies (Jelinek et al., 2009; Kleim et al., 2008) used the Structured Clinical Interview for DSM-IV (SCID; Spitzer, Gibbon, Williams, & First, 1996) and Rubin et al. (2004) used the clinician administered PTSD scale (Blake et al., 1995), alongside self-report questionnaires, to ascertain PTSD symptomology. The advantage of using this type of interview over just self-report measures is the clinically trained interviewer can use other qualitative observations to make a judgement about the quality of the participant's self-report, rather than relying on the participant. This approach is

considered to be the "gold standard" for PTSD diagnosis (Foa, Keane, Friedman, & Cohen, 2010).

Trauma memories

Different methods were used to ask participants about their trauma memories. Hellawell and Brewin (2003) and Peace et al. (2008) asked participants to provide written narratives of their traumatic events. Asking for written narratives can be problematic because it relies on participants being literate and able to express themselves in a written format. Providing a written account may be difficult for individuals whose first language is not the one that they are being asked to write in (i.e., English). It may also be problematic for individuals to provide written accounts if they have a learning disability or difficulty.

To overcome these difficulties, three studies (Jelinek et al., 2010; Jones et al., 2007; O'Kearny & Hunt, 2011) asked for verbal accounts and analysed transcripts of the interviews. However, this method can be open to bias as other factors such as non-verbal behaviour may influence participants' accounts. Two studies (Rubin, Bernsten, & Boals, 2008; Rubin, Feldman, & Beckham, 2004) used items from the Autobiographical Memory Questionnaire (AMQ) to measure a nominated memory. Using this type of measure ensures that there is structure to the information provided by participants. However, with all methods it is difficult to assess the accuracy or reliability of participants' accounts.

Rehearsal effects

Another methodological limitation of these studies is that they do not state how long ago the trauma occurred, and whether participants had received professional help to manage symptoms of PTSD. The NICE (2005) guidelines recommend that Cognitive Behavioural Therapy is used in the first instance to treat PTSD, and this may mean that

participants had told and re-told their narrative many times. This, in turn, may mean that the trauma memories are less fragmented which may affect the validity of the studies.

Group 1 - Comparing memories in those with PTSD

Four studies (Hellawell & Brewin 2004; O'Kearney & Hunt 2011; Peace, Porter, & ten Brink, 2008; Rubin, Feldman, & Beckham 2004) recruited clinical samples of people diagnosed with PTSD, and compared their trauma memories with non-trauma memories, to establish whether there are differences in the way the memories are processed.

O'Kearney and Hunt (2011) recruited 47 participants who had been exposed to a traumatic event. They specifically looked at the integration and organisation of the traumatic memory into personal memory, and PTSD symptom severity. Results showed that although trauma memory was more distant than the non-trauma memory, it was rated as more important. No relationship was found between trauma memory and the severity of post-traumatic stress symptoms. Trauma memories were better integrated into autobiographical memory than non-trauma memories. Therefore, the more important the memory is to the self, the more integrated it is in autobiographical memory, and the quicker it is to retrieve.

Hellawell and Brewin (2004) examined the dual representation theory (DRT) of PTSD. According to DRT, traumatic memory can be stored in different formats, i.e., in the verbally accessible system, where memories can be edited and interact with other processes, or in the situationally accessible system, where memories do not interact with other information, and can occur as dreams or flashbacks. Participants with a diagnosis of PTSD ($N = 62$) were recruited, and a written narrative of their trauma was analysed for the content and language used in flashback memories, and compared to ordinary

memories. Results showed that participants reported more detail, greater use of present tense, and emotions such as fear and hopelessness when describing a flashback. In contrast, ordinary memory descriptions showed more emotions that expressed guilt and anger.

Two of the studies recruited participants who had been through a specific traumatic event. In the first study, Rubin, Feldman, and Beckham (2004) examined four narratives, (pre-service, non-combat, combat and intrusive) of 50 male war veterans. Narratives were analysed to examine experiences of reliving, emotions and fragmentation, within each narrative. Results showed that trauma memories were not more fragmented than non-traumatic memories; however, they did differ emotively, such that visceral emotions were higher in trauma memory. Furthermore, the results found that severity of PTSD symptomology was related to increased reliving. Both studies reinforced that although trauma memory is not-fragmented, it does differ in content and emotion.

In the second study, Peace, Porter, and then Brinke (2008) recruited 44 female participants who had a sexually traumatic experience. They compared this memory with another traumatic experience that was not sexually related, and a positive experience. They hypothesized that sexually traumatic experiences are processed differently, and therefore recalled differently. The results showed that sexually traumatic experiences were not fragmented or impaired when compared to other memories. However, they found that these trauma memories had higher sensory components and more vivid detail.

Although the aforementioned studies examined slightly different hypotheses and had different samples, the results have many similarities in that they indicate that trauma memories are not disorganised. In fact, they appear to be more organised than

non-traumatic memories. However, trauma memories have more present tense emotions and detail and a centrality to the autobiographical memory store, rather than being disconnected from it.

Group 2 - PTSD vs. no PTSD

Three studies (Jelinek, Stockbauer, Randjibar, Kellner, & Ehring, 2010; Kleim, Wallott, & Ehlers, 2008; Rubin, Dennis, & Beckham, 2011) looked at the memories of trauma survivors, and compared individuals with and without PTSD. Kleim, Wallott, and Ehlers (2008) investigated whether trauma memories are disjointed from other autobiographical memories in those who have PTSD. Participants completed an autobiographical memory task during a taped imagery script, and were asked to describe two types of memory: the assault and another negative event. They also asked participants about the worst moment (hotspot) during the trauma memory. The results indicated that both the PTSD and non-PTSD groups reported dissociative symptoms in the trauma script. However, the PTSD group had greater dissociation and negative emotions in both scripts. The PTSD group also took longer to retrieve autobiographical information when imagining a hotspot in their trauma narrative. This supports the hypothesis that hotspots in trauma are poorly elaborated into autobiographical memory, and are recalled in the present tense with heightened emotional response and a sense of reliving.

Jelinek et al. (2010) compared the worst moments (hotspots) of trauma memory in those with and without PTSD, focussing on intrusions. Single trauma participants (*N* = 81) were recruited and asked to complete measures to assess for PTSD and alexithymia (difficulty expressing and describing emotions verbally), and to provide verbal narrative accounts of the traumatic event. The narratives were analysed for emotional expression, cognitive processes and linguistics. The results showed that in

the non-PTSD group, participants provided longer trauma narratives, as well as providing more anxiety words. In contrast, participants in the PTSD group displayed higher levels of threat, higher levels of alexithymia, and their narratives had fewer words and disorganisations in hotspots. Both groups showed physiological responses, but the type of response was different in both groups. For example, the non-PTSD group used more words indicating anxiety, and the PTSD group used words connected to fear. Although anxiety and fear can be interlinked, they do have different neurological pathways. Therefore, the results indicate that memories of those with PTSD may be different physiologically, and the presence of PTSD symptoms indicates disruption in the processing and retrieval of traumatic memories.

The most recent study, conducted by Rubin, Dennis, and Beckham (2011), compared positive and most stressful memories in participants with and without PTSD. They also looked at voluntary versus involuntary memory, to find out whether they differed in content. When most stressful memories were compared between PTSD and non-PTSD participants, the results indicated that having PTSD does affect some properties (i.e., more rehearsal, frequent involuntary recall, remembering sensory detail such as smell and a sense of reliving) of autobiographical memory. When comparing voluntary memories with involuntary memories, those with PTSD rated all memories as more emotionally intense, they showed a greater physiological reaction, and they reported that the memory was more central to their life story regardless of the type of memory (i.e., voluntary or involuntary). Furthermore, they also showed higher rehearsal in their voluntary and involuntary recall. However, trauma related memories were not higher in involuntary recall.

All studies have reported similar findings when comparing the worst point of traumatic memories in those with and without PTSD. In particular, hotspots were highlighted as central to the life story, with more emotional intensity and heightened

physiological reaction in those with PTSD. This pattern of findings supports Ehlers and Clark's (2000) cognitive model, which maintains that trauma memory in PTSD is poorly elaborated into autobiographical memory. In addition, these studies found that the content in traumatic memory is more likely to be in present tense, and have stronger emotional reactions. This also provides support for theories of PTSD, which maintains that memory in PTSD has strong perceptual priming and reactions to stimuli that are associated with the trauma (Ehlers & Clark, 2000), and that trauma memories do not use a verbally assessable system, but rely on a situationally accessed system (Brewin, 1996).

Group 3 - High vs. Low PTSD

Rubin, Berntsen, and Boals (2008) examined the differences in involuntary, voluntary, non-traumatic and traumatic autobiographical memory in PTSD, comparing people who had high levels of PTSD symptom severity with people who had low levels of PTSD symptom severity. They tested two models: the basic, and the special system models. The basic system model proposes that emotion and personality traits affect autobiographical memories and increase the likelihood of PTSD. The special system model proposes that:

(i) autobiographical memory can be affected by peri-traumatic dissociation,

(ii) voluntary access is impaired and

(iii) involuntary access is enhanced.

The study recruited and screened (for PTSD) undergraduate students (N =115). It consisted of three parts. The first focussed on voluntary memories, the second focussed on involuntary memories, and the third examined affect intensity. The results of the first part of the study found that those with higher levels of PTSD reported more emotion, their narrative was more central to their life story, and scored higher on certain

personality traits (i.e., neuroticism) and lower on others (i.e., conscientiousness). These findings support the basic system model, as personality traits and negative emotions account for PTSD symptom severity. In the second part of the study, participants recorded involuntary memories using a diary. The results were identical to the first part of the study, regarding differences between individuals with high and low PTSD scores. However, differences were found between voluntary and involuntary memories. Involuntary memories involved more reaction, mood changes were less coherent and used a first person perspective. The third part of the study used the sample from part one, and also recruited participants ($N = 533$) using a web-based design. Participants completed a number of measures to examine affect intensity, depression, centrality of memory and PTSD scores. The results supported the basic system model, that personality and intense emotional states (e.g., negative mood) are predisposing factors for PTSD.

This study is the only one that has explored the contribution of personality traits in PTSD symptom severity. The results are consistent with the other studies in this review in terms of the content for involuntary memories being more emotive. However, this study recruited a non-clinical undergraduate sample, and this may affect the generalisability of the results to clinical populations.

Group 4 - Brain injury

Jones, Harvey, and Brewin (2007) examined the content and organisation of trauma memories in participants who had a road traffic accident, and potential traumatic brain injury (TBI). Survivors of road traffic accidents ($N = 131$) were recruited from medical centres in the UK. The participants completed trauma narratives one week, six weeks and three months post-trauma. The narratives were rated by an independent rater, and the results showed that the narratives of survivors who had PTSD or acute stress disorder (ASD) were less coherent at one week and six weeks post- trauma;

however, at three months their narratives had more non-consecutive chunks, contained more repetitions, and exhibited more dissociation than those without PTSD or ASD. No link was found between TBI and PTSD symptom severity; however, those with TBI contained more confusion in their narratives.

By examining trauma narratives soon after the traumatic event, this study provided an insight into how narratives change over time following the initial traumatic event. The results highlighted that whilst trauma narratives in PTSD/ASD do change over a three month time frame, they always remain different from the narratives of those without PTSD/ASD. The results also indicate that there is a difference in the way traumatic events, for those with PTSD, are incorporated into autobiographical memory because, although improvement was highlighted at three months, the narratives of ASD/PTSD were still different to those participants who did not go on to develop ASD/PTSD. Clinically, this may indicate that even soon after trauma, narratives could be examined to determine if PTSD/ASD is likely to develop.

Discussion

The review aimed to firstly examine how trauma memories are assimilated and retrieved from autobiographical memory, and secondly, to examine whether retrieval of traumatic memories is different from non-traumatic memories in people with PTSD, compared to those individuals who do not develop symptoms of PTSD following a traumatic event. The main findings of the review indicate that trauma memories are dissimilar to non-traumatic memories; they are retrieved in present tense, have more detail, and are viewed as more central to autobiographical memory. Those individuals who had PTSD had heightened physiological responses when recalling, and used language which indicated fear. The review also highlighted that trauma memories are seen through the first person perspective rather than the third person perspective (Rubin,

1995). Studies in this review found that when the 'hotspots/flashbacks' occurred during the narrative accounts, the most traumatic part changed from the third person to the first person perspective, and more emotional language was used.

The cognitive model of PTSD by Ehlers and Clark (2000) describes the development of PTSD following a trauma event due to two key processes which maintain the individual's current sense of threat. The first part maintains that there are individual differences in how the trauma is appraised. The second part of the model proposes that there are individual differences in the memory links to the autobiographical memory store. Trauma memories that are poorly elaborated, and have strong perceptual priming, and strong association memory, will disrupt the normal autobiographical memory processes and this, in turn, may lead to PTSD symptoms and their maintenance. Within this review studies have reported that trauma memory appears to be organised and central to autobiographical memory. However, when the content has been examined, the terminology used by participants during highly traumatic events has tended to be emotionally salient, vivid, and in the first person perspective. This supports the memory aspect of Ehlers and Clark's (2000) cognitive model, which maintains that certain processes during trauma, such as perceptual priming, will lead to disruption of how trauma memory is stored within the autobiographical memory store.

The reviewed studies showed that when comparing memories of individuals with PTSD, trauma memories are not fragmented or disorganised. In fact, they seem to be central to the autobiographical memory system, and highly significant to the self-system. Studies that looked at memory differences between individuals with higher and lower levels of PTSD showed that the trauma memories of those individuals with higher levels of PTSD were more central to their story, and had higher emotional intensity.

