AN INVESTIGATION INTO POST-TRAUMATIC STRESS DISORDER FOLLOWING STROKE

by

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Submitted to The University of Edinburgh as part fulfilment of the degree of Doctorate in Clinical Psychology (2001).

CONTENTS

ACKNOWLEDGEMENTS

I would like to thank my supervisors, Ruth Thomson and Tony Prior, for their assistance and support during the past year. I would also like to thank the staff at the Astley Ainslie Hospital for their help in recruiting participants. This thesis would not have been possible without the individuals who gave up their time to participate in this project, and I am especially grateful to them. I would also like to thank my family and friends who have provided invaluable support over the past months in particular.

For Jonny.

This thesis has been composed by myself and the work contained herein is my own.

Elizabeth Carmel Flynn

ABSTRACT

There is now increased recognition that Post-traumatic Stress Disorder (PTSD) can occur after Traumatic Brain Injury (McMillan, 1996; Bryant & Harvey 1999). Recent literature highlights the occurrence of traumatic incidents which, although accompanied by brain injury, result in symptoms consistent with PTSD. Furthermore, this raises the question of whether PTSD can occur after non-traumatic brain injury, for example, stroke (Sembi et al. 1998). Memory for the event appears to be important in the development of PTSD symptoms particularly in relation to re-experiencing the event. The role of memory for the event in the development of PTSD following acquired brain injury remains unclear (Sbordone & Liter 1995).

Following on from work by Berry (1998), this study aimed to confirm whether it is possible for PTSD to occur following both haemorrhagic and ischaemic stroke. The effect of loss of consciousness on memory for the event, as well as the consequence of this on subsequent psychological distress is explored. The implications of a stressor which disrupts brain function is considered with particular emphasis on the frequency and type of symptoms reported.

The thirty-two individuals who participated in this study had recently experienced either a haemorrhagic or an ischaemic stroke. All participants were screened to exclude those who had severe cognitive impairment. Participants completed self-report measures describing current psychological distress including symptoms of post-traumatic stress disorder. Semi-structured interviews were conducted to diagnose PTSD. Data was collected on type, location and severity of stroke as well as demographic details. The results are discussed with reference to previous research findings.

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CHAPTER 1

INTRODUCTION

Post-traumatic stress disorder (PTSD) is usually associated with natural disasters, deliberately caused disasters or accidents. However, more recent studies have started to investigate the presence of this disorder following medical events. One particular area of investigation which continues to be controversial is co-morbid post-traumatic stress disorder and acquired brain injury. This thesis will review the existing research with particular attention given to the role of memory and consciousness in development of PTSD and the neurobiology of PTSD. This thesis will argue that PTSD is not incompatible with brain injury. It will also describe the mechanisms which could explain development of PTSD in brain injured individuals. The specific typology associated with post-traumatic reactions which present with co-morbid brain injury will also be described.

As most of the research to date has involved traumatically brain injured participants, this thesis aims to investigate whether post-traumatic stress disorder can occur following cerebrovascular accident. The emphasis will be on whether consciousness and memory are required for an individual to develop PTSD symptoms. A brief review of the literature on lesion location and mood disorders will be considered as the study also aims to investigate whether there is a link between lesion site and PTSD. Finally, the thesis aims to establish whether the PTSD typology associated with traumatic brain injury is also representative of the traumatic reaction following the experience of stroke.

1:1 Post -traumatic Stress Disorder

1:1:1 Definition

Post-traumatic stress disorder (PTSD) is the diagnostic category applied to individuals who develop particular psychological symptoms following a traumatic experience. The American Psychiatric Association DSM IV criteria for diagnosis are given in Appendix 1. The main diagnostic requirements are that the person has been exposed to an event which involved actual or threatened, death or injury, and that their response was one of intense fear, horror or helplessness. The symptoms of this disorder are grouped into three main categories: the individual repeatedly relives the event in some way, they avoid cues which remind them of the event and there is a numbing of their general emotional responsiveness despite increased physiological arousal. The prevalence of PTSD is difficult to ascertain as rates are influenced by the occurrence of disasters and they vary considerably depending on the assessment measures used. While the generally accepted rate of PTSD lies between 5 and 15 per cent of the general adult population (Yule, 1999), an estimated 69 per cent of the population are thought to be exposed to a traumatic event at some point during their lifetime (Sbordone, 1999).

1:1:2 What constitutes a stressor?

Post-traumatic Stress Disorder became a diagnostic category of the 3rd edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM III) in 1980. To be diagnosed as having PTSD under DSM III-R (1987) it was required that the individual had been exposed to an event which could be considered *'outside the range of usual human experience'* (DSM III-R p 247). Since the original entry both the definition and the criteria have evolved in response to epidemiological data which indicated that PTSD could occur in response to events which were not infrequent. As Kilpatrick, Edmunds & Seymour (1992) estimated that 13 per cent of women have been sexually assaulted, it could be concluded that

for women, sexual assault is not an event 'outside the range of usual experience'. Despite this, almost half of reported rape cases result in PTSD (O'Shea, 2001). In DSM-IV (1994), the definition of what constitutes a stressor was revised and there is now general consensus on the types of experiences which are associated with the subsequent development of PTSD. These include combat, rape, natural disasters or extreme deprivation. A recent epidemiological survey reported by Andrews, Rocco, Lampe, Hunt & Page (1994) found the most frequently occurring traumatic events which led to development of PTSD were; in males, combat or violent incidents, and in women, rape and sexual molestation. This revision meant the range of events which might constitute a trauma has been broadened.

1:1:3 PTSD and medical events

For a considerable time it had been recognised that a traumatic event, however it was defined, was not sufficient to result in PTSD. Research studies had been emphasising the role of individual differences in the development of PTSD. These included the event specific features, for example, the cognitive appraisal of the event as well as factors which predisposed the individual to develop PTSD in response to trauma. Brewin, Dalgleish & Joseph (1996), described trauma as an event which challenges the assumptions that an individual has about the world. This might include experiences which highlight personal vulnerability, for example being attacked, or those which contribute to the inability to achieve one's life goals. Research into PTSD began to highlight that events which were considered part of normal life experience could, nevertheless, have catastrophic significance. Definitions, including that by Brewin, were recognising the impact that major illness, disability or loss of employment could have on an individual's assumptions about the world. The consequences of these types of events began to be interpreted by some within a trauma framework. One of the earliest of these studies was by Shalev, Schreiber, Galai, & Melmed, (1993) who reported a series of case studies of PTSD in patients who had

undergone, what would be considered, routine medical procedures. These cases highlighted that although the event is an important part of the trauma, the individual's appraisal as well as the significance the event has for them, is crucial in the subsequent development of PTSD. Shalev et al, (1993) described one patient who developed PTSD following diagnosis of and treatment for a brain tumour. Despite making a good physical recovery with no residual disability, the patient experienced distressing intrusive imagery of being permanently disabled, thus suggesting that re-experiencing is not always dependent on memory of the event. O'Carroll, Masterton, Gooday, Cossar, Couston, & Hayes (1999) interviewed a group of patients who had experienced variceal haemorrhages and were surprised to find that only one patient in their sample of thirty, met diagnostic criteria for PTSD. The experience of variceal haemorrhage could be considered highly traumatic and this finding demonstrated that the event itself is not sufficient to result in PTSD. Research into psychological reactions to physical illness and medical events has continued. Ballard, Stanley, & Brockington (1995) described PTSD following childbirth, an event not 'outwith usual experience', and Bennett & Brooke (1999) described a PTSD prevalence rate of 10 per cent following myocardial infarction.

1:2 Acquired Brain Injury

1:2:1 What is acquired brain injury?

Acquired brain injury is the generic term applied to brain injuries which can occur as the result of a number of different mechanisms. Common mechanisms associated with brain injury include trauma, infection or vascular disruption. Brain injury which has been acquired as the result of trauma is relatively common. King (1997b) claimed that the annual incidence for hospital admission due to brain injury is between 250-300 per 100,000 of the population. The majority of these, approximately 75 per cent, will be classified as mild head injuries, with a Glasgow Coma Scale score on admission to hospital of between 13 and

15, or a post-traumatic amnesia (PTA) of one hour or less. The mechanism of injury provides information on the type of potential damage and the neuropsychological sequelae likely to be reported. A traumatic brain injury (TBI) resulting from a road traffic accident or a fall, tends to involve what is termed acceleration-deceleration injury. This means that as the individual has either stopped suddenly or as their head has hit a stationary object, the brain sustains a coup and contre-coup injury. This is the consequence of the sudden force exerted, causing the brain to be thrown about inside the skull. A coup injury affects the area below the site of impact while a contra-coupe injury describes lesions diametrically opposed to the point of impact (Walsh & Darby, 1999). Injury is not confined to points of direct impact. When the brain is shaken violently, as in acceleration-deceleration injury, the axons stretch and shear to the point where the connections within the brain substance are disrupted and damaged. Loss of consciousness can occur due to a number of reasons including brain stem or midbrain damage and perhaps "brain shock" (Lezak, 1995). The most commonly affected sites in acceleration-deceleration or concussional injuries are the frontal and the anterior temporal areas. Secondary injuries including, for example, cerebral oedema are potentially serious complications. As the brain is contained within the skull there is no surplus space to accommodate the expansion associated with cerebral swelling. This can lead to raised intracranial pressure, intracerebral haemorrhage and secondary damage (Lezak, 1995). Brain injured patients who sustain concussional or diffuse injuries are likely to experience neuropsychological and neurological sequelae referred to as post-concussional syndrome (King, 1997b). This is one factor which complicates the issue of PTSD and brain injury. In reviews of literature reporting dual diagnosis, some authors propose that what the clinicians and researchers diagnose as PTSD is actually post-concussional syndrome (PCS) (Hickling, Gillen, Blanchard, Buckley & Taylor 1998).

The main features of PCS are described by Miller (1999) as:

- attention or concentration problems
- learning and memory difficulties
- concrete thinking
- psychomotor retardation
- mood disturbance including depression and anxiety
- increased irritability and agitation
- impulsive behaviour
- inertia or reduced motivation or initiation

It is clear that there is considerable symptom overlap between PCS and PTSD which will make dual diagnosis extremely difficult. Post-concussional syndrome is believed to be extremely common after brain injury. King (1997b) reports figures of 50 per cent in cases following even mild and moderate traumatic brain injury and this might have led to symptoms being attributed to PCS which might have represented a post-traumatic reaction. The overlap with PCS was clearly illustrated by McMillan, (1991) who published a case study of a severely brain injured patient who presented with symptoms consistent with post-concussional syndrome. It was only following a more detailed investigation of the symptoms that it became apparent to him the patient was also experiencing the defining characteristics of PTSD, namely intrusive thoughts as well as both cognitive and physical avoidance.

1:2:2 Could traumatic brain injury result in PTSD?

Investigations of psychological distress following road traffic accidents have been frequent and some report PTSD prevalence rates as high as 39 per cent (Mayou, Bryant, & Duthie 1993; Blanchard, Hickling, Taylor & Loos 1995). Although relatively common, road traffic accidents frequently expose those involved to risk of death or serious physical injury. As the most common cause of traumatic brain injury is road traffic accident, it is reasonable to

conclude that patients who have experienced a traumatic brain injury might also be at risk of developing PTSD (Hickling et al 1998). Other common causes of traumatic or acquired brain injury are assaults, falls or industrial accidents, all of which are events which are sudden, could be life threatening and are often associated with helplessness or fear. The DSM III-R includes car accidents amongst the types of event which might result in PTSD. Furthermore, both the third and fourth revisions of DSM acknowledge the possibility of co-morbid head injury:-

> *"sometimes there is a concomitant physical component of the trauma which may involve direct damage to the central nervous system (e.g. malnutrition or head injury)"* (DMS III-R 1987 p 248).
>
> *"general medical conditions may occur as a consequence of the trauma (e.g. head injury) "* (DSM IV p 426).

Despite this, the issue of whether an event which has caused a brain injury could also result in PTSD, remains contentious. However, it is not that brain injury per se is incompatible with PTSD, but whether it is possible for someone to have PTSD if they have no memory of the event. Some authors have even argued that the absence of memory resulting from brain injury protects individuals from becoming traumatised by their experience. In 1943, Adler (cited in O'Brien & Nutt 1998) first suggested that brain trauma might protect the individual from developing the symptoms that we would recognise now as PTSD. She described individuals who had been involved in the Coconut Grove Fire disaster in 1940's America and reported better neuropsychiatric outcome for those patients who had lost consciousness during the incident. O'Brien & Nutt (1998) described another of Adler's studies which reported the same phenomenon in a different group of patients. She had observed that of those with retrograde amnesia only a small number had subsequently developed any psychogenic symptoms. The main reason why brain injury could be considered to protect the individual from developing PTSD is if the brain shuts down then memory of the event cannot be stored or encoded. In the discussion that follows the author intends to describe

theory and research relating to both memory in general, and the neurobiology of fear and trauma. This suggests that traumatic memory is acquired in such a way that it might not be incompatible with brain injury.

1:2:3 Role of memory

Discussion of memory usually relates to memory which has been explicitly processed and which has required an awareness of the environment. The information stored via this route is then consciously accessible and would include autobiographical and semantic memories. It has been concluded from a number of investigations of memory impairment that the explicit memory system is within the domain of medial temporal area and the limbic system, specifically the hippocampus (Krikorian & Layton 1998; Kolb & Wishaw 1990). As well as the hippocampus being important in constructing explicit memory it also stores the temporal features of the memory and makes connections to any other stored information which might be relevant (Bechara, Tranel, Damasio, Adolphs, Rockland, & Damasio 1995). Establishing an explicit memory requires the individual to be conscious and would be disrupted by brain injury. It is this which has led some authors, including Mayou et al (1993), to conclude that only patients who have remained conscious during a traumatic event could develop memory of it and therefore go on to develop symptoms associated with PTSD. Sbordone & Liter, (1995) categorically state that traumatic brain injury and PTSD are '*mutually incompatible and different disorders"* (Sbordone & Liter, 1995, p411). Their evidence for this conclusion comes from a study in which participants who were diagnosed with either mild TBI or PTSD were assessed. In the course of the interviews the researchers were struck by the contrasting reports given of the events and symptoms. All of the participants with PTSD were able to recall their experiences in detail and they all reported having intrusions, flashbacks and nightmares. In contrast, none of the participants with TBI were able to describe the event or reported having any of the re-experiencing symptoms. As a result the

authors concluded that none of the TBI group had PTSD. O'Brien & Nutt (1998) state that as loss of consciousness leads to the absence of memory, patients:-

> *"will not have any horrific memories, flashbacks or nightmares and so will not re-experience the incident repeatedly"* (O'Brien & Nutt 1998, p102).

Furthermore, it could be argued that the absence of memory for the event, means reminders of the event do not evoke distress. The individual does not therefore feel the need to avoid cues which are associated with the incident. It is not thought possible for re-experiencing or avoidance to be among the psychological symptoms which could occur subsequent to TBI. However, both O'Brien & Nutt (1998) and Sbordone & Liter (1995) appear to present the view that PTSD has a specific symptom profile which will be the same for all patients and furthermore that explicit processing is the only way that traumatic memory can be acquired.

In addition to the conscious processing of information, memory can also be processed implicitly. This refers to the acquisition and storage of information which has not been conscious and despite it being inaccessible it nevertheless influences our conscious awareness (Bradshaw & Mattingley 1995). This type of processing is thought to store primarily perceptual representations and because it is not related to conscious awareness, there is no connection between these implicit aspects of memory and previous experience. Bradshaw & Mattingley (1995) reported that this type of processing is mediated by the cerebellum. It was one of the classic cases of neuropsychology reported by Scoville & Milner (1953), that described an example of intact implicit processing despite impaired explicit processing (cited in Walsh & Darby 1999). Their patient, HM, had a bilateral, medial temporal lobe resection and subsequently lost his ability to explicitly learn new information. However, he continued to demonstrate the ability to learn information implicitly. This was the first of many neuropsychological studies to demonstrate that these two memory processes are differentially affected by brain injury. During brain injury the

system responsible for the explicit processing of information is disrupted and this will clearly have an effect on how the event is encoded. However, implicit memory means that despite the disruption to the explicit system, information can and does continue to be encoded and stored (Bradshaw & Mattingley 1995), even if this occurs outwith our conscious awareness.

Current cognitive models have taken this dual processing of information into account when explaining development and symptoms of PTSD. Brewin (2001), suggested that when faced with trauma, information is processed into a verbally accessible system (compatible with explicit processing) and a situationally accessible system (compatible with implicit processing). The verbally accessible system is capable of storing information which would inhibit the fear response. Post-traumatic stress disorder symptoms develop as the verbally accessible information, which usually competes with the situationally accessible system to inhibit fear, is disrupted which means the information stored in the situationally accessible system predominates. If this occurs, the fear response is repeatedly triggered by situational cues which are not mediated by the verbally accessible system or influenced by conscious awareness. The person might consciously 'know' that the event is not recurring but the trigger at a perceptual or sensory level which activates their physiological reaction, does not have access to this information. Brewin proposed that the PTSD symptoms facilitate the transfer of information to the verbally accessible system where it can be fully processed.

1:3 Neurobiology of fear and PTSD

Not only does implicit processing have an important role in storing trauma memory in non-brain injured but discussion of the neurobiology of fear and PTSD suggests that the acquisition of trauma memory is not the same as normal memory and therefore might not be disrupted by brain injury. The group of structures, which includes the hippocampus and the

amygdala, known collectively as the limbic system is important in both memory and emotional responding (Walsh & Darby 1999). When faced with a threat, sensory information is projected from the thalamus to the amygdala where it is then appraised before a decision is made about necessary action. The amygdala is directly responsible for triggering the fear response. The fear or stress response initiates a chain of biochemical and behavioural changes. This includes; activation of the Sympathetic Nervous System which is responsible for the release of catecholamines; the Autonomic Nervous System and finally the Hypothalamic-Pituitary-Adrenal Axis which is responsible for activating corticotropin releasing factor which stimulates the adrenal gland to release cortisol (Yehuda 2000). The catecholamines, including norepinephrine are responsible for energising the body and in moderate amounts they facilitate the consolidation of explicit memories (Yehuda 2000; Charney, Deutch, Krystal, Southwick, & Davis 1993). The cortisol which is released will eventually inhibit the fear response through a negative feedback loop which connects the hippocampus, hypothalamus, pituitary-adrenal systems before returning to the hippocampus. In this way the effect the catecholamines have on memory are modulated by levels of cortisol.

