VOLUME ONE

Poor memory as a predictor of poor treatment response in adults diagnosed with Posttraumatic Stress Disorder

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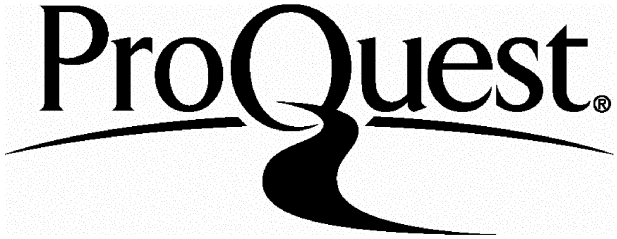

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ABSTRACT

Current research has highlighted impaired learning and memory processes in adults diagnosed with Posttraumatic Stress Disorder (PTSD). Although some clients respond favourably to psychological therapy, outcome studies indicate that treatment rarely leads to a full remission of the disorder. Neuroimaging studies of adults with chronic PTSD have suggested altered brain morphology in regions associated with memory functioning, specifically the hippocampus. It is possible that impaired learning and memory processes adversely affect the capability of clients with PTSD to respond to treatment. This study investigated the memory, attention and learning profiles of 27 adults diagnosed with PTSD who presented at a specialist treatment centre prior to commencing cognitive-behavioural therapy. Measures of PTSD, anxiety, depression, and past and current history of alcohol/substance use were obtained on assessment. A neuropsychological test battery was then administered to assess baseline cognitive functioning, memory, learning, attention, and executive function. Twenty-three adults were followed up at session eight of treatment, and their PTSD diagnosis was re-evaluated. Clients who did not improve with treatment had significantly poorer performance on intake measures of verbal memory. In particular, a measure of encoding meaningful verbal material was found to independently predict outcome. Differences were not accounted for by performance on tasks of attention and executive function. Further, severity of PTSD symptomatology, severity of anxiety and depression, length of time since trauma, and alcohol and substance use were not related to memory functioning. The theoretical, clinical, and research implications of this were discussed.

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1. INTRODUCTION

Current research has highlighted impaired learning and memory processes in adults diagnosed with Posttraumatic Stress Disorder (PTSD). Cognitive-behavioural therapy (CBT) is currently the most widely practised psychological treatment for this disorder (O'Brien, 1998). Although some clients respond favourably to CBT, research indicates that it rarely leads to a full remission of the disorder (e.g. Shalev, Bonne & Eth, 1996). Impaired memory functioning, common in PTSD, may contribute to poor treatment outcome. To date, no study has looked at pre-treatment learning and memory functioning in adults diagnosed with PTSD and therapeutic outcome.

The goal of this study is to evaluate the contribution of cognitive and memory functioning to therapeutic outcome in adults diagnosed with PTSD.

The Introduction is divided into three sections. The first presents an overview of PTSD, the literature on treatment outcome and factors currently thought to predict chronicity. The second section presents an overview of memory, learning and related neuropsychological processes with specific consideration to PTSD and the possible mechanisms of impairment. The third section concludes the Introduction with the study questions and hypotheses.

1.1 POSTTRAUMATIC STRESS DISORDER

1.1.1 Definition

Posttraumatic Stress Disorder (PTSD) is a psychiatric disorder that gained formal recognition in the diagnostic nomenclature in 1980 when it was included in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-III;* American Psychiatric Association, 1980). It describes the psychological symptoms that develop in some people following exposure to extreme trauma. Prior to 1980, psychological symptoms relating to a traumatic event were described and named according to the inciting event. For example, combat fatigue, shell shock, and the rape trauma syndrome (Golier & Yehuda, 1998).

PTSD is classified as an anxiety disorder and shares many symptoms in common with panic disorder, phobic anxiety, generalised anxiety disorder, and obsessive-compulsive disorder, although none of these diagnoses covers the whole of the PTSD syndrome. PTSD encompasses three broad categories of symptoms: (a) the re-experiencing of intrusive memories, (b) avoidance behaviour and numbing of emotional responsiveness and, (c) autonomic hyperarousal. These are described in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association, 1994).

(i) The Re-experiencing of Intrusive Memories

These symptoms include intrusive thoughts and/or images, nightmares, visual or other sensory hallucinations, and vivid flashback memories in which there is an element of dissociation from the present surroundings. Situational cues, such as

smells, people, places, and activities, often trigger the re-experiencing symptoms. The individual may act or feel as though the traumatic event is recurring.

(ii) Avoidance Behaviour and Numbing of Emotional Responsiveness

These symptoms refer to behavioural and/or cognitive avoidance of reminders of the trauma, such as avoiding people, places, conversations, and thoughts. They also include features of depression, such as emotional numbing, restricted range of affect, and diminished interest in activities. For this group of symptom criteria, DSM-IV notes possible memory difficulty with respect to an inability to recall an important aspect of the trauma.

(iii) Autonomic Hyperarousal

These symptoms refer to heightened arousal as indicated by difficulty falling asleep or staying asleep, irritability, difficulty concentrating, hypervigilance, and exaggerated startle responses.

1.1.2 DSM-IV Diagnosis[1]

The DSM-IV diagnosis of PTSD consists of six criteria. Criterion A specifies that an individual must have been exposed to a traumatic event in which he/she witnessed, experienced or was threatened with serious physical injury or death, resulting in intense fear, helplessness or horror. If this criterion is not met, a diagnosis cannot be made despite the nature of the individual's symptoms.

[1] The tenth edition of the International Classification of Diseases (ICD-10) (World Health Organisation, 1992) also outlines diagnostic criteria for PTSD. However, they are less specific than DSM-IV criteria, and are therefore not discussed in this thesis.

Criteria B, C and D refer to the clinical features of PTSD. These include the symptoms of re-experiencing, avoidance and hyperarousal. In order to meet a diagnosis of PTSD, an individual must experience at least one re-experiencing symptom, three symptoms of avoidance, and two hyperarousal symptoms.

Criterion E specifies that the symptoms must endure for one month.

Criterion F states that the symptoms must cause significant distress or impairment in social, occupational or other important areas of functioning.

When the symptoms endure between one and three months post-trauma, a diagnosis of Acute PTSD is given. When the symptoms endure for more than three months, a diagnosis of Chronic PTSD is given. Additionally, DSM-IV recognises 'delayed onset' PTSD. This requires that the symptoms appear at least six months after the individual experienced the traumatic event.

Appendix 1 shows the DSM-IV diagnostic criteria for PTSD.

1.1.3 Prevalence

DSM-IV places the lifetime prevalence of PTSD from 1 to 14% in community populations. For high-risk populations, such as combat veterans or rape victims, the prevalence rates range from 3% to 58%. In an extensive review of the literature, Green (1994) reported that approximately 25-30% of the general population develop PTSD in response to a traumatic event. In most trauma survivors, the intensity of

symptoms declines with time. However, a third will develop chronic PTSD (Kessler, Sonnega, Bromet, Hughes & Nelson, 1995; Solomon, 1997).

1.1.4 Comorbidity

Individuals with PTSD are at risk of developing secondary affective, alcohol/substance abuse, panic and phobic disorders (Marmar, Weiss & Pynoos, 1996). Typically, 50% to 90% of PTSD sufferers experience one other co-morbid psychiatric disorder (Freedy, Shaw & Jarrell, 1992; Kulka, Schlenger, Fairbank, Hough, Jordan, Marmar & Weiss, 1990).

Depression is the most common comorbid diagnosis among women with PTSD, followed by alcohol use disorders, drug abuse, phobia, and conduct disorder (Kessler *et al.*, 1995). Alcohol use disorders are the most common comorbid diagnosis in men with PTSD, followed by depression, conduct disorder, drug abuse, and simple phobias (Kessler *et al.*, 1995).

There are three main hypotheses regarding the reason for the high comorbidity associated with PTSD: (1) the diagnosis of PTSD includes symptoms of anxiety and depression. There is therefore a high degree of symptom overlap between PTSD and other anxiety disorders, and depression (Keane & Wolfe, 1990), (2) alcohol and substance use disorders may develop as a consequence of trying to manage the distress associated with the symptoms of PTSD (Keane & Kaloupek, 1997), and (3) the comorbid disorders may reflect a possible genetic vulnerability to the development of psychiatric disorders in general (Keane & Kaloupek, 1997).

Regarding the high degree of symptom overlap between PTSD and other disorders, in particular major depression, Yehuda, Southwick, Krystal, Bremner, Charney & Mason (1993) suggest that the depressive disorder that often accompanies PTSD is a different biological abnormality than primary major depression. In a study of cortisol suppression in adult men with PTSD, Yehuda *et al.* (1993) found that PTSD-only subjects showed greater suppression of cortisol in response to administration of dexamethasone (DEX), an exogenous steroid, compared to normal controls. They also found that PTSD subjects with comorbid major depression showed greater suppression of cortisol following DEX administration compared to normal controls. This is in contrast to studies of cortisol suppression in subjects with depression only in which the usual response is non-suppression of cortisol, rather than enhanced suppression following DEX administration (e.g. Arana, Baldessarini & Ornsteen, 1985; Carol, 1982). This led the group of investigators to conclude that the depression seen in PTSD sufferers may be a different dysthymic disorder unique to PTSD.

The second hypothesis regarding comorbidity and PTSD suggests that PTSD and comorbid disorders, such as substance abuse, are initiated concurrently (Bremner, Southwick, Darnell & Charney, 1996; Davidson, Kudler, Saunders & Smith, 1990). This hypothesis proposes a model of self-medication in which alcohol or other substances are used to manage symptom-related distress. That is, individuals who develop PTSD may use alcohol or substances to alleviate some of the distress associated with their symptoms (Keane & Kaloupek, 1997).

The third hypothesis incorporates the idea of a pre-existing vulnerability to the development of psychiatric disorders. It is possible that individuals with psychiatric disorders, such as alcohol and substance use disorders, experience genetic, social or familial vulnerabilities which may predispose them to the development of PTSD following exposure to traumatic stress (Keane & Kaloupek, 1997).

A review of the literature suggests that, in general, rates of comorbidity appear to develop over time (Yehuda & McFarlane, 1995) representing a complication that emerges in the context of PTSD. This is consistent with the self-medication model which proposes that individuals who develop PTSD use alcohol and/or other substances to alleviate the distress associated with their symptoms.

1.1.5 The Course of PTSD

Most cases of recovery from PTSD occur in the first twelve months (Kessler *et al.*, 1995; Rothbaum, Foa, Riggs, Murdock & Walsh, 1992). This is the same for those who seek treatment and those who do not. The mean time to remission in individuals seeking treatment is three years, and six years for those who do not seek treatment (Kessler *et al.*, 1995). One third of individuals will not recover from PTSD regardless of whether or not they seek treatment (Kessler *et al.*, 1995). That is, they will continue to experience chronic PTSD for many decades.

The long-term course of PTSD may result in enduring personality change and symptoms of hostility, distrust towards the world, social withdrawal, feelings of emptiness, hopelessness, being on edge and estrangement from others (World Health Organization, 1993) as well as neurobiological changes. These changes likely

interact, affect the individual's capacity to recover, and prolong the course of the disorder.

Research suggests that symptoms change over time (e.g. Freedman, Brandes, Peri & Shalev, 1999). This reflects the complex interplay between neurobiological changes, comorbid conditions, and the psychological and social impact of ongoing symptoms. Neurobiological changes include heightened sensitivity to the stress hormone, cortisol, which influences the body's response to stress, as well as the body's system of arousal. Further, increased sensitivity appears to lead to atrophy in the brain region associated with learning and memory, specifically the hippocampus[2]. The neurobiological changes, the psychiatric comorbidity, and the psychological and social impact of experiencing PTSD may prolong the course of the disorder. These factors may also increase vulnerability to a range of psychiatric disorders, including reactivation of recovered PTSD. A study of combat veterans has shown that recovered PTSD is subject to reactivation upon exposure to similar conditions (e.g. Solomon, Garb, Bleich & Grupper, 1987).

1.1.6 Factors Associated with Outcome

A number of studies have looked at factors predicting the onset and outcome of PTSD. These include variables associated with the traumatic event, the individual, and the recovery environment. They are summarised as follows and described below:

[2] These changes are discussed in more detail in Section 1.2.2.5.3: Reduced Volume of the Hippocampus.

Traumatic Event – Exposure Variables:

1. Trauma of human design
2. Personal injury
3. Life threat

Personality Factors:

1. History of psychological or behavioural problems
2. Prior experience of traumatic events
3. Peri-traumatic dissociation
4. External locus of control

Post-trauma factors:

1. Low level of social support
2. Low level of cortisol
3. Depression
4. Avoidant coping behaviour
5. Fragmented memory
6. Alcohol misuse
7. Intrusive symptoms
8. Litigation

1.1.6.1 Precipitating Event: Exposure Variables

Studies of the prevalence rates of PTSD in survivors of different traumatic events indicate that events of human design, such as rape and torture, are associated with a high incidence of PTSD (e.g. Kilpatrick, Saunders, Amick-McMullan & Best, 1989; Mellman, Randolph, Brawman-Mintzer, Flores & Milanes, 1992). Janoff-Bulman (1985, 1989, 1992) proposed that PTSD following victimisation is largely due to the shattering of basic assumptions that victims hold about themselves and the world. These assumptions are: (1) the world as benevolent; (2) the world as meaningful; and (3) the self as worthy[3]. Victimisation, in particular, may lead to a questioning of all three assumptions (Joseph, Williams, & Yule, 1997). This rests on the notion of the world as meaningful. If one deserves what one gets, then the experience of victimisation would suggest that one is not a worthy person, the world is not a

[3] This theory is discussed in more detail in Section 1.1.7.3: (ii) The Schema Approach.

benevolent place, and the world is no longer meaningful. It is possible that traumatic events of non-human design, such as natural disasters, are less likely to shatter these assumptions, particularly the self as worthy, and perhaps, for this reason are associated with a lower incidence of PTSD.

Other important variables regarding the traumatic event include the degree of life threat and physical injury (e.g. Gleser, Green & Winget, 1981; Green, Grace & Gleser, 1985). Perceived life threat has been shown to consistently predict PTSD across stressors (e.g. Blanchard, Hickling, Taylor & Loos, 1995; Resnick, Kilpatrick, Best & Kramer, 1992).

1.1.6.2 Personality Factors

Studies of personality factors predicting PTSD have led to inconsistent results. The majority of studies suggest that prior history of psychological or behavioural problems (e.g. Atkeson, Calhoun, Resick & Ellis, 1982; Breslau, Davis, Andreski & Peterson, 1989; Frank, Turner, Stewart, Jacob & West, 1981), prior experience of traumatic events (e.g. Breslau *et al.*, 1991; Burgess & Holmstrom, 1978; Kilpatrick, Veronen & Best, 1985; Roth, Wayland & Woolsey, 1990), peri-traumatic dissociation (Shalev, Peri, Canetti & Schreiber, 1996), and external locus of control (McCormick, Taber & Kruedelbach, 1989; Frye & Stockton, 1982; Solomon, Mikulincer & Benbenishty, 1989) are strong predictors of PTSD development. The predictive value of external locus of control appears to fluctuate across stressors. For example, among combat veterans, external locus of control is associated with poorer outcome (e.g. Frye & Stockton, 1982; Solomon *et al.*, 1989), but among survivors of transportation accidents, external locus of control is associated with better outcome

(e.g. Joseph, Brewin, Yule & Williams, 1991; Joseph, Brewin, Yule & Williams, 1993)

1.1.6.3 Post-Trauma Factors

Post-trauma factors include person variables (psychological and physiological) and environmental variables that develop or are present in the trauma aftermath.

An individual's post-trauma method of coping is a psychological variable that affects the probability of suffering from PTSD. For example, in their prospective study of trauma survivors recruited from a general hospital's emergency room, Freedman *et al.* (1999) found that early symptoms of behavioural and/or cognitive avoidance were associated with chronic PTSD one year later. Cognitive avoidance in the form of thought suppression results in the recurrence of intrusive trauma cognitions (Wegner, Shortt, Blake & Page, 1990). Intrusions have been associated with the development of PTSD (Freedman *et al.*, 1999; Joseph, Yule & Williams, 1994, 1995; McFarlane, 1992). Some studies have not found this (e.g. Creamer, Burgess & Pattison, 1992). The discrepant findings may relate to when intrusive activity was initially assessed. McFarlane (1992) suggests that intrusive activity immediately following trauma is common and therefore unlikely to be predictive of later pathology.

Misuse of alcohol has been found to predict later development of PTSD (e.g. Blanchard *et al.*, 1996) and this may indicate difficulties engaging with and organising the traumatic memory. Fragmented memories are associated with ongoing PTSD (Foa, Molnar & Cashman, 1995).

Depression in the aftermath of trauma predicts long-term PTSD (e.g. Freedman *et al.*, 1999). This may reflect depressive cognitions and negative appraisal of the traumatic event which is associated with chronic PTSD (Ehlers & Clark, 2000).

Neuroendocrine alterations are a physiological variable associated with the development of PTSD. For example, low cortisol levels in the immediate aftermath of trauma predict the later development of PTSD (e.g. McFarlane, Atchinson & Yehuda, 1997; Resnick, Yehuda, Pitman & Foy, 1995). These may reflect a prior history of trauma and/or an overly sensitive biological stress response system, both of which are associated with chronic PTSD.

Studies have shown that environmental variables predict PTSD. For example, greater social support is associated with better outcome following a range of traumatic events, such as rape (e.g. Kilpatrick *et al.*, 1985), toxic exposure (e.g. Bromet, Parkinson, Schulberg, Dunn, & Gondek, 1982), combat (e.g. Fontana & Rosenheck, 1994), and civilian disasters (e.g. Cook & Bickman, 1990).

Finally, some studies have found that those involved in litigation are likely to have a worse outcome (e.g. Kuch, Cox & Evans, 1996). One explanation for this is that litigation proceedings may provide constant reminders of the trauma which exacerbates PTSD symptoms. It is also possible that those involved in litigation are more severely traumatised and this adversely affects recovery. It is also possible that litigation proceedings encourage sufferers to exaggerate their symptoms. However, studies suggest that PTSD symptoms remain even after completion of proceedings, suggesting that this is unlikely to be the case (Mendelson, 1995). Other studies have

not found a relationship between litigation and the development or duration of PTSD (e.g. Mendelson, 1984; Mendelson, 1991).

1.1.7 Psychological Models of PTSD Used To Guide Treatment

A number of psychological models have been put forth to explain the development and maintenance of PTSD. All models view at least some of the symptoms of PTSD as problems of anxiety. The emphasis and role of anxiety in the development and maintenance of symptoms differs from model to model.

Both behavioural and cognitive models guide the widely practised cognitive-behavioural interventions and cognitive models, in particular, have good explanatory power. Behavioural models are based on learning theory and support the use of exposure therapies in the treatment of PTSD. The behavioural approach to understanding PTSD is described briefly below. Cognitive models are described in Section 1.1.7.3.

1.1.7.1 Behavioural Models

(i) Mowrer's Two Factor Learning Theory (Mowrer, 1960)

Keane, Zimmerling & Caddell (1985) applied Mowrer's two factor learning theory (Mowrer, 1960) to the onset and maintenance of PTSD. Two factor theory asserts that the onset of PTSD is via classical conditioning whereas the condition persists because of instrumental conditioning. Traumatic events serve as unconditional stimuli that become associated with cues (conditioned stimuli) present at the time of the trauma. These conditioned stimuli then elicit conditioned responses similar to the unconditioned responses such as fear and anxiety which the traumatic episode

elicited. Escape from conditioned stimuli reduces fear. Hence, escape or avoidance of conditioned stimuli is negatively reinforced. In other words, a trauma sufferer learns avoidant behaviours as a way of terminating or reducing the presence of aversive stimuli.

(ii) Higher Order Conditioning

To explain why individuals with PTSD avoid stimuli that were not present at the time of the trauma, Keane *et al.* (1985) use principles of higher order conditioning and stimulus generalisation. That is, stimuli (i.e., sounds, time of day, odours) originally conditioned to the trauma may become paired with other similar stimuli and eventually come to elicit the same fear, anxiety and physiological responses. These cues elicit trauma-related memories and again, avoidant strategies become negatively reinforced as a way to reduce the fear, anxiety and hyperarousal.

1.1.7.2 Limitations: Behavioural Models

The learning models explain generally why PTSD sufferers experience distressing recollections of the trauma and why they develop avoidant behaviours. However, many symptoms of PTSD cannot be easily conceptualised as unconditioned responses that have become conditioned responses. For example, the unconditioned response of fear at the time of the trauma does not involve having flashbacks or nightmares and hence these symptoms cannot accurately be conceptualised as conditioned responses to the unconditional trauma stimuli. Thus, behavioural models have trouble accounting for specific symptoms of intrusion (i.e., nightmares, and acting or feeling as if the event is recurring), dissociation (i.e., flashbacks, psychogenic amnesia), physiological overarousal (i.e., startle responses, sleep

disturbance, and concentration difficulties), and emotional numbing (i.e., restricted range of affect and chronic feelings of detachment from others). Further, they specifically fail to account for the presence of PTSD in some trauma victims and not others.