These findings support models proposed by Ehlers and Clark (2000), and Brewin, Dagleish, and Joseph (1996), who maintain that multiple memory processes function simultaneously when traumatic situations occur. Studies examining the difference in memory between people with and without PTSD found that those with PTSD rated memories as more emotionally intense, and had higher rehearsal than those without PTSD. However, there was no difference in the accuracy of the recall in both groups.

Only one study examined the effect of physical trauma on the brain and the development of PTSD. Although the findings of this study did not support the hypothesis that those with traumatic brain injury are more likely to develop PTSD, they showed that those who developed PTSD had differences in their recall of information, and were likely to be less coherent and dissociative. This was the only study that examined the development of PTSD shortly after trauma (six-weeks and three months later), thus showing the trajectory of PTSD following a traumatic event.

Limitations of Studies

There are a number of methodological limitations in some of the reviewed studies. The majority of the studies focused on one type of trauma for the individual, but did not assess whether those individuals had other traumas in their life within the same time period, or at a different time. Peace et al. (2008) asked about childhood sexual trauma, as well as adult trauma, and found differences in the types of remembering. However, they did not ask if more than one trauma occurred for the individual over the lifetime. Other traumatic events in the person's life may influence the narrative accounts of the participants and, in turn, compromise the overall validity of the results. Studies (Follette, Polusny, Bechtle, & Naugle 1996; Green et al., 2000) have examined traumatic events over an individual's lifetime and found that people who experience multiple traumas over a single trauma are at higher risk of mental distress.

Only Jones et al. (2007) looked at TBI following road traffic accidents, and the development of ASD/PTSD symptoms. Although their results showed that TBI did not cause the development of PTSD, the memories of those with and without PTSD were different at three months post-trauma. Other studies failed to report how long ago trauma had occurred. It may be the case that a recent trauma might produce different narrative accounts than more distant traumas. The social interaction hypothesis (Hudson, 1990) highlights that autobiographical memory is not fixed and new information can be consolidated into a memory through discussions and interactions with other people. Therefore, if the traumatic event happened a long time ago, individuals may have had the opportunity to build their narrative of the event, and consequently fill in missing information. As a result, this may strengthen the disorganisation and structure of the trauma narrative.

Another limiting factor is that the studies reviewed all have used relatively small samples (ranging from 44 to 131). Small samples raise questions about the generalisability of the results to the wider population. Furthermore, having small samples can also introduce biases into the statistical analyses, and increase the probability of making type II errors.

The majority of studies used self-report measures (i.e., Posttraumatic Diagnostic Scale; Foa, 1995) when asking about memories, PTSD symptoms and the trauma narrative. Although this enables the participant to tell their story and express it in their own words, it has some drawbacks. First, it does not consider the role of avoidance in PTSD, and the impact distress can have on recall. Second, it also relies on the participants to have a certain level of intellectual ability and educational level to write down their narratives. Third, cultural differences may mean that people use an alternative language to describe emotions and trauma.

The majority of studies in this review used a cross-sectional design. The main advantage of this method is that it is quick way to determine prevalence which can then be followed-up later by longitudinal studies. However, it is more difficult to establish causal relationships, or the direction of relationships between variables.

Although systematic review methods were employed, only one author reviewed the studies and extracted the data. The method was also time-consuming and relied on abstracts and titles to provide initial screening which may not be reliable.

Even though the studies reviewed had some methodological weaknesses, as detailed above, the results were consistent across the groups of studies considered. The results found that different processes are used when assimilating traumatic memories into autobiographical memory, and this is shown in the different types of information that are provided during retrieval. Trauma memories are more emotionally intense, recalled from the first person viewpoint, and are much more vivid that non-trauma memories.

Implications for Clinical Practice

Memory processes play an important role in the development and maintenance of PTSD symptoms following a traumatic event. The results of this review found that trauma memory is not more likely to be fragmented or disorganised. Therefore, an individual with PTSD may be missed if this type of presentation is considered typical in PTSD. It may be more useful to look at the narratives and examine whether traumatic memories are central to the autobiographical memory store and self-system, and to examine whether parts of the traumatic event are recalled from the first person perspective and are more emotionally intense. This may provide more reliable, qualitative information alongside the DSM criteria when making a decision on PTSD presentation. The second clinical implication of this review is the importance of talking

about traumatic experiences to help consolidate information into autobiographical memory. Therefore, it is important to signpost individuals to support groups following such events. It may also be useful to provide GPs with information about self-help narrative techniques in order to encourage individuals to write, draw, or verbalise their trauma experiences. Information should also be available for family and friends about which type of support is useful and not helpful (i.e., not avoiding talking about the event).

Future Directions

The review highlighted that none of the studies indicated whether participants had treatment for their PTSD symptoms. It would be useful in future research to compare the narratives of those individuals who had gone through therapy, and reconstructed their trauma narrative, and those individuals who had not had any formal therapy for their PTSD symptoms. Similarly, it would also be useful to examine the effects of multiple traumas on memory, and compare this with a single trauma for PTSD symptom severity.

Jobson and O'Kearney (2006) found that following trauma an individual's culture can influence the direction of self-change, as sociolinguistic factors are central to narrative construction within autobiographical memory. It would be useful to examine further differences in education, socio-economic background and different languages, as well as other cultures, in order to determine whether there are any differences in the recall of trauma memories.

Only one study in the review examined physical trauma on the brain and whether it can cause vulnerabilities in developing PTSD. It would be useful to look at other conditions including medical events such as stroke, in order to examine whether a

traumatic event and physical damage in certain parts of the brain can be linked to developing PTSD.

Conclusion

To conclude, the review highlights that trauma memories are incorporated and retrieved differently to non-trauma memory in autobiographical memory. Trauma memories appear to be central to the autobiographical memory store, and this review highlights that they are not always fragmented and disorganised, even in people who have PTSD symptoms. What differentiates a traumatic moment from other information within the narrative is the content used when describing that moment. The tense changes to present tense and the content is more emotive. Therefore, it may be more useful to examine the content of traumatic memory for tense used, language related to fear and a sense of reliving, alongside other diagnostic methods when assessing for PTSD.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders, 4th Ed.* APA, Washington, DC.

Bedard-Gilligan, M., & Zoellner, L.A. (2012) Dissociation and memory fragmentation in post-traumatic stress disorder: An evaluation of the dissociative encoding hypothesis. *Memory*, *20*, 277-299. doi:10.1080/09658211.2012.655747

Blake, D., Weathers, F., Nagy, L., Kaloupek, D., Klauminzer, G.,Charney, D., & Keane, T. (1992). *Clinician administered PTSD scale*. Boston: National Centre for Post-Traumatic Stress Disorder.

Brewin, C.R. (2001). A cognitive neuroscience account of post-traumatic stress disorder and its treatment. *Behaviour Research and Therapy*, *39,* 373-393. doi:10.1037/a0018113

Brewin, C.R. (2007). Autobiographical memory for trauma: Update on four controversies. *Memory*, *15*, 227–248. doi:10.1080/09658210701256423

Brewin, C.R., Dalgleish, T., & Joseph, S. (1996). A dual representation theory of post-traumatic stress disorder. *Psychological Review*, *103*, 670-686. Retrieved from: http://homepage.psy.utexas.edu

Butler, G., Wells, A., & Dewick, H. (1995). Differential effects of worry and imagery after exposure to a stressful stimulus: A pilot study. *Behavioural and Cognitive Psychotherapy, 23*, 45–56. doi:10.1017/S1352465800017628

Downs, S.H., & Black, N. (1998).The feasibility of creating a checklist for the assessment of the methodological quality both of randomised and non-randomised studies in health care intervention**.** *Journal of Epidemiol Community Health*, *52*, 377-384. doi:10.1136/jech.52.6.377

Ehlers, A., & Clark, D.M. (2000). A cognitive model of post-traumatic stress disorder. *Behaviour Research and Therapy*, *38*, 219-245. doi:10.1016/S0005-7967(99)00123-0

Foa, E.B. (1995). *The posttraumatic diagnostic scale (PDS) manual*. Minneapolis, MN: National Computer Systems.

Foa, E.B., Keane, T.M., Friedman, M.J., & Cohen, J.A. (2010). *Effective treatments for PTSD: Practice guidelines from the international society of traumatic stress studies.* New York, NY: Guildford Press.

Hellawell, S.J., & Brewin, C.R. (2004). A comparison of flashbacks and ordinary autobiographical memories of flashbacks and ordinary autobiographical memories of trauma: Content and language. *Behaviour Research and Therapy*, *42*, 1-12. doi:10.1016/S0005-7967(03)00088-3

Holmes, E.A., & Bourne, C. (2008). Inducing and modulating intrusive emotional memories: A review of the trauma film paradigm. *Acta Psychologica, 127*, 553–566. doi:10.1016/j.actpsy.2007.11.002

Hudson, J.A. (1990). The emergence of autobiographical memory in mother-child conversation. In R. Fivish & J.A. Hudson (Eds.), *Knowing and remembering in young children* (pp. 166-196). New York: Cambridge University Press.

Jelinek, L., Stockbauer, C., Randjbar, S., Kellner, M., Ehring, T., & Moritz, S. (2010). Characteristics and organization of the worst moment of trauma memories in posttraumatic stress disorder. *Behaviour Research and Therapy*, *48*, 680-685. doi:10.1016/j.brat.2010.03.014

Jobson, L., & O' Kearney, R. (2006). Cultural differences in autobiographical memory of trauma. *Clinical Psychologist*, *10*, 89–98. doi:10.1080/13284200600939892

Jones, C., Harvey, A.G., & Brewin, C.R. (2007). The organisation and content of trauma memories in survivors of road traffic accidents. *Behaviour Research and Therapy*, *45*,151–162. doi:10.1016/j.brat.2006.02.004

Kleim, B., Wallott, F., & Ehlers, A. (2008). Are trauma memories disjointed from other autobiographical memories in PTSD? An experimental investigation. *Behavioural and Cognitive Psychotherapy*, *36*, 221–234. doi:10.1017/S1352465807004080

Landis, J.R., & Koch, G.G. (1977). The measurement of observer agreement for categorical data. *Biometrics*, *33*, 159-74. doi:10.2307/2529310

Lazarus, R.S., & Alfert, E. (1964). The short-circuiting of threat by experimentally altering cognitive appraisal. *Journal of Abnormal and Social Psychology*, *69*, 196–205. Retrieved from: APA PsychNET

Lazarus, R.S., & Opton, E.M. (1964). The study of psychological stress: A summary of theoretical formulations and experimental findings. In C. D. Spielberger (Ed.), *Anxiety and behaviour* (pp. 225–262). New York: Academic Press.

Moher, D. Liberati, A. Tetzlaff, J. Altman, D.G. (2009). *P*referred *R*eporting *I*tems for *S*ystematic Reviews and *M*eta-*A*nalyses: The PRISMA Statement. PLoS Med 6, doi:10.1371/journal.pmed1000097

Nelson, K.D., & Fivush, R. (2004). The emergence of autobiographical memory: A social cultural developmental theory. *Psychological Review*, *111*, 486 –511. doi: 10.1037/0033-295X.111.2.486

NICE guidelines (2005: March 15). *The management of PTSD in adults and children in primary and secondary care.* Retrieved from www.nice.org.uk/CG026NICEguideline.

O'Kearney, R., Hunt A., & Wallace N. (2011). Integration and organisation of trauma memories and posttraumatic symptoms. *Journal of Traumatic Stress, 24*, 716-725. doi:10.1002/jts.20690

Peace K.A., Porter, S., & ten Brinke, L. (2008). Are memories for sexually traumatic events "special"? A within-subjects investigation of trauma and memory in a clinical sample*, Memory*, *16*, 10-2. doi:10.1080/09658210701363583

Rubin, D.C. (1995). *Memory in oral traditions: The cognitive psychology of epic, ballads, and counting-out rhymes.* New York: Oxford University Press.

Rubin, D.C., Berntsen, D., & Boals, A. (2008). Memory in posttraumatic stress disorder: Properties of voluntary and involuntary, traumatic and non-traumatic autobiographical memories in people with and without PTSD symptoms. *Journal of Experimental Psychology*, *137*, 591-614. doi:10.1037/a0013165

Rubin, D.C., Dennis, M.F., & Beckham, J.C. (2011). Autobiographical memory for stressful events: The role of autobiographical memory in posttraumatic stress disorder. *Consciousness and Cognition, 20*, 840-856. doi: 10.1016/j.concog.2011.03.015

Rubin, D.C., Feldman, M.E., & Beckham, J.C. (2004).Reliving, emotions, and fragmentation in the autobiographical memories of veterans diagnosed with PTSD. *Applied Cognitive Psychology*, *18*, 17-35. doi:10.1002/acp.950

Spitzer, R.L., Williams, J.B.W., Gibbon, M., & First, M.B. (1996*). Structured clinical interview for DSM-III-R, patient edition/non-patient edition, (SCID-P/SCID-NP)*, Washington, D.C.: American Psychiatric Press, Inc.

Stuart, A.D.P., Holmes, E.A., & Brewin, C.R. (2006). The influence of a visuospatial grounding task on intrusive images of a traumatic film. *Behaviour Research and Therapy*, *44*, 611–619. doi**:**10.1016/j.brat.2005.04.004

Williams, H. L., Conway, M.A., & Cohen, G. (2008*).* Autobiographical memory. In G. Cohen & M. A. Conway (Eds.), *Memory in the real world* (3rd ed., pp. 21-90). Hove, UK: Psychology Press.