In considering PTSD, it is useful to understand why, in some circumstances, the fear response results in a post-traumatic syndrome and what effect this has on memory. At a neurobiological level there appears to be a difference between the normal fear response and a PTSD response despite them both being mediated by the same structures. At a biological level once a stressor has been removed the fear response is inhibited through cortisol production and this allows the body to return to its normal state, the process described as extinction. This process is facilitated by explicit memory as features of the current situation are integrated into and then influenced by pre-existing information. It is in this way that previous experience will influence the fear response and eventually leads to inhibition. In

those individuals who develop PTSD this process is disrupted, as the way in which some of the memory has been acquired does not seem to connect to information related to past experience. Van der Kolk (1994) hypothesised that this is the result of the cortisol failing to inhibit the production of catecholamines. The excess catecholamines result in the memory being 'overconsolidated' and it does not acquire spatial or temporal associations and it cannot be influenced by previous experience. One manifestation of this is, that despite being removed from the stressful situation and knowing that the stressor is no longer present, the individual continues to react as though the event was recurring. When they are exposed to reminders of the event they re-experience it with the same intense level of arousal. It appears as though, in those people who develop PTSD, the process of extinction has not been completed. This results in an individual's 'fear network' being constantly activated and easily triggered (Yule 1999).

Current descriptions of the neurobiology of fear and PTSD suggest that, during intense stress, memory is processed in a different way. This is evident by what is described as psychogenic amnesia experienced by some, contrasting with the intrusive re-experiencing reported by others. For some, during a traumatic experience, both explicit and implicit aspects of memory appear to be stored in a more robust way. Despite having knowledge about the way in which brain injury disrupts the explicit system it is not known if, or how, brain injury would affect the acquisition of traumatic explicit memory. As the encoding of explicit memory is enhanced during intense stress, and because implicit processing contributes to trauma memory, it is possible that some features of the traumatic experience could be stored despite brain injury.

1:4 PTSD and Brain Injury

1:4:1 PTSD through implicit memory

There is still a considerable amount that we do not know about how memory is acquired and stored. What is clear from the discussion so far, is that memory is not processed in the usual way during extreme stress. McNeil & Greenwood (1996) proposes that the emotional significance of an event, which could produce a sense of helplessness or horror and involve a threat to life, could even be strong enough to override the defect in explicit memory associated with brain injury. Even if it did not, it is still probable that sensory and perceptual representations of the event are encoded at an implicit level. Krikorian & Layton (1998) described a patient who developed PTSD following anoxic brain injury. The patient had been buried under sand for a period of 15 minutes following a construction accident. The anoxic injury resulted in a coma which lasted for two days and post-traumatic amnesia which lasted several weeks. As well as being amnesic for the accident the patient had retrograde amnesia for approximately 15 years prior to the accident, which could indicate extensive hippocampal disruption. It was not until some weeks after being discharged from hospital that the patient's wife reported that his personality had changed. Although this type of report is not uncommon following brain injury (Brooks, et al 1986), it became apparent to the authors that the patient was experiencing a post-traumatic reaction. Despite having no conscious recall of the accident he described symptoms which were representative of it. The authors concluded that although the patient's declarative memory had been disrupted due to the reduced oxygen to his brain, his implicit system had processed certain aspects of the event, including the fear and some sensory images. Although it could be argued that the patient developed PTSD vicariously the authors believe this to be unlikely as his symptoms developed while he was still in a period of confusion. This provides some evidence for the suggestion that non-declarative or implicit memory is a crucial mechanism in PTSD after brain injury. Despite this, Sbordone & Liter (1995) are of the opinion that if implicit

processing of trauma during brain injury was possible there would have been some evidence of PTSD in their study. However, their study had a number of limitations, in particular as the sample was very small and taking into account prevalence rates in general, it might simply have been that they have failed to find examples of PTSD because their sample was too small to detect them.

1:4:2 Traumatic islands of memory

The evidence indicating that PTSD does occur after brain injury continues to accumulate. In addition to implicit memory there are other mechanisms by which PTSD symptoms could occur. Post-traumatic amnesia refers to the length of time which elapses before a patient who has been unconscious regains their ability to lay down continuous new memories. During the period of PTA, islands of memory occur in at least one third of all mild head injury cases (Gronwall & Wrightson 1980). Post-traumatic amnesia is a period during which consciousness fluctuates and where an individual might be able to acquire some new memories although this is not being done in a continuous way. King (1997a) suggested that the period of variable consciousness which occurs during post-traumatic amnesia would be sufficient to allow some 'islands' of memory to be stored. He presented a case study describing a man who, after being struck from behind by a car while he was walking, went on to develop PTSD. The patient had only a single memory relating to the incident, which had been laid down when he was in a period of intermittent consciousness. It was this memory that had become the source of his re-experiencing. King (1997a) hypothesised that these 'islands' occur because, as discussed above, the intensity of the emotional and physiological arousal has been intense enough to establish an explicit memory of the event. This is the mechanism sometimes known as creating 'flashbulb memories' (Brown & Kulik 1977) which are particularly vivid and long-lasting memories, which have been encoded more strongly due to the suddenness or significance of the event.

1:4:3 Post injury memories

McNeil & Greenwood (1996) described a case study of a patient who had been the pedestrian victim of a road traffic accident which had been an intentional attack on his life. He experienced a severe head injury and also developed symptoms consistent with PTSD diagnosis. In this case it was suggested that the PTSD developed in response to information given after the event. Once the patient had regained consciousness it appears that he was vicariously traumatised by information given to him about the attack. DSM-IV permits diagnosis even if the event has not been directly experienced. It allows that other people can be traumatised by witnessing or hearing about a life threatening event which has occurred to a close friend or family member. O' Carroll et al, (1999) in their study of PTSD subsequent to having variceal haemorrhage noted that a number of participants had commented that the experience had been more distressing for their partners and family members. Vicarious experience is therefore consistent with the diagnosis of PTSD.

An elaboration of this is the construction of pseudo memory, based on what the individual imagines to have occurred. McMillan (1996) reported that the period of amnesia for some patients is itself traumatic, and the gaps in memory might be filled by creating a narrative, or by the patient visualising themselves being in the accident. The usual experience for brain injured patients is that they regain consciousness in hospital, often having severe physical injuries, perhaps being told how close to death they had been, yet having no recollection of how they came to be in that situation. They may then develop ideas and beliefs about the event which can subsequently become the source of the traumatic re-experiencing.

Developing PTSD symptoms on the basis of the mental construction of a traumatic event is consistent with PTSD in other populations (Shalev et al, 1993). Miller (1999) discussed four case studies of individuals who had no loss of consciousness, who constructed scenarios which reflected hypothetical outcomes of their traumatic experiences. In one case,

the patient had fallen from a roof only narrowly missing a large metal spike. He later reported intrusive images which centred around what might have happened had he landed a couple of inches closer to the spike. The idea that a patient may construct an image which then becomes the source of traumatic re-experiencing is not an unusual feature of PTSD symptoms.

The images, whether constructed or accurate recollections of events, can be equally traumatic as shown by research by Bryant & Harvey (1998). They compared three groups on the intrusive imagery they experienced. The groups were PTSD patients who had accurate recall of the trauma, PTSD patients who had amnesia for the event and a group of PTSD symptom simulators who acted as a control group. The hypothesis was that the group without recall of the trauma, would report the same degree of belief in the accuracy of their images as the group with accurate recall. This hypothesis was based on source monitoring theory which describes that a belief in the accuracy of memories is attributed to those which are accompanied by perceptual and contextual detail (Johnson, Hashtroudi, & Lindsay 1993). The study supported the hypothesis that there was no difference between the intrusions reported by PTSD patients with and without accurate recall of the event, on the variables of imagery, intrusiveness and the affect associated with the images. The explanation proposed by the authors was consistent with the theory of Brewin et al (1996) of dual representation. They suggested that the patients who had been amnesic for the event had actually encoded a non-verbal memory of the event, which then provided the source of the re-experiencing. The importance of this type of research is that, whatever mechanisms are operating, patients with brain injury do report PTSD. They have also been found to re-experience the event consistently and Bryant & Harvey's (1998) study demonstrated that this is accompanied in some patients with the belief that what is being re-experienced is an accurate account of the event. Whether the recall is accurate or not may not be entirely relevant to the debate of whether or not PTSD can occur.

On reviewing the literature it is apparent that there is more evidence to suggest that PTSD can occur after brain injury than that it cannot. In some studies symptoms of PTSD have been reported to have been found in as many as 84 per cent of patients with co-morbid mild brain injuries (Feinstein, Hershkop, Jardine, & Ouchterlony 2000). Bryant & Harvey (2000) reported a PTSD prevalence of 27 per cent in a sample of patients who had severe brain injuries. Although the mechanisms involved in acquisition of traumatic memory and development of PTSD might be different in those with and without brain injury, it is clear from these studies that there is evidence PTSD can occur following both mild and severe brain injury.

1:4:4 If the mechanisms are different is it still PTSD?

It could be that as a consequence of different mechanisms the symptom profile may be different although McMillan (1996) reported that his dual diagnosis patients experienced the full range of PTSD symptoms, including intrusive thoughts and nightmares. However, in a later study, Bryant & Harvey (2000) reported that only 19 per cent of those brain injured patients with co-morbid PTSD had intrusive imagery, although 96 per cent reported symptoms of emotional and physiological reactivity. Perhaps it is not as Adler suggested in 1943 that loss of consciousness protects from the development of PTSD in its entirety, but that some symptoms are likely to be less prevalent than others. It might be as Bryant & Harvey (2000) conclude, that loss of consciousness is protective in reducing the likelihood that PTSD patients will experience intrusive imagery. This might also help explain the lack of PTSD found in the study of Sbordone & Liter (1995) and Mayou et al (1993). However, it does not mean that because the symptom profile is different that it is not PTSD. The DSM IV (American Psychiatric Association, 1994) gives equal weighting to all symptoms with none being more important than any other (Solomon, Neria, Ohry, Waysman, & Ginzburg, 1994). Furthermore, it is consistent with other trauma research for some symptoms to be

reported more frequently than others and there is no profile of symptoms that is considered to reflect 'typical' PTSD. In the general trauma literature different types of trauma are already found to be associated with different post-traumatic reactions. The PTSD reaction associated with combat for example, has been found to differ from that associated with civilian traumas. North, Smith & Spitznagel (1994) reported that the most frequently reported symptoms of survivors of a mass shooting in the US was intrusive recollections. Solomon, et al (1994) also reported intrusions as the most frequently reported symptoms of combat veterans. However, Blanchard et al (1995) reported that in their study the most frequently reported symptoms following car accidents were behavioural and cognitive avoidance. Heffernan & Cloitre (2000) in their study described the victims of childhood sexual abuse as reporting arousal and avoidance symptoms more than re-experiencing. A different profile of PTSD symptoms in those with co-morbid brain injury would not be inconsistent with diagnosis.

1:4:5 Is there evidence for a unique symptom profile?

As brain injury could be considered to be a unique type of stressor, it is possible that the symptom profile might also have unique features. Turnbull, Campbell & Swann, (2001) concluded in their study that brain injury did seem to alter the presentation of post-traumatic symptoms. However, accepting different clinical subtypes of PTSD or even subsyndromal presentations would not be incompatible with DSM IV diagnosis. Within the category of PTSD, distinctions are drawn between acute versus chronic subtypes, and acute or delayed onset. It is also generally accepted that despite the manifestation being different in other groups it can nonetheless be classified as PTSD, for example, with children (Miller 1999). Some studies have suggested that subsyndromal or partial forms of PTSD, where the number of criteria met might fall short of that required by DSM IV, might instead indicate a different subtype of the disorder. Schnyder, Moergeli, Klaghofer & Buddeberg (2001)

provided a definition of subsyndromal PTSD as those cases where criterion B was met as well as either, criteria C or D.

A number of authors are of the opinion that PTSD is more accurately considered on a continuum rather than as dichotomous (Yule, 1999). Consistent with this view Alarcon, Deering, Glover, Ready, & Eddleman (1997) believe that it is more useful to describe PTSD as a clinical typology with clinical subtypes. They suggest six subtypes of PTSD which are based on the symptoms most prominent in the presentation. For example, the 'neurotic' subtype presents with heightened psychomotor tension, phobic behaviour and evidence of avoidant strategies, in addition to the core symptoms. The 'organomorphic' subtype includes manifestation of cognitive impairments, including reduced information processing ability and impaired concentration. In the Sbordone & Liter (1995) study, both the PTSD group and the TBI group reported the same frequency and type of cognitive symptoms but significant differences on re-experiencing indices. This might be suggestive of a different profile of PTSD symptomatology in patients who have brain injury. There are a number of reasons why it is likely that the manifestation of post-traumatic distress is different in the acquired brain injury population:

1. Firstly, the event is processed differently and therefore there may be more emphasis on some symptoms as opposed to others. This is consistent with studies which have reported that intrusive symptoms are less frequently reported in trauma patients who have also sustained brain injury. These patients have also tended to report feeling less afraid and less helpless than non-brain injured patients during the traumatic event (Bryant & Harvey 1995; Bryant & Harvey 1999). As well as the acute symptom presentation being different other research has suggested that the longitudinal course might also be different. Bryant & Harvey (1999) compared PTSD patients with and without brain injury at two time points. They found that the TBI group experienced less intrusive

imagery than the non-TBI group in the acute stage but, that at six months, there was no significant difference in rates of intrusions. In fact, the TBI group reported more intrusions while the non-TBI group reported less.

2. The physical impairments and disabilities which can accompany brain injury could also result in a different manifestation of the disorder. As PTSD is associated with increased stress it might be that the increased stress associated with psychological adjustment to a brain injury, is the final precipitant (Miller, 1999). The symptoms might not develop until the individual has returned home and is forced to confront any limitations, they may have. This could suggest an increased risk of delayed onset PTSD in this group. King (1997a) described a case study where the patient did not present with symptoms which met diagnostic criteria for PTSD until four months after being discharged. McMillan (1991) also described delayed onset of PTSD following severe brain injury.
3. In addition to point 2, it is possible that avoidance of reminders of the event might not be feasible immediately following the injury. The individual is likely to be hospitalised and is therefore not able to avoid reminders, which could include the hospital, medical interventions or the physical injuries which were consequences of the event. As this may produce a flooding effect rather than the usual avoidance, it is possible the symptoms will present differently. The low PTSD prevalence reported by O' Carroll et al, (1999) was explained as the participants being unable to avoid reminders of the incident due to the haemorrhage having occurred in the patients' own homes. The same may apply to brain injured patients.
4. It could also be that events subsequent to the stressor contribute to a different presentation of symptoms, for example the focus following brain injury tends to be medically orientated, with the emphasis on physical recovery. This might also result in delayed onset of the symptoms (Miller 1999).

5. The features of post-concussional syndrome will mean than certain symptoms of PTSD are presented differently or more frequently, thus contributing to an altered presentation of symptoms. For example, concentration impairment is a common sequelae of brain injury while also being a feature of PTSD. Due to the overlap in PTSD and post-concussional symptoms it would be easy to attribute some PTSD symptoms to an organic basis even when they are exacerbated by the post trauma reaction.
6. As discussed earlier, the lack of recall of the event might not be a protective factor but an exacerbating one. The impaired recall and 'absence' which many brain injured individuals report, may exacerbate feelings of dissociation and distress. Inability to recall the event has also been associated with poor adjustment (Bryant & Harvey 1999).

A specific presentation would also be consistent with Brewin's (2001) model of PTSD. As it could be argued that brain injury interferes with the production of verbally accessible memories, it may increase the likelihood that PTSD will develop as the information stored within the situationally accessible system predominates. Brewin (2001) argues that:

> *"any factor that interferes with the construction of a detailed, consciously accessible memory for intense moments of the trauma would be predicted to lead to a worse outcome"* (Brewin 2001, p384).

It may be that due to the inability to recall the event, PTSD is more likely to occur following brain injury. It is not uncommon for people with no recall of their brain injury to become preoccupied with trying to find out what happened to them.

Ohry, Rattock, & Solomon (1996) reported that 33 per cent of their traumatically brain injured sample met full diagnostic criteria for PTSD. They explored the pattern of symptoms which had been reported, and concluded that the most prevalent were amnesia for the event, poor concentration and physiological reactivity. The least endorsed symptoms included intrusive re-experiencing. Scores from the Impact of Events scale indicated that

the TBI group had higher levels of avoidance than intrusions, a finding contrary to that in studies involving combat survivors, which indicate that intrusion sub-scale scores are usually higher than the avoidance scores (Solomon, Neria, Ohry, Waysman, & Ginzburg 1994). This finding raise raises two questions: a) why are avoidance scores high when there is no recollection of the event, and b) what could the function of avoidance be? In other PTSD populations, avoidance is considered to be a strategy adopted to reduce exposure to reminders of the event which result psychological distress and heightened physiological arousal. If there is no memory to be reminded of, it opens a debate on why avoidance would be high. This evidence further supports the contention that there is a clinical typology of PTSD associated with brain injury.

It seems therefore, that there is considerable evidence to suggest that, during a TBI, it is possible to have processed sufficient information to allow PTSD to develop. Furthermore, there is evidence from a number of studies that explicit memory is not required to develop PTSD (Ohry et al 1996).

1:5 Cerebrovascular Accident and PTSD

1:5:1 Cerebrovascular accident

Traumatic brain injury affects approximately 90,000 people in Scotland every year (Currie, Ritchie, & Stott 2000). The number of people affected by cerebrovascular accident (CVA) or stroke, as it is more commonly known, is less than TBI at roughly 15,000 people each year (SIGN Guidelines, 2001).

Cerebrovascular accident (CVA) is brain injury which is due to a disruption to the blood supply in the brain caused by two main mechanisms:

1. Ischaemia - which is the result of an artery being obstructed. This can be thrombotic when blood flow is reduced due to a change in the walls of the blood vessel, or embolic when the occlusion is due to material formed elsewhere breaking off and then lodging in the vessel. If brain tissue is sufficiently deprived of oxygenated blood, permanent injury can result, referred to as infarction (Lezak 1995). Certain arteries are more likely to be affected than others. Blockage most commonly occurs at points where vessels bifurcate. The outcome of an ischaemic stroke depends on which vessel was occluded, the extent to which it was blocked and whether a collateral vessel to continue to supply blood, was available.