Finally, the models focus on fear and anxiety as motivational states for avoidance and symptom development. They fail to consider the role and activation of the full range of distressing emotional states in response to conditioned reminders of the traumatic event. These include anxiety, fear, as well as grief, guilt, shame, rage and anger (Joseph, Williams & Yule, 1997). Thus, the models ignore the importance of cognitive and information processing which precedes these states.

1.1.7.3 Cognitive and Information Processing Models

Cognitive and information processing theories conceptualise PTSD as indicating the presence of unprocessed trauma-related information in memory. According to Foa & Kozak (1986), the traumatic event is represented in a different form in the memory of a PTSD sufferer compared to a victim who has recovered. The re-coding of the trauma memory into an organised memory record facilitates emotional processing of the traumatic event. Anxiety and re-experiencing symptoms are a measure of incomplete emotional processing. That is, the traumatic memories remain coded in a form different to the recovered trauma victim.

Emotional processing is a largely conscious process in which representations of past events and associations as well as representations of future events repeatedly enter into and are actively manipulated within working memory (Brewin, Dalgleish, &

Joseph, 1996). Foa and her colleagues (e.g. Foa & Kozak, 1986; Foa, Steketee & Rothbaum, 1989; Foa & Riggs, 1993) have made extensive use of the concept of emotional processing. Foa & Kozak's (1986) model of PTSD and other cognitive and information processing models are described below.

(i) The Cognitive Information Processing Approach

Fear Network Model (Foa & Kozak, 1986)

In this model, PTSD reflects impaired emotional processing as a result of problematic memory integration. This is due to the formation of a fear network at the time of the trauma. The network encompasses stimulus information about the traumatic event, information about cognitive, behavioural, and physiological reactions to the trauma, and interoceptive information that links these stimulus and response elements. The fear network is disjointed and consequently difficult to integrate with existing, organised models. Activation of this fear network by triggering stimuli causes information in the network to enter consciousness (the intrusion symptoms of PTSD). Attempts to avoid and suppress such activation lead to the cluster of avoidance symptoms. Integration of information in the fear network with existing memory structures leads to adaptive emotional processing. This requires activation of the fear network and integration of incompatible information with this network.

(ii) The Schema Approach

There are two theories of PTSD which fall under the schema approach: Janoff-Bulman's (1985, 1989, 1992) cognitive appraisal theory of PTSD and Horowitz's

(1975, 1976, 1979, 1982, 1986a, 1986b) information processing theory of PTSD. They are described briefly below.

Janoff-Bulman's Theory of PTSD (1985, 1989, 1992)

This theory focuses on basic assumptions (schemas) about the self and world that exist prior to traumatisation. These are: (1) the world is benevolent, (2) the world is meaningful, and (3) the self is worthy (Janoff-Bulman, 1985; 1989; 1992). Traumatic experiences shatter these basic assumptions leading to post-traumatic stress. The theory has been criticised because it does not explicate what processes are involved when the assumptions are shattered, and why individuals with a premorbid psychiatric history are more likely to develop PTSD after a trauma (Brewin *et al.*, 1996). However, it was one of the first theories to underscore the role of cognitive appraisal in the development of PTSD and this process is incorporated in current cognitive and information processing models of the disorder.

Horowitz's Theory of PTSD (Horowitz, 1975, 1976, 1979, 1982, 1986a, 1986b)

This theory is based on the idea that individuals have schemas of the world and of themselves that they use to interpret incoming information. New information is integrated with current schema because there is a psychological drive for completion, called the 'completion tendency.' A traumatic event presents information which is incompatible with existing schemas. This gives rise to a stress response in which there is an information overload. Thoughts, memories, and images of the trauma cannot be reconciled with current schema. As a result, a variety of defense mechanisms come into operation to keep traumatic information unconscious and the individual experiences a phase of numbing, denial, and avoidance. The tension between the completion tendency and the psychological defense mechanisms causes

oscillation between phases of intrusion, and avoidance and denial as traumatic material is gradually integrated with long-term schemas. PTSD is viewed as a failure of information processing in which traumatic information remains in active memory without ever being integrated (Brewin *et al.*, 1996).

This theory has a number of limitations. It does not explain why some individuals develop PTSD and others do not. It is unable to explain delayed onset PTSD. It does not explain the observation that not all individuals experience oscillation between denial and intrusion (Brewin *et al.*, 1996; Creamer, Burgess & Pattison, 1992). Finally, the theory does not incorporate the effect of an individual's attributions and interpretations of the traumatic experience on outcome (Brewin *et al.*, 1996).

(iii) Integration: Cognitive, Information and Emotional Processing

Dual Representation Model (Brewin *et al.*, 1996)

This model suggests that representations of trauma in memory take two forms: conscious verbally accessible memories (VAMs) and non-conscious, situationally accessible memories (SAMs). PTSD symptoms result when SAMs intrude from the non-conscious to the conscious in response to reminders. Emotional processing after any trauma has three potential endpoints. The first is completion/integration where memories of the trauma have been fully processed, worked through, and integrated with memories of the self and world view. There are two maladaptive endpoints: chronic emotional processing and inhibited emotional processing. The first is associated with severe trauma in which the discrepancy between prior assumptions and trauma information is too great to effectively integrate. Thus, trauma-related

VAMs and SAMs are chronically processed leading to preoccupation with the trauma consequences and intrusive memories. The second endpoint is a result of sustained efforts to avoid the reactivation of unpleasant SAMs and VAMs. This state inhibits further emotional processing and is associated with symptoms of cognitive, behavioural and affective avoidance.

1.1.8 Treatment of PTSD

There are a range of treatment approaches to PTSD: behavioural, cognitive, cognitive-behavioural, psychodynamic, pharmacological and a combination of these approaches. Cognitive behavioural therapy (CBT) is the most widely practised and widely studied psychological treatment for PTSD, and has the best evidence for efficacy (O'Brien, 1998). Numerous controlled studies support the use of CBT techniques, such as exposure therapies, in the treatment of PTSD (e.g. Cooper & Clum, 1989; Boudewyns, Hyer, Woods, Harrison, & McCranie, 1990; Foa, Rothbaum, Riggs, & Murdock, 1991; Keane, Fairbank, Caddell, & Zimmerling, 1989). CBT draws on behavioural, cognitive and information processing models. CBT for PTSD includes exposure procedures, cognitive restructuring procedures, anxiety management programs, and their combinations. CBT is often practised in conjunction with pharmacotherapy.

1.1.8.1 Exposure Therapy

Exposure therapy procedures aim to undo the fear and arousal that have been conditioned to stimuli cues paired with the stressful exposure. This involves having patients confront their fears and to learn to pair new responses (such as relaxation) to cues associated with the stressful exposure. This necessitates activation of the fear

network (stimulus information about the traumatic event formed at the time of trauma) which allows an opportunity for corrective information to be integrated, and thus to modify the pathological elements of the trauma memory. The repeated trauma reliving generates a more organised memory record which can be more readily integrated with existing schemas (Foa & Rothbaum, 1998). Individuals who fail to organise this memory exhibit more trauma-related disturbances, suggesting that the process of recovery involves the organising and streamlining of the traumatic memory (Foa & Riggs, 1993).

Exposure therapies are often effective in reducing the intrusion symptoms of PTSD. These treatments require at the very least, the ability to focus and sustain attention, intact episodic memory processes, as well as an ability to articulate memories. There are three types of exposure therapies used in the CBT treatment of PTSD: systematic desensitisation, flooding, and direct therapeutic exposure. They are further classified according to the medium of exposure. That is, imaginal versus in vivo.

1.1.8.1.i Systematic Desensitisation

Wolpe (1958) pioneered systematic desensitisation. This technique pairs imaginal exposure to feared stimuli with relaxation in a graded, hierarchical fashion (Foa & Meadows, 1997). Outcome studies (e.g. Bowen & Lambert, 1986; Frank, Anderson, Stewart, Dancu, Hughes & West, 1988; Peniston, 1986; Schindler, 1990) looking at this type of therapy in PTSD have led to inconclusive results due to methodological problems, such as lack of a control condition, failure to assess PTSD symptom severity, and/or failure to assess comorbid conditions.

1.1.8.1.ii Flooding

Stampfl and Levis (1967) introduced this learning-based therapy to reduce anxiety and avoidance in clients who suffered from these problems. The aim is to achieve habituation to the stimulus by prolonged exposure to an imaginal or in vivo stimulus where high levels of arousal are induced in the patient. Flooding treatments begin with exposure to highly feared rather than innocuous stimuli.

Keane *et al.* (1989) compared two groups of Vietnam veterans with PTSD. One group received imaginal flooding, the other was the wait-list control. The treatment group showed reduced depression, fear, state anxiety, and intrusive symptoms six months post-treatment, but no reduction in emotional numbing or avoidant symptoms of PTSD.

Cooper and Clum (1989) compared two groups of treatment conditions to evaluate the effectiveness of flooding therapy. The subjects were Vietnam veterans who were troubled by re-experiencing symptoms of PTSD. One group received flooding therapy in addition to a 'standard' treatment; the other group received the 'standard' treatment alone which consisted of weekly individual and group therapy. The flooding group experienced a greater reduction in weekly nightmares, a reduction in self-rated arousal, sleep disturbances, hypersensitivity to sound, state anxiety and 'psychotic-like' symptoms compared to the control group. However, there was no effect on depression, trait anxiety and proneness to violence.

Contrasting with the above, however, in an open trial study, Pitman, Altman, Greenwald, Longpre, Macklin, Poire & Steketee (1991) reported an exacerbation of

depression, panic, anxiety, increased alcohol consumption, and mobilisation of negative appraisal during flooding therapy in a group of 15 war veterans.

1.1.8.1.iii Direct Therapeutic Exposure

Richards & Rose (1991) and Rothbaum & Foa (1992) describe this form of therapy. It is also referred to as prolonged exposure. Direct therapeutic exposure (DTE) requires the client to recount the traumatic incident in the first person and present tense with the client being encouraged to hold particularly difficult scenes in his/her mind until anxiety levels have subsided. Sessions are audiotaped and the client is instructed to listen to the tape whilst imagining the traumatic scene, several times a week. Imaginal exposure is supplemented by in vivo desensitisation to situations representing those circumstances in which the original trauma occurred (Hughes & Thompson, 1994).

Foa *et al.* (1991) compared rape victims with PTSD randomly assigned to one of four treatment conditions: stress inoculation training (SIT), prolonged exposure (PE), supportive counselling (SC), or a wait-list control (WL). All conditions produced improvement on all measures immediately post-treatment and at 3.5 months follow-up. However, SIT produced significantly more improvement on PTSD symptoms than did SC and WL immediately following treatment. At follow-up PE produced superior outcome on PTSD symptoms. The SC and WL conditions improved arousal symptoms of PTSD, but not intrusion and avoidance symptoms.

In an open-trial study, Thompson, Charlton, Kerry, Lee, & Turner (1995) found that DTE for a group of 23 patients with PTSD led to 50% recovery. That is, half of the subjects no longer met diagnostic criteria for PTSD at post-treatment.

1.1.8.2 Cognitive Restructuring

Cognitive restructuring formally addresses negative automatic thoughts and the dysfunctional beliefs about oneself and the world that these thoughts reflect (Foa & Rothbaum, 1998). Cognitive restructuring requires intact episodic memory and cognitive processes, as well as an ability to articulate thoughts.

Brewin *et al.* (1996) and Thrasher, Lovell, Noshirvani & Livanou (1996) advocate the use of cognitive techniques, such as cognitive restructuring, to manage negative emotions, reassign meaning and promote cognitive change. This involves discourse in which the client, after having identified the thoughts and beliefs underlying fear, examines whether or not these accurately reflect reality, and replaces mistaken thoughts or beliefs with more realistic, helpful ideas (Foa & Rothbaum, 1998). The aim is to reattribute meaning to memories, attempting to correct the common problems of inappropriate guilt and self-recrimination (O'Brien, 1998). Like exposure techniques, cognitive restructuring aims to reduce symptoms via correcting erroneous elements of the pathological fear structure underlying PTSD (Foa & Rothbaum, 1998). Additionally, cognitive restructuring aims to deal with the thoughts and beliefs underlying anger, guilt and shame.

Cognitive restructuring is usually used as part of a combined treatment package which includes exposure therapy. An outcome study comparing cognitive processing

therapy, a combination of exposure and cognitive restructuring, to a wait-list control group of sexual assault survivors showed that the treatment group improved from pre-to posttreatment on PTSD and depression measures (Resick & Schnicke, 1992).

Tarrier, Pilgrim, Sommerfield, Faragher, Reynolds, Graham & Barrowclough (1999a) conducted a randomised trial to compare cognitive therapy and imaginal exposure in the treatment of chronic PTSD. Both treatment approaches resulted in symptom reduction, but neither led to complete recovery.

1.1.8.3 Anxiety Management Techniques

Anxiety management techniques (AMT) aim to help clients improve their management of high anxiety and other symptoms (Joseph, Williams, & Yule, 1997). One of the most commonly used anxiety management treatments for PTSD is stress inoculation training (SIT) (Foa & Meadows, 1997). This program incorporates a number of educational and skills components such as relaxation, thought stopping, and guided self-dialogue. AMT are effective in reducing PTSD severity in the short-term (Foa *et al.*, 1991; Foa, Riggs, & Gershuny, 1995) and require an ability to focus and sustain attention, as well as an ability for new learning.

1.1.8.4 Pharmacotherapy

Pharmacological studies of PTSD are varied. There are few controlled studies, and most of the research has focused on male Vietnam veterans who have complex and chronic histories of PTSD. This makes it difficult to generalise across populations and stressors. Nevertheless, recent studies (e.g. Van der Kolk, Dreyfuss, Michaels, Shera, Berkowitz, Fisler, & Saxe, 1994; Nagy, Southwick & Charney, 1993; Shay,

1992; Kline, Dow, Brown, & Matloff, 1994) suggest that selective serotonin re-uptake inhibitors (SSRIs) ameliorate symptoms of intrusion and avoidance to some extent. No study, however, has shown full remission of PTSD symptoms through pharmacotherapy alone (Shalev *et al.*, 1996). Prescribed medication may reduce the tendency of PTSD patients to self-medicate, as well as the risk of violent behaviour and suicide which may facilitate participation in psychotherapy (Shalev *et al.*, 1996). Outcome data (e.g. Bleich, Siegel, Garb and Lerer, 1986) suggest that a combination of pharmacotherapy and psychotherapy is beneficial in up to 70% of patients with PTSD.

1.1.8.5 Treatment Limitations

Studies of treatment intervention in PTSD show that it is a difficult condition to treat, and that for a sizeable proportion of clients, treatment is not particularly effective. Most of the outcome studies have involved small sample sizes and open trials. It is therefore necessary to draw on meta-analytic reviews which have attempted to integrate research findings to further look at the efficacy of treatment.

Sherman (1998) reported a meta-analysis of 17 controlled clinical trials for PTSD. The predominant treatment modality was behavioural or cognitive-behavioural with extensive use of exposure techniques. Results indicated a significant impact of psychotherapeutic intervention on PTSD symptomatology both immediately post-treatment and at follow-up. However, the author reported that the effect size of almost half the studies may be inflated. Eight studies used wait-list, no-treatment control groups, rather than psychological placebo control groups. Placebo treatments show superior outcomes to wait-list controls (McConaghy, 1990).

Six of the 17 studies included in the Sherman (1998) review assessed patients for PTSD diagnosis after treatment. Of the 103 patients with a full PTSD diagnosis before treatment, more than half (57%) still met diagnostic criteria for PTSD after treatment. Nevertheless, 43% of clients with the diagnosis improved, suggesting that treatment is effective for some PTSD sufferers.

Another widely-sited meta-analysis of PTSD treatment concluded that several treatment protocols reduce PTSD symptoms and improve the patient's quality of life, but the magnitude of results is limited and remission is rarely achieved (Shalev *et al.*, 1996). This meta-analysis qualitatively reviewed 65 treatment studies, and as such includes studies of variable quality.

Thus, meta-analytic reviews of treatment outcome for PTSD are inconclusive. It appears that treatment reduces PTSD symptoms, but does not necessarily lead to a long-term remission of the diagnosis. This may reflect the duration of the disorder (Shalev, 1997). Prolonged arousal produces significant alteration in neuronal functioning which treatment interventions may fail to redress (Shalev, 1997). Treatment resistance in chronic PTSD may also result from the presence of comorbid disorders (Shalev, 1997). Approximately 80% of PTSD sufferers are diagnosed with an additional psychiatric disorder at the time of assessment (Green, 1994).

1.1.8.6 Factors Affecting Treatment Outcome

Factors that influence the ability to engage in treatment are likely to affect outcome. Studies have shown that poor attendance (Tarrier *et al.*, 1999a; Tarrier, Sommerfield, Pilgrim, & Faragher, 2000), poor motivation (Tarrier *et al.*, 1999a),

and high levels of expressed emotion (EE) in the support environment (Tarrier, Sommerfield, & Pilgrim, 1999b) negatively influence CBT outcome for PTSD. Furthermore, individuals with PTSD often have high levels of shame (Andrews, Brewin, Rose, & Kirk, 2000) and shame drives individuals to hide and withdraw (Gilbert & McGuire, 1998). Therefore, some PTSD sufferers may avoid discussing their problems because of shame. It is also possible that co-morbid disorders, such as anxiety, depression, and substance abuse may interfere with treatment and recovery. Depression affects motivation and this can influence treatment engagement, and therefore treatment outcome.

Finally, it is possible that memory impairment and other related difficulties, such as attention and learning, affect the ability to engage in treatment and to maintain therapeutic gains. Memory and learning difficulties have been found in PTSD sufferers, as well as those with anxiety, depression and/or substance abuse.

1.1.8.7 The Importance of Learning and Memory Processes

Several recent studies have suggested that PTSD sufferers demonstrate deficits in memory function (Barrett, Green, Morris, Giles, & Croft, 1996; Bremner, Scott, Delaney, Southwick, Mason, Johnson, Innis, McCarthy, & Charney, 1993; McNally, Lasko, Macklin, & Pitman, 1995; Yehuda, Keefe, Harvey, Levengood, Gerber, Geni, & Siever, 1995) and new learning (Jenkins, Langlais, Delis & Cohen, 1998). These deficits may occur before the onset of the disorder or as a result.

Short-term psychological treatments require intact cognitive functioning, specifically learning and episodic memory processes. Factors that adversely affect learning and

memory processes have implications for the ability of these clients to respond to treatment and may be associated with poor outcome. No study to date has looked at neuropsychological functioning in PTSD sufferers and treatment outcome, despite the overwhelming evidence of learning and memory deficits in this population of patients and the variable efficacy of psychological treatment. This study aims to do this.

The next section reviews the literature on learning and memory deficits in patients with PTSD, and the proposed mechanisms of impairment.

1.2 LEARNING AND MEMORY

1.2.1 Summary of Neuropsychological Findings and Methodological Difficulties

The majority of studies examining neuropsychological performance of PTSD samples have found difficulties with memory function, as well as related processes, such as attention, learning, and executive function. Some studies have found evidence of actual impairment in these areas, while others have found significant differences in the performance of PTSD subjects when compared to controls. A few studies have found no differences or evidence of impairment.

1.2.1.1 Pattern of Deficits

Specific patterns of memory performance have varied in individuals with PTSD. Deficits have been found in verbal memory and/or learning (Bremner *et al.*, 1993; Jenkins *et al.*, 1998; Vasterling, Brailey, Constans & Sutker, 1998; Yehuda *et al.*, 1995; Barrett *et al.*, 1996), non-verbal learning (Bremner *et al.*, 1993; Vasterling *et al.*, 1998; Barrett *et al.*, 1996), attention (Uddo, Vasterling, Brailey & Sutker, 1993;

Vasterling *et al.*, 1998; Beckham, Crawford & Feldman, 1998), autobiographical memory (McNally, Litz, Prassas, Shin & Weathers, 1994; McNally *et al.*, 1995; Kuyken & Brewin, 1995), and processing bias for disaster-specific information (Thrasher, Dalgleish & Yule, 1994).

1.2.1.2 Inconsistent Findings

Only a few studies of PTSD sufferers have found no evidence of impairment (e.g. Gil, Calev, Greenberg, Kugelmass & Lerer, 1990; Zalewski, Thompson & Gottesman, 1994; Stein, Hanna, Vaerum & Koverola, 1999). These studies highlight the importance of considering the nature of the traumatic stressor and other factors which may influence cognitive functioning.