Can disturbances in autobiographical memory processes during stroke explain the severity of posttraumatic stress disorder symptoms?

Abstract

Objectives: To examine whether disturbances in autobiographical memory during stroke contribute to the severity of Post-Traumatic Stress Disorder (PTSD) symptoms.

Methods: Stroke survivors ($N = 80$) were recruited from six-week follow-up appointments across three outpatient stroke clinics. The Posttraumatic Diagnostic Scale (Foa, 1995) was completed to assess PTSD symptom severity. Memory of the trauma was assessed by the trauma memory questionnaire (Halligan, Michael, Clark, & Elhers, 2003) and cognitive processing (e.g., data driven, dissociation and self-referent processing) was measured using the processing styles questionnaire (Ehlers & Clark, 2000)

Results: Significant correlations with PTSD symptom severity were found for all memory variables and for age and time since stroke. After controlling for age and time since stroke, the memory measures were found to explain 40% of the variance in the severity of PTSD symptoms, with self-referent processing and intrusion making significant contributions to the regression equation. Further analyses revealed that the effect of self-referent processing on PTSD symptom severity was mediated by intrusion.

Conclusions: This study provides support for Ehlers and Clark's cognitive model which proposes that disruptions in trauma memory and cognitive processing during a traumatic event such as stroke can predict PTSD symptom severity. The limitations and clinical implications of the study are discussed.

Recently, it has become widely recognised that posttraumatic stress disorder (PTSD) is not just an illness of war veterans but can happen following a range of traumatic events, including life-threatening medical events, such as cancer (Smith, Redd, Peyser, & Vogl, 1999) and human immunodeficiency virus (HIV) (Martin & Kagee, 2011). Theories of PTSD (Brewin, Dagleish, & Joseph 1996; Ehlers & Clark, 2000; Foa & Kozak, 1986) have linked the development of PTSD to a range of negative thoughts about the event and a disruption in memory processes during the event. These disruptive memory processes (i.e., data driven processing, dissociation and lack of self-referent processing), which take place during the time of the trauma, can affect how memories are organised into the autobiographical memory store, causing intrusive reliving in the form of flashbacks or vivid images/dreams and disorganisation in the trauma narrative. For example, a recent review highlighted that trauma memories tend to be more central to autobiographical memory, relived in the present tense and associated with overly emotional responses. In addition, previous research (Halligan, Michael, Clark, & Ehlers, 2003) has examined these peri-cognitive memory processes following road traffic accidents and found that peri-cogntive processes (i.e., data-driven, dissociation and lack of self-referent processing) are related to memory disorganisation and can predict current and later onset of PTSD symptoms. This study will examine if disturbances in autobiographical memory processing during a stroke are related to the severity of PTSD symptoms.

Autobiographical Memory

Autobiographical memory is central to the experience of self-identity. William, Conway and Cohen (2008) have stated that autobiographical memories are based on two memory systems: episodic (personal experiences) and sematic (general knowledge) memories. They suggest that there are four aspects of autobiographical memories: 1) biographical information about oneself; 2) information that can be stored as either

copies or reconstructions – hence, they are either reconstructed from new information following the event, or they are based on perceptual and visual information; 3) memories that may be stored with specific details about an event, for example, having a certain type of ice-cream at a festival or as general information, such as going to a festival and having ice-cream; and 4) memories within the autobiographical store that may be stored in a first person or a third person perspective.

Conway and Pleydell-Pearce (2000) propose that autobiographical memory is a dynamic process constructed in three main ways: lifetime periods (e.g., school), general events (e.g., childbirth) and event specific knowledge (e.g., specific images about an event). These different levels of information form the general knowledge base within autobiographical memory. Furthermore, the authors highlight the self as being fundamentally related to autobiographical memory. The self is part of working memory and incorporates goal hierarchies which limit beliefs and cognitions. The goals of the self, determine access to the knowledge base; therefore, memories are initially encoded and later constructed and re-acted when remembering. However, during a traumatic event there is a threat to the self and there are no active goals to mediate the integration of the traumatic experience into autobiographical memory and, as a result, this can lead to either traumatic amnesia or event specific knowledge representations (images or flashbacks) with no autobiographical knowledge.

As autobiographical memories have many different components and are personal to the individual, certain situations can interfere with encoding and integration of memories into autobiographical memory. Feldman-Barrett (1997) found that personality traits can affect autobiographical memory, for example, those with higher self-esteem were positively biased and the opposite was true for those with lower self-esteem. Furthermore, stress hormones which overflow in the brain during a trauma event can cause disruption in memory processes. When the hippocampus is flooded

with cortisol, it is prevented from being able to form new memories or retrieve existing ones (Kuhlmann, Piel, & Wolfe, 2005). Therefore, events that are emotionally distressing and stressful, such as stroke, can cause stress for the individual and interrupt autobiographical memory.

Stroke

Stroke is a generic term denoting a sudden, focal neurological deficit secondary to cerebral arterial or venous disease (Bowman & Giddings, 2003). The Stroke Association (2013), reports that an estimated 150,000 strokes occur in the UK every year. In addition, following heart disease and cancer, stroke is the third most common cause of death and the most common cause of severe disability. There are two types of stroke: ischemic, which is a blockage in an artery carrying blood to the brain, and haemorrhagic, which occurs when a blood vessel bursts causing bleeding into the brain. This can happen in the brain itself or the area between the skull and brain (The Stroke Association, 2013).

Stroke can occur anywhere in the brain. In particular, it may occur in one or both hemispheres and because of this, many different parts of the brain can be affected, leading to physical, cognitive and emotional deficits. The physical effects of stroke may include: paralysis, spasticity, pain and changes in sensations. The cognitive deficits may include: memory difficulties, attention difficulties, and problems with executive function tasks. Cognitive deficits are reported in about one third of stroke survivors and, of those, memory difficulties are the most commonly reported (Doornhein, 1998). In addition, a range of psychological changes can occur following stroke such as apathy, emotional liability, and personality change. Research in this area has highlighted the psychological effects of stroke, such as depression, (Hackett, Yapa, Parag, & Anderson, 2005) and anxiety disorders such as generalised anxiety disorder, agoraphobia, panic, as well as simple phobias (Merriman, 2007). Furthermore, a growing number of studies

have also highlighted a link between stroke and the development of posttraumatic stress disorder (Bruggimann, Annoni, Staub, von Steinbuchel, Van der Linden, & Bogusslavsky, 2006; Field, Norman, & Barton, 2008; Merriman, 2005; Sembi, Tarrier, & O'Neil, 1998).

Post-Traumatic Stress Disorder (PTSD)

In order to consider a PTSD diagnosis following a perceived traumatic event such as a stroke, the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) outlines six criteria that should be met. These are: A) there was actual or threatened death or serious injury to oneself or to others; B) the traumatic event is re-experienced by the individual (e.g., acting or feeling if the event was happening again or persistently having distressing dreams, flashbacks images, etc.); C) the individual avoids stimuli associated with the trauma; D) there are increased arousal symptoms such as difficulty concentrating, hyper-arousal and sleep difficulties that were not present before the traumatic event occurred; E) the symptoms have been happening for longer than one month; and F) the effects of the symptoms are causing clinically significant impairment in different areas of life, such as work (APA, 1994).

Traditionally, research has focused on PTSD following external events such as accidents and physical assaults (Mayou, Ehlers, & Bryant, 2002). However, over the last ten years there has been a shift to examining internal events such as life threatening medical conditions. Studies have highlighted that PTSD can develop following myocardial infarction (Bennett, Conway, & Clatworthy, 2001), diagnosis of human immunodeficiency virus (HIV) (Matinez, Israelski, Walker, & Koopman 2002), cancer (Cordova, Studts, Hann, Jacobsen, & Andrykowski, 2005) and haemorrhage stroke (Bruggimann et al., 2006).

Three key psychological theories have been developed in order to explain the development and maintenance of PTSD following a traumatic event: emotional

processing (Foa & Kozak, 1986), dual representation (Brewin et al., 1996) and cognitive model of PTSD (Ehlers & Clark, 2000).

Emotional Processing Theory

Foa and Kozak (1986) proposed the emotional processing theory and stated that fear underlines anxiety disorders. Furthermore, the fear response present in anxiety disorders is different from normal fear responses because of the excessive response displayed by the individual. Foa and Riggs (1993) and Foa and Rothbaum (1998) later modified the theory and stated that, alongside the heightened fear response, two cognitive dysfunctions also occur: 1) the individual views the world as a dangerous place, and 2) the individual sees themselves as being unable to cope with the traumatic event. In addition, Foa and Cahill (2001) proposed that effective treatment of PTSD means exposing the individual to the avoided activities and the traumatic memory, in order to challenge the fear and relieve the symptoms of PTSD. Although linking the fear response to development of PTSD has been supported by studies looking at heart rate (Pitman et al., 1996), there have been criticisms of this model for it being too simplistic (Teasdale & Barnard, 1993) as it only focuses on one element of emotion and often traumatic events tend to be multifaceted and complicated (Brewin, 2003).

Dual Representation Theory

Brewin et al. (1996) proposed a dual representation theory of PTSD. This model moves away from a solo memory system and proposes that PTSD arises from two separate memory systems which work independently but are linked. Brewin explains that trauma memory becomes dissociated from ordinary memory because of the nature of the traumatic event. The two systems described are the verbally accessed memory (VAM) and situational accessed memory (SAM), which generally work parallel to each other. However, in certain situations one can become more dominant than the other.

VAM memory is the oral and written narratives that are encoded into the autobiographical memory store. It is stored with information about the past, present and the future and consequently can be retrieved deliberately when discussing the trauma with others. In contrast, SAM represents memory that is not consciously encoded as it does not rely on verbal input; rather, it relies on primary senses, i.e., sights, sounds, smells. This type of memory is more challenging to manage because it is difficult to control exposure to these primary senses and associated emotions.

Brewin (2001) proposes that the SAM system processes information at a faster rate than the VAM system because it draws from sensory perceptions and does not interact with other information in autobiographical memory. In normal recovery VAM uses the information provided by SAM, in order to process the memory and place it in context and time in autobiographical memory. However, in PTSD the VAM system makes a poor copy of the information from the SAM system and is unable to encode the information fully. In order for treatment to be successful both these systems need to be addressed.

Hellawell and Brewin (2002) examined cognitive resources and behavioural observations during periods of ordinary memory and flashback memories. They predicted that flashback and ordinary memory would use different systems. They recruited 62 participants who had met the DSM-IV criteria for PTSD. Participants were presented with a series of cognitive and verbal tasks and were tested using the dual task paradigm (i.e., where examination on a basic primary task is done simultaneously with a second task, and the results are compared with a situation where the primary task is completed alone). The results supported the dual representation theory and found that flashbacks were associated with a higher degree of autonomic and motor behaviours, and only flashbacks competed for visuo-spatial resources. In addition, using the same

sample, Hellawell and Brewin (2004) also examined the differences between ordinary trauma and flashback trauma memories in terms of their content and language. The results indicated that flashback memories contained more detail, were more interconnected, had a greater mention of fear, horror and helplessness, and used the present tense more when compared with ordinary trauma memories. Both these studies provide support for the dual representation theory that two memory systems compete with each other when processing traumatic events.

Cognitive Model of PTSD

Ehlers and Clark (2000) developed a cognitive model to explain the development and persistence of PTSD. They argue that PTSD can occur if a traumatic event is processed in a way which produces a sense of current serious threat. This occurs because of 1) making negative assessments of the trauma and/or its sequelae, and 2) disturbances in memory processes and their relationship to other autobiographical memories. These processes keep the individual in a state of current serious threat, maintaining PTSD symptomatology and preventing change.

Individuals with PTSD may hold several negative appraisals about the trauma and its sequelae. Firstly, they may be unable to see the trauma as a time limited one-off event and may overgeneralize to other situations (e.g., not going out anymore because the world is dangerous), leading to fear and a change in their behaviour (e.g., feeling fearful about being in social settings and avoiding contact with friends). Secondly, the individual may have negative appraisals about their behaviour or feelings during the trauma (e.g., I should have shouted louder). Thirdly, negative appraisals may include interpretations about one's own PTSD symptoms (i.e., flashbacks, memory loss, physical arousal) and feeling that the change may be permanent and that one is unable to cope, or negatively interpreting other people's reactions following the trauma (i.e.,

due to a change in physical appearance). Fourthly, negative appraisals about emotional responses following trauma, such as feeling guilt (e.g., I should have gone a different route), anger (e.g., why me?), shame (e.g., how will I face my family?) and feeling sadness about perceived loss (i.e., life will never be the same again). In addition, these negative feelings are interlinked with the memory disturbances in PTSD.