2. Haemorrhage - which can be due to one of three main mechanisms; a ruptured aneurysm, arteriovenous malformation or an intracerebral bleed. In the case of the first two the ruptured artery results in blood being released into the sub-arachnoid space, allowing it to leak onto the brains surface before it penetrates the brain substance. An aneurysm is a weakness in the wall of the blood vessel which can 'balloon' out, filling with blood to the point where it ruptures. An arteriovenous malformation is less common accounting for only 1 per cent of haemorrhagic strokes (Lezak 1995). It refers to a developmental malformation of a vessel which means the arteries and veins are not formed properly, as a result of which they are prone to recurrent rupture. With both types of precipitant the blood flow into the brain is rapid and is often accompanied by sudden raising of intracranial pressure. The third mechanism of haemorrhagic brain injury is intracerebral bleeding where blood goes directly into the brain's substance. The blood destroys the brain tissue and also causes pressure effects, large haemorrhages frequently cause death in this way by displacing vital structures. This bleeding tends to be in a localised region and is often associated with hypertension or trauma.

The prevalence, symptoms and course of these different types of stroke vary. Ischaemic strokes are more common and account for approximately 80 per cent of all strokes (Caplan & Stein 1986). Thrombotic strokes usually occur when the individual's circulatory system is least active, meaning it often occurs at night or on first waking. The embolic and haemorrhagic strokes occur when circulation is more active and when blood pressure rises. It could be that people have more awareness that something is wrong with embolisms or haemorrhages, a factor which could prove to be important in the development of PTSD. During an ischaemic stroke, loss of consciousness is rare, occurring in only 15 per cent of patients (Caplan & Stein 1986). This is because there is usually a collateral blood supply which is available to provide the minimum level of oxygen required to maintain consciousness. It is also very unusual that the brain stem, which controls the level of consciousness, would be affected by ischaemia unless it's own vessels are those which are disrupted. As there is no sudden release of blood or raised intracranial pressure, few patients will experience a headache. In contrast, a sub-arachnoid haemorrhage (SAH) is usually accompanied by an excruciating headache with only 5 per cent of patients having no accompanying headache (Chicoine & Dacey 1997). The patient will usually vomit and then experience a reduced level of consciousness. Patients who suffer a SAH are more than four times as likely as ischaemic patients to lose consciousness (Caplan & Stein 1986). This is the result of blood escaping into the brain tissue which, by various mechanisms including vasospasms (sudden contraction of vessels) often results in impaired consciousness. With an intracerebral haemorrhage the patient tends to decline more slowly, they may experience a headache and vomiting in the later stages before their consciousness reduces.

1:5:2 Cerebral circulation and mood disorders

The brain receives its blood supply from the internal carotid and the vertebro-basilar arteries. The internal carotid artery gives off a number of minor branches, before splitting

into the anterior and middle cerebral arteries. The vertebro-basilar artery serves the posterior cerebral arteries. The junction of a number of the main arteries known as the Circle of Willis, includes the anterior communicating, anterior cerebral, internal carotid, posterior communicating and posterior cerebral. Strokes can affect any of these arteries. Frequent sites for ischaemia include the origin of the internal carotid artery, the lower portion of the basilar artery, the stem of the middle cerebral artery and posterior cerebral artery. Approximately 80 per cent of SAH are due to a ruptured aneurysm occurring somewhere within the Circle of Willis (Chicoine & Dacey 1997), with the most common site of rupture being the anterior communicating artery or the middle cerebral artery bifurcation. With an intracerebral haemorrhage it is more likely that brain stem structures, for example, basal ganglia or pons will be affected.

As cerebral infarction usually affects a relatively specific area of the brain a number of investigations have attempted to relate mood disorders to specific brain locations especially as mood disorders are known to be common following stroke (Schramke, Stowe, Ratcliff, Goldstein, & Condray 1998; Sharpe, Hawton, House, Molyneux, Sandercock, Bamford & Warlow 1990). MacHale, O'Rourke, Wardlaw & Dennis (1998) reported that in their sample 26 per cent met DSM criteria for either an anxiety or a depressive disorder. Sharpe et al (1990) reported an overall prevalence rate for depression of 18 per cent in their sample of stroke patients. Although most research has concentrated on post-stroke depression, Sharpe et al (1990) concluded that anxiety is probably more common post-stroke than depression. Robinson, Starr, & Price (1984) reported a higher prevalence of depression in stroke patients where the left hemisphere had been affected. In a later study, Starkstein & Robinson (1989) reported that left hemisphere strokes which were located in the frontal and basal ganglia regions were associated with more frequent cases of depression. They also concluded from scan data that the closer the lesion was to the frontal pole the greater the severity of depression. In contrast, Sharpe et al (1990) found no evidence of a link between

depression and site of lesion. The most recent review of all of the literature relevant to depression and lesion location concluded that studies which had suggested a link between location and depression were methodologically flawed and that no relationship has yet been confirmed (Carson, et al 2000).

Some recent investigations into PSTD have proposed the presence of a similar link between PTSD and specific brain areas. Freeman & Kimbrell (2000) reported that in their patient who had PTSD, right frontal damage reduced the frequency of his intrusive symptoms. This finding is supported by the reports of the right hemisphere being important in storage and retrieval of traumatic memories (Berthier, Posada, & Puentes 2001). However, Berthier et al (2001) described the case of a combat veteran who did not develop PTSD symptoms until after he sustained a traumatic brain injury. They proposed that the affected right frontal area identified on the scan, had functioned prior to injury, to inhibit his intrusive symptoms. However, although both studies implicate the right anterior region, one suggests it had the effect of reducing intrusive re-experiencing while the other suggests lesions to this area precipitated these symptoms.

1:5:3 Stroke and PTSD

There is a paucity of literature on post-trauma reactions following stroke, despite criterion A from DSM IV for post-traumatic stress disorder being met by the experience of stroke per se. The individual has been exposed to an event in which injury is likely to have occurred and where the response could feasibly involve intense fear, helplessness or horror. The issue of whether individuals can experience criterion B, persistent re-experiencing of the event, is an issue that this study addresses. It may be necessary to exclude organic reasons for the criteria of section C, in particular inability to recall the event and impaired concentration, both of which could be attributable to brain injury. In research of PTSD and traumatic brain injury it is the symptoms of criterion D which have proved most difficult to

separate from those of the post-concussional syndrome. In stroke, the absence of post-concussional syndrome makes it possible to attribute symptoms to PTSD per se, rather than to post-concussional symptoms. Therefore, by considering a population of stroke patients this confounding variable will be removed and the pitfalls of dual diagnosis of brain injury and PTSD, as described by McMillan (2001), will be avoided.

In a literature review only two papers were found to have concentrated on PTSD following stroke. Berry (1998) investigated the nature of the psychological distress reported by patients who had experienced SAH. All of the participants in her study complained of anxiety, loss of confidence and impaired memory. Some also reported being preoccupied with the suddenness of their experience. Thirty-two percent of the sample reported experiencing recurrent intrusive imagery, nightmares, and flashbacks and the entire sample reported behavioural avoidance and increased physiological arousal. The study concluded a prevalence rate for PTSD of 32 per cent. The fear of recurrence was reported to be a particularly salient feature in this population, as was catastrophic misinterpretation of normal physiological sensations. This contributes further evidence that the typology of PTSD following brain injury might be different. Sembi et al (1998) aimed to establish whether a syndrome, which would meet either full or partial diagnostic criteria for PTSD, existed in a sample of patients who had ischaemic strokes. They estimated a post-stroke prevalence of PTSD of 7 per cent using the Penn Inventory, and 21 per cent using the Impact of Events scale, although neither of these is diagnostic. A diagnostic interview for those patients who had met cut-off levels on self-report measures established the prevalence rate at approximately 10 per cent. No correlation was found in this sample between the avoidance scale on the IES, and a measure of physical disability, suggesting that avoidance behaviour was more likely to be a consequence of cognitive processes rather than a result of physical limitations. The authors concluded that the patients in the study presented with

symptoms of arousal, intrusion and avoidance, which they interpreted as suggesting that the patients had, "*appraised the event as extremely traumatic and had subsequently developed a traumatic reaction*" (Sembi et al, 1998 p321). Both studies provide evidence for the existence of PTSD following stroke. The role that loss of consciousness and memory of the event has on the development of PTSD, was not discussed in either paper. As the type of brain impairment associated with stroke tends to be more focal than in a concussional brain injury, this means it is more likely that individuals will recall the event, even when they have subsequently lost consciousness. Furthermore, as the onset is more gradual than in TBI, some information about the experience might be stored. By including both ischaemic and haemorrhagic patients it will be possible to compare one group that is more likely to lose consciousness and lack explicit memory of the event with another group that is less likely to lose consciousness. Furthermore, it will be possible to compare the effect the type of stroke has on prevalence of PTSD. By exploring the symptoms reported it will also be possible to contribute to the growing evidence of a different manifestation of PTSD following brain injury.

1:6 Aims and hypotheses of the study

The main aim of this study was to ascertain whether the experience of cerebrovascular accident could precipitate a post-traumatic reaction. Furthermore, the study considered whether consciousness and memory of the event are required to develop PTSD. As some studies suggest an association between PTSD and the right hemisphere, the study aimed to investigate the presence of this type of relationship in a stroke population. This study also aimed to investigate whether the symptoms reported after CVA are the same as in other groups, in particular to consider whether there is a typology of PTSD which could be said to be specific to acquired brain injury populations.

Study Hypotheses

Further to the review of the literature and consistent with the aims of the study the hypotheses were:-

- Hypothesis One: It is hypothesised that a cerebrovascular accident can result in PTSD as defined by DSM IV. It is predicted that PTSD will be present in this sample.
- Hypothesis Two: It is hypothesised that loss of consciousness during cerebrovascular accident does not prevent the development of PTSD. In this study it is predicted that the participants who have both lost consciousness and developed PTSD would not be equal to zero.
- Hypothesis Three: It is hypothesised that following CVA memory of the event is not required to develop PTSD. In this study it is predicted that the number of participants who have no memory of the event and who also developed PTSD would not be equal to zero.
- Hypothesis Four: It is hypothesised that PTSD will be diagnosed more in the group of individuals who had right-sided lesions than in the group who had non right-sided lesions.
- Hypothesis Five: It is hypothesised that following CVA participants will experience less re-experiencing symptoms than avoidance or arousal symptoms.

CHAPTER 2

METHOD

2:1 Ethical approval

Ethical approval was sought and obtained from the Lothian Region Ethics Committee. Approval was applied for in December 2000 and given in January 2001. The only stipulation was that neither medical nor psychological case notes were to be reviewed prior to the individuals concerned having given their consent to participate in the project. This necessitated a change to the proposed recruitment process.

The planned recruitment procedure would have involved screening all patients discharged from the Brain Injury Service at the Astley Ainslie Hospital over the preceding year prior to contacting them regarding participation. The screening would have involved the researcher reviewing the medical records of potential participants who met the inclusion criteria. Those patients who had severe cognitive deficits or dysphasia would have been excluded from the study prior to them being contacted. However, as the Ethics committee did not give permission for medical notes to be reviewed prior to the patient having given consent, the recruitment procedure was altered. It would not have been feasible in the time-scale of the project to recruit participants, gain consent and then exclude anyone with severe language problems or cognitive impairments. It was decided instead to rely on consultants and other psychologists to identify those participants who met the inclusion criteria. Once this initial screen had been completed the researcher would approach the remaining individuals to invite them to participate.

2:2 Participants

2:2:1 Recruitment

A total of 32 patients agreed to participate in this study. They were recruited from the rehabilitation services provided at Astley Ainslie Hospital, Edinburgh. This service is a

national resource and as such provides assessment and rehabilitation to patients from all over the country. Within the rehabilitation service, potential participants were drawn from two sources. Firstly, the Scottish Brain Injury Rehabilitation Service which provided patients who had a diagnosis mainly of sub-arachnoid haemorrhage and secondly, from the Stroke Rehabilitation Service which provided patients who had experienced ischaemic strokes. It was decided to use two sources; firstly to investigate the role type of stroke might have on development of PTSD; secondly to increase the total sample size; and thirdly to ensure the sample was representative of the general stroke population. All participants had received either assessment and/or rehabilitation from the rehabilitation services at the Astley Ainslie Hospital.

Initially, three consultants within the hospital were approached and asked to identify potential participants who met the inclusion criteria. The inclusion criteria for the study were as follows:

Inclusion criteria:	**Exclusion criteria:**
• cerebrovascular accident of either ischaemic or haemorrhagic origin • out-patient • aged 18 - 65 years • at least three months since injury	• severe cognitive deficits • dysphasia

The potential participants were drawn from those who had been discharged from the service in the year 2000. This yielded a total sample of 72 potential participants. A further 5 patients were identified via out-patient clinics at the psychology department. Consultants or psychologists reported significant cognitive deficits or dysphasia in 18 of the 77 potential participants: this reduced the potential participants to 59. A further 5 people were excluded as they no longer attended the hospital for rehabilitation and they lived too far from the study base for the researcher to visit them at home. In accordance with the inclusion criteria

a further 6 patients were excluded due to age or type of injury which resulted in the total number of participants approached being 48. All of these potential participants were sent an information sheet (Appendix 2) which provided details about the project as well as explaining what the interview session would entail. Forty-two were followed up by telephone to provide each participant with the opportunity to ask any questions they may have had as well as to establish whether or not they wanted to participate in the study. The 6 who could not be contacted by telephone were sent a second letter. Only one participant responded to this. Of those followed up 10 declined to participate. No information is available about their reasons for this. Due to the restrictions imposed by the Ethics board case notes pertaining to these patients could not be reviewed, therefore no conclusions can be drawn regarding any similarity or difference to the obtained sample. One person who had agreed to participate was unable to do so due to re-admission to hospital. This meant the total sample was 32, which equated to a 67 per cent take up rate.

2:2:2 Demographics

The mean age of the sample was 51 years (sd =9 years) and ranged between 35 and 65 years. The sample included 14 males and 18 females. The mean time since injury was 14 months (sd=18 months) and ranged between 3 months and 9 years. With the outlier removed the mean time since stroke was 11 months (sd=5). The sample included; 8 patients who had infarct strokes, 4 who had intracerebral haemorrhages (ICH) and 20 who had sub-arachnoid haemorrhages (SAH). The ICH and SAH groups were combined to make a single group of haemorrhagic stroke participants. Glasgow Coma Scale (GCS) scores were available for 26 of the participants. The scores ranged from 3 to 15 with the mean being 12 (sd=4). Based on GCS score the majority of the sample would be classified as having had a brain injury of mild-moderate severity. Duration of post traumatic amnesia (PTA) was calculated for all participants. This was based on patient's self-report and confirmed,

whenever possible, using information documented in the case notes. The mean PTA was 15 days (sd=17 days) and ranged from 0 to 84 days. Using assessment of PTA as an index of injury severity the majority of the sample would be classified as having had severe brain injuries. The discrepancy with classification based on GCS only can partly be explained by the period of fluctuating consciousness which is included in PTA assessment but not generally recorded in case notes as part of a GCS assessment.

2:3 Design

The aims and hypotheses of this study required that it be conducted in two stages, with each stage having a different design. The first stage was to determine whether Post-traumatic Stress Disorder (PTSD) can occur after stroke and the prevalence of PTSD in the entire sample of 32 participants was assessed using descriptive statistics. As this was a prevalence study it did not require a control group and the rates were compared with other published PTSD prevalence rates. The second stage of the study was a between-subjects design, and as it essentially compared two experimental groups, a control group was not required for this stage either. Hypotheses two, three and four required that participants be allocated to different groups depending on one of three grouping variables. This meant that participants could belong to a different group for each condition. The variables used to allocate participants to the groups were loss of consciousness, recall of the event and site of stroke. As the group allocation was not fixed it was not possible to match the groups.

2:4 Power analysis

In order to assess prevalence of PTSD post-stroke the entire sample was used. Power analysis was not required at this point as only descriptive statistics were to be used. For the secondary hypotheses, inferential statistics were used and the power analysis was as follows:- from previous trauma literature a medium to large effect size was predicted

(Cohen's d = 0.6-0.8). To achieve statistical power of 0.85 each group would need 30 participants, therefore the total sample required was 60. For the data which was analysed using Chi-squared the power and effect sizes were as follows:- again, predicting a medium to large effect size (Cohen's w = 0.4-0.6) with an anticipated sample size of 60, the results would have power of 0.87-0.97.

However, due to circumstances outwith the researcher's control the obtained sample size was smaller than was required to ensure the study had sufficient power. During the study, the designated ward for patients who have had infarct strokes experienced staffing difficulties. Unfortunately, this resulted in potential participants not being identified for the study. The number of participants obtained was less than expected and unfortunately, less than had been available. Although the total sample size obtained falls short of that predicted and required to ensure power, it is not untypical in brain injury research which has generally used small samples or single case design (see McMillan 1996; Ohry et al 1996). Studies which use a haemorraghic stroke sample are likely to be smaller as this type of injury only comprises 20 per cent of the total stroke population (Caplan & Stein 1986). Additionally, the survival rate for patients who have experienced a haemorrhagic stroke is small, with a 30-day fatality rate of 50 per cent (Caplan & Stein 1986). It could therefore be concluded, that despite being below that predicted, the sample size is reasonable.

Retrospective power calculations were produced for the three main hypotheses. The harmonic mean was used due to the unequal sample sizes.

Power Calculations for the Three Main Hypotheses

Measures	Harmonic n	Effect size	Power
Hypothesis two (I)	14	0.5 (medium)	0.35
Hypothesis two (A)	14	0.8 (large)	0.66
Hypothesis two (T)	14	0.9 (large)	0.75
Hypothesis three (I)	12	0.5 (medium)	0.3
Hypothesis three (A)	12	0.6 (medium)	0.4
Hypothesis three (T)	12	0.6 (medium)	0.4
Hypothesis four (I)	15	0.07 (small)	0.01
Hypothesis four (A)	15	0.3 (small)	0.19
Hypothesis four (T)	15	0.4 (small)	0.27

I = Intrusions
A = Avoidance
T= Total Impact of Events scale score.

Clark-Carter (1997) suggests using those effect sizes which were initially described by Cohen. A small effect is 0.20, a medium effect is 0.5 and 0.8 is a large effect. The power analysis indicates that this study was under powered. To have achieved generally accepted power of 0.80 the group sizes would have required to have been as follows:

I. For hypothesis two : Loss of consciousness
   - A. to test intrusion a sample of 140 would be required
   - B. to test avoidance a sample of 35 would be required
   - C. to test the total IES score a sample of 25 would be required.