For example, Stein *et al.* (1999) compared adult women with a history of childhood sexual abuse (CSA) to healthy comparison subjects on measures of learning, memory, and executive function. Seventy-seven percent of the CSA group met criteria for current PTSD. Results revealed no differences in performance between the two groups. As the majority of neuropsychological studies have been conducted with Vietnam veterans with PTSD, the results led the authors to conclude that different traumatic stressors may lead to different neurocognitive sequelae. However, adults who develop PTSD following sexual assault do show deficits in memory function (e.g. Jenkins *et al.*, 1998). This highlights the importance of considering the developmental stage at which the trauma occurred. Childhood trauma is associated with neuroendocrine alterations (Golier & Yehuda, 1998). However, these change over time, and the effect on development and the interactions with behavioural and cognitive symptoms is unknown (Golier & Yehuda, 1998). It is

possible that neuroendocrine alterations associated with learning and memory difficulties in adults with PTSD have less of an impact in childhood due to the developmental stage of the trauma, as well as the educational input at that time. If children develop learning and memory difficulties, it is possible that they are more likely to learn to overcome these through their ongoing education, and as such, may not exhibit such difficulties at a later age.

In their study, Stein *et al.* (1999) measured current PTSD. There was no information regarding the duration of the PTSD symptoms in the CSA group beyond a time period of one month. The specificity of their findings to acute, chronic or lifetime PTSD is therefore unclear.

Although Stein *et al.* (1999) found no differences, their study suggests that the following factors may interact to influence cognitive functioning in PTSD samples: the nature of the trauma (e.g. sexual abuse vs. combat exposure), gender (men vs. women), the developmental stage at which the trauma occurred (e.g. childhood vs. adulthood), and the duration of symptoms (e.g. current vs. lifetime).

1.2.1.3 Methodological Difficulties

The inconsistencies in the neuropsychological findings point to the past and current methodological difficulties inherent in conducting research with PTSD samples. Most of the research has been done on Vietnam veterans with a history of symptoms enduring for more than 20 years. Such a lengthy duration of symptoms may increase the likelihood of comorbid psychological, health-related, and social factors that may confound study findings (Vasterling *et al.*, 1998). Further, the heterogeneity of

symptoms allowed within the PTSD diagnosis contributes to within-group differences in the expression of PTSD both in the range of symptoms experienced, as well as their severity. Most studies do not comment on the frequency and severity of intrusive, avoidance, and hyperarousal symptoms of their PTSD samples. Thus, some studies may have more homogeneous PTSD samples than others and this may lead to different expressions in neuropsychological performance. Another difficulty has been the small sample sizes. Given the debilitating impact of PTSD symptoms, it is difficult to recruit subjects for two to three hours of neuropsychological testing. Further, given the high rate of comorbidity, it is difficult to recruit a PTSD-only sample. Hence, samples have tended to be small, and this can influence data interpretation.

Another difficulty is that studies have used different inclusion criteria regarding loss of consciousness. Even relatively brief periods of unconsciousness can result in diffuse axonal injury (Gennarilli, Thibault, Adams, Graham, Thompson, & Marcincin, 1996). Some studies have stipulated an inclusion criteria of ten minutes or less (e.g. Bremner *et al.*, 1993), while others have stipulated 30 minutes or less (e.g. Vasterling *et al.*, 1998). These differences may account for some of the discrepant findings.

Finally, few studies have reported on the compensation-seeking status of their subjects. Bellamy (1997) and Frueh, Gold & de Arellano (1997) found that seeking compensation encouraged sufferers to exaggerate their symptoms. This may relate to neuropsychological performance as well. Individuals with PTSD who are also seeking compensation may exaggerate poor performance on neuropsychological

measures, particularly if they believe that doing so could be helpful to their litigation proceedings.

Despite the inconsistencies, however, the majority of studies do support deficits in memory function across populations and stressors.

1.2.2 Memory and the Nature of Impairment in PTSD

1.2.2.1 Basic Concepts

Memory refers to knowledge that is stored in the brain and to the processes of acquiring and retrieving such knowledge (Tranel & Damasio, 1995). Memory can be divided into two broad systems: explicit (declarative) and implicit (non-declarative) memory. Explicit memory refers to the ability to learn about and remember information, objects and events (Tranel & Damasio, 1995). Implicit memory refers to types of learned responses, such as conditioned reflexes and motor skills, which are not available for conscious reflection, such as driving a car (Hodges, 1994).

Explicit memory is further divided into two systems: short-term memory and long term memory. Short-term memory is a concept more accurately referred to as working memory. It is further divided into verbal and spatial components. That is, immediate memory for small amounts of verbal material, and immediate memory for spatial material. Baddeley and Hitch (1974) propose a model of working memory which consists of three subsystems: the phonological loop, the visuo-spatial sketchpad, and the central executive. The phonological loop is responsible for the immediate repetition of words; the visuo-spatial sketchpad is responsible for the immediate repetition of numbers. The central executive is the attentional controller.

It is associated with frontal lobe function (Hodges, 1994) and is assumed to be responsible for the selection and operation of strategies, and for maintaining and switching attention as the need arises (Baddeley, 1995). The central executive forms an interface between long-term memory and the other two subsystems in the model.

Long-term memory consists of two components: episodic and semantic memory. Episodic memory refers to the system involved in recollecting particular experiences or episodes, such as remembering what one ate at breakfast (Baddeley, 1995). Semantic memory refers to knowledge of the world (Baddeley, 1995). Knowing the frequency of radio waves, or how many centimetres there are in a metre are examples of semantic memory. Figure 1 shows the major divisions of memory and the neural substrate associated with each system (adapted from Hodges, 1994). It is not a theoretically driven model, however, and is therefore not theoretically accurate. For example, implicit memory is shown as separate from long-term memory, even though the sub-divisions of implicit memory (i.e. conditioning, priming, and motor skills) can be conceptualised as part of a long-term memory system.

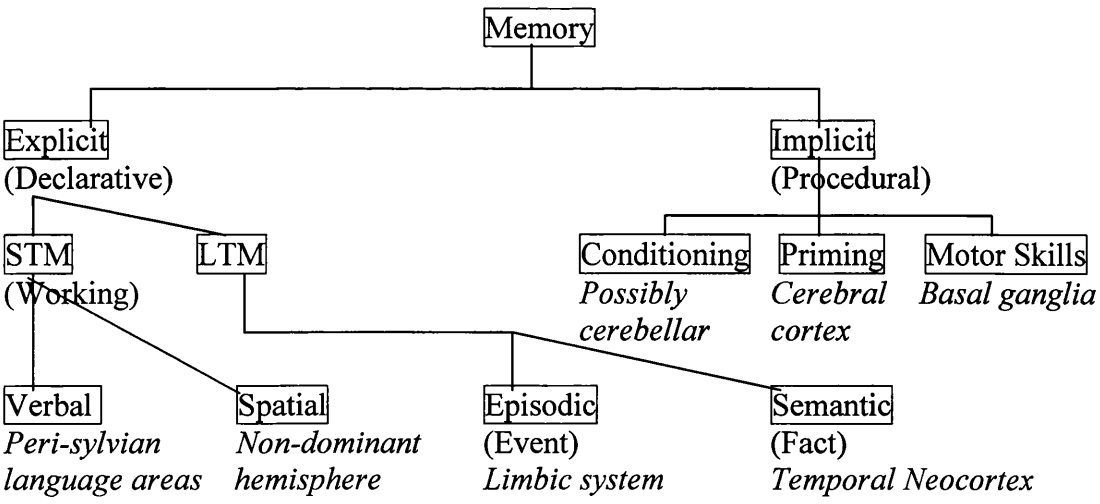

Figure 1.1: Major divisions of memory and their neural substrates (adapted from Hodges, 1994)

1.2.2.2 Memory Formation

The process of forming memory involves three basic steps: (1) acquisition, (2) consolidation, and (3) storage.

Acquisition refers to the process of bringing knowledge into the brain and into a first-stage memory 'buffer', via sensory organs and primary sensory cortices (Tranel & Damasio, 1995). Consolidation is the process of rehearsing knowledge and building a robust representation of it in the brain. Storage refers to the creation of a relatively stable memory trace or record of knowledge in the brain (Tranel & Damasio, 1995).

Retrieval is the process of reactivating knowledge in such a way that it can become available to consciousness (as in recall and recognition) or translated into a motor output (movement in a limb or in the vocal apparatus, autonomic activity) (Tranel & Damasio, 1995).

1.2.2.3 Explicit Memory Function

The majority of studies of PTSD samples point to deficits in explicit memory function. These include difficulties with verbal and non-verbal memory and learning which indicate problems with the verbal and spatial components of working memory, as well as the episodic component of long-term memory. Autobiographical memory difficulties indicate problems with episodic memory.

1.2.2.3.i Verbal and Non-Verbal Memory and Learning

Studies of PTSD samples have looked at verbal and non-verbal memory performance, as well as verbal and non-verbal learning. Most neuropsychological measures that tap verbal and non-verbal learning are also a measure of working memory since they test immediate recall of verbal and non-verbal material.

Bremner *et al.* (1993) found that compared to control group subjects, PTSD subjects had significantly lower scores on verbal memory tasks of immediate and delayed recall. This means that subjects had difficulty immediately recalling a story that was read to them, and they also had difficulty recalling this story after a delay period. The performance of the PTSD subjects fell in the impaired range. They also had significantly lower scores on the total recall, long-term retrieval, long-term storage, and delayed recall measures of verbal learning. This means that they had difficulty learning 12 words over a number of trials, and difficulty recalling these words after a delay period. Significant differences were also found on the long-term retrieval, long-term storage, and continuous recall measures of visual learning. This means that they had difficulty learning and recalling 12 designs over a number of trials. These results suggest difficulty with both the visual and spatial components of working memory which reflect disruption in one or more of the stages of memory processing. That is, disruption in the stage of encoding, consolidation and/or retrieval.

The drawback of this study was that Bremner *et al.* (1993) did not control for or evaluate comorbid diagnoses other than alcohol abuse. Given the prevalence of

depression in PTSD samples, it is likely that the control and PTSD groups differed significantly on this variable, and this may have inflated the findings.

Nevertheless, other studies of PTSD samples have found impairment in the verbal component of working memory when comorbid diagnoses have been controlled for (Jenkins *et al.*, 1998; Vasterling *et al.*, 1998; Yehuda *et al.*, 1995), and in the spatial component (Vasterling *et al.*, 1998; Barrett *et al.*, 1996). Barrett *et al.* (1996) found that memory and learning performance was worse in PTSD subjects with comorbid diagnoses of depression, anxiety, and/or alcohol/substance abuse compared to PTSD subjects without comorbid diagnoses.

1.2.2.3.ii Autobiographical Memory

Autobiographical memory refers to the capacity to recollect facts and incidents from one's own life. In response to cue words, the retrieval of autobiographical memories falls into two categories: specific or overgeneral. Specific autobiographical memories refer to specific, detailed memories which include details of when the episode or event occurred. For example, 'I had a good time at Jane's party last week' is an example of a specific memory. Overgeneral memories refer to memories in which details are vague, and exclude information indicating the date the event or episode occurred. For example, 'I enjoyed the party' is an example of an overgeneral memory.

Much of the research on specificity of autobiographical memory has focused on depressed patients. Many studies have found that individuals with depression retrieve significantly more overgeneral memories in response to positive and

negative cue-words compared to controls (e.g. Williams & Scott, 1988; Moore, Watts, & Williams, 1988).

Studies of PTSD have shown that compared to non-PTSD samples, individuals with PTSD demonstrate difficulties retrieving specific autobiographical memories in response to negative and positive cue words (e.g. McNally *et al.*, 1994; McNally *et al.*, 1995). Thus, PTSD is associated with deficits in retrieving specific memories. This may reflect problems with allocating attentional resources to the task at hand. The findings may also reflect compromised anatomical structures which adversely influence the ability to recall details from the past (McNally, 1997). Further the findings may reflect the influence of cognitive avoidance, a feature of PTSD. Kuyken & Brewin (1995) found that cognitive avoidance was associated with overgeneral memories in response to both positive and negative cue words in individuals with a history of physical and sexual abuse.

1.2.2.4 Related Processes: Attention, Information Processing, and Executive Function

The processes of attention, information processing and executive function are involved in and overlap with memory performance.

1.2.2.4.i Attention

Attention refers to an individual's ability to attend to and grasp all of a specific stimulus (Howieson & Lezak, 1995). Mirsky, Anthony, Duncan, Ahearn & Kellam (1991) proposed a model of attention which includes the following four components: (1) Focus-Execute: This refers to the ability to focus on specific environmental cues

from an array and respond appropriately to them; (2) Sustain: This refers to the ability to maintain optimal levels of focused attention or vigilance over time; (3) Shift: This refers to the ability to change the focus of attention in an adaptive manner; and (4) Encode: This refers to the ability to register, recall, and mentally manipulate information sequentially. This component overlaps with the construct of working memory.

Reductions in attention produce marked limitations on memory (Howieson & Lezak, 1995). Distinguishing between memory impairment and more general impairment in attention or concentration is difficult. Administration of attention and memory measures is needed in order to look at the relative contribution of both.

In a study of attention and verbal memory performance in a sample of Persian Gulf War veterans with and without PTSD, Vasterling *et al.* (1998) found that the PTSD group showed performance deficiencies on the Encode and Sustain aspects of attention. They also demonstrated working memory difficulties of verbal material.

Beckam *et al.* (1998) found that veterans with PTSD performed significantly worse than veterans without PTSD on measures of sustained attention, concentration and complex processing.

These results suggest that attentional processes are affected in subjects with PTSD and that they may contribute to memory dysfunction.

1.2.2.4.ii Information Processing

Information processing refers to the ability to extract the meaning of the stimulus based on past experiences (Howieson & Lezak, 1995). Information processing applies to both verbal and non-verbal material. Slow information processing can contribute to memory dysfunction (Howieson & Lezak, 1995).

To date, studies have looked at trauma-related information processing in PTSD samples. Using a modified Stroop procedure, a number of studies have shown increased response latency for trauma-related words in PTSD samples compared to controls (e.g. Bryant & Harvey, 1995; Cassiday, McNally & Zeitlin, 1992; Foa, Feske, Murdock, Kozak & McCarthy, 1991; McNally, Kaspi, Riemann & Zeitlan, 1990; Thrasher *et al.*, 1994).

Thrasher *et al.* (1994) looked at severity of PTSD and information processing. They compared two groups of PTSD subjects with high and low levels of symptomatology with a non-traumatised control group. They found that PTSD subjects with high levels of symptomatology evidenced a significantly longer response latency for colour-naming disaster-related words compared to PTSD subjects with low levels of symptomatology and non-traumatised controls. They concluded that the selective processing bias for trauma-specific material was specific to those survivors with chronic PTSD. They postulated that this was because presentation of trauma-related material in the Stroop paradigm activated the fear structure within memory and this interfered with performance.

To date, no study has looked at non-trauma related information processing and whether or not this contributes to the observed memory difficulties in subjects with PTSD.

1.2.2.4.iii Executive Function

Executive functions refer to motivating, control and regulatory behaviours necessary to formulate goals and carry them out effectively (Lezak, 1982; Stuss & Benson, 1987). The major categories of executive behaviours are (1) volition, (2) planning, (3) executing activities, and (4) self-monitoring (Lezak, 1995). Difficulty in any of these task-oriented behaviours can adversely affect memory performance.

Few studies have looked at executive function in PTSD. Beckham *et al.* (1998) used the Trail Making Test of the Halstead-Reitan test battery (Reitan & Wolfson, 1993) to compare the performance of veterans with and without PTSD. Part A measures simple attention, motor function, attention/concentration, and manual dexterity. Part B measures sustained attention, concentration, and complex processing (Beckham *et al.*, 1998). Part B is hypothesised to reflect the ability to self-monitor. That is, the ability to monitor and self-correct performance spontaneously and reliably (Howieson & Lezak, 1995). Results revealed significant differences in performance between the two groups on both parts of the Trail Making Test. Analyses excluding participants on anti-anxiety or cardiac medication and those with comorbid diagnoses and/or positive compensation seeking status, revealed a significant group difference for Trail B only. The authors concluded that neuropsychological deficits in combat veterans may extend beyond specific memory deficits. It is possible that difficulty

with executive functions, specifically self-monitoring behaviour, may contribute to the observed memory difficulties in PTSD.

Vasterling *et al.* (1998) used the Wisconsin Card Sorting Test (WCST; Berg, 1948) as one of their measures of attention when comparing the performance of PTSD and non-PTSD veterans. The WCST is also a measure of executive behaviour, specifically the ability to execute activities (Howieson & Lezak, 1995). That is, the ability to initiate behaviour and modify that behaviour through switching, maintaining or stopping behaviour in an integrated manner according to an analysis of appropriate actions (Lezak, 1982). Vasterling *et al.* (1998) found no significant differences between the two groups on this measure.

To date, research shows executive function difficulty related to self-monitoring behaviour in PTSD samples. Further research is needed to look at both executive function and memory performance in PTSD samples.

1.2.2.5 Mechanisms of Memory Impairment

The observed memory difficulties in subjects with PTSD may reflect (1) the influence of anxiety, depression, and/or alcohol-substance misuse, (2) severity of PTSD symptomatology, particularly symptoms of intrusion, (3) hippocampal impairment (Bremner, Randall, Scott, Bronen, Sebyl, Southwick, Delaney, McCarthy, Charney & Innis, 1995; Bremner, Randall, Vermetten, Staib, Bronen, Mazure, Capelli, McCarthy, Innis & Charney, D.S., 1997; Stein, Hanna, Koverola, Torcha & McClarty, 1997) (4) poor premorbid memory status, (5) global

impairment, and/or (6) disrupted system of arousal (e.g. Vasterling *et al.*, 1998). These are discussed in detail below.

1.2.2.5.1 The influence of anxiety, depression and alcohol-substance misuse on memory

(i) Anxiety

Studies of anxiety have looked at its influence on working memory, learning, and information processing. In summary, working memory is adversely affected by state anxiety (e.g. Dunn, 1968; Eysenck, 1979), but not by trait anxiety (Watts, 1995). On measures of learning, anxiety is associated with enhanced performance (Eysenck, 1977), particularly for high ability subjects (e.g. Hodges & Durham, 1972). However, on difficult learning tasks, anxiety is associated with poorer performance (Standish and Champion, 1960). Finally, anxious subjects show processing strategies that are less structured and less efficient and performance that is more effortful than non-anxious subjects (Watts, 1995).

Eysenck and Calvo (1992) suggested that anxiety, through worry and other mechanisms, reduces the amount of processing capacity that is available for task performance and subjects may compensate for this by more effortful performance. MacLeod & Mathews (1991) found that subjects with Generalised Anxiety Disorder (GAD) showed high processing priorities to threat-related options compared to subjects without GAD. It is possible that this processing bias reduces processing efficiency needed for optimal task performance.

Thus, anxiety typically impairs processing efficiency (e.g. Eysenck & Calvo, 1992) and task performance when demands on working memory capacity increase (e.g. Calvo & Ramos, 1989; Eysenck, 1982).

(ii) Depression

Studies of depression have looked at working memory, learning, information processing and executive function. In general, depressed subjects show relatively intact performance on tasks of working memory, such as measures of immediate recall, but are more consistently impaired on longer-term episodic memory tasks, such as delayed recall and new learning (Watts, 1995). They show difficulty with memory tasks that require effortful processing (e.g. Calev & Erwin, 1985; Calev, Nigal & Chazan, 1989; Golinkoff and Sweeney, 1989; Roy-Byrne, Weingartner, Bierer, Thompson & Post, 1986; Watts & Sharrock, 1987; Zakzanis, Leach & Kaplan, 1998), and some tasks of executive function, such as multiple scheduling (Channon & Green, 1999).

In order to determine whether depression is associated with difficulty engaging in any effortful mental activity versus difficulty with effortful processing, Zakzanis *et al.*, (1998) looked at studies of depressed and control subjects that used demanding tests of mental effort and studies that used memory measures of effortful processing. They found that the effect sizes for studies using the former measures were small compared to the effect sizes of studies using the latter measures. They concluded that the effect of effort in depressed subjects is specific to memory processes and not a general effect of effortful mental activity.

The memory difficulties exhibited by depressed subjects have both cognitive and physiological explanations. At a cognitive level, they may reflect fewer available processing resources due to preoccupation with negative thoughts, as well as inefficiency or difficulty appropriately applying available processing resources (Ellis & Ashbrook, 1988). Some studies have shown that when depressed subjects are provided with cues to aid their memory performance, their performance shows no deficit (e.g. Hertel & Hardin, 1990; Hertel & Rude, 1991). Others (e.g. Channon & Green, 1999) have found that the provision of strategy hints improves the use of performance strategies, but does not improve overall memory performance.