Considering the role of memory in PTSD, Ehlers and Clark (2000) propose that memories are affected in four different ways. First, trauma memories are often poorly elaborated and integrated into autobiographical memories. The aim of autobiographical memory is to store memories based on meaning so that stimulus triggers do not interfere with everyday tasks. However, in traumatic situations memories are poorly elaborated and insufficiently incorporated into the general memory store, which places experiences in context, place and time. As a consequence, there is no link to the 'here and now', and there is a higher threat perception triggered by harmless cues which are similar to the trauma experience. Second, the encoding and retrieval of trauma memories can also produce a strong emotional response because they are poorly integrated. As a result, individuals may not be aware that their highly aroused emotions may be due to reactivating the trauma memory. Third, there is strong perceptual priming; that is, stimuli associated with the trauma for a short amount of time will also reactivate trauma memories. Hence, due to a lower perceptual threshold, cues that are associated with the trauma are more likely to be noticed and trigger trauma memories. Fourth, when an individual recalls the trauma there can be a bias based on their appraisal of the trauma. If these are overly negative then the memory recall will also be overly negative and impact on feelings about the trauma, maintaining the perception of threat. The above disrupted memory processes impact on people's autobiographical memory base, such that general knowledge about oneself may become disorganized and retrieval may be less semantic and more cue driven. The model therefore offers an insight into how

disruption of different memory processes creates difficultly for individuals to place the trauma event in a correct autobiographical context, thus sustaining the sense of current serious threat.

Halligan et al. (2003) conducted two studies examining the development of PTSD in relation to trauma memory, cognitive appraisals and peri-traumatic variables following assault. Three peri-traumatic variables were examined: dissociation, data-driven and self-referent processing. Dissociation consists of a lack of connection with oneself during the traumatic event and includes emotional numbing, depersonalisation and a reduced awareness of one's surroundings. Data-driven processes are those that occur at a surface level, taking information from sensory cues. Self-referent processing occurs when information is processed and related to other memories in the autobiographical memory store. An inability to process traumatic memory from a self-referent viewpoint may affect the autobiographical memory base as a whole.

The first study employed a cross-sectional design looking at (i) disorganized trauma memories and the onset and maintenance of PTSD, and (ii) the impact of peri-traumatic processing on disorganised trauma memories and how this impacts on the development of PTSD. The findings indicated that all peri-traumatic variables (data driven, lack of self-referent processing and dissociation) were correlated with disorganisation in trauma memories and were related to PTSD. The second, a longitudinal study, examined on-going dissociation and negative appraisals of memories and whether these factors maintain the PTSD symptomatology. Memory processes (dissociation, data-driven, self-referent) were found to be associated with disorganised trauma memories. In turn, disorganised trauma memories predicted current and future levels of PTSD.

To conclude, Ehlers and Clark's (2000) model highlights that trauma memories that are poorly elaborated are likely to be poorly integrated with other autobiographical

memories and, therefore, are likely to be easily triggered by associated cues and perceptual priming. A number of studies have examined the influence of negative cognitive appraisals on the development of PTSD after stroke (Field et al., 2008). However, none to date have focused on the influence on memory processes and the development of PTSD after stroke.

The Present Study

Over the last ten years research has shown that PTSD can occur following medical events which are traumatic for the individual as they can represent a threat to one's integrity or possible death. Stroke can be a frightening experience and, as well as the possibility of death, it can leave an individual with a range of difficulties which may be emotional (such as depression), physical (such as paralysis) and cognitive (such as memory difficulties). Studies have shown that PTSD can occur following stroke (Bruggimann et al., 2006; Field, Norman, & Barton, 2008; Merriman, Norman, & Barton, 2007; Sembi, Tarrier, O'Neill, Burns, & Faragher, 1998).

Theories (Brewin, 1996; Ehlers & Clark, 2000; Foa et al., 1986) devised to aid understanding of why PTSD can occur after a traumatic event have linked this phenomenon to disturbances in autobiographical memory processes at the time of the trauma. Ehlers and Clark's cognitive model describes in detail how peri-cognitive processes (i.e., data driven, dissociation and self-referent processes) can create disorganisation in trauma memory which, in turn, may lead to the development of PTSD.

The present study is a test of Ehlers and Clark's (2000) model which proposes that, as well as negative cognitive appraisals, disturbances in autobiographical memory processes during stroke can contribute to the development and persistence of PTSD. Therefore, the proposed study will examine the ways in which trauma memories are

processed (e.g., being elaborated) and organised as well as looking at their impact on the severity of PTSD symptoms in stroke survivors.

Aims

The main aim of the study was to explore the effect of peri-traumatic experiences (dissociation, data-driven and self-referent processing) during stroke on autobiographical memories and the severity of PTSD symptoms.

Hypotheses

1. Cognitive processing during trauma (dissociation, data-driven and self-referent processing) will be associated with poorly integrated trauma memories.

2. Cognitive processing and disorganised trauma memories will be associated with the severity of PTSD symptoms.

Method

Ethical Considerations

Ethical approval for the research was given by the NHS Research Ethics Committee for Leeds. In order to ensure participant safety and confidentiality, access to personal information was not given to the researcher until informed consent was provided by the participant. Confidential information was kept on an electronic database which was password protected. Completed questionnaires were kept in a locked filing cabinet at the university, separate from any identifying information.

Power Analysis

Field et al. (2008) were able to explain 47% of the variance in PTSD symptom severity at baseline and 24% variance at follow-up in a study on cognitive appraisals. In contrast, Halligan et al. (2003) explained 22% of the variance of PTSD symptoms following assault (i.e., a medium effect size). These amounts of variance represent medium-to-large effect sizes.

An initial power analysis indicated that a sample of 114 would be required to detect a medium effect size (f^2=.15) in a regression analysis consisting of 9 independent variables (i.e., 5 memory variables described below, and 4 control variables) with power set at .80 and alpha at .05. Only 54 respondents would be required to detect a large effect size (f^2=.35). It was therefore proposed to recruit between 54 and 114 participants during the recruitment period to allow the detection of a medium-to-large effect size in the regression analysis. .

Participants

Opportunistic sampling was used to recruit and select potential participants from three out-patient clinics in the stroke unit at the Royal Hallamshire Hospital in Sheffield. Two were consultant led geriatric clinics and one was a consultant nurse led specialist stroke clinic. A total of 80 participants were recruited into the study. Table 1 provides a breakdown of sample characteristics.

Table 1 *Sample Characteristics (N = 80)*

Characteristic	N	%
Gender		
Male	38	47.5
Female	42	52.5
Ethnicity		
White British	74	93
Black British	3	4
Irish	1	1.3
Other	1	1.3
Marital Status		
Married	36	45
Single	11	13.8
Divorced	10	12.5
Widowed	23	28.8
Employment status		
Employed	14	17.5
Unemployed	4	5
Retired	62	78
Previous trauma		
Yes	34	42.5
No	46	57.5
Previous mental health Problem		
Yes	11	13.8
No	69	86.3
	M	**SD**
Age	72.27	12.41
Time since stroke	15.64	20.89
Number of strokes	1.41	.77

Materials

Measures were used to assess peri-traumatic processes and the nature of trauma memories. One measure was used to assess the severity of post-traumatic stress disorder symptoms.

Measures

Cognitive Processing Questionnaire (Elhers & Clark, 2000).

This questionnaire examines cognitive processing of trauma memories. It includes three components which assess data-driven processing, dissociation and self-referent processing during the traumatic event. All three components are scored using a Likert scale ranging from zero to four where individuals score their level of agreement with the statements. Eight items assess data-driven processing (e.g., "I was overwhelmed by sensations and couldn't put everything together") ($\alpha = .88$). Scores on the items are summed to provide a measure of data-driven processing that can range from 0 - 32. Nine items examine dissociation, adapted from the State Dissociation Questionnaire (Murray et al., 2002), measuring peri-traumatic experiences such as depersonalization (e.g., "My body felt as if it was not really mine") ($\alpha = .91$). Scores on the items are summed to provide a measure of dissociation ranging from 0 - 36. Third, eight items assess self-referent processing (e.g., "I felt as if it were happening to somebody else") ($\alpha = .88$). Scores on the items are summed to provide a measure of self-referent processing ranging from 0 - 32. These scales have been shown to be related to PTSD symptomatology following assault (Ehlers & Clark, 2000).

Trauma Memory Questionnaire (Halligan, Michael, Clark, & Elhers, 2003).

This questionnaire consists of two sub-scales: disorganisation and intrusion. The disorganisation scale comprises five items and examines how much of the trauma memory is incomplete or disorganized ($\alpha = .88$). Scores on the items are summed to provide a measure of disorganisation ranging from 0 - 20. The intrusion scale

comprises eight items and examines how easily the traumatic event is triggered and if these memories are accompanied by a sense of reliving the event (α = .90). Scores on the items are summed to provide a measure of intrusion ranging from 0 - 32. These sub-scales have shown to be related to autobiographical memory processes and PTSD symptomatology following road traffic accidents (Halligan et al., 2003).

Post-traumatic Diagnostic Scale (PDS; Foa, 1995).

The PDS is a 49 item self-report measure based on the DSM-IV diagnostic criteria (A-F) that can be used to assign a PTSD diagnosis. It has good internal consistency (α = .92) and good test–retest reliability (Foa, 1995). In addition, the PDS has good agreement with the structured clinical interview for the DSM-IV, with good sensitivity (.89) and specificity (.75).

As well as PTSD screening it also assesses the severity of PTSD symptoms using a 17 item self-report questionnaire. Each response is viewed over the past month and is rated on a four point Likert scale ranging from zero to three. Five items assess re-experiencing the trauma (e.g., having bad dreams or nightmares), seven items assess avoidance (e.g., trying not to think about, talk about or have feelings about the traumatic event) and five items assess arousal (e.g., being jumpy or easily startled). Items are summed to produce a symptom severity score which ranges from 0 - 51. For all three areas, a score of 1 or higher is viewed as indicating that the symptom is present. Respondents meeting the standard criteria of PTSD as outlined in DSM –IV (i.e., criteria A1 and criteria A2) and having a score of 15 or higher on symptoms are considered positive for PTSD (Sheeran & Zimmerman, 2002).

Control Variables

A number of additional variables were assessed and controlled for in the analyses. Some of this information was included in the questionnaire (such as number of strokes) and other information was obtained from patients' notes (such as hemisphere

of stroke). Control variables included demographic information (i.e., age, gender, marital status, education, ethnicity) and clinical information (i.e., anxiety and depression scores which are routinely administered after stroke, type of stroke, hemisphere of stroke).

Procedure

Potential participants were identified through follow-up outpatient stroke clinics at the Royal Hallamshire Hospital in Sheffield. Nurses supporting the clinics were briefed and initially identified potential participants from their clinical history and notes using the inclusion/exclusion criteria of the study. When suitable potential participants were identified, a note highlighting their suitability was put into their clinical notes for the doctor or nurse consultant's clinic. In addition, the potential participant was given an information sheet outlining in summary the details of the study to read in the waiting area. Once in their appointment, the doctor or nurse consultant asked them if they had considered the information they had read and if they would like to participate in the study or meet the researcher for further information.

When potential participants agreed to take part in the study or get more information they were then seen by the researcher. The researcher went through the information sheet verbally with participants (see Appendix III). If patients agreed, consent was obtained from the participants and either participants went through the questionnaire alone in the room, took away the questionnaires to complete at a later date, or completed the questionnaire with the researcher reading it to them aloud whilst recording their responses. Some participants took consent forms and questionnaires home with them as they wanted more time to consider whether or not to participate. On the consent form personal details were recorded and consent was obtained to access patients' medical notes in order to obtain information such as location and type of stroke. Once all participants were recruited into the study the researcher went through

the medical files and extracted details of the date of the stroke, location of the stroke and what type of stroke had occurred.

Statistical Analysis

The data were analysed in three stages. First, the prevalence of PTSD post-stroke, as assessed by the PDS, was determined. Second, correlations were computed between each of the memory measures and the severity of PTSD symptoms. Associations were also examined between socio-demographic and clinical variables and the severity of PTSD symptoms. Third, hierarchical regression analyses were conducted to assess the predictors of PTSD symptom severity. The independent variables were entered in three blocks. In the first block, any socio-demographic or clinical variables that had significant associations with PTSD symptom severity were entered. In the second block, memory processing measures (i.e., data driven processing, dissociation, self-referent processing) were entered. In the third block, trauma memory variables (i.e., disorganisation and intrusion) were entered.

Results

Descriptive results

A total of 80 participants were recruited. The sample predominately comprised older adults with a mean age of 72 and, consequently, the majority were retired (78%). The majority of participants (93%) reported their ethnicity as White British and 53% were female. Just under half (43%) reported that they had been through a previous trauma and 14% of participants reported having had a previous mental health problem before having the stroke. A small minority (11%) of participants were classified as having PTSD on the basis of the PDS. The clinical notes showed that the majority of participants (85%) had an ischemic type of stroke and 36% of these affected the left side of the brain.

Data screening

Raw data were screened for missing data and any outliers. There was no missing data for the PDS, trauma memory questionnaire (TMQ) and cognitive processes questionnaire (CPQ). However, there was significant data missing for some of the control variables (i.e., localisation of the stroke, anxiety and depression scores and type of stroke). Due to the large amount of missing data on these variables it was deemed inappropriate to conduct analyses with these variables and they were therefore excluded from subsequent analyses. The distributions of the remaining variables were checked and no outliers were present in the data. The mean and standard deviations for the main study variables are shown in Table 2.

Table 2. *Means and Standard Deviations of Main Study Variables*

Variable	Mean	SD	Alpha
PTSD			
1. Symptom Severity	7.77	7.55	.82
Memory Processing			
2. Data-Driven	9.88	8.83	.90
3. Dissociation	6.06	7.03	.86
4. Self-referent	5.02	6.00	.82
Trauma Memory			
5. Disorganisation	5.77	6.63	.90
6. Intrusion	2.28	3.78	.79

Associations between demographic and clinical factors and PTSD symptom severity

Associations between the demographic/clinical factors and PTSD symptom severity were examined using ANOVA, t-tests and correlations as appropriate. Tables 3 and 4 summarise the result of these analyses.