II. For hypothesis three: Memory of the event
   - A. to test intrusion a sample of 140 would be required
   - B. to test avoidance a sample of 80 would be required
   - C. to test the total IES score a sample of 80 would be required

III. For hypothesis four: Right-sided lesions
   - A. to test intrusion a sample of >1000 would be required
   - B. to test avoidance a sample >1000 would be required
   - C. to test the total IES score a sample of 400 would be required

This suggests that as the effect sizes found for hypotheses three and four were very small, the sample sizes required to detect a significant difference would have been outwith the scope of this study. However, the effect size for hypothesis two was large and a sample of 35 per group would have been large enough to increase the power in this study.

2:5 External validity

The response rate in this study was high at 67 per cent. It is therefore unlikely that the sample could be said to be self-selected. The study only includes those patients who had mild-moderate cognitive deficits and, those who were not dysphasic. This means patients with left hemisphere strokes and stroke which resulted in severe cognitive impairments are not represented by this sample. The sample includes participants aged 65 years or less and is therefore not representative of the general population of infarct stroke patients which tends to be older. However, it is representative of the SAH population which tends to be younger with the majority of haemorrhages being associated with 45-60 year olds (Kaplan & Cerullo 1986). It was considered necessary to concentrate on younger stroke patients as it would allow participants with different types of stroke to be compared.

2:6 Materials

Galveston Orientation and Amnesia Test (GOAT; Levin, O'Donnell & Grossman 1979)

This is a brief measure which is administered as a semi-structured interview (Appendix 3). It is based on the individual's self-report and provides a global score which represents the patient's current level of disorientation. The items which comprise the GOAT include; memory for the event, which allows evaluation of Post-traumatic Amnesia (PTA); and memory prior to the event, which allows assessment of retrograde amnesia. The scale was included in this study for three reasons; firstly to ensure all participants were orientated to time and place; secondly to estimate their PTA and thirdly to gather information about the

individual's recall of the event. Although the PTA duration was based on retrospective self-report this is considered as reliable as PTA which has been measured prospectively (McMillan, Jongen, & Greenwood 1996).

Post-traumatic Amnesia (PTA; Russell & Smith 1961)

Post-traumatic amnesia is considered to be the 'gold standard' in assessing severity and predicting outcome after brain injury (McMillan et al 1996). Post-traumatic amnesia is defined as the length of time between the injury and the point when continuous memory for day to day events is restored. The duration of PTA includes both the period when the patient is in a coma and the period where they experience 'islands of memory'. The original classifications were provided by Russell & Smith (1961) cited in Levin et al (1979). Although these were revised by Jennett & Teasdale (1981) into the current classifications. The definitions are as follows:

- Less than five minutes: 'very mild injury'
- Five to sixty minutes: 'mild injury'
- One to twenty-four hours ' moderate injury'
- One to seven days: 'severe injury'
- One to four weeks: 'very severe injury'
- More than four weeks:' extremely severe injury'

Glasgow Coma Scale (GCS; Teasdale & Jennett 1974)

This is a widely used quantitative scale which assesses the severity of the injury by measuring depth of unconsciousness. It comprises three sections; eye-opening, motor response and verbal response. Patients are given a score according to their best response on each section. The scoring is as follows; eye-opening 1-4; motor response 1-6 and verbal response 1-5. The assessment provides a score between 3 and 15. In terms of severity of injury, 3-5 is very severe, 6-8 is severe, 9-12 is moderate and 13-15 is mild. In this study the

GCS score provides objective confirmation of whether a patient had lost consciousness. This information was obtained from either medical or psychological case notes. Participants were also asked whether they had lost consciousness and whether they had recall of the event. The self-report method has been found to be generally accurate (Mayou, Black & Bryant 2000).

Clock Drawing Test (Borod, Goodglass & Kaplan 1980)

The Clock Drawing Test was used so that participants with severe cognitive impairments could be excluded from the study. It is a measure widely used in assessment of dementia but was included in this study because it is quick to administer and complete. In order for the task to be completed successfully a number of cognitive abilities are used. They include, auditory comprehension, abstract thinking and concentration. All of these abilities were considered necessary to participate in this study. This measure correlates highly with the Mini-Mental State Examination ($r = 0.61$) which is a measure used in screening for cognitive impairment (Tombaugh & McIntyre 1992).

There are a number of alternative procedures for scoring this test. Shulman (2000) provided a comprehensive review and the procedure adopted by this study was the standardised approach for administration and the five point scoring system recommended by Shulman, Gold, Cohen, & Zucchero (1993). Each participant was presented with a pre-drawn circle which was 10 centimetres in diameter. The following instruction was given - ' *This circle represents a clock-face. Please put the numbers in so it looks like a clock and then set the time to 10 minutes past 11'*. The scoring system used was:

- five for a perfect representation
- four for minor visuo-spatial errors
- three for an inaccurate representation of the time

- two for moderate visuo-spatial disorganisation and inaccurate presentation of the time
- one for severe visuo-spatial disorganisation
- zero for inability to make a reasonable representation.

This scoring system was chosen as it was simple and yet provided assessment of comprehension, executive ability and would highlight impulsivity.

Hospital Anxiety and Depression Scale (HAD; Zigmond & Snaith 1983)

This is a well known self-report measure which comprises 14 questions designed to detect current levels of anxiety and depression in a non-psychiatric population. As it does not rely on somatic symptoms it is less likely to be affected by co-morbid physical illness. In this study, anxiety and depression were to be assessed in a population which was likely to have residual physical symptoms. It was therefore considered to be the most appropriate measure. The HAD has two sub-scales, each of which is scored separately. The measure only assesses current symptoms as the patient is required to rate the symptoms they have experienced over the past seven days. In this study the scores were used both as a measure of psychological distress as well as to group participants into 'cases' and 'non cases'. The recommended cut-off (score >8) was used to define participants as reaching caseness levels of anxiety and depression.

Impact of Events Scale (IES; Horowitz, Wilner & Alvarez 1979)

This is a self-report measure of an individual's response to a traumatic event (Appendix 4). It has 15 questions which separately measure symptoms of intrusion and avoidance. Responses are scored on a four point scale. A response of 'not at all' would score zero, a response of 'rarely' scores one, 'sometimes' three and 'often' is given five. The maximum score is 60 and a higher scores reflects a report of more frequently experienced symptoms. It is designed to assess the patient's current symptoms by assessing frequency over the

preceding week. It has been widely used in both clinical and research practice but is not a diagnostic tool and can be used only to report on PTSD symptomatology. The intrusion sub-scale provides a seven item measure of the extent to which the event intrudes into the person's consciousness. The avoidance sub-scale is eight items and measures the extent to which the person engages in cognitive avoidance. Although this measure is intended to be used descriptively, norms are available from the original study by Horowitz et al (1979). This measure has previously been used with both traumatically brain injured and stroke populations (Schnyder et al 2001 and Sembi et al 1998).

Structured Clinical Interview for DSM-IV Axis 1 Disorders (SCID; First, Spitzer, Gibbon & Williams 1997).

This is a semi-structured clinical interview used to diagnose DSM IV Axis 1 disorders (Appendix 5). The use of a standardised diagnostic interview to ascertain whether any of the sample had PTSD ensured the prevalence rate would be reliable. The interview includes questions which allow the clinician to explore the areas necessary to conclude if an individual meets the diagnostic criteria for PTSD. The diagnostic criteria are given in Appendix 1. Criterion E specifies that symptoms have been present for a minimum of one month. In this study, the duration of symptoms was taken as the time since the stroke, therefore all participants would be classified as meeting criterion E. It is possible to distinguish between acute and chronic PTSD depending on whether the symptom duration is greater or less than three months. The DSM IV structured clinical interview has been used with a brain injured population (Hibbard et al 1998) and the DSM III-R version has been used with a stroke population (Sharpe et al 1990). As the study was not assessing the prevalence of all DSM Axis 1 disorders it was decided to use the clinician's version rather than the research version. Furthermore, to reduce the time required for participants to

complete the study battery only the questions necessary to make a diagnosis of PTSD were asked.

2:7 Procedure

2:7:1 Participation

In the patient information sheet participants were not informed that the purpose of the study was to assess for PTSD. Instead it was decided to describe the study in general terms as an investigation into the emotional consequences of having a stroke. There was a minimum of two weeks between participants being sent the information sheet and the follow-up telephone call being made. Those potential participants who were not contactable by telephone were sent a letter which asked them to contact the researcher if they were interested in participating in the project. The purpose of the follow-up telephone call was to describe the purpose of the study in more detail and to explain what the session would entail. It also gave participants the opportunity to ask questions. If participants consented to take part in the study an appointment time was arranged.

2:7:2 Venue

For those participants who were already attending the hospital as out-patient's the appointments for the study were arranged to coincide with other scheduled hospital visits. Fifteen participants chose to be interviewed at home. This was due to either work commitments or their mobility problems.

2:7:3 Interview

The data collection process took between one and two hours. The interview time for those who were seen at home tended to be longer. All of the participants seen at home took more time to complete the measures as they appeared more relaxed and talked more freely about

their experiences. The majority of the domicillary visits involved, on average, two hours travelling time.

The format of the interview was as follows; all participants were given a few minutes to read and sign the consent form (Appendix 6) before being asked the questions required to complete the GOAT. They were then asked to complete the Clock-Drawing Test , the HAD and lastly the IES. The session was concluded by the researcher administering the SCID. In the case of two participants the questionnaires were read aloud by the researcher due to their visual impairments. To conclude the session the aims of the research were re-iterated and participants were given another opportunity to ask any questions or to add any information they thought was relevant which had not been covered in the session. If requested, arrangements were made to provide additional feedback once the project had been completed. Participants were also reminded that they could contact the researcher at any time if they did have any further questions. Finally, all participants were given a photocopy of the consent form they had signed.

A small number of the participants became distressed in the course of the interviews. When this occurred the interview was suspended and the participant was given an opportunity to discuss their personal circumstances. Only when the researcher was sure that they were no longer distressed were they asked whether they would like to continue. As stated in the application for Ethical Approval, for those participants who were identified as being significantly distressed a follow-up telephone call was made to the individual's GP or to any other involved agencies. The aim of this was to highlight the need for the patient to be reviewed or referred on to appropriate services. In all cases where the researcher considered it necessary to contact the GP the participant was aware and had agreed that this could be done.

2:7:4 Case note review

Once the interviews were completed and consent had been given the patient's case notes were reviewed. Psychology case notes were available from the department in which the research was conducted. All participants had been assessed by the service, however four sets of psychology case notes were missing. If following the review of the psychology notes it was necessary to complete the data set with any further information the medical case notes were also reviewed. Medical case notes were obtained from the Astley Ainslie medical records department. The case note review provided demographic information, as well as information relating to the circumstances surrounding admission to hospital, type and severity of stroke, Glasgow Coma Scale scores and the duration of any loss of consciousness. At this point copies of the consent form were filed in the patients case notes.

All of the participants gave their permission for their GPs to be notified of their decision to take part in the project. Although a number of participants had already discussed the project with their GPs a standard letter which provided a brief summary of the aims of the project was sent to the GPs of all participants in the study.

2:8 Data analysis

The data was analysed using the Statistical Package for the Social Sciences version 10 (SPSS 10) for Windows computer package. The prevalence of PTSD within the entire sample was assessed using descriptive statistics. On completing the descriptive statistics the data was divided into two groups depending on particular variables allowing the hypotheses which were based on each variable to be tested. Further analysis compared total IES scores, avoidance and intrusion scores of each of the two groups. Between-groups t-tests were used as the Impact of Events and HAD questionnaires are on an interval scale of measurement. Chi squared was used to analyse the categorical data pertaining to PTSD diagnosis, anxiety

and depression. Post-hoc analysis was conducted using a combination of parametric and non-parametric analysis. The variables explored included type of stroke experienced, sex and previous history of exposure to trauma. For all statistical procedures $\alpha = 0.05$.

CHAPTER 3

RESULTS

3:1 Demographics

A total of thirty-two individuals were interviewed for this study. One person diagnosed as having PTSD was excluded. This individual had been the victim of an assault two weeks prior to having his stroke. It was necessary to exclude him as the stressor which precipitated his PTSD could not clearly be identified as having been the stroke. This reduced the sample size to thirty-one for analysis. The mean age of the sample of thirty-one participants was fifty-one years (sd=9) and ranged from thirty-five to sixty-five years. The sample comprised eighteen females and thirteen males. Of the group, twenty-four had suffered haemorrhagic strokes and seven had experienced cerebral infarcts. The mean time since stroke was almost fourteen months (sd=18.8) and was found to range from three months to nine years. The participant who had her stroke nine years earlier had been re-referred to rehabilitation services due to anxiety. When her data was excluded the mean duration was ten months (sd = 4.5 months) and ranged from three to nineteen months. The duration since stroke was significantly different for this participant and it was therefore considered prudent to conduct the analysis both with and without her data being included. In general the inclusion of this participant's data did not significantly alter the overall findings. Any instance where the results did differ as a result of her inclusion will be highlighted.

3:2 Distribution of data

The distribution of data was reviewed for each different pair of groups. In all analyses the Hospital Anxiety and Depression scale (HAD) scores were found to be normally distributed and parametric analyses were conducted. However, the Impact of Events (IES) scale data was only found to be normally distributed when comparing the groups on the basis of sex,

despite this parametric statistics have been used throughout. The reasons for this decision were; it was assumed that the population from which the data was drawn would be normally distributed and it was further assumed that parametric statistics would be robust enough to withstand the degree of skewness and kurtosis found.

3:3 Primary hypotheses

Hypothesis One: It is hypothesised that a cerebrovascular accident can result in PTSD as defined by DSM IV. It is predicted that PTSD will be present in this sample.

The results of this study suggest that it is possible to develop PTSD in response to stroke and therefore the hypothesis is supported. In this sample of stroke patients (n=31), six were diagnosed during interview as having Post-traumatic stress disorder. This is a prevalence of 19.4 per cent in this sample. This is higher than rates reported following other medical events for example, 10 per cent following myocardial infarction (Bennett & Brooke 1999), and is higher than the reported prevalence rate of 5-15 per cent in the general population (Yule 1999). However, this finding is consistent with literature reporting prevalence of PTSD in brain injured samples (Bryant et al 2000; Ohry et al 1996).

Hypothesis Two: It is hypothesised that loss of consciousness during cerebrovascular accident does not prevent the development of PTSD. In this study it is predicted that the participants who have both lost consciousness and developed PTSD would not be equal to zero.

The findings of this study support the hypothesis as all six of the participants who developed PTSD had lost consciousness at some point during the event. Chi-squared analysis was carried out but as one cell was found to have less than five entries the Fisher's exact test is quoted instead. There was no significant relationship between loss of consciousness and PTSD, (Fisher's (1); p=0.14, two-tailed test), loss of consciousness and anxiety, (Fisher's

(1); p=0.24, two-tailed test) or loss of consciousness and depression (Fisher's (1) ; p=1, two-tailed test).

Table 1 shows the IES and HAD anxiety and depression scale scores for the 'loss of consciousness group' (LOC) and 'no loss of consciousness' group (No LOC). The participants who had lost consciousness had higher mean scores on all of the variables than those who had not lost consciousness.

Table 1: *IES and HAD scores for the 'loss of consciousness' (LOC) and 'no loss of consciousness' (No LOC) groups.*

	Intrusions m (sd)	Avoidance m (sd)	Total IES m (sd)	Anxiety m (sd)	Depression m (sd)
LOC (n=21)	8.9 (7.8)	10.5 (10.4)*	19.4 (15.8)**	7.2 (4.6)	6.5 (4.2)
No LOC (n=10)	4.2 (3.9)	3.8 (5.8)*	8 (8.6)**	5.4 (3.1)	5.7 (5.2)

m=mean, sd = standard deviation
* significantly different at p<.05 and ** significantly different at p<.01

Between-groups t-tests were used to analyse the differences between the groups. On the measures of intrusions, anxiety and depression the mean scores did not differ significantly between the groups. On the measure of avoidance, Levene's test for equality of variance showed that the variances differed significantly between the groups (F=6.808; p=0.01) and a Welch's t-test for unequal variances is quoted instead. The mean of the avoidance scores for the 'loss of consciousness' group was found to be significantly higher, (t(28)= 2.301; p=0.03, two-tailed test), than the 'no loss of consciousness' group. For analysis of total IES score a t-test for unequal variances is again reported as the variances differed significantly between the groups (Levene's test, F=4.891; p=0.035). The mean total IES score of the 'loss of consciousness' group was found to be significantly higher (t(28)=2.604; p=0.01, two-tailed test) than the 'no loss of consciousness' group.

All of those who developed PTSD had lost consciousness during the event. Furthermore, the group who had lost consciousness scored significantly higher on avoidance and total IES than the group who did not lose consciousness.

Hypothesis Three: It is hypothesised that following CVA memory of the event is not required to develop PTSD. In this study it is predicted that the number of participants who have no memory of the event and who also developed PTSD would not be equal to zero.

As none of the eight participants who reported having no recall of the event had developed PTSD hypothesis three was not supported. Furthermore, those with no recall of the event were found to have lower scores on the Impact of Events scale and to have lower anxiety scores (Table 2). A between-groups t-test was done. The variances of the two groups total IES score were significantly unequal ($F= 4.316$; $p< 0.05$), and Welch's t-test for unequal variances is reported accordingly. The mean for the total IES scores of the 'recall' group was found to be significantly higher ($t(25)= 2.08$; $p=0.048$, two-tailed test) than the 'no recall' group. However, when the data from the outlier was removed this difference was no longer significant. The other scores were not found to differ significantly.

Table 2: *IES and HAD scores for the 'recall' and 'no recall' groups.*

	Intrusions m (sd)	Avoidance m (sd)	Total IES m (sd)	Anxiety m (sd)	Depression m (sd)
Recall (n=23)	8.3 (7.9)	9.8 (10.2)	18.1 (16.0)*	6.8 (4.6)	6.3 (4.2)
No recall (n=8)	4.8 (3)	4.3 (6.30)	9 (8)*	6 (3.3)	6.3 (5.5)

m=mean, sd = standard deviation
* significantly different at p<.05

The group with recall of the event scored higher on intrusions, avoidance and total IES than the group without recall. This difference was significant for the total IES score only when the outlier was included. The mean scores of the two group on the measures of anxiety and depression were similar.

Hypothesis Four: It is hypothesised that PTSD will be diagnosed more in the group of individuals who had right-sided lesions than in the group who had non right-sided lesions.

To test this hypothesis two groups were defined by stroke location. The study participants were grouped into those with a right-sided stroke and those who had stroke of 'other ' location which comprised left, bilateral and those without lateralisation. Table 3 (a) shows the frequency of diagnosis for these two groups. Diagnosis of PTSD did not differ significantly between these two groups (Fisher's (1); p= 0.653 two-tailed test). No significant association was found between these groups on the occurrence of caseness for anxiety ($\chi 2$ (1) = 1.052, p=0.305, n=31) and no significant relationship was found between diagnosis of depression and site of stroke (Fisher's (1); p=0.065, two-tailed test).