At a physiological level, impaired memory in depressed subjects may reflect frontal lobe dysfunction (Channon & Green, 1999) and/or hippocampal dysfunction (Bremner, Narayan, Anderson, Staib, Miller & Charney, 2000). Functional imaging studies have provided evidence of frontal lobe dysfunction in depression (e.g. Dolan, Bench, Brown, Scott, & Frackowiak, 1994). Further, a number of studies (e.g. Channon, Baker & Robertson, 1993; Channon & Baker, 1994; Channon, 1996) have found that depression is associated with impairment in the central executive component of working memory associated with frontal lobe function.

Depressed subjects also show higher levels of cortisol compared to normals (e.g. Carroll, Curtis, Davies, Mendels & Sugarman, 1976; Sachar, Hellman & Roffwarg, 1973). Cortisol is a glucocorticoid steroid released by the adrenal glands in response to stress. High levels of cortisol have a deleterious effect on memory (McGaugh, Liang & Bennett, 1984). Further, the hippocampus, a brain structure important in learning and memory, is rich in glucocorticoid receptors, and sensitive to these

hormones (Golier & Yehuda, 1998). Some MRI neuroimaging studies have found smaller hippocampal volumes in depressed subjects (e.g. Bremner *et al.*, 2000), while others have not (e.g. Axelson, Doraiswamy, McDonald, Boyko, Tupler, Patterson, Nemeroff, Ellinwood & Krishman, 1993).

(iii) The influence of Drugs and Alcohol

A consideration of drugs and alcohol with respect to memory performance must consider the effects of current use, as well as the effects of chronic abuse.

(iii.i) Current Use: Drugs and Alcohol

While SSRIs, commonly used to treat depression, exert minimal effects on measures of performance and cognition (Bond & Lader, 1996), a number of other psychotropic medications interfere with memory functioning. Benzodiazepines, used in the short-term treatment of anxiety (Bond & Lader, 1996), produce consistent impairments in performance on many types of memory tests, such as recall and recognition tests (e.g. Brown, Lewis, Horn & Bowes, 1982; Clarke, Eccersley, Frisby & Thornton, 1970; Curran, Schiwy, Eves, Shine, & Lader, 1988; Weingartner, Grafman, Herrmann, Molchan, Sunderland, Thompson & Wolkowitz 1992). These drugs interfere with the encoding of new material, but not with the retrieval of previously learned material (Bond & Lader, 1996).

During marijuana usage, there is reduced memory efficiency related to storage, but not retrieval (Darley, Tinklenberg, Roth & Atkinson, 1974; Darley, Tinklenberg, Hollister & Atkinson, 1973), as well as slowed visual processing (Braff, Silverton, Saccuzzo & Janowsky, 1981).

Alcohol also produces deficits in memory performance (Birnbaum, Parker, Hartley & Noble, 1978; Hastroudi, Parker, DeLisi, Wyatt, & Mutter, 1984; Lister, Gorenstein, Risher-Flowers, Weingartner, & Eckardt, 1991). Alcohol has an effect on a number of areas in the central nervous system, resulting from its action on a number of neural transmitters (Lombardi & Weingartner, 1995). It impairs performance on explicit tests of memory, while leaving performance on implicit tests of memory intact (Hastroudi *et al.*, 1984; Lister *et al.*, 1991). Alcohol also interferes with retrieval processes, as measured by performance on general-information questions requiring subjects to access their long-term semantic knowledge base (Nelson, McSpadden, Fromme & Marlatt, 1986).

(iii.ii) Chronic Alcohol and/or Substance Use

Alcohol

Chronic alcohol abuse has been found to adversely affect a number of cognitive functions, although findings are not always universal. Chronic alcohol abuse almost always impairs sensorimotor functioning, particularly colour vision (Mergler, Blain, Lemaire & Lalande, 1988), and visual search and scanning efficiency (Kapur & Butters, 1977; Ryan & Butters, 1986). To a lesser extent, difficulties with memory, learning, executive functions and visuospatial organisation have been found. Some studies report improvement in these processes following a period of abstinence.

With respect to memory functioning, chronic alcoholics tend to show subtle but consistent working memory and learning deficits. These become more evident as task difficulty increases (Ryan & Butters, 1982, 1986). The deficits appear to reflect encoding difficulties (Lezak, 1995), although retrieval difficulties have also been

found (e.g. Nelson *et al.*, 1986). On learning tasks, intrusions (recalling a word that has not been presented in the test trial) appear in greater frequency than is normal (Kramer, Blusewicz & Preston, 1989). Many studies suggest that chronic alcoholics perform normally on verbal learning tests but may do poorly on visual learning assessments (e.g. Bowden, 1988; De Renzi, Faglioni, Nichelli & Pignattari, 1984). However, verbal and visual deficits have also appeared concurrently (Nixon, Kiyawski, Parsons & Yohman, 1987). Overall, Lezak (1995) reports that serious memory and learning deficits are not a regular feature of chronic alcoholism. When former alcoholics are abstinent for five years or more, improvements in working memory approach normal levels (Ryan & Butters, 1982).

On a physiological level, cerebral atrophy has been found among chronic alcoholics (Harper & Blumbergs, 1982; Jernigan, Butters, DiTraglia, Schafer, Smith, Irwin, Grant, Schuckit & Cermak, 1991) and is thought to be due to the toxic effects of alcohol (Walton, 1994). Chronic heavy alcohol ingestion reduces the elaboration of dendrites in the brain, mostly in the hippocampus and cerebellum (Ryan & Butters, 1986). The hippocampus is a brain region crucial in memory functioning (see Section 1.2.2.5.3).

Other Substances: Marijuana, Cocaine, Opiates

Heavy use of marijuana has been associated with affective blunting, mental and physical sluggishness, apathy, restlessness, some mental confusion, and poor recent memory (Lezak, 1995).

Long-term use of cocaine has been associated with memory and concentration difficulties (e.g. Washton & Stone, 1984). The memory problems appear to be due mostly to reduced retrieval efficiency, but a mild storage deficit is also suggested (e.g. Mittenberg & Motta, 1993).

With respect to opiates, such as heroin, reports on mental status with abstinence are conflicting. Some studies have found no lasting deficits even in persons who had long-term addictions (e.g. Fields & Fullerton, 1975; Parsons & Farr, 1981). However, other studies suggest that long-term opiate users do sustain permanent impairments with poor performance on tests involving visuospatial and visuomotor activities (e.g. Carlin, 1986; Grant, Adams, Carlin & Rennick, 1977). Polydrug users, who use central nervous system depressants in particular, such as sedatives, hypnotics and opiates, show neuropsychological impairments within the first several weeks of abstinence (e.g. Carlin, 1986; Grant, Adams & Reed, 1979).

(iv) Teasing out the influence of anxiety, depression and alcohol-substance use on memory performance in adults with PTSD

A number of studies of memory function in PTSD have controlled for comorbid disorders. Nevertheless, the results are conflicting. Some studies (e.g. Jenkins *et al.*, 1998; Beckham *et al.*, 1998) suggest that the observed memory impairments are not attributable to comorbid depression, anxiety and/or alcohol use. While other studies (e.g. Barrett *et al.*, 1996) suggest that they are. It is possible that gender and type of traumatic stressor influence the impact of comorbid disorders on memory functioning. Women survivors of rape with PTSD show memory impairments not due to depression, anxiety or alcohol-substance use (e.g. Jenkins *et al.*, 1998).

Vietnam veterans with a lifetime history of PTSD may be more vulnerable to the impact of comorbid disorders on cognitive functioning.

In an effort to examine this more closely, Barrett *et al.* (1996) conducted a large study of Vietnam veterans with and without PTSD and comorbid disorders on measures of memory, learning, executive function and IQ. They compared four groups: veterans with PTSD only, veterans with only a current diagnosis of major depression, generalised anxiety disorder, and/or alcohol-substance abuse, veterans with PTSD and one or more of these diagnoses, and veterans without any psychiatric diagnoses.

The veterans with both PTSD and a current comorbid diagnosis performed more poorly on all measures of cognitive functioning than the other groups. The authors concluded that the cognitive deficits seen among Vietnam veterans with PTSD may be associated with their concomitant diagnoses.

However, the study has a number of limitations which make it difficult to draw firm conclusions. The comorbid disorders of anxiety, depression and alcohol-substance use were all grouped together and referred to as 'other diagnosis.' It is therefore difficult to evaluate the relative contributions of each disorder on cognitive functioning. This is further complicated by the fact that the exact rates of disorder in the comorbidity group are not reported. There may have been more subjects with PTSD and alcohol abuse than subjects with PTSD and generalised anxiety disorder (GAD), for example, and the findings could reflect this. Further, there is no information on severity or length of onset of each of the comorbid disorders. This

clouds data interpretation. Some veterans may have had lifetime alcohol use, and others may just meet the study's criteria of one month of alcohol use, for example. Further, the PTSD groups may differ in their range of PTSD symptoms as well as their severity. The PTSD-only group may have fewer symptoms that are less severe. Sutker, Vasterling, Brailey & Allain (1995) found that cognitive deficits were correlated with PTSD symptom severity. The PTSD-only group in this study may represent a subgroup of the PTSD diagnosis which is associated with low severity and intact cognitive functioning. Finally, the groups differed on the baseline measure of cognitive functioning (shortened version of the WAIS-R[4]). The group with PTSD and comorbid disorders had significantly lower baseline IQ scores than the other groups, suggesting that the observed differences in memory and learning may be due to differences in baseline cognitive ability.

Despite the limitations, the study highlights the importance of considering the influence of comorbid diagnoses on cognitive functioning in PTSD. Specifically, the study suggests the likelihood of poor cognitive functioning among Vietnam veterans with PTSD and a comorbid diagnosis of depression, anxiety or alcohol-substance use.

1.2.2.5.2 Severity of PTSD symptomatology, particularly re-experiencing symptoms

Few studies have looked at severity of PTSD symptomatology and memory performance. The data to date suggest that symptom severity is positively correlated with memory deficit (e.g. Sutker *et al.*, 1995) and that there is an identifiable

[4] WAIS-R refers to the Wechsler Adult Intelligence Scale – Revised (Wechsler, 1981).

relationship between the severity and pattern of PTSD symptomatology and memory impairment (Vasterling *et al.*, 1998).

Vasterling *et al.* (1998) found that on measures of memory and learning, errors of self-monitoring were associated with PTSD symptom status. These types of errors refer to 'intrusions'. That is, recalling a word that has not been presented in the test trial. They may reflect difficulty with the executive function behaviour of self-monitoring, as well as difficulty in the memory processes of encoding and retrieval. Vasterling *et al.* (1998) found that errors of self-monitoring were positively associated with severity of re-experiencing phenomena, and inversely associated with avoidance-emotional numbing phenomena. They showed no significant relationship to hyperarousal symptoms. Thus, subjects with severe re-experiencing symptoms were more likely to show memory performance marked by errors of self-monitoring. It is unclear whether these subjects also performed significantly worse on memory measures overall when compared to subjects with less severe symptoms.

1.2.2.5.3 Reduced Volume of the Hippocampus

Current research suggests that the hippocampus is reduced in size in individuals with PTSD and that there are neuroendocrine alterations that may account for this.

(i) The Hippocampus

The hippocampus is a structure in the medial temporal lobe of the brain that is crucial in new learning and memory consolidation (Gluck & Myers, 1997; Graham & Hodges, 1997; Squire & Alvarez, 1995). Episodic memory is dependent on the hippocampus, whereas declarative memory depends on the perihippocampal cortical

regions (Graham & Hodges, 1997; Vargha-Khadem, Gadian, Watkins, Connelly, Van Paesschen & Mishkin, 1997; Tulving & Markowitsch, 1998).

There are two hippocampal complexes, one in the left hemisphere and one in the right. Lesion studies suggest that they are specialised for different types of knowledge. Some studies have found that the left-sided complex has an essential role in verbal memory (e.g. Trenerry, Jack, Ivnik, Sharbrough, Cascino, Hirschorn, Marsh, Kelly & Meyer, 1993; Baxendale, Cook, Shorvon, Thompson & Warrington, 1994), and that the right hippocampal complex has a role in learning and consolidation of visuospatial material (e.g. Baxendale *et al.*, 1994). When one of the hippocampal complexes is damaged, the capacity to acquire the aspect of knowledge which relies upon the damaged system is lost or relatively reduced (Tranel & Damasio, 1995).

(ii) Reduced Hippocampal Volumes in Adults with Chronic PTSD

A number of neuroimaging studies have found reduced hippocampal volumes in PTSD sufferers. Using magnetic resonance imaging (MRI), some studies have found reduced right hippocampal volume compared to controls (e.g. Bremner *et al.*, 1995); others have found reduced left hippocampal volume (e.g. Stein *et al.*, 1997; Bremner *et al.*, 1997). One study has found reduced volumes of both left and right hippocampi (Gurvitis *et al.*, 1996). In this study, hippocampal volume was directly correlated with combat exposure, suggesting that traumatic stress may damage the hippocampus. Alternatively, reduced hippocampal volumes may be a risk factor for developing PTSD upon exposure to traumatic stress. In studies of combat-related PTSD, reduced hippocampal volumes were associated with concurrent deficits in

learning and memory (i.e. Bremner *et al.*, 1995; Gurvitis *et al.*, 1996). This was not the case in studies of sexual abuse-related PTSD (i.e. Stein *et al.*, 1997; Bremner *et al.*, 1997).

Unfortunately, the studies have methodological limitations of small sample sizes and high incidence of comorbid diagnoses, particularly history of alcohol abuse. Nevertheless, they suggest that the hippocampus may play a role in the memory-related impairments in PTSD.

(iii) Neuroendocrine Alterations

The findings of low cortisol and increased glucocorticoid receptor numbers in subjects with PTSD suggest that the hypothalamic-pituitary-adrenal (HPA) may be differentially regulated in PTSD. The increased glucocorticoid receptors may be the mechanism of hippocampal atrophy.

(iii.i) The Hypothalamic-Pituitary-Adrenal Axis

The hypothalamic-pituitary-adrenal (HPA) axis is one of the major biological systems involved in co-ordinating the body's response to stress (Yehuda, 1997). During stress, neuropeptides in the brain stimulate the release of corticotrophin-releasing factor (CRF) from the hypothalamus, which in turn initiate the release of adrenocorticotropic hormone (ACTH) from the pituitary and cortisol from the adrenal glands (Golier & Yehuda, 1998). The release of cortisol suppresses the release of ACTH and CRF which in turn reduces the release of cortisol, a process called negative feedback inhibition.

Several studies have demonstrated lower urinary cortisol levels in trauma survivors with PTSD compared to controls (e.g., Mason, Giller, Kosten, Ostroff & Podd, 1986; Yehuda, Southwick, Nussbaum, Wahby, Mason & Giller, 1990; Yehuda, Boisoneau, Mason & Giller, 1993; Yehuda, Kahana, Binder-Brynes, Southwick, Mason & Giller, 1995) lower plasma cortisol levels (e.g., Boscarino, 1996; Yehuda, Teicher, Trestman, Levengood & Siever, 1996), as well as lower salivary cortisol levels (Goenjian, Yehuda, Pynoos, Steinberg, Tashjian, Yang, Najarian & Fairbanks, 1996).

(iii.ii) Glucocorticoid Receptors

Glucocorticoid receptors are found in most neurons and glial cells in the brain. The largest density of receptors is in the CA3 region of the hippocampus (Keenan & Kuhn, 1999). The physiological and behavioural effects of cortisol depend on the ability of cortisol to bind to glucocorticoid receptors (Svec, 1985). Alterations in the sensitivity of glucocorticoid receptors can influence the functioning of the HPA axis. Studies of both combat veterans and adult survivors of childhood sexual abuse have found that the number of basal glucocorticoid receptors is larger in PTSD than in non-traumatised subjects without psychiatric disorders (Yehuda *et al.*, 1993; Yehuda, Boisoneau, Lowry & Giller, 1995; Yehuda, Lowry, Southwick, Shaffer & Giller, 1991).

(iv) Putting it all together: Mechanism of hippocampal atrophy

Studies of cortisol suppression and pituitary activity suggest that the neuro-endocrine profile of PTSD best fits a model of enhanced negative feedback in which the primary deficit is an increased responsiveness of glucocorticoid receptors at several sites along the HPA axis (Golier & Yehuda, 1998). At the time of the trauma, stress

leads to release of catecholamines which increases the release of ACTH and hence glucocorticoids. High levels of catecholamines are associated with deleterious effects on memory (Golier & Yehuda, 1998). They may interfere with memory consolidation at the time of the trauma. Following trauma, the low levels of cortisol and the increased glucocorticoid receptor numbers associated with increased glucocorticoid receptor sensitivity may render the hippocampus more vulnerable to atrophy (Golier & Yehuda, 1998). This may lead to the more general memory impairments seen in adults with PTSD.

(v) The functional effect of atrophy

Atrophy of one or both of the hippocampi may be associated with the observed memory impairments in PTSD. The effect of atrophy may have a wider influence on the duration of the illness. It is widely accepted that the hippocampus is crucial in memory consolidation. A number of models suggest that the hippocampus acts as a temporary store or link for new memories before they are integrated into neocortical networks (Alvarez & Squire, 1994; McClelland, McNaughton & O'Reilly, 1995; Murre, 1997). Hippocampal atrophy may be associated with incomplete memory consolidation of the trauma. The aim of many psychological treatments is to consolidate the fragmented trauma memory (Foa & Kozaks, 1986), and factors associated with consolidation have been linked to successful treatment (e.g. Foa *et al.*, 1995).

Inability to consolidate the trauma memory may prolong the course of PTSD. Neuroendocrine alterations that affect the HPA axis and ultimately reduce the size of the hippocampus may affect hippocampal function. Further, emotional activation of

the amygdala interferes with hippocampal functioning (Van der Kolk, Burbridge & Suzuki, 1997). These factors may prevent the hippocampus from fully consolidating the trauma memory. Research suggests that a memory that has been consolidated is stored in the neocortex (Gluck & Myers, 1997; Graham & Hodges, 1997). In PTSD, it is possible that the trauma memory is under-consolidated and re-presented in the form of intrusions, in a continual attempt to consolidate it and place it within the long-term memory store. It is possible that intrusions represent a failure of the hippocampal complex to consolidate the memory and transfer it to the neocortex.

1.2.2.5.4 Poor premorbid memory status

The observed memory impairments in PTSD may reflect poor memory functioning prior to the onset of trauma. In order to rule this out, prospective studies are needed. Obviously it is hugely difficult to conduct this sort of study. One can draw on clinical reports in order to provide some elucidation on this matter. Clients with PTSD consistently report that their memory problems post-date their traumatic stress exposure. This raises the issue of whether memory problems are related to traumatic stress exposure or PTSD. Some studies (e.g. Jenkins *et al.*, 1996) have compared PTSD samples to trauma exposed non-PTSD samples and have found differences in memory performance, suggesting that it is not traumatic stress exposure that is related to memory difficulties, but PTSD.

1.2.2.5.5 Global impairment

The observed memory impairments in PTSD may reflect overall poor cognitive ability. Most studies of memory functioning have not assessed cognitive ability, and few have looked at pre-morbid ability. However, those that have (e.g. Barrett *et al.*,

1996; Vasterling *et al.*, 1998) found significant differences between the PTSD groups and the controls. Barrett *et al.* (1996) found lower scores on the shortened version of the WAIS-R, and Vasterling *et al.* (1998) found significantly lower scores on the Vocabulary subtest, which was their shortened version of the WAIS-R. However, Beckham *et al.* (1998) found that although the PTSD and non-PTSD groups differed significantly on age and level of education, significant memory differences remained even when these were controlled for.

1.2.2.5.6 Disrupted system of arousal

Vasterling *et al.* (1998) put forward the idea that the observed memory difficulties in PTSD represent a disrupted arousal system associated with frontal-subcortical system dysfunction. They suggest that PTSD-related memory difficulties closely resemble those typically associated with frontal system dysfunction, that is inefficient acquisition and errors of intrusion. Arousal dysregulation disrupts attention and in particular, vigilance (Mirsky *et al.*, 1991; Robbins & Everitt, 1996) and possibly explains some of the observed memory difficulties. However, the observed memory difficulties found generally in PTSD subjects are unlikely to be fully explained by a disrupted arousal system. Vasterling *et al.* (1998) posited that if frontal system functioning is disrupted in PTSD, then the capacity of the frontal lobes to inhibit unwanted information or situation-inappropriate information may be reduced leading to difficulties learning and remembering information. Although the authors stress the involvement of the frontal lobes over the medial temporal lobes in memory-related impairments in PTSD, it is possible that the two interact. That is, a disrupted arousal system could interfere with the functioning of the medial temporal lobes in the process of memory consolidation. Studies which look specifically at PTSD

symptomatology and memory functioning will further inform this area. Vasterling *et al.* (1998) looked at PTSD symptomatology and measures of learning and executive function. They found that high levels of intrusions and low levels of avoidance were associated with poor functioning. Studies are needed to look at symptomatology and specific measures of explicit memory to determine if PTSD sufferers with high levels of avoidance and low levels of intrusion also exhibit memory difficulties.