Table 3. *Associations between the Demographic and Medical factors and PTSD Symptom Severity*

Variable	M (SD)	N	t/F	df	p
Employment					
Employed	10.07 (6.74)	14	1.36	78	.19
Retired	7.28 (7.67)	66			
Gender					
Male	6.55 (7.12)	38	1.39	78	.17
Female	8.88 (7.23)	42			
Previous Trauma					
Yes	8.09 (6.28)	33	.37	77	.71
No	7.48 (8.46)	46			
Previous Mental Health Problem					
Yes	10.44	9	1.99	76	.06
No	7.91	69			
Stroke Type					
Ischemic	7.64 (7.75)	34	1.39	38	.21
Haemorrhage	15 (7.71)	6			
Hemisphere					
Left	8 (8.26)	29	.647	40	.52
Right	10 (9.68)	13			
Marital Status					
Married	9.11 (7.97)	37	.73	3, 76	.54
Single	6.45 (10.52)	11			
Divorced	7.50 (3.75)	10			
Widowed	6.43 (6.47)	23			

Only age, r (78) = .24, p = .03, and the length of time since having the stroke, r (78) = .30, p <. 001, were found to correlate significantly with PTSD symptom severity.

Table 4 *Correlations between Control Variables and PTSD Symptom Severity (N = 80)*

Correlations	r
Age	.24*
Time since stroke	.30**
No. of strokes	.03
Education	.02

*Note. * p <.05. ***p<.001.*

Correlations between memory variables and PTSD symptom severity

Significant correlations were found between memory processing (data driven, dissociation and self-referent memory) and PTSD symptom severity and between trauma memory (disorganisation and intrusion) and PTSD symptom severity (see Table 5). These correlations indicate that peri-cognitive factors in memory processing and trauma memory are associated with PTSD symptom severity. In addition, significant correlations were also found between the memory processing variables (i.e., data driven, dissociation and self-referent processing) and the trauma memory variables (i.e., disorganisation and intrusion). Thus, patients who have had a stroke, who engaged in more data-driven processing, self-referent processing and dissociation during the trauma, had more disorganised and intrusive trauma memories, and reported the more severe PTSD symptoms.

Table 5 *Correlations between Memory Variables and PTSD Symptom Severity (N =80)*

Variables	2.	3.	4.	5.	6.
PTSD					
1.Symptom severity	.35**	.48***	.49***	.27*	.63***
Memory Processing					
2. Data Driven		.71***	.53***	.46***	.38**
3. Dissociation			.68***	.36**	.44***
4. Self-Referent				.27*	.45***
Trauma Memory					
5. Disorganisation					.20
6. Intrusion					

Note. * $p < .05$. ** $p < .01$. *** $p < .001$.

Hierarchical Multiple Regressions

PTSD symptom severity.

In order to examine whether PTSD symptom severity can be explained by cognitive processing during trauma and memory disorganisation, a hierarchical multiple regression analysis was performed (see Table 6). The variables were entered in three steps. In the first step, the control variables of age and time since stroke were entered and were found to account for 16% of the variance, $R^2 = .16$, $F(2, 75) = 6.99$, $p = .002$. Age was the only variable to make a significant contribution to the regression equation.

In step two, the three memory processing variables (data driven, dissociation and self-referent memory) were entered into the model, which increased the amount of variance explained by 13%, $\Delta R^2 = .13$, $F(3, 72) = 4.62$, $p = .005$, such that the total amount of variance explained was 29%, $R^2 = .29$, $F(5, 72) = 5.97$, $p < .001$. Only self-referent processing made a significant contribution to the regression equation.

In the third step, the two trauma memory variables (disorganisation and intrusion) were added into the model. This increased the amount of variance explained by 11%, $\Delta R^2 = .11$, $F(2, 70) = 6.42$, $p = .003$, such that the total amount of variance explained was 40%, $R^2 = .40$, $F(7, 70) = 6.74$, $p < .001$. In the final model, only one of the subscales from the trauma memory variables (i.e., intrusion) significantly contributed to the prediction of PTSD symptom severity.

Table 6 *Summary of Variables Predicting PTSD Symptom Severity (N = 80)*

Variables	B	SE B	B
Step 1			
Age	0.16	0.06	.27*
Time since stroke	0.06	0.03	.21
Step 2			
Age	0.10	0.06	.17
Time since stroke	0.06	0.03	.00
Dissociation	0.15	0.16	.15
Self-referent	0.33	0.16	.27
Step 3			
Age	0.04	0.06	.07
Time since stroke	0.04	0.03	.12
Data Driven	0.06	0.11	.08
Dissociation	0.15	0.16	.14
Self-referent	0.21	0.15	.17
Disorganisation	0.08	0.11	.08
Intrusion	0.78	0.22	.39***

Note **p<.05 ***p<.001

In order to test the second hypothesis (cognitive processing during trauma will be associated with poorly integrated trauma memories), two hierarchical regression

analyses were conducted examining the effects of cognitive processing on the disorganisation and intrusion sub-scales of trauma memory.

Disorganisation.

To examine whether disorganisation in memory at the time of stroke could be explained by cognitive processing variables, a hierarchical regression was performed (see Table 7). The variables were entered in two steps. In the first step the control variables of age and time since the stroke were entered and were found to account for a non-significant amount of variance, R^2 = .05, F(2, 75) = 2.06, p =.14. In the second step, the three cognitive processing variables were entered. This increased the variance explained by 18%, ΔR^2 = .18, F(3, 72) = 5.65, p = .002, such that the total amount of variance explained was 23%, R^2 = .23, F(5, 72) = 4.37, p = .002. Only data-driven processing made a significant contribution to the regression equation.

Intrusion.

A hierarchical regression was also conducted to examine whether intrusive memories could be explained by the cognitive processing variables (see Table 8). The variables were entered in two steps. First, the control variables of time since stroke and age were entered and accounted for 19% of the variance, R^2 = .19, F(2, 75) = 8.77, p < .001. Only age made a significant contribution to the regression equation. In step two, the cognitive processing variables were added to the model and accounted for an additional 10% of the variance, ΔR^2 =. 10, F(3, 72) = 3.35, p = .02, such that the total amount of variance explained was 29%, R^2 = .29, F(5, 72) = 5.85, p < .001. At this step, age was again the only significant contributor to the regression equation.

Table 7 *Summary of Variables Predicting Disorganisation of Trauma Memories*

Variable	B	SE	B
Step 1			
Age	.03	.07	.05
Time since Stroke	.06	.04	.21
Step 2			
Age	.04	.06	.07
Time since stroke	.06	.03	.20
Data driven	.31	.11	.41**
Dissociation	.04	.16	.43
Self-referent	.00	.16	.00

Table 8 *Summary of Variables Predicting Intrusion in Trauma Memories*

Variable	B	SE	B
Step 1			
Age	.09	.03	.35**
Time since Stroke	.03	.02	.16
Step 2			
Age	.08	.03	.27**
Time since stroke	.03	.02	.17
Data driven	.05	.06	.12
Dissociation	.00	.08	.00
Self-referent	.15	.08	.24

*Note ** p<.01*

The regression results suggest that the effect of self-referent processing on PTSD symptom severity may be mediated by intrusive trauma memories, given that the

significant beta weight for self-referent processing became non-significant when the trauma memory variables were entered into the regression equation, and that intrusion trauma memory was the sole significant predictor of PTSD symptom severity.

Mediation Analysis

As recommended by Preacher and Hayes (2008), further analyses were conducted to test whether the trauma memory variables mediated the effect of cognitive processing on PTSD symptom severity. As potential mediators, the trauma memory variables were entered (i.e., disorganisation and intrusion) simultaneously along with the covariates of age and time since stroke and the cognitive processing variables (i.e., self-referent processing, dissociation and data driven processing). The effect of self-referent processing on PTSD was significant, $B = 0.32$, $SE = 0.16$, $p = .05$, but became non-significant when controlling for the trauma memory variables, $B = 0\ .21$, $SE = 0.15$, $p = .16$. Using bootstrapping procedures, the total mediated effect was found to be significant, $B = 0.14$, $SE = 0.11$, $CI = 0.04$ to 0.35. Examination of the individual mediator variables showed that intrusion significantly mediated the effect of self-referent processing, $B = 0.14$, $SE = 0\ .11$, $CI = 0.04$ to 0.36, whereas disorganisation did not, $B = 0.00$, $SE = 0.02$, $CI = -0.05$ to 0.04.

Discussion

This study examined associations between disturbances in autobiographical memory processes and the severity of PTSD symptoms following stroke. Within the sample PTSD caseness was found in a small percentage of individuals (11%). In their review of PTSD following stroke, Norman, O'Donnell, Creamer and Barton (2012) reported PTSD prevalence rates ranging between 3 and 31%. More recent studies have reported PTSD caseness in between 18 and 25% of stroke survivors (Baisch, Schenk, & Noble, 2011; Favrole, Jehel, Levy, Descombes, & Muresan, 2013; Kronish,

Edmondson, Goldfinger, Fei, & Horowitz, 2012). Therefore, the current study had a slightly lower percentage of individuals reporting PTSD symptoms following stroke than reported in most other studies.

The study tested a key component of Ehlers and Clark's (2000) cognitive model of PTSD, which states that individual differences in memory and the link to other autobiographical memories can lead to a current sense of threat and, in turn, persistent PTSD. The aim of the study was to examine whether cognitive processing during trauma (dissociation, data-driven and self-referent processing) would be associated with poorly integrated trauma memories and, in turn, the severity of PTSD symptoms following stroke. When exploring cognitive processing variables and trauma memory variables in relation to PTSD symptom severity, significant correlations were found between all variables (i.e., data driven, dissociation, self-referent, disorganisation and intrusion) and PTSD symptom severity. However, a hierarchical multiple regression analysis showed that, after controlling for age and time since stroke, the main cognitive processing variable to make a significant contribution to the explanation of PTSD symptom severity was self-referent processing, i.e., thinking or feeling that the trauma is happening to somebody else and feeling disconnected to the traumatic experience.

Hierarchical multiple regressions were also conducted to test the second hypothesis which examined whether trauma memory mediated the effects of cognitive processes on PTSD. The first model tested the relationship between cognitive processing and disorganisation of trauma memories. The results showed that data-driven processes were significantly associated with disorganised trauma memories. Thus, the results highlight that bottom up data-driven processes (such as sensory input of smell, sound, sight) may lead to disorganisation in trauma memory and contribute to the severity of PTSD. In addition, the second model investigated the relationship

between cognitive processing and the trauma memory variable of intrusion. The results indicated that self-referent processing had a significant effect, such that engaging in self-referent processing was associated with high levels of PTSD symptoms. The present results therefore support previous research (Halligan, Michael, Clark, & Elhers, 2003) that has found that peri-cognitive processing during a traumatic event (i.e., road traffic accident) is related to disorganisation in trauma memory and consequently can predict concurrent and subsequent PTSD.

Considering the control variables, age and time since stroke were significantly correlated with PTSD symptom severity. This is in line with Merriman, Norman and Barton (2007) who found that age and the time since stroke predicted the severity of PTSD symptoms. Similarly, Field et al. (2008) examined cognitive appraisals and PTSD severity after stroke and found that age was the only control variable that correlated with PTSD symptom severity. In contrast, non-significant correlations were found for length of education, gender, marital status, and number of previous strokes. However, previous studies have shown significant correlations with gender (Bruggiman et al., 2006), previous strokes and time since stroke (Merriman, Norman & Barton, 2007).

As self-referent processing and intrusion both contributed significantly to the explanation of PTSD symptom severity, a mediation analysis was conducted to test whether the effect of self-referent processing on PTSD symptom severity was mediated by intrusive memories. The analysis indicated that the effect of self-referent processing was mediated by intrusion. However, as intrusion is also one of the symptom clusters that define PTSD, it is perhaps not surprising that intrusive memories were strongly predictive of the severity of PTSD symptoms. Thus, the measure of intrusion may be confounded with criterion B of the DSM-IV (APA, 1994 p.242) which lists “persistent re-experiencing of the traumatic event” as a key criterion for diagnosing PTSD.

This study examined one aspect of Ehlers and Clark's (2000) cognitive model, that is, the nature of trauma memory and its connection to other memories in the autobiographical memory store. Trauma memories that are problematic are those which produce a sense of current threat for the individual, even though the event occurred in the past. In turn, this fear leads to persistent PTSD. The model emphasises that the quality of processing during the trauma will affect the nature of the trauma memory. During the time of trauma, different processes at the peri-cognitive level (i.e., data-driven, self-referent and dissociation) can influence the processing of trauma memories which can lead to disorganisation in autobiographical memories and unwanted intrusive recollections of the traumatic event. The results of this study showed that although all peri-cognitive factors did correlate with the severity of PTSD symptoms, subsequent regression analysis indicated that self-referent processing was the most important factor.

Limitations

There are a few limitations of the study that should be noted. First, the sample size of the study was small ($N = 80$) and this may have led to some of the regression analyses being under-powered. However, the study was sufficiently powered to detect medium-to-large effects. Moreover, the sample size is comparable or larger than other studies on PTSD following stroke which have recruited samples of 34 (Sharkey, 2007) to 65 (Eccles, House, & Knapp, 2008).