Table 3 (a): *Percentage of participants classified with PTSD, anxiety and depression grouped by site of stroke.*

	PTSD	No PTSD		Anxiety	No anxiety		Depression	No depression
Right (n=19)	16	84		32	68		53	47
Other (n= 12)	25	75		50	50		17	83

The group defined as having non right-sided strokes was found to have higher ratings than the non right-sided group on the intrusions, avoidance and total IES scales as well as on the HAD anxiety scale (Table 3 (b)). Using between-groups t-test none of these group differences was found to be significant. The right-sided group scored higher (m= 7.8, sd=4.5) on their ratings of depression on the HAD in comparison to the 'other' group (m= 3.8, sd=3.4). Further analysis found this difference to be significant (t(29)= 2.709; p=0.01, two-tailed test) than the non right-sided stroke group.

Table 3 (b): *IES and HAD scores for the 'right sided stroke' and 'other' groups*

	Intrusions m (sd)	Avoidance m (sd)	Total IES m (sd)	Anxiety m (sd)	Depression m (sd)
Right (n=19)	6.6 (7.4)	7.1(8.2)	13.7 (14.2)	6.4 (4.3)	7.8(4.5)*
Other (n=-12)	8.6 (6.8)	10.3(11.6)	18.9 (15.8)	7 (4.3)	3.8(3.4)*

m= mean, sd = standard deviation
* significantly different at $p<.05$

Twenty-one participants were grouped according to whether their stroke involved anterior cerebral arteries, middle cerebral arteries or posterior cerebral arteries. The remaining ten could not be classified and so were excluded from this analysis. None of the differences were found to be significant using Fisher's exact test. A one-way, between-subjects Analysis of Variance was used to compare the differences across and within these three groups on the HAD and IES and no significant differences were found to exist in this sample.

There were no significant differences between the groups on diagnosis of PTSD, anxiety or depression. The group with non right-sided strokes scored higher on intrusions, avoidance, total IES and anxiety, although the differences were not significant. The right-sided stroke group did score significantly higher on depression. No difference was found between those who had anterior, middle or posterior region strokes.

Hypothesis Five: It is hypothesised that following CVA participants will experience less re-experiencing symptoms than avoidance or arousal symptoms.

The study considered whether PTSD symptoms reported by a stroke population are; a) consistent with those defined by DSM-IV and b) indicative of a specific profile. Table 4 (a) shows the numbers who met the individual DSM-IV criteria for PTSD. Although the percentage diagnosed with PTSD in this sample was 19 per cent, the frequency with which

some symptoms were endorsed was much higher for example, criterion B was met by 52 per cent. The criteria B, C and D reflect the frequency with which symptoms were reported. Forty per cent of the entire sample met all three criteria levels, only twenty-six per cent did not meet any of them.

Table 4 (a): *DSM-IV criteria met by participants.*

DSM-IV Criteria	A	B	C	D	E	F
Met by (n)	8	16	16	19	31	6
% of sample	26	52	52	63	100	19

Table 4 (b) reports the frequency with which the 17 individual PTSD symptoms were endorsed by the sample. This illustrates the profile of symptoms which were reported for the entire sample and confirms the hypothesis that re-experiencing symptoms would be less frequently reported than arousal and avoidance symptoms.

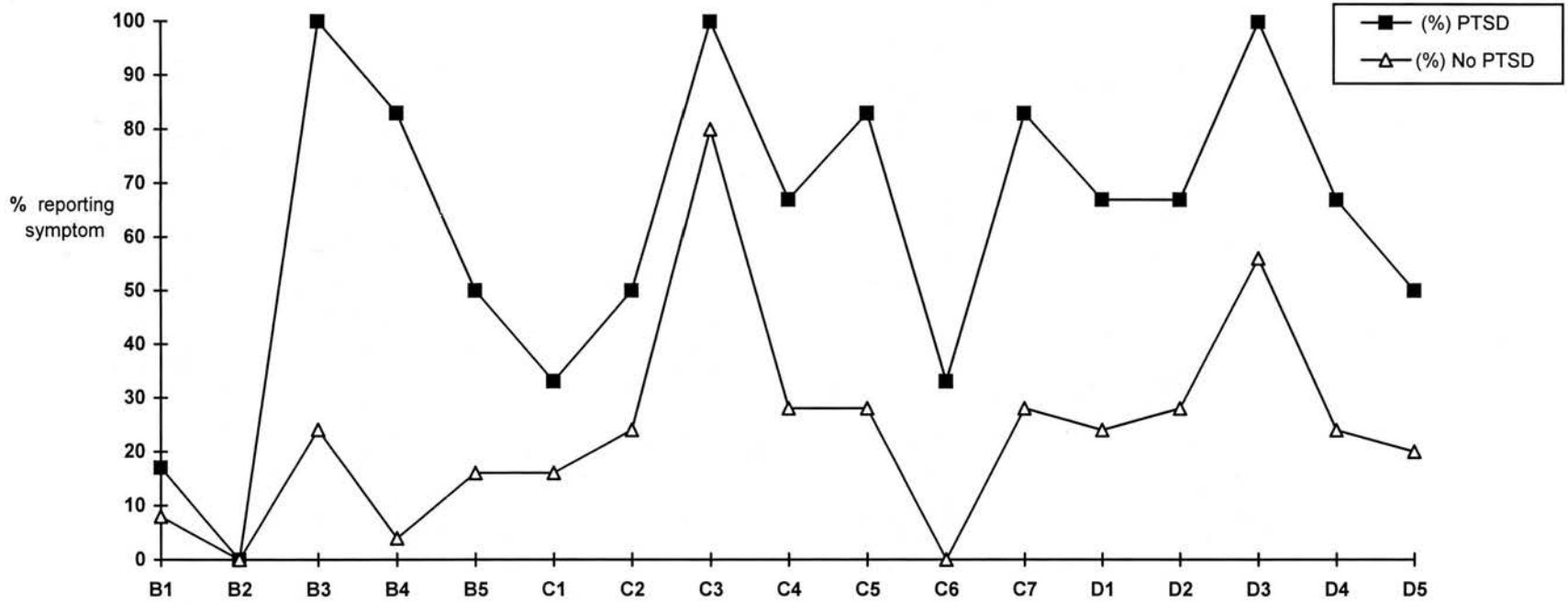
Table 4 (b): *PTSD symptoms endorsed by participants.*

SYMPTOM	No. of sample who endorsed (n)
Criterion A:	
Trauma	31
Fear	8
Criterion B:	
Intrusive thoughts	3
Nightmares	0
Recurring	12
Distress at reminders	6
Arousal at reminders	7
Criterion C:	
Avoid thoughts	6
Avoid activities	9
Cant remember part of event	26*
Diminished interest	11
Detached	12
Affect	2
Future	12
Criterion D:	
Sleep disturbance	10
Anger	11
Concentration	20*
Hypervigilance	10
Heightened Startle	8

* indicates symptoms which could be endorsed due to brain injury rather than PTSD.

Figure one illustrates the similarities in the symptoms which were reported by the group who were diagnosed with PTSD and those who did not meet the DSM IV criteria.

Figure 1: Comparison of the Symptom Profiles for those with and without PTSD diagnoses.



DSM IV Symptoms

B1= Intrusive thoughts
B2= Nightmares
B3= Recurring
B4= Distress at reminders
B5= Arousal at reminders

C1= Avoid thoughts
C2= Avoid activities
C3= Can't remember part of event
C4= Diminished interest
C5= Detached

C6= Affect
C7= Future

D1= Sleep disturbance
D2= Anger
D3= Concentration
D4= Hypervigilant
D5 =Heightened Startle

As shown in Figure 1 there are similarities in terms of the symptoms reported by both groups. The symptoms which were not reported at similar levels by both groups were, (B3) 'feeling as though the event is recurring', (B4) 'distress at reminders', (C5) 'feeling detached' and (C7) 'sense of a foreshortened future'. Post-hoc analysis was conducted to see if the differences between the groups were significant on these symptoms.

Table 4 (c): *Frequency of symptoms reported by PTSD and no PTSD groups.*

	PTSD		No PTSD	
	symptom yes	symptom no	symptom yes	symptom no
B3	6	0	6	19
B4	5	1	1	24
C5	5	1	7	18
C7	5	1	7	18

The PTSD and 'no PTSD' groups were found to differ significantly on the frequency with which all of these symptoms were reported. For 'feeling that the event is recurring', (Fisher's (1); p=0.001, two-tailed test), for 'distress at reminders' (Fisher's (1); p<0.001, two-tailed test), for 'feeling detached' (Fisher's (1); p=0.022, two-tailed test) and 'sense of foreshortened future', (Fisher's (1) ;p=0.022, two-tailed test).

Table 4 (d) shows the mean scores of the two groups on the IES and the HAD. A between-subjects t-test was used to compare the PTSD and 'no PTSD' groups. Variances between the groups were significantly unequal for total IES (F= 9.648; p= 0.004) and intrusions (F= 18.425; p< 0.000). Accordingly, analysis of the means of these two groups used Welch's t-test for unequal variances. The groups were not found to differ significantly on intrusions or total IES score. On the measure of avoidance the PTSD group was found to have scored significantly higher (t(29)= 2.570; p=0.016, two-tailed test) than the 'no PTSD' group.

The PTSD group also scored significantly higher on anxiety, (t(29)= 3.597; p=0.001, two-tailed test) than the 'no PTSD' group. However, although the PTSD group scored higher on depression than the 'no PTSD' group this difference was not found to be significant (t(29)= 1.598; p=0.121, two-tailed test).

Table 4 (d): *IES and HAD scores for the 'PTSD' and 'no PTSD' groups.*

	Intrusions m (sd)	Avoidance m (sd)	Total IES m (sd)	Anxiety m (sd)	Depression m (sd)
PTSD (n=6)	13.5 (11.8)	16.7 (13)*	30.2 (22)	11.3 (5)**	8.8 (4.4)
No PTSD (n=25)	5.9 (4.7)	6.4 (7.7)*	12.3 (10.5)	5.5(3.2)**	5.6 (4.4)

m= mean, sd = standard deviation
* significantly different at p<.05 and ** significantly different at p<.01

Almost 20 per cent of the sample were diagnosed as having PTSD. This increased to 48 per cent when those who meet the criteria for subsyndromal PTSD were also included. Those with PTSD reported four particular symptoms significantly more than those without PTSD. The PTSD group scores higher on all variables with the differences being significant for scores of avoidance and anxiety.

3:4 Pearson Correlations

As PTSD is an anxiety disorder it was expected that there would be a significant correlation between HAD anxiety and the IES sub-scales. Table 5 (a) show the correlation matrix of the IES and HAD scores for those diagnosed with PTSD. Anxiety ratings on the HAD were not found to significantly correlate with the Impact of Events and the correlation was only slightly higher than that between the depression score and the IES. However, the intrusion and avoidance scales were found to be significantly correlated. When the data for the entire sample was used the IES and the anxiety scores were found to be significantly correlated (table 5 (b)).

Table 5 (a): *Correlations of IES and HAD scores for PTSD group.*

	Intrusions	Avoidance	IES Total	Anxiety	Depression
Intrusion	1.00				
Avoidance	0.549	1.00			
Total IES	0.868*	0.892*	1.00		
Anxiety	0.290	0.370	0.377	1.00	
Depression	0.153	0.143	0.168	0.939**	1.00

** Correlation is significant at the 0.01 level (two-tailed).
* Correlation is significant at the 0.05 level (two-tailed).

Table 5 (b): *Correlations of IES and HAD scores for the entire sample.*

	Intrusions	Avoidance	IES Total	Anxiety	Depression
Intrusion	1.000				
Avoidance	0.561**	1.000			
Total IES	0.843**	0.918**	1.000		
Anxiety	0.457**	0.488**	0.536**	1.000	
Depression	0.298	0.262	0.312	0.488**	1.000

** Correlation is significant at the 0.01 level (two-tailed).

Significant correlations were observed in those diagnosed with PTSD on their intrusion and avoidance scores although no correlation was found with these and the anxiety scores. However, for the entire sample the intrusions and avoidance scores correlated, as well as both correlating with anxiety scores.

As the IES is a measure which has not been widely used with participants who have experienced strokes, the scores for the PTSD group were compared with the published norms for the IES scale (Horowitz et al 1979). A one sample t-test was used to compare the scores. The groups were not found to differ significantly on intrusion scores (t(5)= 1.551;

$p>0.1$, two-tailed test), avoidance scores ($t(5)= 0.251$; $p>.01$, two-tailed test) or total IES score ($t(5)= 1.045$; $p>0.1$, two-tailed test). This appears to indicate that the sample who developed PTSD following stroke reported similar levels of intrusions and avoidance as Horowitz's sample. The results require cautious interpretation as the PTSD post-stroke sample was very small and a retrospective power calculation suggest that the small sample size of the stroke group reduced the power of the study. The effect sizes were 0.8, 0.1 and 0.5 respectively. The sample size required for these effect sizes to give power of 0.80 would have been 25 to assess intrusions, >1000 to test avoidance and 60 to test the total score.

3:5 Post Hoc Analysis

Additional analysis was conducted post-hoc to consider the effect of the following variables on reported symptoms of PTSD, anxiety and depression; 1) type of stroke; 2) sex; 3) duration of post-traumatic amnesia and 4) exposure to previous trauma.

1. Type of stroke

The sample was separated into groups defined by type of stroke, one group of haemorrhagic stroke participants (n=24) and one of ischaemic stroke (n=7) participants. Only one of the ischaemic stroke group developed PTSD compared with five of the haemorrhagic group although this difference was not significant (Fisher's (1) p=0.59, two-tailed test). The haemorrhagic group was found to have higher scores on both sub-scales of the IES and on HAD anxiety, but was found to have lower scores on depression, than the ischaemic group (Table 6). However, when the data from the outlier was excluded the avoidance scores were lower for the haemorrhagic group. Analysis using a between-subjects t-test indicated that the groups did not differ significantly on these measures.

Table 6: *IES and HAD scores for the two groups defined by type of stroke.*

	Intrusions m (sd)	Avoidance m (sd)	Total IES m (sd)	Anxiety m (sd)	Depression m (sd)
Ischaemic (n=7)	5.6 (5)	8 (8.4)	13.6 (11.6)	5.6 (2.7)	7.7 (4.2)
Haemorrhagic (n=24)	7.9 (7.5)	8.5 (10.1)	16.4 (15.8)	6.9 (4.6)	5.8 (4.6)

m= mean, sd = standard deviation

The groups did not differ significantly on IES and HAD scores although the haemorrhagic group scored higher on avoidance, intrusions and anxiety. The haemorrhagic stroke patients reported less depression than the ischaemic stroke group.

2. Sex differences

More women were found to have developed PTSD, to have become anxious and to have become depressed than men post-stroke (Table 7 (a)). A Fisher's exact test was used to compare the incidence of psychological distress in the two groups. The results were non-significant for PTSD, (Fisher's (1); p= 0.36, two-tailed test), and depression, (Fisher's (1); p= 0.48, two-tailed test). But were found to be significant for anxiety, (Fisher's (1); p=0.03, two-tailed test).

Table 7 (a): *Percentage of female and male participants classified with PTSD, anxiety and depression.*

	PTSD	No PTSD	Anxiety	No anxiety	Depression	No depression
Female (n = 18)	28	72	56	44	44	56
Male (n=13)	8	92	15	85	31	69

Furthermore, the female group were found to have higher mean scores on the Impact of Events scale, the anxiety scale and the depression scale (Table 7 (b)). Between-groups t-tests were used to analyse the data. On the HAD anxiety scale, the mean score for the female group was found to be significantly higher ($t(29)= 2.295$; $p=0.03$, two-tailed test) than the mean score of the male group. The other scores were not found to differ significantly.

Table 7 (b): *IES and HAD scores for the two groups defined by sex.*

	Intrusions m (sd)	Avoidance m (sd)	IES Total m (sd)	Anxiety m (sd)	Depression m (sd)
Female (n=18)	8.9 (8.3)	10.6 (11.2)	19.5 (17.1)	8 (4.7)*	7.6 (4.4)
Male (n=13)	5.2 (4.4)	5.3 (6.1)	10.5 (9.1)	4.7 (2.6)*	4.5 (4.1)

m= mean, sd = standard deviation
* significantly different at $p<.05$

More of the female group were diagnosed as having PTSD, anxiety and depression than the males. They also scored significantly higher on the anxiety measure.

3. Severity of injury

Relationship between duration of PTA and prevalence of PTSD, anxiety and depression was considered. The sample was separated into those with PTA of more, and less than seven days duration. These two groups were compared on frequency of diagnosis for PTSD, anxiety and depression (Table 8 (a)). For anxiety there was no significant difference between the two groups, (Fisher's (1); p=0.274, two-tailed test) and neither was there a significant difference on frequency of depression, (Fisher's (1); p=0.717, two-tailed test). But the groups were found to be significantly different on frequency of PTSD, (Fisher's (1); p=0.037, one-tailed test) with the group with longer post-traumatic amnesia having significantly higher diagnosis of PTSD. However, although this difference was not significant at the two-tailed level, (Fisher's (1); p=0.059, two-tailed level) it is approaching significance.

Table 8 (a): *Percentage of participants with PTSD, anxiety and depression grouped by duration of post-traumatic amnesia.*

	PTSD	No PTSD	Anxiety	No anxiety	Depression	No depression
PTA < 7 days (n=12)	0	100	25	75	33	67
PTA>7 days (n=19)	32	68	47	53	42	58

The group with longer duration of PTA scored higher on all variables and the IES and HAD scores for the two groups were analysed using a between-groups t-test. The groups were not found to differ significantly on any variable (Table 8 (b)).

Table 8 (b): *IES and HAD scores for the two groups defined by duration of PTA.*

	Intrusions m (sd)	Avoidance m (sd)	Total IES m (sd)	Anxiety m (sd)	Depression m (sd)
PTA < 7 days (n=12)	5.8 (5.5)	4.9 (8.1)	10.7 (11.5)	5.4 (3.3)	6 (3.8)
PTA > 7 days (n=19)	8.4 (7.9)	10.5 (10.0)	19 (16)	7.3 (4.7)	6.4 (5.0)

m= mean, sd = standard deviation

The group with longer PTA was found to have more participants diagnosed with PTSD than the shorter PTA group. They also scored higher on all measures although not significantly so.