1.2.2.6 Neuropsychological Findings: Concluding Comment

Research suggests that PTSD sufferers experience difficulties in learning and memory and related processes. The previous section put forth various cognitive and physiological mechanisms that may lead to impairment. These are not mutually exclusive, but likely interact to influence memory functioning. For example, cognitive intrusions may limit the processing resources available for optimum memory functioning. Neuroendocrine changes may aggravate memory performance because of their influence on memory-related structures in the brain and further compromise the use of available resources. Difficulties associated with poor memory function may contribute to the onset of comorbid disorders which further aggravate memory ability.

The research to date has shown that the memory-related processes of attention, information processing and executive function are compromised in PTSD and these likely contribute to the observed memory difficulties. However, the studies have extensively assessed one process over the others, and determining the relative contribution of each has not been possible. Further research is needed to address this issue, and also to consider the range of PTSD symptoms and how they relate to the

observed impairments. Doing so will likely shed light on both the etiology of memory impairment in PTSD, and potentially factors related to poor outcome. Thus, an outcome study of PTSD is needed that looks at pre-treatment neuropsychological functioning. The proposed study aims to do this.

1.3 RATIONALE

Neuropsychological research has shown that adults with PTSD exhibit impairment in memory and related processes. Research has also shown poor outcome following intervention for PTSD. It is possible that impaired learning and memory processes adversely affect the capability of clients with PTSD to respond to treatment. Cognitive-behavioural therapy, in particular, exposure therapies and cognitive restructuring, require intact verbal memory processes, such as an ability to articulate memories, and an ability to register and recall relevant CBT material. It is possible that verbal memory difficulties will adversely affect treatment outcome.

To date, no study has examined the impact of poor memory functioning on treatment outcome in adults with PTSD.

This study aims to evaluate the contribution of cognitive and memory functioning to therapeutic outcome in adults with PTSD. This is a clinically relevant study as it may help to clarify factors associated with poor outcome in PTSD, allowing better prediction of the course of the disorder and possible improvement in treatment strategies. The study will also afford the opportunity to look specifically at PTSD symptom profiles and memory function, thus identifying symptoms associated with poor memory. Finally, this study will assess the range of processes affecting

memory function and will shed light on their contribution to memory dysfunction in PTSD.

1.3.1 Model: The Course of PTSD and Treatment Outcome

Figure 1.2 presents a model that summarises the path to recovery versus the onset of PTSD following traumatic stress exposure. Factors associated with recovery include low level of dissociation at the time of the trauma, personality factors and social support (discussed in Section 1.1.6). Factors associated with the onset of PTSD include peri-traumatic dissociation, personality factors, low social support, comorbid disorders, and low cortisol (discussed in Section 1.6). Poor memory functioning may be an additional mediating factor. Factors associated with ongoing and chronic PTSD include comorbid disorders, possible neuronal changes and hippocampal atrophy, and the mediating factor to be considered in this study, poor learning and memory processes. Specifically, this study focuses on the shaded area, the treatment non-responders, and the contribution of learning and memory functioning to treatment outcome.

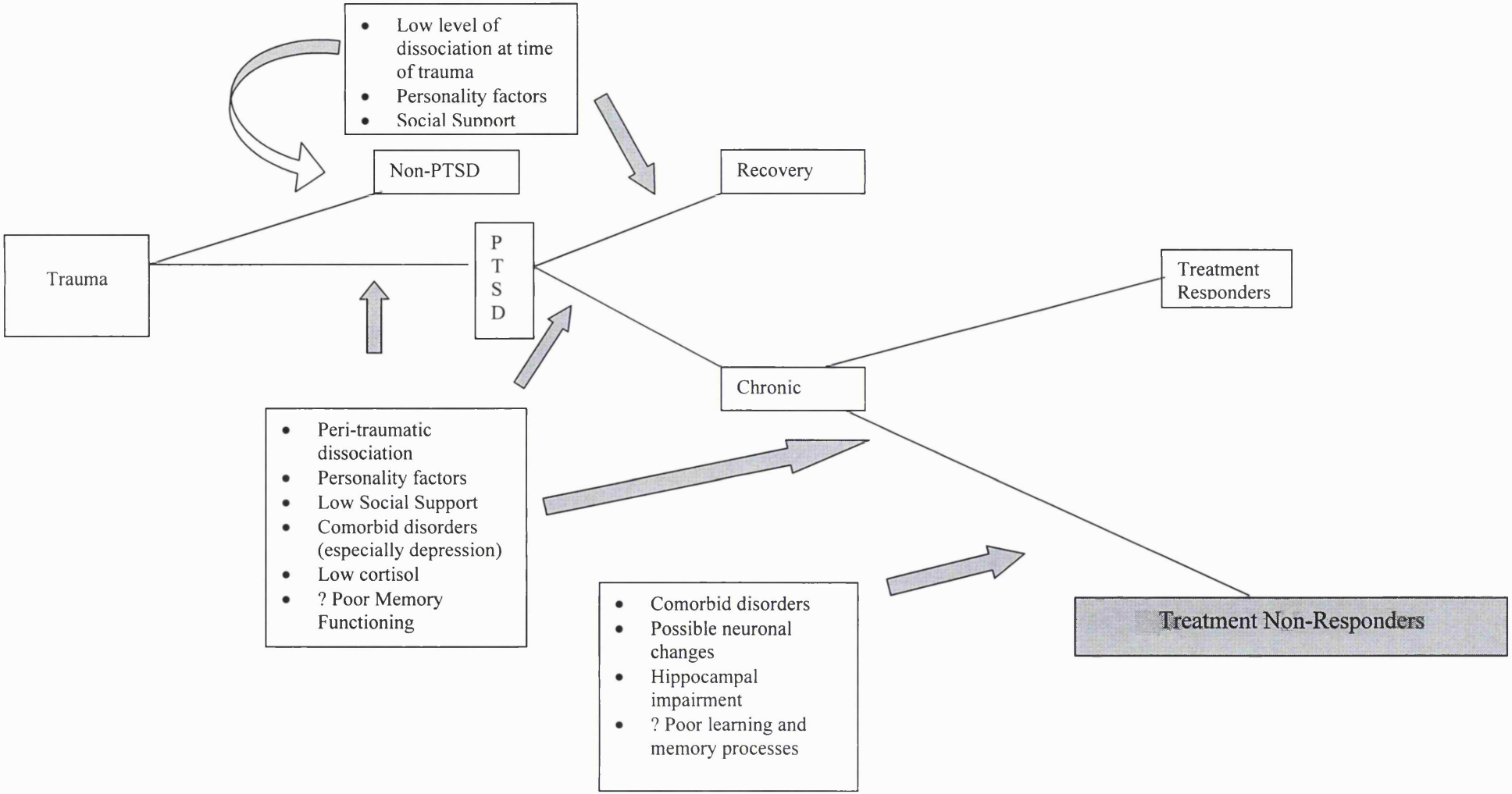

Figure 1.2 Model showing the course of PTSD and highlighting the area to be researched.

1.3.2 RESEARCH QUESTIONS

1. Is severity of PTSD symptomatology related to memory functioning?

2. Is time since trauma related to memory functioning?

3. Are there differences in performance on the information processing task between those who improve with treatment and those who do not?

4. Are intake anxiety and depression scores related to memory functioning (on the AMIPB)? Are they related to specificity of memory (on the AMT)?

5. Is alcohol use measured at intake related to memory functioning?

1.3.3 RESEARCH HYPOTHESES

1. Participants with a DSM-IV diagnosis of PTSD at follow-up will have poorer memory performance compared to participants who do not meet a DSM-IV diagnosis at follow-up.

2. Verbal memory performance will predict outcome.

3. Performance on tests of executive function and attention will not account for differences in memory performance.

4. Based on the research on a disrupted arousal system, intrusions on the verbal learning task will be positively correlated with intrusive symptomatology.

5. Participants with a DSM-IV diagnosis at follow-up will show less specific memories compared to individuals without a diagnosis of PTSD at follow-up.

6. Self-report scores of previous week avoidance and previous week cognitive avoidance will be negatively correlated with specificity memory scores (based on Kuyken & Brewin, 1995).

2. METHOD

2.1 SAMPLE

2.1.1 Recruitment

Subjects were recruited primarily from the Traumatic Stress Clinic, a specialist PTSD clinic within Camden and Islington Community Health Services NHS Trust. In order to increase participant numbers, two further clinical psychology departments within the same trust were approached (Hunter Street Health Centre and the Clinical Psychology Department of the Whittington Hospital). The Local Research Ethics Committee of the Camden and Islington Community Health Services NHS Trust granted ethical permission. Appendix 2 shows a copy of the letter of ethical permission. Appendix 3 shows copies of the information sheet and consent form.

2.1.2 Participants

Participants who met DSM-IV criteria for PTSD on assessment were invited to take part in the research. Participants were excluded if: (1) their first language was not English, (2) if they were considered to have a DSM-IV Axis II disorder, (3) if they had sustained a head injury resulting in post-traumatic amnesia, and/or (4) if they had sustained a head injury resulting in loss of consciousness. Over a period of 12 months, 27 participants were identified and agreed to participate. They formed the intake sample.

Psychological and memory data were obtained for this sample prior to CBT treatment. Four participants dropped out of treatment shortly after assessment. Follow-up data were obtained for 23 participants.

All participants met full DSM-IV criteria for PTSD. One participant had received previous help for non-trauma related psychological difficulties (20 years previously for depression). There were 13 women and 14 men with an average age of 33.87 years (SD = 8.49 years, range 20.08-50.00 years). Five participants had university qualifications, two had college qualifications, seven had completed 'A'-levels, 12 had completed GCSEs or equivalent, and one had no paper qualifications, leaving school at the age of 12 years. Fifteen subjects were employed (three had senior management positions, four had middle management positions, four had clerical positions, and four had manual work positions). Three participants were students; four were on sick leave, and five were unemployed. The demographic characteristics of the sample are presented in Table 3.3 in Chapter 3.

2.2 DESIGN

The study was a within-subjects repeated measures design. Suitable participants were identified during routine clinical assessments. The prospective participants were shown information sheets about the research (see Appendix 3) and invited to attend an appointment for memory assessment before they began CBT therapy. They were all asked to give written informed consent (see Appendix 3 for copies of blank consent forms). Subjects completed outcome measures when they were discharged from therapy or at session eight, which ever came first (this is explained in more detail below in Section 2.4.1 'General Research Procedure').

2.3 MEASURES

2.3.1 Symptom Measures

Six questionnaire measures were selected to assess symptoms of PTSD, depression and anxiety, and alcohol and drug use. Three questionnaires assessed symptoms of PTSD. They were selected because they have good psychometric properties and they were already administered as part of the routine clinical assessment procedure at the Traumatic Stress Clinic. One questionnaire was selected to assess alcohol and drug use. This was selected because it was also part of the routine clinical assessment procedure. Two questionnaires assessed severity of anxiety and depression. They were not part of the clinical assessment procedure and were selected because of their good psychometric properties. These questionnaires are discussed below.

2.3.1.1 Impact of Event Scale-Revised (IES-R) (Weiss & Marmar, 1997)

The IES-R is a revised version of the Impact of Event Scale (IES; Horowitz, Wilner, & Alvarez, 1979). The IES is one of the most widely used self-report measures of trauma-related intrusion and avoidance symptoms. It is based on Horowitz's (1976) theory of post-traumatic stress reactions and provides a continuous score for the intrusion and avoidance scales. The IES-R differs from the IES in that it also measures the hyperarousal cluster of PTSD symptoms.

The IES-R is a 22-item questionnaire which takes approximately five minutes to complete. Each item is a statement which corresponds to a symptom of PTSD. The client notes the frequency of occurrence in the last seven days on a 4-point scale: 0=not at all, 1=rarely, 3=sometimes, and 5=often. Fifteen items are drawn from the

IES and measure symptoms of intrusion and avoidance. There are seven additional items, six of which measure hyperarousal symptoms, and one which measures an additional intrusive symptom to parallel the DSM-IV criteria. Weiss & Marmar (1997) reported good internal consistency for all three scales. The Intrusion scale alphas ranged from .87 to .91; the Avoidance scale alphas ranged from .84 to .86, and the Hyperarousal scale alphas ranged from .79 to .90.

2.3.1.2 The Self-Rating Scale for PTSD (SRS-PTSD) (Carlier, Lamberts, Van Uchelen & Gersons, 1997)

The SRS-PTSD is a self-report and abridged version of the Structured Interview for PTSD (SI-PTSD; Davidson, Smith & Kudler, 1989). The SI-PTSD records the presence and severity of DSM-III-R criteria for PTSD and has shown good internal consistency for the separate questionnaire items (Cronbach's alpha=0.93) as well as the PTSD symptom clusters (α=.79 for re-experiencing, .85 for avoidance, .87 for hyperarousal) (Carlier *et al.*, 1997).

The SRS-PTSD is a much shorter instrument than the SI-PTSD with good psychometric properties. It takes approximately 10 minutes to complete. It consists of 17 items in the form of statements that correspond to the 17 DSM-III-R (APA, 1987) diagnostic criteria for PTSD.[1] The client must record the frequency of occurrence in the past four weeks on a 3-point Likert scale: 0=not at all, 1=a little bit/once/less than four times a week, 2=very much/almost constantly, four or more

[1] The DSM-III-R and the DSM-IV criteria of the clinical features of PTSD are similar. The one main difference is that DSM-III-R groups one criterion (i.e. physiological reactivity on exposure to events that are similar to the traumatic event) under the hyperarousal grouping, whereas DSM-IV has modified this criterion and placed it within the re-experiencing grouping. The modified criterion includes physiological reactivity to either internal or external triggers.

times.

The SRS-PTSD shows good internal consistency for the 17 separate items (Cronbach's alpha=0.96) as well as for each of the symptom clusters (alpha=.88 for re-experiencing, .88 for avoidance, .93 for hyperarousal). The SRS-PTSD also demonstrates good inter-rater reliability (Cohen's κ=.98) (Carlier *et al.*, 1997). Further, the SRS-PTSD demonstrates good sensitivity (86%) and specificity (80%) (Carlier *et al.*, 1997), suggesting that it discriminates between respondents with and without a diagnosis of PTSD.

The SRS-PTSD provides a score for each of the three symptom clusters of PTSD. This score reflects the number of symptoms that meet the DSM-III-R criteria per cluster. It can be used as a diagnostic instrument.

2.3.1.3 The Clinician Administered Posttraumatic Stress Disorder Scale (CAPS) (Blake, Weathers, Nagy, Kaloupek, Gusman, Charney & Keane, 1990)

The CAPS is a structured interview schedule which assesses the frequency and intensity of core and associated symptoms of lifetime and current PTSD. It consists of 23 questions and requires approximately 45 to 60 minutes to administer. The first seventeen questions parallel the DSM-IV criteria for PTSD, and the final five questions assess associated symptoms of guilt and dissociation. Often only the first 17 questions are administered. The CAPS shows good sensitivity (84%) and specificity (95%) (Blake *et al.*, 1996). The CAPS shows good internal consistency (Cronbach's α = .94) and satisfactory inter-rater reliability (Cohen's κ=.78) (Blake *et al.*, 1995). Test-retest reliability ranged from .90 to .98 (Blake *et al.*, 1995).

The CAPS provides a number of scores, five of which were used in this study. They were: DSM-IV Diagnosis of PTSD (Yes/No), Severity of Re-experiencing Symptoms, Severity of Avoidance Symptoms, Severity of Hyperarousal Symptoms, and Subjective Distress.

2.3.1.4 The Clinician Administered Posttraumatic Stress Disorder Scale – Self-report Version (Unpublished)

Because the CAPS (Blake *et al.*, 1990) is time-consuming, it was not always possible for the clinician to re-administer it at the interval required for the purposes of this study. For this reason, a self-report version of the CAPS was constructed. This is very similar to the interview version of the CAPS and requires approximately 10 to 15 minutes to complete. It consists of the same 23 questions presented in non-technical language. When it was not possible for the clinician to re-administer the CAPS, the participants were given the self-report version. Appendix 4 shows a blank copy of this questionnaire. Psychometric properties of the scale are reported in Section 3.1.2 of Chapter 3.

2..3.1.5 The Alcohol and Drugs Questionnaire (ADQ) (Unpublished)

The staff at the Traumatic Stress Clinic developed this self-report questionnaire in 1995 to assess clients' current use of alcohol and substances. The ADQ provides a weekly measure of alcohol intake in units. The ADQ also assesses whether alcohol and drug related behaviour has changed since the trauma. The ADQ requires approximately five minutes to complete. Appendix 5 shows a blank copy of the ADQ.

2.3.1.6 The Beck Anxiety Inventory (BAI) (Beck, 1990)

The BAI measures the severity of self-reported anxiety. It consists of 21 descriptive statements of anxiety symptoms which the client may have experienced in the past week. They are rated on a 4-point Likert scale: 0=Not at all, 1=Mildly, 2=Moderately, 3=Severely. It takes approximately five minutes to complete.

The BAI has shown good internal consistency (Cronbach's alpha=.94) in studies of anxiety disorder samples (Fydrich, Dowdall & Chambless, 1990) as well as more mixed samples which included subjects with anxiety disorders, and subjects without diagnosed anxiety disorders, but with academic problems or adjustment disorders (Cronbach's alpha=.92) (Beck, Epstein, Brown & Steer, 1988). The BAI shows good test-retest reliability (r=.75) (Beck *et al.*, 1979).

The BAI provides an overall score out of 63. A score that falls under 10 suggests minimal or no anxiety. A score that falls between 10-18 suggests mild to moderate anxiety. A score that falls between 19-29 suggests moderate to severe anxiety. A score that falls between 30-63 suggests severe anxiety.

2.3.1.7 The Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock & Erbaugh, 1961)

The BDI measures the severity of self-reported symptoms of depression. It consists of 21 descriptive statements which the client may have experienced in the last seven days. They are rated on a 4-point scale ranging from 0 to 3 in terms of severity. The BDI requires approximately 5 to 10 minutes to complete.

Reliability studies (Beck, Steer & Garbin, 1988) have shown a high degree of internal consistency.

The BDI provides an overall score out of 63. A score that falls under 10 suggests minimal or no depression. A score that falls between 10-18 suggests mild to moderate depression. A score that falls between 19-29 suggests moderate to severe depression. A score that falls between 30-63 suggests severe depression.

2.3.2 Neuropsychological Measures

2.3.2.1 The Wechsler Adult Intelligence Scale – Revised UK (Shortened Version) (WAIS-R) (Wechsler, 1981)

In order to assess general level of cognitive ability, a shortened version of the WAIS-R was administered. The WAIS-R is one of the most widely accepted and employed measures of cognitive function in adults. A UK version is available, although it has not yet been standardised on a UK population. Therefore the scores obtained on the UK version are age-scaled according to the American sample. Verbal, Performance and Full-Scale IQs are calculated based on the American norms.

The WAIS-R consists of 11 subtests (6 verbal and 5 performance). There are a number of shortened versions of the WAIS-R which yield satisfactory regression coefficients with Verbal, Performance and Full-Scale IQs.

For the purpose of this study, six subtests (4 Verbal and 2 Performance) were selected to provide a baseline measure of cognitive function. This shortened version required one hour to administer.

For Verbal IQ, Canavan, Dunn & McMillan (1986) recommended a short-form consisting of Vocabulary, Comprehension and Similarities. In addition to these subtests, the Digit Span subtest was also selected. It has been widely used as a measure of auditory working memory and was selected for this purpose. Therefore, in this study, the following subtests were selected from the Verbal Scale: Vocabulary, Comprehension, Similarities, and Digit Span.

Reynolds, Wilson & Clark (1983) recommended the use of Block Design and Picture Completion as measures of the Performance scale. These were the two performance subtests administered in this study. Together they have good predictive value of Performance IQ (r=.90) (Crawford, Allan & Jack, 1992).

2.3.2.1.i The Verbal Subtests

Vocabulary

The Vocabulary subtest of the WAIS-R consists of 35 words arranged in increasing order of difficulty. The subject is asked to define each word. The list begins with familiar words, such as 'bed' and 'winter' and concludes with infrequent words, such as 'audacious' and 'tirade.' The subtest continues until the subject fails five consecutive words or completes the list.

The Vocabulary subtest can also be used as a measure of pre-morbid intelligence (Lezak, 1995) and as a single ability measure (Feingold, 1982). The Vocabulary subtest alone has good predictive value of Verbal IQ (r=.84) (Crawford *et al.*, 1982).