Second, due to time and practical constraints, convenience sampling was employed with recruitment of participants from three local stroke clinics at the Royal Hallamshire Hospital. This type of sampling method can create bias in the representativeness of the sample to the population of stroke survivors and, as a consequence, may limit the generalisability of the findings. For example, due to the nature of PTSD symptoms and presentation (i.e., avoiding talking about the trauma, or avoiding trauma feelings), some individuals may have been more likely to opt out of

consenting in the study. In addition, potential participants with severe cognitive impairments were also excluded from the study; hence, the results may not be representative of the post-stroke PTSD population as a whole.

Third, data about the type of stroke, location of the stroke, depression and anxiety scores were taken retrospectively from clinical notes. However, it was not possible to access all of these data as patients' clinical notes were often incomplete. Consequently, there was a large amount of missing data relating to these factors. This is problematic as the literature on stroke and PTSD (Sampson, Kinderman, Watts, & Sembi, 2003) highlights that depression and anxiety following a traumatic event are related to the development PTSD.

Fourth, Ehlers and Clark's (2000) cognitive model proposes that dissociation at the time of trauma can cause disruption in memory which can lead to persistent PTSD. Although this study measured dissociation, consciousness at the time of stroke was not taken into account. However, Field et al. (2007) found that consciousness at the time of stroke was not related to PTSD symptom severity.

Fifth, data obtained about the symptoms of PTSD were based on patients' self-reports, as opposed to a clinical interview. While the PDS is a valid and reliable measure of PTSD, clinical interviews, such as structured clinical interviews (SCID) for DSM-IV axis I disorders PTSD module (Spitzer, Gibbon, Williams & First, 1996) or the clinician administered PTSD scale, (CAPS) (Blake, Weathers, Nagy, Kaloupek, Charney & Keane, 1995), are widely regarded as the "gold standard" for assessing PTSD.

Implications for clinical practice

Notwithstanding these limitations, this study has some important implications for clinical practice. Age and time since stroke were found to be significantly

associated with the severity of PTSD symptoms. Therefore, clinicians need to be aware that younger adults may be particularly prone to developing PTSD symptoms. In addition, clinicians working in the six-week post-discharge clinic should also be aware that patients who experienced their stroke further in the past than other patients may also be at risk of heightened PTSD symptoms. This could reflect the experience of severe stroke where individuals spend a longer time in hospital before being discharged.

Given that 11% of patients in this study could be classified as having PTSD, and prevalence rates as high as 31% have been reported in the literature (Bruggiman et al., 2006), there is a clear need for clinicians to screen for PTSD following stroke. An example of assessing for PTSD following a medical event is presented by a study conducted by Twigg, Humphris, Jones, Bramwell and Griffiths (2008), who noted that cases of PTSD following a stay in intensive care units are becoming more widely recognised. The authors highlighted that whilst there are measures to assess PTSD, such as the PDS or the Impact of Events Scale (IES), they are not wholly suited to a medical population, due to their length (PDS) or to them not providing a complete assessment of PTSD as outlined by the DSM-IV (IES). They modified the Post-traumatic stress scale 10 (PTSS-10) to create the UK post-traumatic stress scale -14 (UK-PTSS-14) and tested it on 44 patients discharged from intensive care unit (ICU). They found preliminary support for the UK-PTSS-14 with patients being discharged following a medical event. The measure had good test-retest and internal validity and the authors concluded that this tool may be a reliable screen to use with discharged ICU patients in order to ensure early diagnosis and treatment of PTSD. These findings highlight that screening for PTSD following a medical event is useful; a similar tool could prove beneficial for patients post-stroke.

Studies have shown that PTSD can impair adherence to treatment in medical conditions such as in myocardial infarction (Shemesh, Rudnick, Kaluski, Milovanov, Salah, & Alon, 2001), as well as following a HIV diagnosis (Boarts, Buckley-Fischer, Armelie, Bogart, & Delahanty, 2009). Furthermore, a recent study by Kronish, Edmondson, Goldfinger, Kezhen, Fei and Horowitz (2012) found that those individuals who develop PTSD following stroke or transient ischemic attacks were at higher risk of poor treatment adherence. For this reason it is important to find ways to identify those at risk from developing PTSD following stroke and offer appropriate psychological support such as trauma-focused cognitive behavioural therapy (CBT) or eye movement desensitisation and reprocessing therapy (EMDR).

Future research

This study only examined one component of Ehlers and Clark's (2000) cognitive model of PTSD. As well as accounting for disruption in memory processes during a traumatic event, the model also proposes that negative appraisals of the trauma and the events that occur around the trauma (i.e., treatment by emergency care professionals) can also feed into a sense of current threat and the onset of PTSD. There have been a range of studies examining the effects of negative appraisals and the persistence of PTSD (e.g., Dunmore, Clark, & Ehlers 2001; Field et al., 2007; Halligan et al., 2003). Furthermore, Ehlers and Clark (2000) also highlight that trauma memory has a reciprocal relationship with the types of appraisals about the trauma and its sequelae. They report that those who have persistent PTSD will selectively retrieve information which is negative; this idea is supported by studies of PTSD and depression, as depressed individuals may be biased to more negative factors (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Shalev, Freedman, Peri, Brandes, Sahar, Orr, & Pitman, 1998). Future research on post-stroke PTSD should examine Ehlers and

Clark's model as a whole in order to test whether both disruption in trauma memory and negative appraisals following stroke lead to persistent PTSD symptoms.

As stated earlier, the sample of this study was largely homogenous and mainly from a white British background. However, previous research into stroke prevalence (Stansbury, Huanguang, Williams, Voigel & Duncan, 2005; Trimble & Morganston, 2008) has shown that there is a higher occurrence of stroke in minority communities. Therefore, given that prevalence of stroke is higher in minority communities and incidence of PTSD following stroke is also common, it is important for future research to examine whether PTSD is also prevalent in minority communities to ensure that they are not overlooked in terms of psychological treatment post-stroke.

This study did not find a link between type of stroke (i.e., ischemic or haemorrhage) or the location (left or right hemisphere) of the stroke and disruption of memory processes or PTSD symptom severity. However, this may be due to the small sample of participants meeting the criteria for PTSD. Neuropsychological research has demonstrated that damage in different parts of the brain such as the medial pre-frontal cortex, which regulates the amygdala, and impairment to hippocampus can contribute to symptoms of PTSD. For example, dysfunction in this area may create insufficiencies in working memory which may promote the development of PTSD (Elzinga & Bremner, 2002). Dysfunction in the hippocampus may contribute to trauma memories that are fragmented (Bremner et al., 1996). Furthermore, Schouten, Schiemanck, Brand and Post (2009) examined hemispheric lesions following ischemic stroke and found that those who had lesions in the left hemisphere performed poorly on verbal memory tasks. In addition, lesion level and volume were also predictors of verbal memory performance. Thus, future studies that seek to address this question will require larger samples of

post-stroke PTSD participants. Any link would be able to provide vital screening information to the vulnerability of developing PTSD post-stroke.

Finally, while this study provided support for Ehlers and Clark's model of memory disturbances during a traumatic event (i.e., stroke) and a link to PTSD symptom severity, future qualitative research would be useful to examine these peri-cognitive factors in more detail by asking individuals who have developed PTSD following stroke to talk about memories from both at the time of the stroke and at the present time.

Conclusion

To conclude, caution should be exercised when interpreting the results of this study, due to the small number of individuals meeting the criteria of PTSD. Nonetheless, this study indicates that PTSD may develop following stroke and provides support for one component of Ehlers and Clark's (2000) cognitive model, which states that disruptions in cognitive processes and trauma memory are linked to PTSD symptom severity.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders, 4th (Ed).* Washington, DC: American Psychological Association

Baisch, S.B., Schenk, T., & Noble, A.J. (2011). What is the cause of post-traumatic stress disorder following subarachnoid haemorrhage? Post-ictal events are key. *Acta Neurochirurgica*, *153*, 913–922. doi: 10.1007/s00701-010-0843-y

Bennett, P., Conway, M., Clatworthy, J., Brooke, S., Stuart, M., & Owen, R. (2001). Predicting posttraumatic symptoms in cardiac patients. *Heart and Lung, 30,* 458-465. Retrieved from: Pubmed.gov

Blake, D., Weathers, F., Nagy, L., Kaloupek, D., Klauminzer, G., Charney, D., & Keane, T. (1992). *Clinician administered PTSD scale*. Boston: National Centre for Post-Traumatic Stress Disorder.

Boarts, J.M., Buckley-Fischer, B.A., Armelie, A.P., Bogart, L. M., & Delahanty, D.L. (2009). The impact of HIV diagnosis-related vs. non-diagnosis related trauma on PTSD, depression, adherence, and HIV disease markers. *Journal of Evidence-Based Social Work, 6*, 4-16. doi:10.1080/15433710802633247

Bowman, P., & Giddings, F.D. (2003). *Strokes: An illustrated guide to brain structure, blood supply, and clinical signs.* NewYork: Prentice Hall.

Bremner, J.D., Randall, P., Scott, T.M., Bronen, R.A., Seibyl, J.P., Southwick, S. M…, & Innis, R. B. (1995). MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. *American Journal of Psychiatry*, *152*, 973-981. Retrieved from: The American Journal of Psychiatry

Brewin, C.R., Dagleish, T., & Joseph, S, (1996). A dual representation theory of post-traumatic stress disorder. *Psychological Review*, *103*, 670-686. doi: 10.1037/0033-295X.103.4.670

Brewin, C.R. (2001). A cognitive neuroscience account of posttraumatic stress disorder and its treatment. *Behaviour Research and Therapy*, *39*, 373-393. doi:org/10.1016/S0005-7967(00)00087-5

Brewin, C.R. (2003). *Posttraumatic stress disorder: Malady or myth?* New Haven, CT: Yale University Press.

Bruggimann, L., Annoni, J.M., Stuab, F., von Steinbuchel, N., van der Linden, M., & Bogousslavsky, J. (2006). Chronic posttraumatic stress symptoms after non-severe stroke. *Neurology, 66,* 513-516. doi:10.1212/01.wnl.0000194210.98757

Cordova, M.J., Studts, J.L., Hann, D.M., Jacobsen, P.B., & Andrykowski, M.A. (2000). Symptom structure of PTSD following breast cancer. *Journal of Traumatic Stress*, *13*, 301-319. DOI: 10.1023/A:1007762812848

Conway, M.A., & Pleydell-Pearce, C.W. (2000). The construction of autobiographical memories in the self-memory system. *Psychological Review, 107*, 261-288. doi: 10.1037/0033-295X.107.2.261

Doornhein, K., & deHaan, E.H.F. (1998). Cognitive training for memory deficits in stroke patients. *Neuropsychological Rehabilitation.* 8, 393-400. doi:10.1080/713755579

Dunmore, E., Clark, D.M., & Ehlers, A. (2001). A prospective study of the role of cognitive factors in persistent posttraumatic stress disorder after physical or sexual assault. *Behaviour Research and Therapy*, *39*, 1063–1084**.** Retrieved from: PubMed

Eccles, S., House, A., & Knapp, P. (2008). Psychological adjustment and self-reported coping in stroke survivors with and without emotionalism. *Journal of Neurology, Neurosurgery, and Psychiatry*, *67*,125-126. doi:10.1136/jnnp.67.1.125

Ehlers, A., & Clark., D.M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy, 38,* 319-345. doi: 10.1016/s0005-7967(99)00123-

Elzinga, B. M., & Bremner, J. D. (2002). Are the neural substrates of memory the final common pathway to PTSD? *Journal of Affective Disorder,70*, 1–17. doi:10.1016/S0165-0327(01)00351-2

Favrole, P., Jehel, L., Levy, P., Descombes, S., Muresan, I., Manifacier, M., & Alamowitch, S. (2013). Frequency and predictors of posttraumatic stress disorder after a stroke: A pilot study. *Journal of Neurological Sciences, 327*, 35-40. doi:10.1016/j.jns.2013.02.001

Feldman Barrett, L. (1997). The relationships among momentary emotion experiences, personality descriptions, and retrospective ratings of emotion. *Personality and Social Psychology Bulletin*, *23*, 1100–1110.

Field, E.L., Norman, P., & Barton, J. (2008). Cross-sectional and prospective associations between appraisals and posttraumatic stress disorder symptoms following stroke. *Behaviour Research and Therapy, 38, 319-345.* doi:10.1016/j.brat.2007.10.006

Foa, E.B., & Cahill, S.P. (2001). Psychological therapies: Emotional processing. In Smelser, N.J, & Bates, P.B. (Eds.). *International Encyclopaedia of Social and Behavioural Sciences.* Oxford: Elsevier.

Foa, E.B. (1995). *The posttraumatic diagnostic scale (PDS) manual.* Minneapolis, MN: National Computer Systems.

Foa, E.B., & Kozak, M.J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin, 99*, 20-35. doi: 10.1037/0033-2909.99.1.20

Foa, E.B., & Riggs, D.S. (1993). Post-traumatic stress disorder in rape victims. In Oldham, J., Riba, M.B., & Tasman, A. (Eds.), *American psychiatric press review of psychiatry. Vol. 12.* Washington, DC: American Psychiatric Press.

Foa, E.B., & Rothbaum, B.O. (1998). *Treating the trauma of rape: A cognitive behavioural therapy for PTSD.* New York: Guilford.