4. Previous trauma experience

The sample was separated into two groups depending on whether participants had reported a previous history of a traumatic event. Table 9(a) shows frequency of diagnosis of PTSD, anxiety and depression. The groups were compared using Fisher's exact test and the group of participants who had reported previous trauma history had significantly higher frequency of depression following their stroke, (Fisher's (1); p=0.05, one-tailed test). Although this was not significant at the two-tailed level, (Fisher's (1); p=0.06, two-tailed test). However, when the data for the outlier was removed this result was significant using a two-tailed test. The results were non-significant for PTSD, (Fisher's (1); p= 0.65, two-tailed test) and anxiety, (Fisher's (1); p=0.717, two-tailed test).

Table 9 (a): *Percentage of PTSD, anxiety and depression in participants grouped by previous trauma experience.*

	PTSD	No PTSD	Anxiety	No anxiety	Depression	No depression
Previous trauma (n= 12)	25	75	33	67	17	83
No previous trauma (n= 19)	16	84	42	58	53	47

Table 9 (b) shows the mean scores of these two groups for IES, anxiety and depression. Analysis was conducted using between-subjects t-tests and indicated that the groups did not differ significantly on any of the variables.

Table 9 (b): *IES and HAD scores for the two groups defined by previous trauma experience.*

	Intrusions m (sd)	Avoidance m (sd)	Total IES m (sd)	Anxiety m (sd)	Depression m (sd)
Previous trauma (n=12)	9.3 (9.3)	8.6 (9)	17.9 (17.3)	6.7 (4)	5.1 (3.6)
No previous trauma (n=19)	6.2 (5.1)	8.2 (10.2)	14.4 (13.3)	6.6 (4.5)	7.00 (4.9)

m= mean, sd = standard deviation

Participants in the group who reported previous experience of trauma were more frequently diagnosed with depression than those who did not report previous experience of trauma.

3:6 Summary of Key Results

Symptoms reported after stroke are consistent with those which define PTSD. Furthermore, they were not only reported by those who were diagnosed with PTSD. In this sample of stroke patients the prevalence of PTSD was 19.4 per cent which suggests it is possible for stroke to precipitate PTSD. The numbers who could be classified as subsyndromal are closer to 50 per cent. The PTSD group scored higher on all variables with the scores differing significantly on ratings of avoidance and anxiety. A key finding was that the PTSD group reported DSM IV symptoms which are not confounded by brain significantly more than the 'no PTSD' group. This suggests a way of distinguishing PTSD from non PTSD in brain injured populations.

The results also indicate the requirement for further investigation into the role type of stroke might have on post-stroke symptoms as only one of the participants in this study diagnosed with PTSD had experienced an ischaemic stroke compared to five from the group who had experienced haemorrhagic strokes. However no definite conclusions can be drawn due to the unequal group sizes. On self-report measures of anxiety and symptoms of PTSD, the haemorrhagic group scored higher than the ischaemic stroke group although the differences were not significant. The ischaemic group scored higher depression although again this was not a significant finding. However, the variable of depression was found to be influenced by the outlier's score.

The group with longer post-traumatic amnesia was found to have significantly higher rates of diagnosis of PTSD suggesting that PTSD could be associated with severity of injury. The more severely injured group were also found to score higher on all variables than the less severely injured group.

All of the participants who developed PTSD had lost consciousness at some point during the event which suggests that in some circumstances loss of consciousness does not preclude development of PTSD. Furthermore, those who had lost consciousness reported higher

scores on anxiety, depression and the Impact of Events Scale. This difference was significant for total IES and on the avoidance sub-scale. All of the participants who developed PTSD had recall of the event. Furthermore, recall of the event was associated with higher levels of psychological distress as measured by the IES and the HAD. Those who had recall of the event scored significantly higher on total Impact of Events.

The prevalence with which anxiety, PTSD and depression were diagnosed was not found to be related to site of stroke. However, the right-sided stroke group scored significantly higher on the HAD depression scale than the 'other' group. This finding could be even more significant as the non right group had higher ratings on all of the other variables. No significant differences were found when groups were defined by the cerebral artery disrupted during stroke and compared on frequency of caseness or degree of symptoms reported. Female participants were found to be more anxious and depressed as well as being more likely to have developed post-stroke PTSD. The mean female score on the anxiety sub-scale of the HAD was found to be significantly higher than the mean score of the male group. The group who had reported a previous trauma scored higher on intrusions, avoidance, total IES and anxiety although this was not significant. The previous trauma group also appeared to have more caseness levels of depression post-stroke, although this result was only significant at the one-tail level except when the outlier's data was removed.

For those who were diagnosed with PTSD, the sub-scales of the IES were significantly correlated. The IES scores only had a slightly higher correlation with the HAD anxiety scale than with the HAD depression scale, neither of which were found to be significant. Although, Impact of Events scores correlated significantly with anxiety when the data for the entire sample were included.

CHAPTER 4

DISCUSSION

Study Hypotheses

Hypothesis One: It is hypothesised that a cerebrovascular accident can result in PTSD as defined by DSM IV. It is predicted that PTSD will be present in this sample.

Hypothesis Two: It is hypothesised that loss of consciousness during cerebrovascular accident does not prevent the development of PTSD. In this study it is predicted that the participants who have both lost consciousness and developed PTSD would not be equal to zero.

Hypothesis Three: It is hypothesised that following CVA memory of the event is not required to develop PTSD. In this study it is predicted that the number of participants who have no memory of the event and who also developed PTSD would not be equal to zero.

Hypothesis Four: It is hypothesised that PTSD will be diagnosed more in the group of individuals who had right-sided lesions than in the group who had non right-sided lesions.

Hypothesis Five: It is hypothesised that following CVA participants will experience less re-experiencing symptoms than avoidance or arousal symptoms.

4:1 Prevalence

It could be concluded from the results of this study, that it is possible for some individuals who have experienced a stroke, to develop Post-traumatic stress disorder. In this sample of 31 stroke patients, PTSD was diagnosed, by interview in six cases, at a prevalence rate of almost 20 per cent. This rate is high when compared with prevalence of 23 per cent reported in prisoners of war (Solomon et al, 1994) and 33 per cent found in a group of the injured survivors of a terrorist attack (Shalev, 1992). The prevalence rate found in this study does differ from those reported by other investigations into post-stroke PTSD for example, 32 per

cent reported by Berry (1998) and 10 per cent reported by Sembi et al (1998). Some of the issues which might have influenced the reliability of the prevalence rate will be discussed.

The prevalence rate reported here could have been influenced by the time since the event. Sbordone, (1999) reports that the DSM-IV predicts that 50 per cent of cases will resolve within three months of the event without any intervention. This conclusion has been supported by studies including that of Schnyder et al, (2001) who reported a 3 per cent reduction in the prevalence rate of their sample over a twelve month period and that of Solomon et al, (1994) who reported that their prevalence rate reduced by 10 per cent over a twenty year time period. As the mean time since stroke in the current study was fourteen months, it is possible that in this sample the prevalence rate reflects chronic cases.

However, although it is possible that time since injury could have reduced the reliability of this study's findings, other studies of brain injured participants have concluded that time since injury was not related to reports of psychological distress (Turnbull et al, 2001). Sembi et al, (1998) reported finding no relationship between the time since injury and PTSD in their sample of stroke patients. However, their group was assessed a maximum of 18 months post event, whereas in the current study the time since stroke ranged from three months to nine years. Unfortunately, Berry (1998) did not report time since injury so it is difficult to ascertain whether this could explain the variation in prevalence rates between hers and the current study. During the interviews for the current study, a number of participants made comments which led the researcher to believe that prevalence might have been higher if people had been interviewed closer to the event. Although this could be considered to be a limitation of the study it suggests that psychological distress post-stroke is not exclusively limited to the acute stages of recovery.

The revision of DSM IV criterion A for PTSD makes comparison with other studies difficult and may have contributed to an underestimation of PTSD in this stroke sample. The current

criterion A stipulates that the individual's response must involve 'horror, fear or helplessness'. This could be considered more restrictive than the DSM III-R definition which was:-

> *"an event outside the range of usual human experience...and that would be markedly distressing to almost anyone"* (DSM III-R, 1987 p247).

A number of research studies continue to use DSM III-R criteria (e.g. Berry 1998; Brewin et al 1999). As the study reported by Berry and the present study had similar sample composition and size, her use of DSM III-R criterion could account for her study's higher prevalence rates. This is further supported by the finding in the current sample, that only eight participants met criterion A which suggests that the DSM III-R definition could be more appropriate with stroke patients. One possible explanation for the reduced fear and helplessness reported by stroke patients, is a reduced awareness that the symptoms being experienced were representative of having a stroke. Some stroke patients might have been unaware that they were having a stroke, for example one of the participants in this study attributed his symptoms to flu and did not contact his GP until two days after the event.

The view that PTSD is a continuum also suggests that the 20 per cent prevalence might be an underestimate. Schnyder et al, (2001) found subsyndromal PTSD prevalence of 21 per cent at their initial point of assessment and 13 per cent twelve months later. In the current study, on considering each DSM criterion separately they were found to be endorsed by more than double the participants that were diagnosed as having PTSD. Using Schnyder et al's definition of subsyndromal PTSD would give a prevalence rate of 48 per cent for those diagnosed with full or partial PTSD. Sembi et al, (1998) also found that symptoms consistent with PTSD were reported frequently in those participants who did not meet full diagnostic criteria. It is important to consider those who fall below the diagnostic threshold because some authors argue that subsyndromal levels can develop into clinical levels

through a positive feedback loop which strengthens the conditioned fear response (Pitman 1989).

Diagnostic factors could have influenced the prevalence rate in this study in two ways. Firstly, the author made all of the diagnoses and it was not possible to have the validity of these confirmed during the project. However, the author is familiar with diagnosis of PTSD and self-report measures were used in addition to the semi-structured interview. Secondly, it is easy to overestimate PTSD in a brain injured population by not taking into account the endorsement of symptoms which could be attributed to the brain injury. In this study, the most frequently endorsed symptoms were impaired concentration and lack of recall of the event both of which could be related to brain injury. Although, with a stroke population presentation is not confounded by post-concussional symptoms the consequences of brain injury cannot be dismissed as having no contribution to symptoms. Despite this, symptoms which could not be attributed to brain injury, but which are consistent with PTSD, were also frequently reported.

4:2 Presentation of post-stroke PTSD.

Hypothesis five predicted that re-experiencing symptoms would be less frequently reported than avoidance or arousal symptoms. The hypothesis was supported as the most frequently met criteria was the arousal criterion met by 63 per cent of the entire sample although re-experiencing and avoidance were met equally by 52 per cent. However, comparing the numbers who endorsed the criteria can give a slightly false impression of the frequency with which symptoms were reported as each of the diagnostic criteria (B,C and D) require a different number of items to be endorsed within each, to say that criteria has been met. When the symptoms reported by the entire sample were considered, hypothesis five was more strongly supported. The most frequently endorsed symptoms were the arousal symptoms and the least frequently endorsed were the re-experiencing symptoms.

Comparison of the PTSD and non-PTSD groups found a difference in reported symptoms as the PTSD group endorsed arousal symptoms most frequently while the non-PTSD group reported avoidance symptoms most frequently. Neither group reported having nightmares and very few reported intrusive and distressing recollections or numbness of affect. Aside from the symptoms which could be attributed to brain injury the most frequently endorsed for those diagnosed with PTSD were; 'feeling as though the event was recurring'; 'sense of foreshortened future'; 'feeling detached' and 'distress on exposure to reminders'. A key finding of the study was that these symptoms could be used to distinguish patients who are more likely to develop chronic PTSD as analysis showed that the groups differed significantly in terms of the frequency with which these symptoms were reported. Given that psychological resources are limited in the area of stroke rehabilitation, it could be that if patients report any of these symptoms it is taken as an indication that more assessment and monitoring of their mood might be required.

As there was a virtual absence of nightmares and intrusions it could be questioned whether the symptom profile was PTSD. However, different profiles are consistent with general trauma research. Studies comparing participants who have been exposed to different traumatic experiences have found that certain symptom patterns might be associated with particular types of trauma. For example, combat survivors have been found to report intrusive symptoms more than avoidance (Solomon et al, 1994). This has lead some authors to conclude that it is more useful to consider PTSD as having distinct clinical typologies (Alarcon et al, 1997). It could be that brain injury is associated with a particular clinical typology as the symptoms presented by participants in this study are consistent with the clinical subtype described by Alarcon et al (1997) as the "neurotic" subtype with the predominant symptoms being anxiety, hyperarousal and avoidance.

The present study confirms that the post-stroke PTSD profile is consistent with that found in other brain injured populations where the least endorsed are the re-experiencing symptoms

of criterion B (Bryant et al, 2000). In brain injured populations the reduced incidence of re-experiencing symptoms is often attributed to the absence of memory for the event. However, in this study only eight people had no recall of the event but they still experienced both intrusive and avoidance symptoms. This suggests that despite having no memory the experience was still re-experienced. Bennett & Brooke (1999), reported that increased intrusions were associated with awareness during the event. In this sample, people reported being unaware that they were having a stroke and this could, in conjunction with having less fear, have contributed to less re-experiencing.

The predominant symptoms in this sample of heightened arousal and avoidance appeared to be associated with particular cognitive themes. The themes which emerged were similar to those found in the general PTSD population while also reflecting specific concerns of this population. A number of the participants were preoccupied with the fear that they could have another stroke. This finding was also reflected in Berry's (1998) sample. She reported that patients tended to misinterpret normal physiological changes as indicating another stroke. This is consistent with current understanding of PTSD as individuals who have experienced trauma over-estimate the risk of another traumatic event occurring (Yule 1999). Participants also made comments that they had accepted that the event could recur or that they had come to terms with their own mortality. This may reflect that for some, the experience had been assimilated and they were able to resume their life accepting the presence of a degree of risk.

Being preoccupied with 'missing time' was another theme which emerged and which is also consistent with other PTSD populations. PTSD in the general population can be accompanied by an inability to remember part of the event. This can create distress as the individual gradually fills in the gaps and constructs an accurate narrative of the event they have been through. In all cases of brain injury there will be an absence of memory. This

means lack of recall, contrary to the widely held belief, might not provide protection from PTSD but might exacerbate features of it.

In the general population, PTSD is categorised as one of the anxiety disorders and in this study the participants with PTSD were found to be more anxious than those not diagnosed with PTSD. Despite this, the IES scores of those diagnosed with PTSD were not found to correlate with their scores on the anxiety sub-scale of the HAD, a finding which would be considered atypical. However, when data for the entire sample were included anxiety correlated highly with the IES scale. This suggest the anomalous finding in the PTSD group might be a reflection of the small sample size.

A further finding was that the group with PTSD reported more depression than the non-PTSD group although issues relating to guilt and grief were noted during interviews with participants from both groups. These findings are similar to those reported by Sembi et al, (1998) who found a highly significant difference between their PTSD and no PTSD groups on anxiety and depression scores. Furthermore, there was a strong correlation between the HAD anxiety and depression scales for both the PTSD group and the entire group. This high level of co-morbid anxiety and depression could reflect chronic distress in this group and might be related to disability. There was no correlation found between intrusions, avoidance and depression, a finding which had been reported in the stroke sample assessed by Sembi et al, (1998). However, it is unclear whether those correlations were for only for participants diagnosed with PTSD or the entire sample which means an accurate comparison of these findings is not possible.

4:3 Type of stroke

Five out of the six participants diagnosed with PTSD had haemorrhagic brain injuries. Although the groups sizes were unequal this finding has identified an area which would benefit from further investigation in subsequent studies. In her study, Berry (1998) reported

a PTSD prevalence rate of 32 per cent, in a sample comprised of participants who had experienced haemorrhagic strokes. Sembi et al, (1998) reported only 10 per cent prevalence in a sample comprised mainly of participants who had cerebral infarcts. The difference in the reported prevalence rates in these studies suggests that the type of stroke might influence prevalence of PTSD. A higher prevalence of PTSD following sub-arachnoid haemorrhage could be explained by the sudden symptoms which accompany it, or the invasive medical procedures which often follow it. However the findings of the present study were that the two groups did not differ significantly in terms of reported psychological distress and the haemorrhagic group scored only slightly higher on the IES and the HAD anxiety scale than the infarct group. In terms of the frequency of PTSD diagnosis in the groups, 21 per cent of the haemorrhagic sample developed PTSD compared with 14 per cent of the infarct sample although this difference was not found to be significant. However these findings have to be interpreted cautiously due to the small and unequal sample sizes and further research is required to explore whether type of stroke has any role in development of PTSD.

Levels of both anxiety and depression were found to be high in this sample. Using the recommended cut-off, 39 per cent of the total sample met caseness for anxiety and the same for depression (Zigmond & Snaith, 1983). A small number of the participants were receiving anti-depressant medication or were still in contact with rehabilitation services although the majority were not. These high levels of anxiety and depression are similar to those reported by Berry (1998) who found anxiety in 50 per cent of her sample. However, a 39 per cent prevalence rate of anxiety and depression is higher than the levels reported by both Sembi et al, (1998) and Sharpe et al, (1990) who reported prevalence rates of 10 per cent and 14 per cent respectively.

4:4 Impact of loss of consciousness & memory on psychological distress post-stroke.

One of the important findings of this study was that all of those individuals who developed PTSD had lost consciousness at some point during the event. The conclusion which can be drawn from this is that loss of consciousness per se does not always protect the individual from developing PTSD and therefore hypothesis two was supported. However, it may be that an absence of memory of the event, for some, does reduce the degree of psychological distress thus explaining the lower levels of PTSD found in some other studies (e.g. Bryant & Harvey 2000). The consequence of this for clinicians is that it cannot be assumed that because the event also involved a brain injury that the occurrence of co-morbid PSTD can be excluded. The finding that PTSD was more common in the study participants who had definitely lost consciousness appears surprising although it is consistent with other reports (Mayou et al, 2000).

Loss of consciousness was also associated with higher levels of psychological distress on all of the measures. Although those with no recall of the event had lower intrusions and avoidance scores than those with recall, both types of symptoms were still reported. Hypothesis three could not be supported but the findings do suggests that memory is not required to traumatically re-experience the event. This was also reported by Turnbull et al, (2001) who concluded that those with traumatic memories scored similarly on the IES to those with no memory of the event, and both of these groups scored higher than the group with non-traumatic recall. This could suggest that for some the absence of recall might be more distressing than having recall of a traumatic event which can eventually be assimilated into pre-existing belief structures. In this study, participants comments reflected two different coping strategies. Some participants said that having no memory made the event easier to deal with while others reported finding the gap distressing. Horowitz's 1986 (as cited in Yule 1999) model of trauma was based on the concept that following trauma the individual goes through a process in which the traumatic experience is integrated into their

pre-existing schema the 'completion tendency'. This process would be interrupted if there is lack of memory which means the event is not emotionally processed.