Digit Span

Digit Span is a widely used test of immediate verbal memory. The subtest is presented orally. It also reflects the efficacy of phonological and attentional processes (Hodges, 1994).

The Digit Span subtest comprises two different tests: Digits Forward, and Digits Backward. Both tests consist of seven pairs of random number sequences that the examiner reads aloud at the rate of one per second. For Digits Forward, the subject must recall the numbers exactly as the examiner has presented them. For Digits Backward, the subject must recall the numbers in the reverse order to which they were presented.

Comprehension

This subtest includes two kinds of open-ended questions: 11 test common-sense judgement and practical reasoning and the other three ask for the meaning of proverbs. The questions are presented orally to the subject in increasing order of difficulty.

Similarities

This is a test of verbal concept formation and an excellent test of general mental ability (Lezak, 1995). Fourteen word pairs are presented orally to the subject. The

subject must explain what each of a pair of words has in common. The word pairs range in difficulty from the simplest ('orange-banana') to the most difficult ('praise-punishment'). The test concludes after four consecutive failures, or when the subject reaches the end of the list.

2.3.2.1.ii The Performance Subtests

Two performance subtests were selected: Picture Completion and Block Design.

Picture Completion

This subtest measures the ability to reason about visually presented material. The subject is shown 20 incomplete pictures of human features, familiar objects or scenes arranged in order of difficulty with instructions to tell what important part is missing. Twenty seconds are allowed for each response. The pictures are presented in increasing order of difficulty.

Block Design

This subtest involves visual-spatial-motor integration. It is a construction test in which the subject is presented with red and white blocks, four or nine, depending on the item. Each block has two white and two red sides, and two half-red half-white sides with the colours divided along the diagonal. The subject is required to use the blocks to construct replicas of two block designs made by the examiner and seven designs printed in smaller scale. This is a timed subtest and the subject earns higher scores for completing the design within specific time-bands.

The Block Design subtest alone has acceptable predictive value of Performance IQ (r=.76) (Crawford *et al.*, 1992).

2.3.2.1.iii Obtaining the Verbal, Performance and Full-Scale IQs

The raw scores were converted to standard scaled scores for each subtest. For Verbal IQ, the mean of the four Verbal subtests was calculated to provide an estimate of the possible scaled scores of the two subtests not administered (Information and Arithmetic). The obtained and estimated scores were then summed to obtain a standard scaled score of the Verbal Scale. The Table of IQ Equivalents of Sums of Scaled Scores by Age Group (Wechsler, 1981) was consulted to obtain the Verbal IQ score adjusted for age.

For Performance IQ, an average of the two administered Performance subtests was calculated to represent the possible score obtained on the subtests which were not administered (Digit Symbol, Picture Arrangement and Object Assembly). The obtained scores and estimated scores were then summed to obtain the prorated Performance Scale score. The Table of IQ Equivalents (Performance Scale) of Sums of Scaled Scores by Age Group (Wechsler, 1981) was consulted to obtain the Performance IQ score adjusted for age.

In order to obtain Full-Scale IQ scores, the standard scaled scores for the Performance and Verbal scales were summed. The Table of IQ Equivalents of Sums of Scaled Scores by Age Group (Wechsler, 1981) was consulted to obtain the Full-Scale IQ score adjusted for age.

2.3.2.2 The Adult Memory and Information Processing Battery (AMIPB) (Coughlan & Hollows, 1985)

The AMIPB is a memory battery which tests verbal and non-verbal memory and learning. There are eight subtests in total, six of which test memory and learning and two which look at numerical information processing. The AMIPB was developed and standardised using a British population. It takes approximately 45 minutes to administer.

2.3.2.2.i Verbal Memory and Learning

There are two subtests which tap verbal memory and learning. They are Story Recall and List Learning.

Story Recall

In the Story Recall task, the subject is read a passage and asked to recall as many ideas as possible from a maximum of 60, both immediately after the presentation and 30 minutes later. This yields three scores: Immediate Recall, Delayed Recall and Percentage Retained.

List Learning

In the List Learning task, the subject is read a list of 15 unrelated words and asked to recall as many of them as possible over five trials. The subject is then read a second list of unrelated words and asked to recall as many as possible. Following this, the subject is required to recall as many words from the first list as possible. This yields four scores: A1-A5 (total number of correct words for trials A1-A5), B (number of

correct words on List B), A6 (number of correct words recalled after distraction) and Intrusions (number of incorrect words summed over all the trials).

2.3.2.2.ii Non-verbal Memory and Learning

There are two subtests which tap non-verbal memory and learning. They are Figure Recall and Design Learning.

Figure Recall

The Figure Recall task requires the subject to copy a complex figure. After doing so, the original is taken away, and the subject must copy it from memory immediately and after 30 minutes. This subtest yields 4 scores: Copy, Immediate Recall, Delayed Recall, and Percent Retained.

Design Learning

In this task, the subject is presented with an abstract design on a dot grid. The design is removed after 10 seconds, and the subject must reproduce it on a dot grid. This is continued for up to five trials or until the subject recalls the design correctly for two consecutive trials. The subject is then shown another abstract design and is required to copy it after the presentation period of 10 seconds. Following this distraction period, the subject is required to reproduce the original abstract design. This subtest yields four scores: A1-A5 (total number of correct features for trials A1-A5), B (number of correct features on Design 2), A6 (number of correct features recalled after distraction) and Intrusions (number of incorrect features summed over all the trials).

2.3.2.2.iii Information Processing

There are two non-verbal information processing tasks: Task A and Task B. Both require the subject to work through a list of numerical items for a period of four minutes. Both are similar in terms of level of difficulty. For the purposes of this study, Task A was used as the measure of information processing. This task is described below.

Task A

In this task, the subject is presented with 105 rows of 7 two-digit numbers. The rows are grouped in blocks of five. The subject must delete (i.e., cross out) the second highest number in each row. After a demonstration and practice exercise, the subject is given four minutes to delete as many as possible. Following this, the subject is presented with three columns of 30 numbers each. The numbers are all the same (i.e., all the number '11'). The subject must cross out as many as possible in 20 seconds. This is a test of motor speed and the score on this task is used to adjust the score on the information processing task. This subtest yields four scores: Total (total number completed), Errors (percentage of errors calculated from the total number completed), Motor Speed (total number of elevens crossed out), and Adjusted Score (information processing score after adjusting for motor speed).

The raw scores obtained on each of the AMIPB subtests are adjusted for the subject's age. Percentile ranges based on normative data are available. The AMIPB does not compute memory quotients. For the purposes of this study, seven scores were selected from the AMIPB as measures of verbal and non-verbal memory, and one

score was selected as a measure of information processing. They are summarised in Table 2.1 below:

TABLE 2.1 AMIPB Scores Selected For Analysis

Verbal Memory	
• Story Recall:	(1) Immediate Recall (2) Percent Retained
• List Learning:	(1) Total A1-A5
	(2) List Learning Intrusions
Non-Verbal Memory	
• Figure Recall:	(1) Immediate Recall (2) Percent Retained
• Design Learning:	(1) Total A1-A5
Information Processing	
• Task A:	(1) Adjusted Score

2.3.2.3 The Test of Everyday Attention (TEA) (Robertson, Ward, Ridgeway & Nimmo-Smith, 1994)

The TEA is the first non-computerised test of attention to provide norm-referenced scores of subtypes of attention. It consists of eight subtests. Two subtests each measure selective attention, sustained attention, attentional switching and auditory-verbal working memory. The standardisation sample consisted of 154 normal volunteers. Additionally, data on a sample of 74 unilateral stroke patients were obtained.

For the purposes of this study, three subtests were chosen to look at attention and executive function. They were: Map Search, Visual Elevator and Lottery. They measure selective attention, attentional switching and sustained attention respectively. They were selected to reflect the Focus-Execute, Shift, and Sustain components of Mirsky *et al.*'s (1991) model of attention. Visual elevator is also thought to measure executive behaviour (Robertson *et al.*, 1994). This three-subtest version of the TEA required 30 minutes to administer.

Map Search

In this TEA subtest, subjects have to search for symbols, such as a knife and fork, on a colour map of the Philadelphia area for two minutes. Subjects are stopped after one minute, and are given a different coloured pen to continue the task. There are two scores: the number out of 80 found in 1 minute, and the number out of 80 found in 2 minutes. This subtest is age-sensitive. It measures selective attention. Raw scores were converted to scaled scores according to normative data. Percentile ranges were available per age group. Poor scores on this subtest reflect a difficulty ignoring irrelevant information and picking out targets in complex visual arrays.

Visual Elevator

In this TEA subtest, subjects have to count up and down as they follow a series of visually presented 'floors' in the elevator. This reversal task is a measure of attentional switching and therefore taps the executive function of cognitive flexibility. It is self-paced and the number correct score loads on the same factor as the number of categories on the Wisconsin Card Sorting Test (WCST; Berg, 1948).

There is also a time per switch measure derived from this subtest. It loads on the attentional switching factor (r=0.78) (Robertson *et al.*, 1994).

This subtests yields two scores, an accuracy score and a time per switch score. Both raw scores are converted to scaled scores according to normative data. Percentile ranges are available per age group.

Overall the Visual Elevator subtest measures the ability to change a train of thought. Those who do poorly on this test also tend to do poorly on the Wisconsin Card Sorting Test which is thought to measure frontal lobe/dysexecutive problems (Robertson *et al.*, 1994).

Lottery

In this TEA subtest, subjects must listen to a 10 minute series of audio-tape-presented numbers which consist of three letters, followed by two numbers, such as 'LB678.' Subjects have to listen for their winning number, which they are told ends in '55.' The task is to write down the two letters preceding all numbers ending in 55, of which there are 10. This subtest loads on the sustained attention factor (r=0.70).

The Lottery subtest yields an accuracy score out of 10 (the number of partially correctly identified letters). This score is converted to an age-graded scaled score. Age-graded percentile ranges are also available.

The Lottery subtest is sensitive to the ability to keep one's mind on a relatively unchanging, boring task. Subjects who do poorly on these tasks may 'drift off'

during therapy and forget what it is they are supposed to be practising (Robertson *et al.*, 1994).

2.3.2.4 The Autobiographical Memory Test (AMT) (Williams & Broadbent, 1986)

The AMT is a widely used method of assessing personal event memory in people with emotional disturbance. The AMT has many variations which differ according to the selection of cue words as well as the timing of response latencies. Most investigators use a version of the Williams & Broadbent (1986) task in which five positive and five negative words are presented in a fixed order, with positive and negative words alternating: *happy, sorry, safe, angry, interested, clumsy, successful, hurt, surprised, and lonely.* Subjects are given 60 seconds to retrieve a specific memory.

For the purposes of this study, subjects' responses were scored as specific or overgeneral according to Williams & Broadbent (1986). Specific responses obtained a score of one, and overgeneral responses obtained a score of zero. Thus, subjects received a score out of five for responses to positive cue-words (Positive Specificity Score), and a score out of five for responses to negative cue-words (Negative Specificity Score). These two scores were summed to compute an overall score out of 10 (Total Specificity Score).

The subjects were given the following instructions:

I am interested in your memory for events that have happened in your life. I am going to read to you some words. For each word, I want you to think of an event that happened to you which the word reminds you of. The event could have happened recently (yesterday, last week) or a long time ago. It might be an important event, or a trivial event.

Just one more thing: the memory you recall should be of a specific event. So if I said the word 'good' – it would not be okay to say 'I always enjoy a good party,' because that does not mention a specific event. But it would be okay to say 'I had a good time at Jane's party' (because that is a specific event).

Three words were given as practice. When it was clear that the subject understood the instructions, the 10 cue words were presented one at a time as in the instructions above. The AMT required 15 to 20 minutes to complete.

2.4 PROCEDURE

2.4.1 General Research Procedure

The research procedure incorporated the assessment process of the Traumatic Stress Clinic. Prior to attending clinical assessment at the Traumatic Stress Clinic, clients were sent a number of self-report questionnaires by post. Two of these questionnaires included the self-report questionnaires used in this study to assess symptoms of PTSD, and one was the questionnaire used to assess alcohol and drug use. Clients brought these completed questionnaires to their assessment session.

Assessment took place with either me or other clinical psychologists practising CBT for PTSD at the Traumatic Stress Clinic. The assessment sessions established whether the client suffered PTSD. At this time the CAPS was normally administered. Clients who met criteria for PTSD were informed of the research and invited to participate. The assessing clinician also screened for loss of consciousness and post-traumatic amnesia. In order to assess the possibility of loss of consciousness at the time of the trauma, clients were asked if: (1) they were admitted to hospital for head injury, (2) if they had a skull X-ray or CT scan, and (3) if they were required to return to hospital within one week post-discharge for head injury complaints. Participants were excluded if they answered in the affirmative to any of the above.

In order to assess for post-traumatic amnesia, clients were first asked if they had a clear memory for the whole event. They were then asked if there was any period following the trauma that they could not remember. If they answered in the affirmative to this particular question, the possibility of post-traumatic amnesia was considered and they were excluded.

Participants who agreed to take part in the research were either telephoned or sent a letter with an appointment time for memory assessment. Twenty-eight clients attending the Traumatic Stress Clinic for treatment agreed to participate. Two clients were excluded because they did not meet full DSM-IV criteria for PTSD. There were no clients who agreed to participate who were also considered to have experienced loss of consciousness or post-traumatic amnesia.

One participant was identified by a clinical psychologist working in the Clinical Psychology Department at the Whittington Hospital. For this client, PTSD assessment took place following the administration of the memory battery, both in the same session.

At the beginning of the memory assessment, the research was explained to participants. They were again given an information sheet and asked if they had any questions. Participants were then asked to sign a consent form.

2.4.1.1 Neuropsychological Assessment

Subjects were asked their age, date of birth, educational attainment and employment status. The memory assessment began with the administration of the shortened WAIS-R. This was followed by the AMIPB, the TEA, and the AMT. Subjects were then asked to complete the self-report questionnaires (BAI and BDI). Self-report questionnaires were administered following the memory assessment to prevent subjects from becoming distracted or pre-occupied with their possible symptoms of anxiety and depression prior to the assessment.

2.4.1.2 Treatment

Subjects attended between 3 and 20 sessions of cognitive-behavioural therapy. This consisted of exposure therapy, cognitive restructuring and anxiety management. The number of sessions offered varied according to the availability of the clinical psychologist, the recency of the traumatic stress exposure, and the nature of the trauma. A client who had experienced traumatic stress within the previous eight weeks could be offered an immediate course of four sessions. When the time since

trauma was greater than eight weeks, clients could be offered between eight to ten sessions of CBT or between 16 and 20 sessions. A client who had experienced traumatic stress involving intense shame was more likely to be offered 16 to 20 sessions of CBT, while a client who had experienced traumatic stress involving intense fear was more likely to be offered between eight and ten sessions of CBT. However, the final decision on the number of sessions offered was at the discretion of the clinical psychologist.

The client who received treatment at the Clinical Psychology Department of the Whittington Hospital was seen by a clinical psychologist practising CBT. The psychologist used a combination of exposure therapy and cognitive restructuring. Ten sessions were offered to this client. However, the client dropped out of treatment after session three and was included in the attrition sample.

2.4.1.3 Outcome: Repeated Measures

Outcome measures were administered to assess symptoms of PTSD when clients were discharged from therapy or at session eight, which ever came first. Session eight was chosen as the outcome session in order to capture those clients discharged at session eight as well as those clients who continued on in therapy.

The outcome measures were part of the clinical re-assessment normally administered upon discharge. For this study, they included the CAPS and the SRS-PTSD. The CAPS in interview form was re-administered if clients were discharged at session eight or sooner (N=15). For subjects who continued in treatment beyond the eighth session (N=8), the self-report version of the CAPS was completed.

Figure 2.1 summarises the data collected at the various stages of the research procedure.

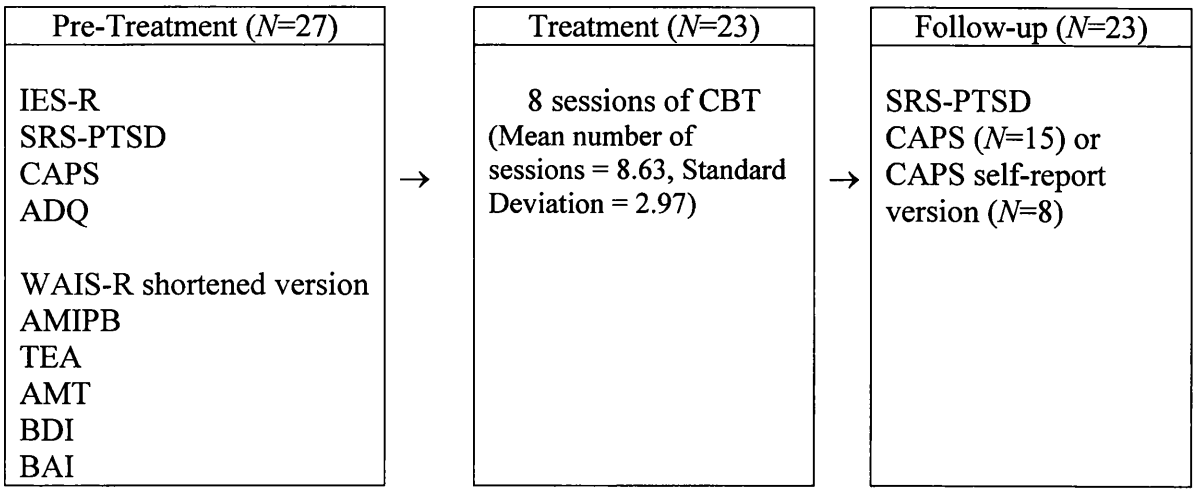

Figure 2.1: Flow chart of the data collected during the research procedure.

2.5 STATISTICAL ANALYSIS

The study set out to answer five questions and to test six hypotheses. These are reproduced below with the statistical analyses used to examine them. Data were analysed using SPSS for Windows Version 6.0.

2.5.1 QUESTIONS

1. Is severity of PTSD symptomatology related to memory functioning?

Pearson's bivariate correlations were calculated to examine the relationship between the CAPS severity scores and the six AMIPB verbal and non-verbal memory scores (Story Recall Immediate, Story Percent Retained, List Learning, Figure Immediate, Figure Percent Retained, and Design Learning).

2. Is time since trauma related to memory functioning?

Pearson's bivariate correlations were calculated between time since the index trauma (in months) and the six AMIPB memory scores listed in question one.

3. Are there differences in performance on the information processing task between those who improve with treatment and those who do not?

In order to answer this question, the subjects who had a DSM-IV diagnosis of PTSD at follow-up were compared to the subjects without a DSM-IV diagnosis of PTSD at follow-up. An Analysis of Covariance (ANCOVA) was performed with Full-Scale IQ entered as a covariate. The Information Processing Task A Adjusted Score was entered as the dependent variable.

4. Are intake anxiety and depression scores related to memory functioning (on the AMIPB)? Are they related to specificity of memory (on the AMT)?

Pearson's bivariate correlations were calculated between intake anxiety scores and nine memory scores: six verbal and non-verbal memory scores on the AMIPB, and the three specificity scores of the AMT (Positive, Negative and Total Specificity Scores). Pearson's bivariate correlations were also calculated between intake depression and these same scores

5. Is alcohol use measured at intake related to memory functioning?

Pearson's bivariate correlations were calculated between the intake alcohol use score and the six memory scores listed in question one.

2.5.2 HYPOTHESES

1. **Participants with a DSM-IV diagnosis of PTSD at follow-up will have poorer memory performance compared to participants who do not meet a DSM-IV diagnosis at follow-up.**

A multivariate analysis of variance with Full-Scale IQ entered as a covariate was selected to look at the effect of diagnostic outcome (PTSD versus No PTSD) on the dependent variables of memory: Story Recall Immediate, Story Percent Retained, List Learning, Figure Recall Immediate, Figure Percent Retained, and Design Learning.

2. **Verbal memory performance will predict outcome.**

In order to test this hypothesis, a direct logistic regression was performed with diagnostic outcome entered as the dependent variable, and the verbal memory scores entered as the independent variables.

3. **Performance on tests of executive function and attention will not account for differences in memory performance.**

In order to tests this hypothesis, multiple regression was run separately for each of the memory variables (Story Recall Immediate, Story Recall Percent Retained, List Learning, Figure Recall Immediate, Figure Recall Percent Retained, and Design Learning) with the attention (Focus and Sustain) and executive function variables (Shift) entered as independent variables.

4. **Based on the research on a disrupted arousal system, intrusions on cognitive tasks will be positively correlated with intrusive symptomatology.**

Pearson's bivariate correlations (one-tailed) were calculated between the intrusion score on the List Learning verbal memory task and the three measures of intrusive symptomatology: (1) the CAPS Intrusion Frequency Score, (2) the CAPS Intrusion Severity Score, and the (3) IES-R Intrusion Frequency Score.