Follette, V.M., Polusny, M., Bechtle, A.E., & Naugle, A.E. (1996). Cumulative trauma: The impact of child sexual abuse, adult sexual assault, and spousal abuse. *Journal of Traumatic Stress*, *9*, 25–35. doi:10.152/psyc.67.3.267.48986

Hackett, M.L., Yapa, C., Parag, V., & Anderson, C.S. (2005). Frequency of depression after stroke: A systematic review of observational studies. *Stroke, 36*, 1330-1340. doi: 10.1161/01.STR.0000165928.19135.35

Halligan, S.L., Michael, T., Clark, D.M., & Ehlers, A. (2003). Posttraumatic stress disorder following assault: The role of cognitive processing, trauma memory, and appraisals. *Journal of Consulting and Clinical Psychology, 71,* 4419-4431. doi:10.1037/0022-006X.71.3.419

Hellawell S.J., & Brewin C.R. (2002) A comparison of flashbacks and ordinary autobiographical memories of trauma. Cognitive resources and behavioural observations. *Behaviour Research and Therapy, 40*, 1143–1156. doi:org/10.1016/S0005-7967(01)00080-8

Hellawell, S.J., & Brewin, C.R. (2004). A comparison of flashbacks and ordinary autobiographical memories of flashbacks and ordinary autobiographical memories of trauma: content and language. *Behaviour Research and Therapy*, *42*, 1-12. doi:10.1016/S0005-7967(03)00088-3

Kessler, R.C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C.B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry, 52*, 1048-1060. doi:10.1001/archpsyc.1995.03950240066012

Kronish, I.M., Edmondson, D., Goldfinger, J., Fei, K., & Horowitz, C. (2012). Posttraumatic stress disorder and adherence to medications in survivors of strokes and transient ischemic attacks. *Stroke, 43*, 2192–2197. doi:10.1161/strokeaha.112.655209

Kuhlmann, S., Piel, M., & Wolf, O.T. (2005). Impaired memory retrieval after psychosocial stress in healthy young men. *Journal of Neuroscience*, *25*, 2977-2982. doi: 10.1523/JNEUROSCI.5139-04.2005

Martin, L., & Kagee, A. (2011). Lifetime and HIV-related PTSD among persons recently diagnosed with HIV. *AIDS and Behaviour*, *15*, 125-131. doi:10.1007/s10461-008-9498-6

Martinez, A., Israelski, D., Walker, C., & Koopman, C. (2002). Posttraumatic stress disorder in women attending Human Immunodeficiency Virus outpatient clinics. *AIDS Patient Care and STDS*, 6, 283-291. doi:10.1089/10872910260066714

Mayou, R.A., Ehlers, A., & Bryant, B. (2002). Posttraumatic stress disorder after motor vehicle accidents: Three year follow-up of a prospective longitudinal study. *Behaviour Research and Therapy, 40,* 665-675. doi.org/10.1016/S0005-7967 (01)00069-9

Merriman, C., Norman, P., & Barton, J. (2007). Psychological correlates of PTSD symptoms following stroke. *Psychology, Health and Medicine, 12, 592-602.* doi:10.1080/13548500601162747

Murray, J., Ehlers. A., & Mayou, R.A. (2002). Dissociation and post-traumatic stress disorder: Two prospective studies of road traffic accident survivors. *British Journal of Psychiatry, 180,* 363-368. doi:10.1192/bjp.180.4.363

Norman, P., O'Donnell, M., Creamer, M., & Barton, J. (2012). Posttraumatic stress disorder after stroke: A review of quantitative studies. In Ovuga, E. (Ed.), *Post traumatic stress disorders in a global context.* Rijeka: InTech Open Access Publisher. ISBN 978-953-307-825-0. Available from: http://www.intechopen.com/articles/show/title/posttraumatic-stress-disorder-after-stroke-a-review-of-quantitative-studies

Pitman, R.K., Orr, S.P., Altman, B., Longpre, R.E., Poire, R.E., & Macklin, M.L. (1996). Emotional processing during eye movement desensitization and reprocessing therapy of

Vietnam veterans with chronic posttraumatic stress disorder. *Comprehensive Psychiatry, 37*, 419-429. doi: 10.1016/S0010-440X(96)90025-5

Preacher K.J., & Hayes, A.F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behaviour Research Methods*, *40*, 879-891. doi:10.3758/BRM.40.3.879

Sampson, M.J., Kinderman, P., Watts, S., & Sembi, S. (2003). Psychopathology and autobiographical memory in stroke and non-stroke hospitalized patients. *International Journal of Geriatric Psychiatry, 1,* 23-32. doi:10.1002/gps.763

Schouten, E.A., Schiemanck, S.K., & Brand, M.W.M. (2009). Long-term deficits in episodic memory and ischemic stroke: Evaluation and prediction of verbal and visual memory performance based on lesion characteristics. *Journal of Stroke and Cerebrovascular Diseases,* 18, 128-138. doi:10.1016/j.jstrokecerebrovasdis.2008.09.017

Sembi, S., Tarrier, N., O'Neill, P., Burns, A., & Faragher, B., (1998). Does post-traumatic stress disorder occur after stroke?: A preliminary study. *International Journal of Geriatric Psychiatry, 13,* 315-322. *doi:*10.1002/(SICI)1099-1166

Shalev, A.Y., Freedman, S., Peri, T., Brandes, D., Sahar, T., Orr, S.P., & Pitman, R.K. (1998). Prospective study of posttraumatic stress disorder and depression following trauma. *American Journal of Psychiatry, 155*, 630-637. Retrieved from: http://psychiatryonline.org/data/Journals/AJP/3688/630.pdf

Sharkey, M. (2007). Post-traumatic stress symptomology following stroke. *PSIGE Newsletter, 99,* 14-17.

Sheeran, T., & Zimmerman, M. (2002). Screening for posttraumatic stress disorder in a general psychiatric outpatient setting. *Journal of Consulting and Clinical Psychology*, *70,* 961–966. doi:10.1037//0022-006X.70.4.000. 961

Shemesh, E., Rudnick, A., Kaluski, E., Milovanov, O., Salah, A., Alon, D… & Cotter, G.A. (2001). Prospective study of posttraumatic stress symptoms and non-adherence in

survivors of a myocardial infarction (MI). *General Hospital Psychiatry, 23*, 215-222. doi: 10.1016/S0163-8343(01)00150-5

Smith M.Y., Redd, W.H., Peyser C., & Vogl D. (1999). Post-traumatic stress disorder in cancer: A review. *Psychooncology, 8*, 521-37. doi:10.1002/(SICI)1099-1611

Spitzer, R.L., Williams, J.,B.,W., Gibbon, M., & First, M.B. (1996*). Structured clinical interview for DSM-III-R, patient edition/non-patient edition,(SCID-P/SCID-NP)*. Washington, D.C: American Psychiatric Press, Inc.

Stansbury, J.P., Huanguang, J., Williams, L.S., Voigel, B.W., & Duncan, P.W. (2005). Ethnic disparities in stroke epidemiology, acute care, and postacute outcomes *Stroke*, *36*, 374-86. doi:10.1161/01.STR.0000153065.39325.fd

Teasdale, J.D., & Barnard, P.J. (1993). *Affect, cognition and change: Remodelling depressive thought*. Hove: Lawrence Erlbaum Associates.

The Stroke Association. (2013). *About stroke*. Retrieved from http://www.stroke.org.uk/about-stroke

Trimble, B., & Morganston, L.B. (2008). Stroke in minorities. *Neurologic Clinic, 26*, 1117-1190. doi:10.1016/j.ncl.2008.05.010

Twigg, E., Humphris, G., Jones, C., Bramwell, R. & Griffiths, R.D. (2008). Use of a screening questionnaire for post-traumatic stress disorder (PTSD) on a sample of UK ICU patients. *Acta Anaesthesiologica Scandinavica*, *52*, 202–208. doi:10.1111/j.1399-6576.2007.01531.x

Williams, H.L., Conway, M.A., & Cohen, G. (2008). Autobiographical memory. In G. Cohen & M. A. Conway (Eds.), *Memory in the real world* (3rd ed., pp. 21-90). Hove, UK: Psychology Press.

Appendix

Appendix A – Modified Quality Rating Scale (Downs & Black, 1998)

Paper Title: _____

_____

Authors: _____

_____

Reporting

1. Is the hypothesis/aim/objective of the study clearly described?

Yes	1
No	0
Unable to determine	0

2. Are the main outcomes to be measured clearly described in the Introduction or Methods section?

Yes	1
No	0

3. Are the characteristics of the patients included in the study clearly described?

Yes	1
No	0

4. Are the interventions of interest clearly described?

Yes	1
No	0

5. Are the distributions of principal confounders in each group of subjects to be compared clearly described?

Yes	2
Partially	1
No	0

6. Are the main findings of the study clearly described?

Yes	1
No	0

7. Does the study provide estimates of the random variability in the data for the main outcomes?

Yes	1
No	0

8. Have all the important adverse events that may be a consequence of the intervention been reported?

Yes	1
No	0

9. Have the characteristics of patients lost to follow-up been described?

Yes	1
No	0

10. Have actual probability values been reported (e.g.0.035 rather than <0.05) for the main outcomes except where the probability value is less that 0.001?

Yes	1
No	0

External Validity

11. Were the subjects asked to participate in the study representative of the entire population from which they were recruited?

Yes	1
No	0
Unable to determine	0

12. Were those subjects who were prepared to participate representative of the entire population from which they were recruited?

Yes	1
No	0
Unable to determine	0

13. Were the staff, places and facilities where the patients were treated, representative of the treatment the majority of the patients receive?

Yes	1
No	0
Unable to determine	0

Internal Validity –bias

14. Was an attempt made to blind study subjects to the intervention they have received?

Yes	1
No	0
Unable to determine	0

15. Was an attempt made to blind those measuring the main outcomes of the intervention?

Yes	1
No	0
Unable to determine	0

16. If any of the results of the study were based on “data dredging”, was this made clear?

Yes	1
No	0
Unable to determine	0

17. In trials and cohort studies, do the analyses adjust for different lengths of follow-up of patients, or in case-control studies, is the time period between intervention and outcome same for cases and controls?

Yes	1
No	0
Unable to determine	0

18. Were the statistical tests used to assess the main outcomes appropriate?

Yes	1
No	0
Unable to determine	0

19. Was compliance with the intervention/s reliable?

Yes	1
No	0
Unable to determine	0

20. Were the main outcome measures used accurate (valid and reliable)?

Yes	1

No	0
Unable to determine	0

Internal Validity –confounding

21. Were the patients in different intervention groups (Trial and cohort studies) or were the cases and controls (case-control studies) recruited from the same population?

Yes	1
No	0
Unable to determine	0

22. Were study subjects in different intervention groups (trials and cohort studies) or were the cases and controls (case-control studies) recruited over the same period of time?

Yes	1
No	0
Unable to determine	0

23. Were study subjects randomised to intervention or groups?

Yes	1
No	0
Unable to determine	0

24. Was the randomised intervention assignment concealed from both patients and health care staff until recruitment was complete and irrevocable?

Yes	1
No	0
Unable to determine	0

25. Was there adequate adjustment for confounding in the analyses from which the main findings were drawn?

Yes	1
No	0
Unable to determine	0

26. Were losses of patients to follow-up taken into account?

Yes	1
No	0
Unable to determine	0

Power

27. Did the study have sufficient power to detect a clinically important effect where the probability value for a difference being due to chance is less than 5%?

	Size of smallest intervention group	
A	$<n_1$	0
B	$n_1 - n_2$	1
C	n_3-n_4	2
D	n_5-n_6	3
E	n_7-n_8	4
F	n_8+	5

Appendix B – Approval letter

NHS
National Research Ethics Service
NRES Committee Yorkshire & The Humber - Leeds East
Yorkshire and Humber REC Office
First Floor, Millside
Mill Pond Lane
Meanwood
Leeds
LS6 4RA

Telephone: 0113 3050122
Facsimile:

24 August 2011

Ms Safeena Ghufran
Trainee Clinical Psychologist
Sheffield Health and Social Care
Clinical Psychology Unit
Western Bank
Sheffield
S10 2TN

Dear Ms Ghufran

Study title:	**Memory Processes and Post Traumatic Stress Disorder(PTSD)after Stroke**
REC reference:	**11/YH/0247**
Protocol number:	**N/A**

Thank you for your letter of 20 July 2011 responding to the Committee's request for further information on the above research and submitting revised documentation.

The further information was considered in correspondence by a sub-committee of the REC A list of the sub-committee members is attached.

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, subject to the conditions specified below.

Ethical review of research sites

NHS sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion" below).

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission ("R&D approval") should be sought from all NHS organisations involved in the study in accordance with NHS research governance arrangements.

Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at http://www.rdforum.nhs.uk.

Where a NHS organisation's role in the study is limited to identifying and referring potential participants to research sites ("participant identification centre"), guidance should be sought from the R&D office on the information it requires to give permission for this activity.

For non-NHS sites, site management permission should be obtained in accordance with the procedures of the relevant host organisation.

Sponsors are not required to notify the Committee of approvals from host organisations

It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).

Approved documents

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

Document	*Version*	*Date*
Covering Letter		20 July 2011
Investigator CV		
Other: CV for Paul Norman		
Participant Consent Form	2	2 May 2011
Participant Information Sheet	2	2 May 2011
Protocol	3	20 June 2011
Questionnaire: First questionnaire	1	17 June 2011
Questionnaire: Second questionnaire	1	17 June 2011
Questionnaire: First Questionnaire	2	
Questionnaire: Second Questionnaire	2	
REC application		13 June 2011
Response to Request for Further Information	2	

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.