In the current study, the difference in scores between the group who lost consciousness and those who did not was found to be significant for total IES scores and avoidance. This finding could reflect either that intrusions scores were low in both groups or that avoidance was higher in the group who had lost consciousness. Low intrusions scores would be consistent with the view that the brain injury disrupts encoding and memory of the event, resulting in less re-experiencing symptoms being reported (Bryant et al 2000). When data for the entire sample was considered, the few re-experiencing symptoms reported were those of re-experiencing through psychological and physiological distress rather than intrusive thoughts or nightmares. However, they were reported more by those who had lost consciousness and there are a number of possible explanations for this. Firstly, the content of the intrusions might not have been related to the experience of stroke. Secondly, a number of participants were distressed by the time which they had 'lost' due to loss of consciousness. It is possible that having no memory of the event resulted in attempts to fill in the missing period. Due to the preoccupation with the gap in their memory they might have become more likely to 're-experience' the event.

The finding of higher avoidance in the loss of consciousness group is very interesting as, if there is no memory, the function of avoidance may not to be the same as that in non-brain injured samples. It has also been reported by other studies, e.g. Bryant et al, (2000) reported high levels of avoidance in a sample of severely brain injured patients with PTSD. Furthermore, in the present study although less so than intrusions, avoidance was still reported by those without recall. This finding is contrary to the widely held belief expressed

by O'Brien & Nutt (1998) as:-

> *" the lack of memory means that there is less reason to avoid the relevant cues to the event and this probably results in less avoidance"* (O'Brien & Nutt 1998 p102).

There are plausible explanations as to why avoidance might be higher in a group who lost consciousness during the event and these are again related to the absence of recall. It is possible that avoidance occurs due to the heightened fear of stroke recurring. If there is a belief that the stroke could have been prevented then there may be a sense that by avoiding particular events and activities that the likelihood of it happening again is reduced. Furthermore, the loss of consciousness group might not be avoiding reminders of the stroke itself but another event which was traumatic, for example, having been told about the event. A number of those who participated in the study described having been told after the event that they had nearly died or that their families had thought they might die. This was distressing for them as they were unable to remember the event and they had difficulty accepting the fact that they could have died and had no awareness that they had been so ill. Information given after the event has been found by others to be associated with significant distress (e.g. McNeil & Greenwood, 1996). For some participants their behaviour after they had regained consciousness became a source of embarrassment to them and avoidance might have been a means of coping with this. This is likely as the coping strategies available to brain-injured patients may be less sophisticated. Instances were described by participants of them having been told by family members that they had behaved in very uncharacteristic and disinhibited ways, which made them feel uncomfortable and embarrassed. Although this behaviour is not uncommon post-brain injury (Lezak 1995), and despite being given reassurance, some of the participants reported finding knowledge of how they behaved to be very distressing. The implications of these findings are that giving greater consideration to how and when, information is given to individuals who have been unconscious might result in reduced distress for those concerned.

As the participants who had lost consciousness during the event were combined with those who lost consciousness subsequent to it to make the 'loss of consciousness' group, it could be said that those who had higher symptoms were the participants who had not lost consciousness until after the event. However, this is unlikely to explain these results as memory encoding and storage would be disrupted regardless of the point at which loss of consciousness occurred.

Furthermore, levels of avoidance might have been underestimated in this study as it is difficult to measure avoidance accurately in a sample who are receiving regular rehabilitation sessions. In receiving rehabilitation patients, are being frequently reminded of the event and would be unable to avoid reminders. O'Carroll et al, (1999) proposed that low prevalence of PTSD following variceal haemorrhage was the result of an inability to avoid reminders of the event. For the majority of participants in their study the traumatic event had occurred at home and as a result participants were de-sensitised to reminders. It is possible that the participants in this study demonstrated less avoidance than they might have if they had not been involved with rehabilitation services.

4:5 Psychological distress and location of stroke.

In investigating the relationship between location of stroke and PTSD this study found that the group with right-sided lesions did not differ significantly from the 'other' group on diagnosis of PTSD or caseness of anxiety. Although this means that hypothesis four is not supported this conclusion could be considered unreliable due to the study only including patients who were not dysphasic. This meant that participants with right hemisphere strokes were over-represented while those in the 'other' category represented strokes which has disrupted areas not associated with language. The right hemisphere lesion group were found to have lower Impact of Events scores although this difference was not significant. The association between right hemisphere and PTSD remains unclear with some authors

reporting that right-sided lesions are associated with an increase in intrusive re-experiencing (e.g. Berthier et al 2001) while others reporting that right hemisphere lesions 'cured' intrusive re-experiencing (e.g. Freeman & Kimbrell 2000).

The sample was also grouped into those who had strokes related to anterior cerebral arteries, middle cerebral arteries and posterior arteries. Of the six with anterior artery disruption, only one instance of caseness emerged and this was of anxiety. Of the seven with middle artery disruption one had PTSD, three had anxiety and four were depressed. Of those with posterior disruption two had PTSD, five were anxious and three had depression. The trend appeared to suggest that when anterior sites are affected it results in least amount of psychological distress. This could be considered consistent with Freeman & Kimbrell's (2000) finding that that right frontal damage reduced intrusive symptoms of PTSD. However, this could also reflect a trend in reporting of symptoms. This study relied solely on self-report data to assess presence of psychological distress and had received no collateral information. It is not uncommon for individuals with anterior lesions to have limited insight into their difficulties (Lezak, 1995). It may be that the group with anterior lesions were found to have less psychological distress because they do not recognise or report their symptoms.

Conclusions drawn from data which suggests relationships between lesion location and psychological symptoms should be tentative particularly as information from scans can be unreliable and because lesions can have wide ranging effects. In previous reports, post-stroke depression has been associated with left anterior strokes (Starkstein & Robinson, 1989) although later community based studies have found no relationship to exist. (Sharpe et al, 1990). A recent review paper examining the existing research on lesion location and depression concluded that the prevalence of depression was slightly higher in right-sided stroke patients (Carson et al, 2000). This was also the finding of the current study as the participants who had right-sided strokes were more often diagnosed with caseness levels of

depression and they scored significantly higher on the HAD depression scale. This finding suggests that current conclusions regarding the association between lesions and mood disorders are tenuous. Furthermore, Carson et al (2000) highlight a methodological flaw which makes data relating to location of stroke lesion and mood disorder unreliable. The majority of studies use samples which exclude dysphasic patients. In the present study one of the exclusion criteria was that participants should not be dysphasic. This criteria was used because the measures used for data collection would have required adaptation to allow assessment of patients with dysphasia. As dysphasia is more usually associated with left-sided lesions this suggests a possible confounding variable as the majority of people included would have had lesions associated with specific areas which are not directly involved in language function. There may be benefits in highlighting the influence of lesion location in PTSD as there could be implications for interventions which rely heavily on left hemisphere language centers.

4:6 Relationship between severity of injury and psychological distress

The more severely injured of this sample experienced more psychological distress. In particular, those with longer PTA were found to have significantly higher diagnosis of PTSD as well as having more instances of depression and anxiety. There was also a trend towards the more severely injured having higher scores on intrusions, avoidance, anxiety and depression. This is consistent with the proposed existence of alternative mechanisms by which traumatically brain injured individuals are believed to acquire 'memory' of the event. In this group, despite long PTA some of the traumatic event might have been encoded either implicitly or prior to losing consciousness (King 1997a). The participant in the current study who had the longest PTA recounted a clear memory of having her haemorrhage and of traveling to hospital before she lost consciousness. The conclusion from these results is that longer periods of disrupted consciousness do not necessarily 'protect' the individual from

subsequent psychological distress. This is especially the case post-stroke as the traumatic event is not restricted to the experience of having the stroke. The individual regains consciousness to find that they have nearly died, that they have acquired, often a significant and life-changing disability, and that their 'life plan' has been turned upside down. For some, the process of regaining consciousness and becoming aware of the implications of the event will have been an additional traumatic experience.

4:7 Influence of sex on symptoms

The female participants were found to have more PTSD, anxiety and depression than the male participants. Their scores on the IES and HAD were consistently higher than the male participants. There has been little research examining sex differences in post brain injury mood disorders, perhaps because brain injured samples are predominantly male. However, the study by Ohry et al (1996) also reported a similar sex difference in post injury PTSD including significant differences in the symptoms reported by men and women. Males reported significantly more 'distress at reminders' and 'avoidance of thoughts and feelings', whereas women reported more 'restricted affect'. In the present study when the symptoms profiles were compared it was found that the female participants had reported more 'feeling as though the event was recurring' and 'sleep disturbance' (see Appendix 7). The frequency with which the other symptoms were reported was not found to differ.

Rather than reflecting a difference between the sexes in adjustment to trauma, the findings could represent a reluctance by men to acknowledge psychological distress. During the interviews, male participants seemed more reluctant to discuss the emotional impact of their stroke and were more likely to be dismissive of the emotional sequelae. They made more comments which indicated a preoccupation with the period of time they could not remember and of their desire to obtain an explanation as to why they had experienced a stroke. Although it is important to be cautious in interpreting the findings from what was a

relatively small sample, the results were consistent with a number of other studies (Schnyder et al, 2001; Brewin et al, 1999; Ohry et al, 1996).

In the general population women are more likely to develop PTSD than men (Andrews et al, 1994). This suggests that although women are often not represented in traumatically brain injured samples, their reactions to the experience might be more severe. In stroke, men and women are equally represented and this is one reason why conclusions from brain injury research might not be entirely applicable to a stroke population.

4:8 Does previous trauma increase likelihood of post-stroke PTSD?

In this study, previous trauma was not found to be associated with diagnosis of PTSD post-stroke. It was however, found to be associated with post-stroke depression. In the general population exposure to trauma is thought to increase the risk of developing PTSD when faced with subsequent stressors (Yule 1999). The finding of this study did not support this in a stroke population but there are a number of issues to consider. The time which had elapsed between the previous stressor and the stroke was unknown and could have influenced the impact of the index traumatic event. In addition, time constraints during the interview did not allow a detailed trauma history to be taken which would have contributed more information about reaction to and frequency of, previous trauma.

Although this finding is not consistent with the general trauma literature it is consistent with PTSD post-stroke. Sembi et al, (1998) found no significant difference in reports of previous adverse life events, when comparing those who did and did not develop PTSD. They did find that pre-morbid psychopathology was a better predictor of psychopathology post-stroke than the individual's cognitive appraisal of the trauma. The reliability of the current study's conclusions could have been enhanced by assessing premorbid levels of psychopathology.

The findings in relation to previous trauma and depression are confounded by the degree of physical and cognitive impairment resulting from the stroke. A link between functional

disability post-stroke and depression has been observed. This relationship is not reported as being causal but one in which disability impairs functioning and recovery by maintaining depression (Eastwood et al 1989; Starkstein & Robinson 1989). Exploring the cognitions which were associated with depression in this sample could have contributed to our understanding of the impact of both previous trauma and disability. Drawing from general trauma literature, one hypothesis is that those who had experienced previous trauma could have developed a sense of vulnerability and powerlessness. Both of which could be reinforced by disability and culminating in chronic depression.

4:9 Study Limitations

4:9:1 Sample

The study sample was representative of the type of strokes that can occur despite being small in size. Although it was predominantly comprised of haemorrhagic stroke participants this was not because patients with infarcts were unavailable or unwilling but was instead due to difficulties in accessing this group. The main difficulty encountered was in recruitment of patients who had infarcts. During the allocated time period for the study the ward which was the source of infarct participants had staffing problems which resulted in potential participants not being screened and referred to the study. Once this became apparent the recruitment procedure was altered to enable the researcher to approach some infarct patients and an additional source was also considered via another hospital. It was not possible to access patients from the other site as they were already being recruited for a similar research project. Although the power of the study was reduced due to the small sample size it has highlighted that post-stroke PTSD requires further investigation. Furthermore, both brain injury and PTSD research often rely on small sample sizes and single-case designs (see Bryant & Harvey, 1998; McNeil, 1996; Berry, 1998; Yule 1999).

Including both haemorrhagic and infarct patients in the study meant that age had to be controlled for. This appears to limit the extent to which findings can be generalised to the general stroke population which tends to be older than participants in this study. However, before being revised the DSM criterion A was based on the event having been 'outwith usual human experience' and some still use this definition as a benchmark. Although stroke is not a normal outcome of ageing it is associated with increasing age, if the study had included all ages it could have been criticised as the event might not be considered to be 'outwith usual human experience' for that age cohort.

4:9:2 Design

The study was based entirely on self-report and interview data. This meant information from those who had left-sided strokes was limited and any participants with limited insight might have underestimated their mood disturbances. The reliance on interview data for diagnosis meant only non-dysphasic patients representing specific left-sided strokes are included and this has implications when interpreting data relating to site of lesion. Future research would benefit from including dysphasic patients and this would be possible by either adapting existing measures so that they do not rely as heavily on language, or by interviewing relatives or other informants. The opportunity to gain collateral information from relatives would also be beneficial in cases where participants might have reduced insight. In Bryant & Harvey's (1995) study, the finding that a head-injured group reported finding their incidents less traumatic, and that they believed they were less seriously injured than the non-head injured group, is probably a reflection of reduced insight. In the current study, the way in which two study participants described their stroke and their current circumstances suggested to the researcher that they had reduced insight. This included a participant who responded "no" indiscriminately and another who was laughing and

giggling inappropriately. Interviewing a significant other would be helpful in distinguishing reduced insight from an avoidant coping strategy.

4:9:3 Measures

The author is not aware of any studies which have used the Structured Clinical Interview for DSM-IV with a stroke population, although it has been used with a head injured sample to identify Axis 1 disorders (Hibbard et al 1998). The version based on DSM III-R has been used with a stroke population by Sharpe et al, (1990) to diagnose anxiety and depression. To comprehensively investigate psychological responses to stroke it will be necessary to include participants with dysphasia. This could be achieved through use of neuropsychological assessment to distinguish expressive and receptive dysphasia and adapt information gathering modes accordingly or, to do as Sharpe et al (1990) did, and rely on informants. Either approach will improve generalisability of future research findings.

Both Sembi et al (1998) and Berry (1998) used the Impact of Events scale, although as yet there are no norms available for brain injured populations. The intrusion and avoidance sub-scales of the IES were not found to correlate significantly in the participants of this study who had PTSD. In general trauma research the scores would be expected to correlate, nevertheless, the lack of correlation is consistent with the avoidance symptoms being more frequently reported by this group than re-experiencing symptoms. When the PTSD and no PTSD groups were compared there was no significant difference between them on depression, total IES or intrusions score. This could reflect an insensitivity of the measure but it could also be explained by high levels of pathology in those who were not diagnosed with PTSD. The latter explanation is consistent with the increased numbers who would be diagnosed with subsyndromal PTSD.

Adaptation of measures could provide further help in distinguishing organic and psychological symptoms. As McMillan, (2001) points out, some DSM symptoms will be endorsed due to sequelae of the brain injury for example, in this study all of those with PTSD and more than half of those not diagnosed reported concentration difficulties. Rather than comparing brain injured with non brain injured on the symptoms reported, it would be more useful to identify which symptoms distinguish brain injured with and without PTSD. In this study, significant group differences were found in a number of symptoms and establishing whether this is specific to stroke or is generalisable to traumatic brain injury could provide indicators of PTSD which are independent of organic brain injury sequelae. Furthermore, when assessing post-stroke PTSD it remains important to distinguish between endorsements of particular symptoms e.g. diminished participation and interest, which could be attributable to physical disability rather than PTSD.

4:9:4 Confounding variables

Although the issue of disability could be said to have been a confounding variable in this study it could also be considered an important part of the traumatic experience. It may not be possible, or appropriate to limit the definition of trauma to the stroke itself while not considering the role of subsequent events. In general trauma research events post-trauma for example, support received or security about the future, have been shown to be very important in preventing chronic PTSD (Yule 1999; Paton & Smith 1998). Two of the participants in this study described feeling more vulnerable since their stroke as their reaction times were slower. Whether the sense of vulnerability is due to disability or fear of the event recurring, the psychological impact is likely to be the same. Sembi et al (1998) concluded in their study, that PTSD was independent of physical disability and that there was no relationship between measures of disability and avoidance. It remains though that in the present study an assessment of the impact of disability was not made and therefore the

levels of anxiety and depression recorded could not be said to be independent of it. When interviewing, the author attempted to distinguish symptoms and limitations which could be attributed to disability from those which were attributable to trauma.

Post-stroke treatment could be considered a confounding variable when considering whether data is representative of the entire stroke population. The experiences of haemorrhagic and infarct strokes patients are different and exploring the impact of this was an important part of the present study. Infarcts are most commonly treated medically, for example anti-coagulants or vasodilators, which will increase the blood flow through the obstructed vessel. Although surgical intervention to clear an obstructed artery is an option, it is usually an elective procedure. In contrast, the intervention for haemorrhage is surgical and is therefore more invasive. Following a haemorrhage, the individual is usually admitted to an intensive care unit to stabilise their condition prior to surgical intervention aimed at reducing the chance of another bleed. The surgery normally involves having a metal clip or coil inserted in the artery to prevent it ballooning out. It could therefore be argued that the patients who have haemorrhages are more likely to develop traumatic symptoms, (despite being more likely to lose consciousness) because of not only the symptoms associated with the bleed, but also the subsequent treatment for it. This study did find higher prevalence of PTSD in the haemorrhagic sample and as such highlights an area for further research.

The estimated prevalence of PTSD in this sample might be lower than would be found if participants had been interviewed closer to the event. DSM IV diagnosis requires that symptoms must be present for at least one month post event to be classified as PTSD. However, the mean time since injury was more than one year which means some cases of PTSD will have resolved as it is usual to expect some remission of symptoms over time (Solomon et al, 1992). Comments made by some participants would be consistent with the conclusion that closer to the event prevalence might have been higher. One participant

stated that he had experienced feelings of dissociation although this was no longer occurred and another participant said she had experienced nightmares although they had stopped at the time of the interview. Despite this however, Yule (1999) reported that the numbers which will remit without intervention are relatively small. This suggests that although the prevalence rate reported here might only reflect those participants with chronic PTSD, the acute levels of PTSD are not likely to be much higher.