5. **Participants with a DSM-IV diagnosis at follow-up will show less specific memories compared to individuals without a diagnosis of PTSD at follow-up.**

In order to test this hypothesis, the performance on the AMT of the subjects who had a DSM-IV diagnosis of PTSD at follow-up was compared to the performance of subjects without a DSM-IV diagnosis of PTSD at follow-up. An analysis of variance was performed with the positive, negative and total specificity scores entered as dependent variables.

6. **Self-report scores of previous week avoidance and previous week cognitive avoidance will be negatively correlated with AMT specificity memory scores (based on Kuyken & Brewin, 1995).**

Pearson's bivariate correlations (one-tailed) were calculated for the whole sample to assess the relationships between the IES-R total Avoidance score and the specificity memory scores (positive, negative and total specificity scores on the AMT). Pearson's bivariate correlations (one-tailed) were also calculated between the two IES-R cognitive avoidance items (Items 11 and 17) and the AMT specificity memory scores.

3. RESULTS

3.1 DEFINING CASENESS

3.1.1 Caseness

At intake, three questionnaires assessed PTSD symptomatology: the IES-R, the CAPS or the CAPS self-report version, and the SRS-PTSD. At outcome of treatment, two questionnaires assessed PTSD symptomatology: the CAPS or the CAPS self-report version, and the SRS-PTSD. Both the CAPS and the IES-R provided a measure of the frequency of PTSD symptoms. The CAPS also provided a measure of the severity of symptoms. The CAPS and the SRS-PTSD reflected the presence or absence of a diagnosis of PTSD.

At both points in time, in order to be considered as having a diagnosis of PTSD, participants had to meet diagnostic criteria on the CAPS, either the clinician-administered version or the self-report version. The psychometric properties of the self-report version are presented below.

3.1.2 Psychometric Properties: CAPS Self-Report Version

Four participants completed the self-report CAPS at intake and eight participants completed it at follow-up. Internal consistency of the scale was good (Cronbach's alpha=.9494). Convergent validity was assessed by calculating Pearson's correlations between the frequency scores on the self-report version and the same scores on the IES-R. It was also assessed by calculating Pearson's correlations between the total number of DSM-IV criteria that were met for each of the three PTSD symptom sub-groups on the self-report version and the total number for each sub-group on the

SRS-PTSD. All resultant correlations were high, suggesting good convergent validity. Tables 3.1 and 3.2 show the correlations and their level of significance.

TABLE 3.1 **Pearson's correlations between the CAPS Self-Report Version DSM-IV Criteria scores and the SRS-PTSD Diagnostic Criteria scores.**

		SRS-PTSD Diagnostic Criteria Score		
		Intrusion	Avoidance	Hyperarousal
CAPS Self-Report Version: DSM-IV Criteria Score per symptom group	Intrusion	r=.7639 p<.023		
	Avoidance		r=.8040 p<.015	
	Hyperarousal			r=.9279 p<.002

CAPS refers to Clinician Administered PTSD-Scale; SRS-PTSD refers to the Self-Rating Scale for PTSD.

TABLE 3.2 **Pearson's correlations between the CAPS Self-Report Version Frequency Scores and the IES-R Frequency Scores.**

		IES-R Frequency Score		
		Intrusion	Avoidance	Hyperarousal
CAPS Self-Report Version: Frequency Score	Intrusion	r=.8208 p<.003		
	Avoidance		r=.8529 p<.002	
	Hyperarousal			r=.9647 p<.000

CAPS refers to the Clinician-Administered PTSD Scale; IES-R refers to the Impact of Events Scale-Revised.

3.1.3 Sensitivity of the Diagnostic Questionnaires

3.1.3.i Intake

The initial intake sample consisted of 29 participants. Of the sample that had CAPS data (either clinician administered N=24 or self-report version N=4), 96.55% (N=27) were identified as having a diagnosis of PTSD.

The SRS-PTSD was available for all 29 participants. This questionnaire identified 93.10 % of the sample as having PTSD (*N*=27).

Twenty-six subjects met criteria on both questionnaires, one subject met criteria on the CAPS only, one subject met criteria on the SRS-PTSD only, and one subject (without CAPS data) did not meet a diagnosis on the SRS-PTSD. Participants were required to show a diagnosis on the CAPS (either clinician administered or the self-report version) in order to be considered as having a diagnosis of PTSD. Thus, the intake sample consisted of 27 subjects.

3.1.3.ii Outcome

There were 23 subjects in the follow-up sample. All participants had CAPS data (*N*=15 clinician administered and *N*=8 self-report version); 30% (*N*=7) were identified as having a diagnosis of PTSD (*N*=6 clinician administered and *N*=1 self-report version).

The SRS-PTSD was available for 19 subjects in the follow-up sample. Of the subjects with SRS-PTSD data, 42.1% (*N*=8) were identified as meeting a diagnosis of PTSD at follow-up.

Four subjects met criteria on both the CAPS and the SRS-PTSD, three subjects met criteria on the CAPS only (these three subjects had no SRS-PTSD data), and four subjects met criteria on the SRS-PTSD only.

As at intake, participants were considered to have a diagnosis of PTSD if they met criteria on the CAPS (clinician administered or self-report version). Thus, seven subjects were considered to have a diagnosis of PTSD at follow-up.

3.2 SAMPLE CHARACTERISTICS

3.2.1 Demographics

The following table (Table 3.3) shows the demographic characteristics of the follow-up sample and the attrition samples.

TABLE 3.3 **Demographic and sample characteristics of the follow-up and attrition groups**

Variable		Follow-up Sample (*N*=23)		Attrition Sample (*N*=4)
		Not PTSD (*N*=16)	PTSD (*N*=7)	
Age: Mean (Standard Deviation)		35.10 (9.27) range=21.01-50.00	34.09 (6.10) range=27.03-43.10	28.57 (8.68) range=20.08-39.10
Gender	**Male**	9	4	1
	Female	7	3	3
Education	**University**	3	1	1
	College	2	0	0
	Age 18	4	1	2
	Age 16	7	4	1
	Age 12	0	1	0
Employment Status	**Student**	2	0	1
	Manual Worker	3	0	1
	Clerical/Other Non-Manual Worker	3	1	0
	Middle Management	2	0	2
	Professional or Senior Management	2	1	0
	Sick Leave	1	3	0
	Unemployed	3	2	0
Time Since Trauma (in months) Mean (Standard Deviation)		13.02 (12.32) range=2.0-37.0	8.23 (7.96) range=1.75-24.0	15.78 (13.04) range=3.25-28.0
Nature of Trauma	**Physical Assault**	2	1	2
	Sexual Assault	3	0	1
	Road Traffic Accident	7	2	0
	Violent death	1	1	0
	Bomb	2	1	0
	Accident (work/home)	1	2	1
Current Litigation	**Yes**	5	2	1
	No	11	5	3
Alcohol (units/week) Mean (Standard Deviation)		17.88 (16.66) range=0-48	16.29 (21.88) range=0-54	11.25 (12.84) range=0-26
Medication	**Yes**	4	4	2
	No	12	3	2
Recreational Drugs	**Yes**	3	2	2
	No	13	5	2
Prior Trauma	**Yes**	9	3	1
	No	7	4	3
Previous Treatment	**Yes**	1	3	1
	No	15	4	3

3.2.3 The Follow-up and Attrition Groups

The non-parametric χ^2 test was used to compare the follow-up and attrition groups on categorical variables, such as education and nature of trauma. Independent samples *t*-tests were used to compare the groups on interval variables, such as age and alcohol use.

There were no significant differences on any variable between the follow-up sample and the attrition sample: age ($t(25)=1.38$, $p<.181$), gender ($\chi^2_{(1)}=.387$, $p<.534$), education ($\chi^2_{(4)}=2.08$, $p<.722$), employment status ($\chi^2_{(6)}=7.85$, $p<.249$), time since trauma ($t(25)=.68$, $p<.503$), nature of trauma ($\chi^2_{(5)}=5.61$, $p<.347$), current litigation ($\chi^2_{(1)}=.00$, $p<1.00$), alcohol use ($t(25)=.65$, $p<.520$), medication ($\chi^2_{(1)}=.00$, $p<.983$), use of recreational drugs ($\chi^2_{(1)}=.00$, $p<1.00$), prior trauma ($\chi^2_{(1)}=.213$, $p<.644$), and previous treatment ($\chi^2_{(1)}=.00$, $p<1.00$).

3.2.3 The Follow-up Sample: PTSD versus No PTSD groups

The non-parametric χ^2 test was used to compare the PTSD versus No PTSD groups on categorical variables, such as education and nature of trauma. Independent samples *t*-tests were used to compare the groups on interval variables, such as age and alcohol use.

There were no significant differences on any variable between the follow-up sample with PTSD and the follow-up sample without PTSD: age ($t(21)=.26$, $p<.795$), gender ($\chi^2_{(1)}=.00$, $p<1.00$), education ($\chi^2_{(4)}=3.66$, $p<.455$), employment status ($\chi^2_{(6)}=7.09$, $p<.312$), time since trauma ($t(21)=.94$, $p<.358$), nature of trauma

($\chi^2_{(5)}$=3.84, p<.572), current litigation ($\chi^2_{(1)}$=.00, p<1.00), alcohol use (t(21)=.19, p<.850), medication ($\chi^2_{(1)}$=1.03, p<.311), use of recreational drugs ($\chi^2_{(1)}$=.00, p<1.00), prior trauma ($\chi^2_{(1)}$=.019, p<.890), and previous treatment ($\chi^2_{(1)}$=2.35, p<.125).

3.3 PTSD Symptomatology

As explained in Section 3.1.1, three questionnaires assessed PTSD symptomatology: the IES-R, the CAPS (clinician administered or self-report version), and the SRS-PTSD. A further two questionnaires assessed associated symptoms of anxiety and depression (the BAI and the BDI respectively). The values obtained by the follow-up and attrition samples are reported below in Table 3.4.

TABLE 3.4 *Mean scores and standard deviations on questionnaires assessing PTSD symptomatology and associated symptoms for the follow-up and attrition groups.*

Questionnaire	Follow-up Sample (*N*=23)				Attrition Sample (*N*=4)	
	Not PTSD (*N*=16)		PTSD (*N*=7)			
	Mean	SD	Mean	SD	Mean	SD
Clinician Administered PTSD Scale (CAPS)						
Intrusion Severity	9.06	3.61	11.00	5.16	13.00	5.77
Avoidance Severity	12.38	3.99	15.57	2.64	16.50	5.45
Hyperarousal Severity	10.88	2.94	12.71	1.79	13.00	.82
Overall Severity	32.31	8.21	39.28	8.26	42.50	11.59
Subjective Distress	2.56	.63	2.71	.76	2.50	.58
Impact of Events Scale-Revised (IES-R)						
Intrusion Score	25.80	10.44	25.86	11.99	28.75	10.99
Avoidance Score	21.13	8.08	23.29	10.53	31.25	11.35
Hyperarousal Score	22.00	5.06	24.71	5.74	23.50	4.66
Self-Rating Scale for PTSD (SRS-PTSD)						
Number of Diagnostic Criteria (maximum=17)	12.38	2.89	14.42	2.23	15.00	3.37
Beck Anxiety Inventory (BAI)						
Total Score	19.69	13.08	23.14	16.84	36.75	8.66
Beck Depression Inventory (BDI)						
Total Score	22.19	7.35	29.00	9.71	36.00	7.53

3.3.1 The Follow-up and Attrition Groups

Independent samples *t*-tests were used to compare the mean intake symptomatology scores of the follow-up and attrition groups.

The attrition sample had significantly more severe anxiety than the follow-up sample as measured by the BAI ($t(25)=2.19$, $p<.038$), and significantly more severe depression as measured by the BDI ($t(25)=2.57$, $p<.016$)[1]. There were no other differences on intake symptomatology between the two groups: CAPS Intrusion Severity Score ($t(25)=1.42$, $p<.168$), CAPS Avoidance Severity Score ($t(25)=1.42$, $p<.168$), CAPS Hyperarousal Severity Score ($t(25)=1.12$, $p<.275$), CAPS Overall Severity Score ($t(25)=1.64$, $p<.114$), CAPS Subjective Distress Score ($t(25)=-.31$, $p<.759$), IES-R Intrusion Score ($t(24)=.50$, $p<.619$), IES-R Avoidance Score ($t(24)=1.91$, $p<.069$), IES-R Hyperarousal Score ($t(24)=.22$, $p<.825$), SRS-PTSD Diagnostic Criteria Score ($t(25)=1.27$, $p<.214$).

3.3.2 The Follow-up Sample: PTSD versus No PTSD Group

Independent samples *t*-tests were used to compare the mean intake symptomatology scores of the PTSD versus No PTSD groups.

There were no differences on intake symptomatology between the follow-up sample with PTSD and the follow-up sample without PTSD: CAPS Intrusion Severity Score ($t(21)=1.04$, $p<.310$), CAPS Avoidance Severity Score ($t(21)=1.93$, $p<.068$), CAPS

[1] These differences were non-significant after application of the Bonferroni correction. This set α at .005.

Hyperarousal Severity Score ($t(21)=1.52$, $p<.143$), CAPS Overall Severity Score ($t(21)=1.87$, $p<.075$), CAPS Subjective Distress Score ($t(21)=.50$, $p<.621$), IES-R Intrusion Score ($t(20)=.50$, $p<.991$), IES-R Avoidance Score ($t(20)=.53$, $p<.603$), IES-R Hyperarousal Score ($t(20)=1.12$, $p<.275$), SRS-PTSD Diagnostic Criteria Score ($t(21)=1.67, p<.111$), BAI ($t(21)=.53, p<.598$), BDI ($t(21)=1.86, p<.077$).

3.4 Neuropsychological Data

Table 3.5 shows the means and standard deviations of the follow-up and attrition samples on the measures of IQ (WAIS-R) and memory (AMIPB). Table 3.6 shows the means and standard deviations of the age-adjusted percentile range scores on the AMIPB. Table 3.7 shows the means and standard deviations on the measures of attention and executive function (TEA) and overgeneral memory (AMT).

TABLE 3.5 *Means and standard deviations on the WAIS-R and the AMIPB for the follow-up and attrition samples, and the AMIPB standardisation sample where appropriate.*

Variable	Follow-up Sample (*N*=23)				*AMIPB Standardisation Sample (N=179-184)*		Attrition Sample (*N*=4)	
	Not PTSD (*N*=16)		PTSD (*N*=7)					
	Mean	**SD**	**Mean**	**SD**	*Mean*	*SD*	**Mean**	**SD**
(1) IQ (Prorated)								
Verbal	106.38	14.04	97.86	13.73			105.25	9.43
Performance	109.81	13.09	103.43	12.21			106.25	12.52
Full-Scale	109.25	14.58	99.86	14.83	*110.0*	*10.2*	106.50	10.66
(2) Verbal Memory								
Digit Span (WAIS-R) Scaled Score	10.81	3.19	8.86	2.97			12.50	2.08
AMIPB Story Recall (Raw Scores)								
Immediate	41.75	7.33	25.28	7.65	*34.3*	*11.1*	38.00	6.05
Delayed	38.31	8.68	22.85	9.29	*32.1*	*11.5*	36.00	4.24
Percent Retained	91.26	7.88	88.42	15.34	*93.1*	*13.9*	95.47	10.30
AMIPB List Learning								
A1-A5	56.31	7.41	47.00	11.68	*52.0*	*9.6*	56.50	3.87
Intrusions	1.06	1.81	1.14	.69	*1.3*	*2.0*	.25	.50
(3) Non-Verbal Memory								
AMIPB Figure Recall (Raw Scores)								
Immediate	66.75	8.36	61.83	8.66	*75.9*	*21.9*	68.00	16.27
Delayed	65.19	8.13	55.27	18.45	*71.3*	*25.7*	68.00	13.44
Percent Retained	99.27	4.65	95.41	8.05	*91.5*	*22.4*	100.97	5.40
AMIPB Design Learning (Raw Scores)								
A1-A5	36.31	6.15	30.86	7.34	*31.6*	*8.3*	38.00	8.37
Intrusions	9.13	5.07	13.57	9.85	*9.3*	*7.2*	3.75	.96

TABLE 3.5 Continued

Variable	Follow-up Sample (*N*=23)				*AMIPB Standardisation Sample (N=179-184)*		Attrition Sample (*N*=4)	
	Not PTSD (*N*=16)		PTSD (*N*=7)					
	Mean	**SD**	**Mean**	**SD**	*Mean*	*SD*	**Mean**	**SD**
(4) AMIPB Information Processing (Raw Scores)								
Task A Adjusted Score	76.73	15.55	60.02	13.76	*62.1*	*16.0*	80.98	29.88

WAIS-R refers to the Wechsler Adult Intelligence Scale-Revised; AMIPB refers to the Adult Memory and Information Processing Battery.

TABLE 3.6 *Age-Adjusted Percentile Range Scores on the Adult Memory Information Processing Battery (AMIPB) for the follow-up and attrition samples.*

Variable	Follow-up Sample (*N*=23)				Attrition Sample (*N*=4)	
	Not PTSD (*N*=16)		PTSD (*N*=7)			
	Mean	SD	Mean	SD	Mean	SD
(1) Verbal Memory						
Story Recall						
Immediate	4.38	1.15	2.14	1.07	4.00	1.16
Delayed	4.06	1.29	2.29	1.25	4.25	1.26
Percent Retained	3.06	1.39	3.00	2.08	4.00	1.83
List Learning						
A1-A5	4.00	1.15	2.29	1.98	3.75	.50
Intrusions	4.00	1.59	3.29	.95	4.50	1.00
(2) Non-Verbal Memory						
Figure Recall						
Immediate	3.75	1.13	2.43	1.13	4.25	2.06
Delayed	3.63	.96	2.86	1.57	3.50	1.29
Percent Retained	4.19	1.11	3.29	1.50	4.25	.96
Design Learning						
A1-A5	4.06	1.34	3.00	1.29	4.50	1.29
Intrusions	3.19	1.28	2.57	1.72	4.25	.50
(3) Information Processing						
Task A Adjusted Score	3.81	1.33	3.14	1.57	3.75	2.23

There were six range scores: 1 refers to 'below the 9.99th percentile,' 2 refers to 'between the 10th and 24.99th percentile,' 3 refers to 'between the 25th and 49.99th percentile,' 4 refers to 'between the 50th and 74.99th percentile,' 5 refers to 'between the 75th and 89.99th percentile,' and 6 refers to 'above the 90th percentile.'

TABLE 3.7 *Means and Standard Deviations on the TEA and the AMT for the follow-up and attrition samples.*

Variable	Follow-up Sample (*N*=23)				Attrition Sample (*N*=4)	
	Not PTSD (*N*=16)		PTSD (*N*=7)			
	Mean	SD	Mean	SD	Mean	SD
(1) Attention						
Focus (Scaled Score)						
Map Search Two Minute Score	9.50	3.46	7.43	2.44	8.75	4.65
Sustain (Scaled Score)						
Lottery Total Accuracy Score	10.94	2.65	8.57	3.31	11.25	2.87
(2) Executive Function						
Shift Attention (Scaled Score)						
Visual Elevator Accuracy Score	11.31	2.68	11.00	3.00	12.75	2.5
Visual Elevator Timing Score	9.00	2.42	7.71	3.04	7.25	3.69
(3) Autobiographical Memory						
Positive Specificity Score	2.81	1.28	2.43	1.27	1.75	2.22
Negative Specificity Score	2.25	1.29	2.28	1.25	2.25	2.26
Total Specificity Score	5.06	2.08	4.71	1.98	4.00	3.56

TEA refers to the Test of Everyday Attention; AMT refers to the Autobiographical Memory Test.

3.4.1 The Follow-up and Attrition Samples

Independent samples *t*-tests were used to compare the neuropsychological scores of the follow-up and attrition groups.

There were no significant differences between the follow-up and attrition groups on the neuropsychological variables: Prorated Verbal IQ ($t(25)=.20$, $p<.845$), Prorated Performance IQ ($t(25)=.23$, $p<.818$), Prorated Full-Scale IQ ($t(25)=.01$, $p<.989$), Digit Span ($t(25)=1.37$, $p<.183$), Story Recall Immediate ($t(25)=.38$, $p<.708$), Story Percent Retained ($t(25)=1.10$, $p<.284$), List Learning ($t(16.47)=.65$, $p<.527$), List Learning Intrusions ($t(25)=.95$, $p<.353$), Figure Recall Immediate ($t(25)=1.20$, $p<.241$), Figure Recall Percent Retained ($t(25)=.50$, $p<.621$), Design Learning ($t(25)=1.02$, $p<.318$), Design Learning Intrusions ($t(25)=1.72$, $p<..097$), Information Processing ($t(25)=.17$, $p<.866$), Focus Attention, ($t(25)=.06$, $p<.950$), Shift Attention ($t(25)=1.05$, $p<.303$), Shift Attention Timing Score ($t(25)=.90$, $p<.374$), Sustain Attention ($t(25)=.64$, $p<.529$), Autobiographical Memory Positive Specificity Score ($t(25)=1.24$, $p<.227$) Autobiographical Memory Negative Specificity Score ($t(25)=.02$, $p<.988$), and Autobiographical Memory Total Specificity Score ($t(25)=.78$, $p<.441$).