After ethical review

Reporting requirements

The attached document *"After ethical review – guidance for researchers"* gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Notification of serious breaches of the protocol
- Progress and safety reports

- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

Feedback

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

Further information is available at National Research Ethics Service website > After Review

11/YH/0247	**Please quote this number on all correspondence**

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

Yours sincerely

[redacted]

pp **Dr Carol Chu**
Chair

Email: Elaine.hazell@nhs.net

Enclosures: *List of names and professions of members who were present at the meeting and those who submitted written comments*

"After ethical review – guidance for researchers"

Copy to: *Ms Ramila Patel, Research Department, STH NHS Foundation Trust*

NRES Committee Yorkshire & The Humber - Leeds East

Attendance at Sub-Committee of the REC meeting on 24 August 2011

Committee Members:

Name	*Profession*	*Present*	*Notes*
Dr Carol Chu	Chair	Yes	
Professor Rob Newell	Professor of Nursing Research and Director of Postgraduate Research	Yes	

Appendix C – Patient information sheet

Department Of Psychology.
Clinical Psychology Unit.

Doctor of Clinical Psychology (DClin Psy) Programme
Clinical supervision training and NHS research training & consultancy.

Clinical Psychology Unit
Department of Psychology
University of Sheffield
Western Bank
Sheffield S10 2TN UK

Telephone: 0114 2226650
Fax: 0114 2226610
Email: **c.harrison@sheffield.ac.uk**

http://www.shef.ac.uk/

PATIENT INFORMATION SHEET

Memory Processes and Post Traumatic Stress Disorder (PTSD) after Stroke

We would like to invite you to take part in a research study. Before you decide you need to understand why the research is being done and what it would involve for you. Please take time to read the following information carefully. Talk to others about the study if you wish.

Take time to decide whether or not you wish to take part.

What is the purpose of the study?

The main purpose of this study is to understand if there is a link with having memory difficulties following stroke and developing trauma-related symptoms. If there is a link then this may help us identify a different care plan for individuals in the future.

Why have I been invited?

The study is examining the role of memory processes following a stroke. It is important that we recruit participants who have recently had a stroke. For this reason it was decided to recruit participants from the six-week follow-up clinics for stroke patients at the Hallamshire Hospital.

Do I have to take part?

Participation within the study is completely voluntary and you are not obliged to take part. Deciding not to take part in the study will not affect your treatment. Your treatment will continue as normal. If you decide to take part we will describe the study and go through the information sheet, which we will then give you to keep. We will then ask you to sign a consent form to show that you have agreed to take part in the following study. You are free to withdraw at any time, without giving reason. This will not affect the standard of care you receive.

What will happen to me if I take part?

If you decide to take part in this study, the researcher will meet with you to explain in more detail and answer any questions you have. She will then go through the questionnaire with you, which should take about 30 minutes to complete.

What will I have to do?

You will have to complete one questionnaire at the clinic which will take about 30 minutes. The questionnaire will be completed with the researcher.

What are the possible benefits of taking part?

The main benefit of taking part in this study is that it may help us to identify those

patients who may be at risk of developing post traumatic stress disorder. We cannot promise that the study will help you, but the information we get from this study may help judge trauma reactions in others who have had a stroke.

What if there is a problem?

If answering the questionnaires brings back any unpleasant feelings for you will be asked to discuss this with the stroke nurse or consultant at the clinic or to contact your GP for further help.

Will my taking part in the study be kept confidential?

We have a duty of confidentiality to you as a research participant and we will do our best to meet this duty. If you join the study, some parts of your medical records and the data collected for the study will be looked at by authorised persons from the research team.

All information which is collected about you during the course of the research will be kept strictly confidential, and any information about you which leaves the hospital will have your name and address removed so that you cannot be recognised.

What if you want to complain?

Any complaints can be addressed to: Dr. Paul Norman at the Clinical Psychology Unit, Department of Psychology, The University of Sheffield, Western Bank, Sheffield, S10 2TN. You can also make a complaint via your local hospital. To do this, please contact the Patient Advice and Liaison Service (PALS) at: 722 Prince of Wales Road, Sheffield S9 4EU, Tel: 0800 085 7539 and provide information about the project.

Contact Information

If you have any questions about the research, you can leave a telephone message with the Research Support Officer on: 0114 222 6650, and she will ask Safeena to contact you.

Appendix D –Consent form

The University Of Sheffield.

Department Of Psychology.
Clinical Psychology Unit.

Doctor of Clinical Psychology (DClin Psy) Programme
Clinical supervision training and NHS research training & consultancy.

Clinical Psychology Unit
Department of Psychology
University of Sheffield
Western Bank
Sheffield S10 2TN UK

Telephone: 0114 2226650
Fax: 0114 2226610
Email: c.harrison@sheffield.ac.uk

http://www.shef.ac.uk/

Consent form

Memory Processes and Post Traumatic Stress Disorder (PTSD) after Stroke

Patient name.....

Patient address.....

I confirm that I have read and understand the information sheet for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.	
I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason, without my medical care or legal rights being affected.	
I understand that relevant sections of my medical notes and data collected during the study, may be looked at by individuals from University of Sheffield, from regulatory authorities or from the NHS Trust, where it is relevant to my taking part in this research. I give permission for these individuals to have access to my records.	
I agree to my GP being informed of my participation in the study.	
I understand that the researcher may need to contact my GP with my permission if I need further assistance.	
I agree to take part in the above study.	

Participant
signature.....Date.....

Appendix E – Questionnaire

Memory Processes and Post Traumatic Stress Disorder (PTSD) after Stroke

About this project

Following a stroke people may experience physical effects. They may also experience problems with remembering what happened during the stroke and this could affect their feelings afterwards. The effects of the stroke will vary from person to person. This research will look into what thoughts occurred during the stroke, what feelings arose during the stroke and how the stroke is remembered. We hope the study will provide information to improve the support and follow up care provided to people after their stroke.

What are you being asked to do?

You will be asked to complete a questionnaire at the clinic with the researcher. You may find that some of the questions are not relevant for you. If so please tick the "not applicable" option for these questions. In addition, you do not have to answer any questions that you don't want to. The questionnaire will take about than 30 minutes to complete.

Thank you for completing this questionnaire

Your help is very much appreciated

PART 1

Could you please complete the following details:

3. Age_____
4. Sex_____
5. Martial Status_____
6. Age that you left full time education? _____
7. Employment status_____
8. If you are employed, what is job title? _____
9. If you are retired what was you job before you retired?

   _____
10. How would you describe your ethnic origin? (please circle)

White British Irish Black British African-Caribbean Asian

Other (please specify) _____

The following questions are about details of your stroke and medical history:

1. How long ago did you have your most recent stroke? _____
2. Have you had more than one stroke? Yes

   No

   If yes, how many strokes have you had? _____

3. Have you experienced any previous traumatic events? Yes

   No
4. Have you had previous psychiatric illness? Yes No

For the following questions about your most recent stroke, please circle Yes/No or N/A (Not Applicable)

1.	Were you physically injured?	Yes	No	N/A
2.	Was someone else physically injured?	Yes	No	N/A
3.	Did you think that your life was in danger?	Yes	No	N/A
4.	Did you think that someone else's life was in danger?	Yes	No	N/A
5.	Did you feel helpless?	Yes	No	N/A
6.	Did you feel terrified?	Yes	No	N/A

PART 2

Below is a list of problems that people sometimes have after experiencing a stroke. Read each one carefully and circle the number (0-3) that best describes how often the problem has bothered you IN THE PAST MONTH. Rate each problem with respect to your stroke.

	Not at all	Once a week/once in a while	2-4 times a week	5 or more times a week	Not applicable
Having upsetting thoughts or images about the stroke that came into your head when you didn't want them to	0	1	2	3	
Having bad dreams or nightmares about the stroke	0	1	2	3	
Reliving the stroke, acting or feeling as if it was happening again	0	1	2	3	
Feeling emotionally upset when you were reminded of the stroke (for example, scared, angry, sad, guilty, etc.)	0	1	2	3	
Experiencing physical reactions when you were reminded of the stroke (for example, breaking out in a sweat, heart beating fast)	0	1	2	3	
Trying not to think about, talk about, or have feelings about the stroke	0	1	2	3	
Trying to avoid activities, people, or places that remind you of the stroke	0	1	2	3	
Not being able to remember an important part of the stroke	0	1	2	3	
Having much less interest or participating much less often in important activities	0	1	2	3	
Feeling distant or cut off from people around you	0	1	2	3	

	Not at all	Once a week/once in a while	2-4 times a week	5 or more times a week	Not applicable
Feeling emotionally numb (for example, being unable to cry or unable to have loving feelings)	0	1	2	3	
Feeling as if your future plans or hopes will not come true (for example, you will not have a career, marriage, children, or a long life)	0	1	2	3	
Having trouble falling or staying asleep	0	1	2	3	
Feeling irritable or having fits of anger	0	1	2	3	
Having trouble concentrating (for example, drifting in and out of conversations, losing track of a story on television, forgetting what you read)	0	1	2	3	
Being overly alert (for example, checking to see who is around you, being uncomfortable with your back to door, etc.)	0	1	2	3	
Being jumpy or easily startled (for example when someone walks up behind you)	0	1	2	3	

How long have you experienced the problems that you reported above? (Circle ONE)

1. Less than 1 month
2. 1 to 3 months
3. More than 3 months
4. Not applicable

How long after the stroke did these problems begin? (Circle ONE)

1. Less than 6 months
2. 6 or more months
3. Not applicable

Indicate if the problems you rated above have interfered with any of the following areas

of your life DURING THE PAST MONTH.	Circle Yes/No or N/A (Not Applicable)		
1. Work	Yes	No	N/A
2. Household chores and duties	Yes	No	N/A
3. Relationships with friends	Yes	No	N/A
4. Fun and leisure activities	Yes	No	N/A
5. Schoolwork	Yes	No	N/A
6. Relationship with family	Yes	No	N/A
7. Sex life	Yes	No	N/A
8. General satisfaction with life	Yes	No	N/A
9. Overall level of functioning in all areas of your life	Yes	No	N/A

PART 3

We are interested in WHAT WENT THROUGH YOUR MIND during the stroke. Please indicate the extent to which the following statements applied to you DURING THE STROKE.

During the stroke…	Not at all	A little	Moderately	Strongly	Very strongly	Not applicable
1. I couldn't really take it all in	0	1	2	3	4	
2. I did not fully understand what was going on	0	1	2	3	4	
3. It was just a like a stream of unconnected impressions following each other	0	1	2	3	4	
4. I could not think clearly	0	1	2	3	4	
5. I was overwhelmed by sensations and couldn't put everything together	0	1	2	3	4	
6. I was confused and could not fully make sense of what was happening	0	1	2	3	4	
During the stroke…	Not at all	A little	Moderately	Strongly	Very strongly	Not applicable

7. My mind was fully occupied with what I saw, heard, smelled, and felt.	0	1	2	3	4	
8. My mind was full of impressions and reactions to them	0	1	2	3	4	
9. I felt dazed, unable to take in what was happening	0	1	2	3	4	
10. The world around me seemed strange or unreal	0	1	2	3	4	
11. My body felt as if it was not really mine	0	1	2	3	4	
12. I felt emotionally numb	0	1	2	3	4	
13. I felt as I was a separate to my body watching it from outside	0	1	2	3	4	
14. I felt as if time was going faster or slower than it really was	0	1	2	3	4	
15. I felt as if I was living a dream or a film, rather than in real life	0	1	2	3	4	
16. Things around me seemed too big or too small, or distorted in shape	0	1	2	3	4	
17. I felt distant from my emotions	0	1	2	3	4	
18. It felt as if the event was happening to someone else	0	1	2	3	4	
19. It felt like I was a different person from the person I used to be	0	1	2	3	4	
20. I was aware that the event was happening, but not so much that it was happening to me	0	1	2	3	4	

During the stroke…	Not at all	A little	Moderately	Strongly	Very strongly	Not applicable
21. I felt cut off from my past	0	1	2	3	4	
22. I felt cut off from my future	0	1	2	3	4	

23. I couldn't imagine anything beyond this experience	0	1	2	3	4	
24. Things that had been important to me before did not matter any longer	0	1	2	3	4	
25. I felt there was no way back to my normal life after this	0	1	2	3	4	

PART 4

The following questions relate to the ways in which people sometimes describe their MEMORIES OF THE STROKE. Please rate the extent to which these statements apply to YOUR MEMORIES OF THE STROKE by circling the appropriate number. If the statement is not relevant for you, please tick 'not applicable.' There are no right and wrong answers to these questions.

Since the stroke…	Not at all	A little	Moderately	Strongly	Very Strongly	Not applicable
1. I feel that my memory for the stroke is incomplete.	0	1	2	3	4	
2. There are periods of time during the stroke that I cannot account for.	0	1	2	3	4	
3. I have trouble remembering the order in which things happened during the stroke.	0	1	2	3	4	
4. My memory of the stroke is muddled.	0	1	2	3	4	
5. I cannot get what happened during the event straight in my mind.	0	1	2	3	4	
6. Many different things trigger memories of the stroke.	0	1	2	3	4	
7. I experience feelings similar to those I had during the stroke even when I am not thinking of it.	0	1	2	3	4	

8. I am reminded of the stroke for no apparent reason.	0	1	2	3	4	
9. I find myself unexpectedly remembering the stroke.	0	1	2	3	4	
10. My memories of the stroke consist of vivid images.	0	1	2	3	4	
11. I experience strong emotions when remembering the stroke.	0	1	2	3	4	
12. The feelings I had during the stroke keep coming back to me.	0	1	2	3	4	
13. When I remember the stroke it is like it is happening again, here and now.	0	1	2	3	4	

Thank you completing this questionnaire