4:10 Recommendations For Future Research

There has been little research in the area of PTSD post-stroke and this study has highlighted a number of areas that would benefit from further investigation. As discussed previously it would be informative to include participants with dysphasia as this would provide an accurate prevalence rate as well as contributing information about the significance of lesion location and PTSD. It would also be useful to explore the content of intrusions reported by this population. In particular, it would be useful to identify what the group who had no recall of the event were re-experiencing. Our understanding of the longitudinal course of PTSD post-stroke is limited and it would be helpful to establish whether acute stress disorder has the same power to predict PTSD in a stroke population as it was recently found to have in a non brain injured sample (Brewin et al, 1999). This type of design might also help to overcome one of the difficulties associated with all PTSD research which is that the most distressed will often decide not to participate in studies. It is not unusual for PTSD patients to be ambivalent about treatment (Yule 1999). Although the symptoms are significantly distressing, the suggestion of treatment or participation in a study which involves thinking and talking about the event they try to avoid, is too difficult. At least two individuals contacted for this study who declined to participate made spontaneous comments relating to the fact that they preferred not to discuss what had happened and would rather put it out of their minds.

It would also be useful to identify the factors which predispose some individuals to develop PTSD after stroke and to consider whether these are the same as in non brain injured groups. There are a number of factors which could have important roles in precipitating and maintaining post-stroke PTSD including premorbid psychopathology, appraisal of the event and social supports available post-stroke. Another area which would benefit from further investigation is that of vicarious traumatisation, especially as comments made by some participants suggested that although they had not been traumatised by the event, their close family members might have been.

4:11 Conclusion

This study can conclude that for some individuals the experience of having a stroke can precipitate a post-traumatic stress reaction. It provides more evidence that brain injury and PTSD are not mutually exclusive conditions. It also highlights a number of implications for clinicians working with stroke patients. Until recently psychological interventions after brain injury have tended to concentrate on neuropsychological assessment and rehabilitation. However, if PTSD is not recognised it is likely to have a detrimental effect on rehabilitation and functional improvement as well as psychological health. Davidson, Hughes, Blazer & George (1991) reported that PTSD patients were fifteen times more likely to attempt suicide than non-PTSD patients. In a sample of participants who had stroke, Sembi et al (1998) found that those diagnosed with PTSD group had higher levels of suicidal ideation than those without PTSD.

It is also likely that PTSD will have on detrimental effect on post-stroke rehabilitation. Post-stroke cognitive deficits, for example reduced motivation, concentration impairments, sleep disturbance will all be exacerbated with co-morbid PTSD (Bryant et al, 2001). Furthermore individuals post-stroke are already functioning with reduced cognitive capacity and any other demands on their reduced cognitive resources e.g. intrusive thoughts, are

likely to inhibit the efficacy of rehabilitation. In this study, levels of avoidance were found to be high, this could mean that patients are at best, unwilling to attend rehabilitation hospitals and at worst, that attending actually contributes to their distress. For individuals with PTSD, it is often crucial in therapy for them to have some sense of control and power over their situation. Post-stroke the individual's world has been "shattered" and they may not be given an opportunity to process this event as they are immediately, (albeit necessarily) immersed in the process of physical rehabilitation. A lack of both power and control are reinforced by this process. Attempts to establish a sense of predictability or certainty are impossible when no one can tell you how much recovery you are likely to make, whether you will have another stroke or why it happened in the first place. Avoidance is not a strategy that could easily be adopted as the individual is constantly faced with reminders of the event as well as being in a ward with others who are either more severely or less severely affected than them. They are constantly exposed to reminders of what they have lost or how "lucky" they have been not to be more disabled. These issues mean that identifying and providing interventions for post-stroke traumatic reactions could be beneficial not only in terms of the psychological health of patients, but also on the progress they make during rehabilitation.

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APPENDIX 1

DSM IV Criteria for Posttraumatic Stress Disorder Diagnosis.

Criterion A. The individual was exposed to a traumatic event in which both of the following were present:

- the person experienced, witnessed or was confronted with an event or events which involved actual or threatened injury to the physical integrity of themselves or others
- their response involved intense fear, helplessness or horror

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

- recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions
- recurrent distressing dreams of the event
- acting or feeling as if the traumatic event were recurring (includes a sense of reliving their experience, illusions, hallucinations and dissociative flashback episodes, including those that occur on awakening or when intoxicated.
- intense psychological distress at exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event
- physiological reactivity on exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.

Criterion C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness. As indicated by three(or more)of the following:

- efforts to avoid thoughts feelings, or conversations associated with the trauma
- efforts to avoid activities, places or people that arouse recollections of the trauma
- inability to recall an important aspect of the trauma
- markedly diminished interest or participation in significant activities
- feelings of detachment or estrangement from others
- restricted range of affect
- sense of foreshortened future.

Criterion D. Persistent symptoms of increased arousal as indicated by two or more of the following:

- difficulty falling or staying asleep
- irritability or outbursts of anger
- difficulty concentrating
- hypervigilance
- exaggerated startle response.

Criterion E. Duration of the disturbance is more than one month.

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

Specify if:

Acute: if duration of symptoms is less than three months

Chronic: if duration is three months or more

Specify if:

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

APPENDIX 2

PSYCHOLOGICAL SYMPTOMS AFTER STROKE : PATIENT INFORMATION SHEET

You are being invited to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with friends, relatives and your GP if you wish. Please ask if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part.

Thank you for reading this.

What is the purpose of the study?
This study will run between January and June 2001 and intends to examine some of the emotional consequences of having a stroke.

Why have I been chosen?
Approximately sixty patients who have had either a stroke or a haemorrhage have been asked to take part in this study.

Do I have to take part?
It is up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to withdraw at any time without having to give a 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 you will be interviewed by a researcher. This will be single session and would last no longer than 90 minutes. This will take place at an out-patient clinic or, if you do not regularly attend the hospital, a researcher could visit you at home.

What do I have to do?
You will be asked to complete two questionnaires. During the interview you will also be asked questions about how you have felt since your stroke or haemorrhage.

What are the possible benefits / disadvantages of taking part?
The information we get from this study may help us to treat future patients who become psychologically distressed after strokes.
The researcher will also ask you before you begin whether or not you would like feedback on the outcome of the interview.
A disadvantage to taking part could be that talking about your stroke and how you have felt since may be upsetting for you. However, if you are experiencing psychological distress, and you would like advice about accessing appropriate services, the researcher will be able to help with this.

Will I get any feedback?
If you would like feedback on the outcome of the project you should ask the researcher and this could be sent out to you once the study is completed. The feedback will however just be general and would not be specifically about you.

Will my taking part in this study be kept confidential?
All information which is collected about you during the course of the research will be kept strictly confidential. Any information about you will have your name and address removed so that you cannot be recognised from it.

What will happen to the results of the research study?
This study is part of the degree of Doctor of Clinical Psychology with the University of Edinburgh. As a result it will be written up and submitted for examination. The study may also be published in a journal. If it were to be published, this would occur within twelve months of the study being completed and you would be able to access a copy of the completed article through the National Library.

Contact for Further Information
Thank you for taking the time to consider participating in this study. If you require any further information regarding this project or if you have any questions please telephone:

Elizabeth Flynn
Trainee Clinical Psychologist
Department of Neuropsychology
Astley Ainslie Hospital
Edinburgh
Tel: 0131-537-9139

You will be contacted by telephone to ask if you wish to take part in this project.

You may also wish to contact the **Independent Advisor** for this project if you would like to discuss participation with someone not directly involved in the project.

Tony Prior
Head of Neuropsychology
Astley Ainslie Hospital
Edinburgh
Tel: 0131-537-9139

This Patient Information Sheet (version 2) is yours to keep. If you agree to participate you will be asked to complete a consent form, a copy of which will also be given to you.

4 January, 2001

Name _____ Date of Test ⌊__⌋⌊__⌋⌊__⌋
mo day yr

Age _____ Sex M F

Date of Birth ⌊__⌋⌊__⌋⌊__⌋
mo day yr

Day of the week s m t w th f s

Time AM PM

Diagnosis _____ Date of injury ⌊__⌋⌊__⌋⌊__⌋
mo day yr

GALVESTON ORIENTATION & AMNESIA TEST (GOAT)

Error Points

1. What is your name? (2) _____ When were you born? (4) _____ ⌊__⌋
   Where do you live? (4) _____
2. Where are you now? (5) city _____ (5) hospital _____ ⌊__⌋
   (unnecessary to state name of hospital)
3. On what date were you admitted to this hospital? (5) _____ ⌊__⌋
   How did you get here? (5) _____
4. What is the first event you can remember after the injury? (5) _____ ⌊__⌋
   Can you describe in detail (e.g., date, time, companions) the first event you can recall after injury? (5) _____
   _____
5. Can you describe the last event you recall before the accident? (5) _____ ⌊__⌋
   _____ Can you describe in detail (e.g., date, time, companions)
   the first event you can recall before the injury? (5) _____
6. What time is it now? _____(1 for each ½ hour removed from correct time to maximum of 5) ⌊__⌋
7. What day of the week is it? _____(1 for each day removed from correct one) ⌊__⌋
8. What day of the month is it? _____(1 for each day removed from correct date to maximum of 5) ⌊__⌋
9. What is the month? _____(5 for each month removed from correct one to maximum of 15) ⌊__⌋
10. What is the year? _____(10 for each year removed from correct one to maximum of 30) ⌊__⌋

Total Error Points ⌊__⌋

Total Goat Score (100-total error points) ⌊__⌋

APPENDIX 4

Impact of Event Scale
(IES)

On (date): _____

You experienced (life event): _____

Below is a list of comments made by people after stressful life events. Please check each item, indicating how frequently these comments were true for you **during the past seven days.** If they did not occur during that time, please mark the "not at all" column.

	Frequency			
	Not at all	Rarely	Sometimes	Often
1. I thought about it when I didn't mean to				
2. I avoided letting myself get upset when I thought about it or was reminded of it				
3. I tried to remove it from memory				
4. I had trouble falling asleep or staying asleep, because of the pictures or thoughts about it that came into my mind				
5. I had waves of strong feelings about it				
6. I had dreams about it				
7. I stayed away from reminders of it				
8. I felt as if it hadn't happened or it wasn't real				
9. I tried not to talk about it				
10. Pictures about it popped into my mind				
11. Other things kept making me think about it				
12. I was aware that I still had a lot of feelings about it, but I didn't deal with them				
13. I tried not to think about it				
14. Any reminder brought back feelings about it				
15. My feelings about it were kind of numb				

F. ANXIETY/OTHER DISORDERS

SCID-CV *Scoresheet*

37

E. Not due to a substance or a general medical condition (check p. 60)
WARNING: A "YES" answer to the interview question equals a "–" rating
Notes:

Check here ___ if criteria have been met in the past month.

? – + F37

F39 below

38 **OBSESSIVE-COMPULSIVE DISORDER CRITERIA A, B, C, D, AND E ARE "+"**
Check here ___ if criteria have been met in the past month.

F38

300.3 Obsessive-Compulsive Disorder

STTRAUMATIC STRESS DISORDER CRITERIA

39

TRAUMATIC EVENTS LIST F39

Brief description	Date (month/yr)	Age
_____	__/__	____
_____	__/__	____
_____	__/__	____
_____	__/__	____
_____	__/__	____
_____	__/__	____
_____	__/__	____

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

F40 (1) the person experienced, witnessed, or was confronted with an event that involved death, serious injury, or a threat to the physical integrity of self or others
Notes:

? – + **F40**

→ **F65** p. 56

F41 (2) response involved intense fear, helplessness, or horror
Notes:

? – + **F41**

→ **F65** p. 56

B. The traumatic event is persistently reexperienced in one (or more) of the the following ways:

F42 (1) distressing recollections of the event
Notes:

? – + **F42**

F43 (2) dreams of the event
Notes:

? – + **F43**

F44 (3) acting or feeling as if the traumatic event were recurring
Notes:

? – + **F44**

Ratings: ? = Inadequate information; – = Absent (or subthreshold); + = Present

F45	(4) intense psychological distress at exposure to internal or external cues *Notes:*	? – +	F45
F46	(5) physiological reactivity on exposure to internal or external cues *Notes:*	? – +	F46
F47	**AT LEAST ONE "B" SYMPTOM IS "+"**	? – + → F65 p. 56	F47
	C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness, as indicated by three (or more) of the following:		
F48	(1) efforts to avoid thoughts, feelings, or conversations *Notes:*	? – +	F48
F49	(2) efforts to avoid activities, places, or people that arouse recollections of the trauma *Notes:*	? – +	F49
F50	(3) inability to recall an important aspect of the trauma *Notes:*	? – +	F50

Ratings: ? = Inadequate information; – = Absent (or subthreshold); + = Present

F51	(4) markedly diminished interest or participation in significant activities *Notes:*	? – +	F51
F52	(5) feeling of detachment or estrangement from others *Notes:*	? – +	F52
F53	(6) restricted range of affect (e.g., unable to have loving feelings) *Notes:*	? – +	F53
F54	(7) sense of a foreshortened future *Notes:*	? – +	F54
F55	**AT LEAST THREE "C" SYMPTOMS ARE "+"**	? – + → F65 p. 56	F55
	D. Persistent symptoms of increased arousal as indicated by two (or more) of the following:		
F56	(1) difficulty falling or staying asleep	? – +	F56
F57	(2) irritability or outbursts of anger	? – +	F57
F58	(3) difficulty concentrating	? – +	F58
F59	(4) hypervigilance	? – +	F59
F60	(5) exaggerated startle response	? – +	F60
F61	**AT LEAST TWO "D" SYMPTOMS ARE "+"**	? – + → F65 p. 56	F61

Ratings: ? = Inadequate information; – = Absent (or subthreshold); + = Present

F62	E. Duration of the disturbance is more than 1 month	? – + → F65 below	F62
F63	F. Clinically significant distress or impairment	? – + → F65 below	F63
F64	**POSTTRAUMATIC STRESS DISORDER CRITERIA A, B, C, D, E, AND F ARE "+"** Check here ____ if criteria have been met in the past month.	+ → **309.81 Post-traumatic Stress Disorder**	F64

OTHER ANXIETY DISORDERS

F65	**300.22 Agoraphobia Without History of Panic Disorder** Check here ___ if present in the past month.	? – +	F65
F66	**300.23 Social Phobia** Check here ___ if present in the past month.	? – +	F66
F67	**300.29 Specific Phobia** Check here ___ if present in the past month.	? – +	F67
F68	**300.02 Generalized Anxiety Disorder** Check here ___ if present in the past month.	? – +	F68

APPENDIX 6

Centre Number:
Study Number:
Patient Identification Number for this trial:

CONSENT FORM

Title of Project: Psychological Consequences of Stroke

Name of Researcher:
Elizabeth Flynn
Trainee Clinical Psychologist
Department of Neuropsychology
Astley Ainslie Hospital
Edinburgh
0131-537-9139

Please tick box

1. I confirm that I have read and understand the information sheet dated 04/01/01 (version 2) for the above study and have had the opportunity to ask questions. ☐

2. 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. ☐

3. I understand that sections of any of my medical notes may be looked at by the researcher, I give permission for this individual to have access to my records. ☐

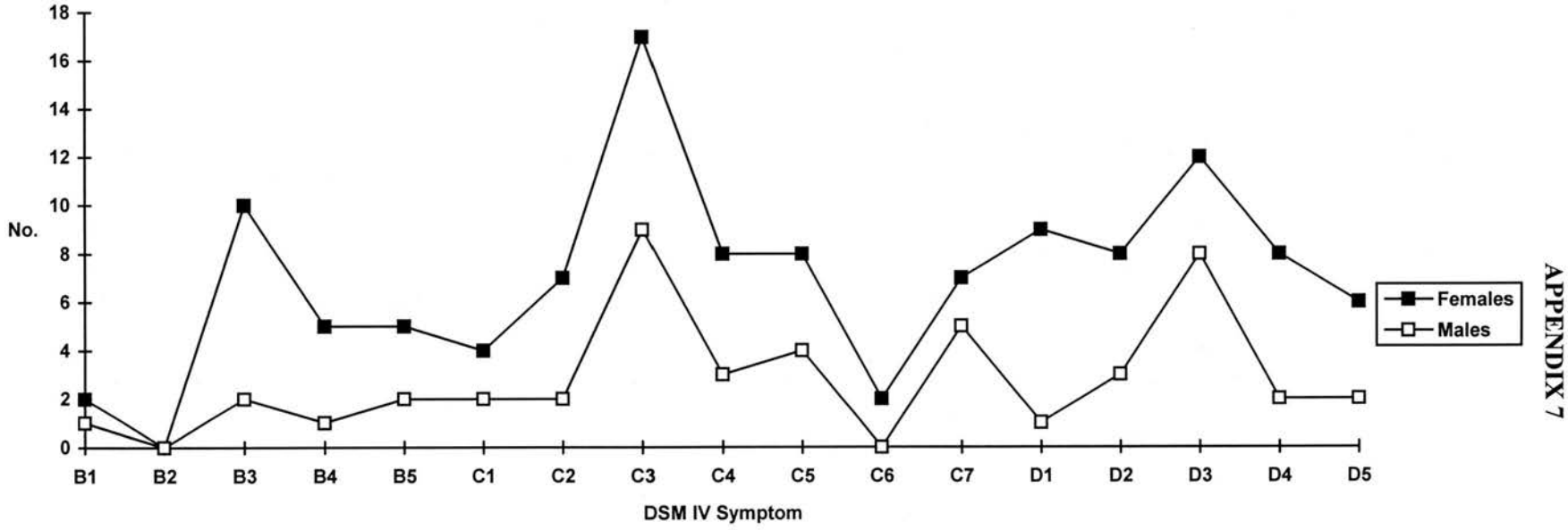
4. I agree to take part in the above study. ☐

5. I give permission for my GP to be notified of my participation in this study ☐

Name of Patient	Date	Signature
Name of Person taking consent (if different from researcher)	Date	Signature
Researcher	Date	Signature

1 for patient; 1 for researcher; 1 to be kept with hospital notes

Male / Female Symptom Profiles



B1= Intrusive thoughts
B2= Nightmares
B3= Recurring
B4= Distress at reminders
B5= Arousal at reminders

C1= Avoid thoughts
C2= Avoid activities
C3= Can't remember part of event
C4= Diminished interest
C5= Detached

C6= Affect
C7= Future

D1= Sleep disturbance
D2= Anger
D3= Concentration
D4= Hypervigilant
D5 =Heightened Startle

APPENDIX 7