3.5 QUESTIONS

1. Is severity of PTSD symptomatology related to memory functioning?

Pearson's bivariate correlations (two-tailed) were calculated for the whole sample ($N=27$) to assess the relationships between the CAPS severity scores and the six AMIPB verbal and non-verbal memory scores. All results were non-significant,

indicating that severity of PTSD symptomatology was not related to memory functioning in this sample. Table 3.8 shows the values of Pearson's *r* and the levels of significance.

TABLE 3.8 Relationship between the CAPS severity scores and the measures of memory.

CAPS Severity Score	Verbal Memory			Non-Verbal Memory		
	Immediate Recall	Percent Retained	List Learning	Immediate Recall	Percent Retained	Design Learning
Intrusion Severity	*r*=-.1069 *p*<.596	*r*=.0603 *p*<.765	*r*=-.1158 *p*<.565	*r*=-.0057 *p*<.977	*r*=-.0556 *p*<.783	*r*=.0729 *p*<.718
Avoidance Severity	*r*=-.2419 *p*<.224	*r*=.1304 *p*<.517	*r*=-.2229 *p*<.264	*r*=.1213 *p*<.547	*r*=-.0465 *p*<.818	*r*=-.0049 *p*<.980
Hyperarousal Severity	*r*=-.2368 *p*<.234	*r*=-.1035 *p*<.607	*r*=-.2983 *p*<.131	*r*=-.1446 p<.472	*r*=-.1610 *p*<.422	*r*=-.0786 *p*<.697

2. Is time since trauma related to memory functioning?

Pearson's bivariate correlations (two-tailed) were calculated between time since the index trauma (in months) and the six memory scores. All results were non-significant, indicating that time since the index trauma was not related to memory functioning in this sample. Table 3.9 shows the values of Pearson's *r* and the levels of significance.

TABLE 3.9 Relationship between the time since trauma and the measures of memory.

	Verbal Memory			Non-Verbal Memory		
	Immediate Recall	Percent Retained	List Learning	Immediate Recall	Percent Retained	Design Learning
Time Since Trauma	*r*=-.1109 *p*<.582	*r*=-.3170 *p*<.107	*r*=-.0291 *p*<.885	*r*=-.0005 *p*<.998	*r*=-.1766 *p*<.378	*R*=-.0287 *p*<.887

3. **Are there differences in performance on the information processing task between those who improve with treatment and those who do not?**

In order to answer this question, the subjects who had a DSM-IV diagnosis of PTSD at follow-up (N=7) were compared to the subjects without a DSM-IV diagnosis of PTSD at follow-up (N=16). As Full-Scale IQ was significantly associated with information processing performance (r=.5830, p<.004), an analysis of covariance was performed with full-scale IQ entered as a covariate.

Results of evaluation of the assumptions of normality, linearity, homogeneity of variance, homogeneity of regression, and reliability of covariates were satisfactory.

There was no main effect of group (F(1,20)=.105, p<.749), suggesting that the information processing performance of the two groups did not differ when IQ was statistically controlled.

4. **Are intake anxiety and depression scores related to memory functioning (on the AMIPB)? Are they related to specificity of memory (on the AMT)?**

Pearson's bivariate correlations (two-tailed) were calculated between intake anxiety scores and nine memory scores: six verbal and non-verbal memory scores on the AMIPB, and the three specificity scores of the AMT. Pearson's bivariate correlations were also calculated between intake depression and these same scores for the whole sample (N=27). All results were non-significant, indicating that severity of anxiety and depression was not related to memory functioning in this

sample. Tables 3.10 and 3.11 show the values of Pearson's *r* and the levels of significance.

TABLE 3.10 Relationship between intake anxiety and the measures of memory.

	Verbal Memory			Non-Verbal Memory		
	Immediate Recall	Percent Retained	List Learning	Immediate Recall	Percent Retained	Design Learning
Anxiety (BAI)	r=-.1502	r=-.2033	R=-.2277	r=-.0371	r=-.0694	R=-.0242
	p<.454	*p*<.309	*p*<.253	*p*<.854	*p*<.731	*P*<..904

	Autobiographical Memory		
	Positive Specificity Score	Negative Specificity Score	Total Specificity Score
Anxiety (BAI)	r=.0979	r=-.0947	r=.0090
	p<.627	*p*<.638	*p*<.964

BAI refers to the Beck Anxiety Inventory.

TABLE 3.11 Relationship between intake depression and the measures of memory.

	Verbal Memory			Non-Verbal Memory		
	Immediate Recall	Percent Retained	List Learning	Immediate Recall	Percent Retained	Design Learning
Depression (BDI)	*r*=-.2928	*r*=-.1689	*r* =-.3114	*r* =-.1275	*r* =-.1286	*R*=-.0331
	p<.138	*p*<.400	*p*<.114	*p*<.526	*p*<.523	*P*<.870

	Autobiographical Memory		
	Positive Specificity Score	Negative Specificity Score	Total Specificity Score
Depression (BDI)	*r*=-.0931	*r*=-.2567	*r*=-.2037
	p<.644	*p*<.196	*p*<.308

BDI refers to the Beck Depression Inventory.

5. Is alcohol use measured at intake related to memory functioning?

Pearson's bivariate correlations (two-tailed) were calculated between the intake alcohol use scores and the six memory scores. All results were non-significant,

indicating that current use of alcohol was not related to memory functioning in this sample. Table 3.12 shows the values of Pearson's *r* and the levels of significance.

TABLE 3.12 Relationship between intake alcohol use and the measures of memory.

	Verbal Memory			Non-Verbal Memory		
	Immediate Recall	Percent Retained	List Learning	Immediate Recall	Percent Retained	Design Learning
Alcohol Use (units/week)	*R*=.0282 *P*<.889	*r*=.0437 *p*<.829	*r*=.0570 *p*<.778	*r*=.0781 *p*<.699	*r*=.3521 *p*<.072	*R*=-.1138 *P*<.572

3.6 HYPOTHESES

1. **Participants with a DSM-IV diagnosis of PTSD at follow-up will have poorer memory performance compared to participants who do not meet a DSM-IV diagnosis of PTSD at follow-up.**

As a prelude to testing this hypothesis, the relationship between Full-Scale IQ and the six measures of memory was assessed. In the follow-up sample (*N*=23), Full-Scale IQ was found to be significantly associated with Story Recall Percent Retained (*r*=.4185, *p*<.047), List Learning (*r*=.4436, *p*<.034), and Design Learning (*r*=.4864, *p*<.019).

A multivariate analysis of variance with Full-Scale IQ entered as a covariate was therefore selected to look at the effect of diagnostic outcome (PTSD versus No PTSD) on the dependent variables of memory: Story Recall Immediate, Story Percent Retained, List Learning, Figure Recall Immediate, Figure Percent Retained, and Design Learning.

Results of evaluation of assumptions of normality, homogeneity of variance-covariance matrices, linearity, and multicollinearity were satisfactory.

With the use of Wilks' criterion, the combined memory scores were significantly affected by diagnostic outcome, $F(6,15)=.400$, $p<.018$. The effect-size estimates of group differences when controlling for Full-Scale IQ indicated a moderate to high effect (.60) for all memory variables considered together.

The pooled-within cell correlations adjusted for Full-Scale IQ among Story Recall Immediate, List Learning and Design Learning were greater than .30. Roy-Bargmann stepdown analysis was therefore performed on prioritised dependent variables. Priority was given to verbal memory variables which were deemed as more important than non-verbal memory variables. Homogeneity of regression was achieved for all components of the stepdown analysis. Results are summarised in Table 3.13.

Story Recall Immediate was found to make a unique contribution to the composite dependent variable that best distinguished between those who improved with treatment and those who did not, $F(1,20)=15.00$, $p<.001$, $\eta^2 = .43$. Participants scoring higher on Story Recall Immediate had better outcome. Forty-three percent of the variance in the adjusted Story Recall Immediate scores was associated with outcome (PTSD versus No PTSD).

TABLE 3.13 Tests of Outcome Group Status on Measures of Memory

IV	DV	Univariate *F*	df	Stepdown *F*	df	*P*
Outcome	Story Recall Immediate	15.00[a]	1/20	15.00	1/20	.001
	Story Recall Percent Retained	.287	1/20	1.19	1/19	.288
	List Learning	4.53[a]	1/20	.53	1/18	.476
	Figure Recall Immediate	4.91[a]	1/20	1.82	1/17	.194
	Figure Recall Percent Retained	1.42	1/20	.091	1/16	.766
	Design Learning	1.46	1/20	2.57	1/15	.130

[a] Significance cannot be evaluated but would reach $p<.05$ in univariate context.

TABLE 3.14 Summary of Adjusted Sums of Squares and η^2 for Effects of Outcome Group Status on Story Recall Immediate

Source of Variance	Story Recall Immediate	
	SS'	η^2
Outcome Status	18.177	.43
Error	24.241	
Total	42.418	

The final analysis for this hypothesis looked at Digit Span which was analysed separately as it was not part of the AMIPB. An independent samples *t*-test was performed on the Digit Span scaled scores. The result was non-significant, indicating that the two groups did not differ on this variable ($t(21)=1.38, p<.182$).

2. Verbal memory performance will predict outcome.

In order to test this hypothesis, a direct logistic regression was performed with diagnostic outcome entered as the dependent variable, and the verbal memory scores entered as the independent variables. A test of the full model with all three

predictors against a constant-only model was statistically reliable, χ^2 (3, N=23)=16.90, p<.0007, indicating that the predictors, as a set, reliably distinguished between those who improved with treatment and those who did not. Prediction success was impressive with 71.43% of non-improvers and 93.75% of improvers correctly predicted, with an overall success rate of 86.96%.

Table 3.15 shows regression coefficients, Wald statistics, p values and odds ratios.

TABLE 3.15 Logistic Regression Analysis of PTSD Outcome Status as a Function of Verbal Memory Variables

Variables	*B*	Wald test (z-ratio)	*p*	Odds Ratio
Story Recall Immediate	2.57	3.03	.08	13.09
Story Recall Percent Retained	-0.92	1.48	.54	0.40
List Learning	0.40	0.37	.22	1.49
(Constant)	-5.92	2.65	.10	

Story Recall Immediate was the only predictor which approached significance. A model run with Story Recall Immediate omitted at Step 1 and entered sequentially at Step 2, significantly improved the model, χ^2 (1, N=23)=7.404, p<.005. This confirms the finding that Story Recall Immediate is the only reliable predictor of outcome status among the three memory variables. The odds ratio suggests that the probability of improving with treatment increases by a multiplicative factor of 13.09 when Immediate Story Recall scores increase from range 3 (25-49.99 percentile) to range 4 (50-74.99 percentile), for example.

To assess the predictive value of the verbal memory variables while controlling for Full-Scale IQ, the model was run with Full-Scale IQ entered at Step 1 and Story Recall Immediate, Story Percent Retained, and List Learning entered at Step 2. The model with Full-Scale IQ entered at Step 1 was non-significant χ^2 (1, N=23)=2.04, p<.153. This suggests that Full-Scale IQ is not a significant predictor of outcome. The model significantly improved with the addition of the verbal memory variables entered at Step 2, χ^2 (3, N=23)=14.99, p<.001. This indicates that the verbal memory variables significantly predict outcome, even after controlling for Full-Scale IQ.

3. Performance on tests of executive function and attention will not account for differences in memory performance.

In order to test this hypothesis, multiple regression was run separately for each of the memory variables (Story Recall Immediate, Story Recall Percent Retained, List Learning, Figure Recall Immediate, Figure Recall Percent Retained, and Design Learning) with the attention (Focus and Sustain) and executive function variables (Shift) entered as independent variables.

For each multiple regression performed, results of evaluation of assumptions of normality, homoscedasticity of residuals, linearity, and multicollinearity were satisfactory. With the use of p<.001 criterion for Mahalanobis distance, no outliers among the cases were found. No cases had missing data and no suppressor variables were found.

None of the regression models were significant and none of the β values for the independent variables were significant in any of the models, suggesting that performance on the attention and executive function measures did not significantly contribute to the prediction of memory performance: Story Recall Immediate: R^2=.21029, F(3,23)=2.04, p<.136, Story Recall Percent Retained: R^2=.21029, F(3,23)=2.04, p<.136, List Learning: R^2=.21029, F(3,23)=2.04, p<.136, Figure Recall Immediate: R^2=.21029, F(3,23)=2.04, p<.136, Figure Recall Percent Retained: R^2=.21029, F(3,23)=2.04, p<.136, and Design Learning: R^2=.21029, F(3,23)=2.04, p<.136.

4. Based on the research on a disrupted arousal system, intrusions on cognitive tasks will be positively correlated with intrusive symptomatology.

Pearson's bivariate correlations (one-tailed) were calculated between the intrusion score on the List Learning verbal memory task and the three measures of intrusive symptomatology: (1) the CAPS Intrusion Frequency Score, (2) the CAPS Intrusion Severity Score, and the (3) IES-R Intrusion Frequency Score. All correlations were non-significant, indicating that intrusive symptomatology was unrelated to intrusions on the List Learning verbal memory task. Table 3.16 shows the values of Pearson's r and the levels of significance.

TABLE 3.16 Relationship between intrusions on verbal memory task and intrusive symptomatology.

AMIPB Intrusion Scores	CAPS		IES-R
	Intrusion Frequency Score	Intrusion Severity Score	Intrusion Frequency Score
List Learning Intrusion Score	r=-.1023 p<.306	r=-.1091 p<.294	r=.2189 p<.141

5. Participants with a DSM-IV diagnosis of PTSD at follow-up will show less specific memories compared to individuals without a diagnosis of PTSD at follow-up.

In order to test this hypothesis, the performance on the AMT of the subjects who had a DSM-IV diagnosis of PTSD at follow-up (N=7) was compared to the performance of subjects without a DSM-IV diagnosis of PTSD at follow-up (N=16). An analysis of variance was performed with the positive, negative and total specificity scores entered as dependent variables. There was no main effect of group for any of the specificity scores: Positive Specificity Score (F(1,21)=.441, p<.541), Negative Specificity Score (F(1,21)=.004, p<.952), and the Total Specificity Score (F(1,21)=.140, p<.712), indicating that the two groups did not differ with respect to specificity of memories.

6. Self-report scores of previous week avoidance and previous week cognitive avoidance will be negatively correlated with AMT specificity memory scores (based on Kuyken & Brewin, 1995).

Pearson's bivariate correlations (one-tailed) were calculated for the whole sample to assess the relationships between the IES-R Avoidance Total Score and the positive,

negative and total specificity memory scores on the AMT. Pearson's bivariate correlations were also calculated to assess the relationships between specific IES-R cognitive avoidance items and the specificity memory scores. All results were non-significant, indicating that the IES-R avoidance scores were unrelated to specificity of memories in this sample. Table 3.17 shows the values of Pearson's *r* and the levels of significance.

TABLE 3.17 Relationship between weekly avoidance scores and specificity of memory scores.

Avoidance Scores	**AMT**		
	Positive Specificity Score	Negative Specificity Score	Total Specificity Score
Total Avoidance			
IES-R Total Avoidance Score	r=.07 p=.367	r=.08 p=.356	r=.09 p=.337
Cognitive Avoidance			
(1) IES-R Item 11 'I tried not to think about it'	r=.1603 p<.217	r=-.0310 p<.440	r=.0849 p<.340
(2) IES-R Item 17 'I tried to remove it from my memory'	r=.13 p=.263	r=.04 p=.430	r=.11 p=.307

IES-R refers to the Impact of Events Scale-Revised; AMT refers to the Autobiographical Memory Task.

4. DISCUSSION

4.1 RESULTS: SUMMARY OF FINDINGS

Verbal memory was found to be a significant predictor of outcome for clients receiving CBT directed at their PTSD. Clients who did not improve with treatment had significantly poorer performance on intake measures of verbal memory. In particular, a measure of encoding meaningful verbal material (Immediate Story Recall) was found to independently predict outcome. Clients who did not improve performed in the Low Average to Abnormal range on this task. Differences were not accounted for by performance on tasks of attention and executive function. Further, severity of PTSD symptomatology, severity of anxiety and depression, length of time since trauma, and alcohol and substance use were not related to memory functioning. There were no differences on the task of information processing. Finally, the sample as a whole showed overgeneral memories consistent with the literature. However, there were no group differences between those who improved and those who did not on this variable.

4.2 DISCUSSION OF RESULTS

4.2.1 Attrition Group

There were no significant differences between the follow-up and attrition samples on measures of learning, memory, attention and executive function. There were also no significant differences on measures of PTSD symptomatology. However, the attrition group was found to have significantly higher scores on measures of anxiety and depression. Although these differences were non-significant when the

Bonferroni correction was applied, they do suggest that severity of anxiety and depression may be a factor affecting treatment attendance. Some follow-up studies (e.g. Kazdin & Mazurick, 1995; Given, Given & Coyle, 1986) have shown that attrition samples have more severe psychological symptoms compared to those who continue on in treatment.

4.2.2 Study Questions

(1) Is severity of PTSD symptomatology related to memory functioning?

This study found that severity of PTSD symptomatology was not related to memory functioning. In contrast to this finding, Sutker *et al.* (1995) found that cognitive deficits were correlated with PTSD symptom severity. However, the Sutker *et al.* (1995) study looked only at prisoners-of-war (POWs). POWs are a very different subject group in that often their trauma is prolonged and can include biological insults and/or prolonged malnutrition. Malnutrition can affect cognitive functioning (e.g. Penland, 1998; Stahelin, 1999).

In samples of subjects with PTSD attending treatment centres who have not experienced prolonged malnutrition, it is possible that severity of symptomatology does not affect memory performance. It is also possible that contact with a treatment centre offsets some of the distress associated with the condition and this has a positive impact on neuropsychological functioning.

(2) Is time since trauma related to memory functioning?

Time since trauma ranged from 1.75 months to 37 months in the study sample. Time since trauma was not related to memory functioning. Other studies have not looked

at this directly. However, Vietnam veterans with PTSD investigated 20 years post-trauma have shown more diverse memory difficulties than Gulf War veterans who were investigated within approximately one to two years post-trauma. It is possible that the two groups differ because the Vietnam veterans have a longer post-trauma period during which they may have experienced a longer duration of PTSD symptoms associated with neuronal alterations which could affect memory performance. However, such conclusions are problematic because PTSD is associated with increased alcohol and drug use, and it is possible that the Vietnam veterans show more global memory problems because they have engaged in these behaviours for longer than Gulf War veterans, for example.

It is also possible that there is a critical post-trauma period when time since trauma becomes important. The longest post-trauma period in this sample was 37 months, a much shorter duration than the 20 years post-trauma period found in studies of Vietnam veterans.

Unfortunately, this is a difficult variable to research because of the many factors that can occur in the post-trauma period which can also affect memory functioning, such as alcohol and drug use, further trauma, and the development of comorbid conditions. Teasing out the relative contribution of time since trauma on memory functioning requires looking at this variable while controlling for all other potential influences. Given the nature of the disorder, this would be very difficult to do.

(3) Are there differences in performance on the information processing task between those who improve with treatment and those who do not?

There were no performance differences on the information processing task between those who no longer met DSM-IV criteria for PTSD at follow-up and those who did. The mean for both groups fell in the Average range on this task, suggesting that PTSD symptomatology does not affect the ability to process non-meaningful material (i.e. a string of numbers) over a period of a few minutes (four minutes on the AMIPB). Further, ability on this task was unrelated to therapeutic outcome. This is not surprising given that the task was developed to assess rapid but repetitive mental activity that is relatively insensitive to problems of memory, reasoning or visual perception.

(4) Are intake anxiety and depression scores related to memory functioning (on the AMIPB)? Are they related to specificity of memory (on the AMT)?

The anxiety and depression scores of the follow-up sample fell in the moderate to severe range, and the scores were not related to memory functioning. This result is in contrast to Barrett *et al.* (1996) who found that adults with PTSD and comorbid diagnoses of depression, anxiety and/or substance abuse performed more poorly on tasks of memory functioning compared to PTSD subjects without comorbid disorders.

However, the Barrett *et al.* (1996) study has a number of limitations which make it difficult to draw firm conclusions. The comorbid disorders of anxiety, depression and alcohol-substance use were all grouped together and referred to as 'other diagnosis.' It is therefore difficult to evaluate the relative contributions of each disorder on

cognitive functioning. This is further complicated by the fact that the exact rates of disorder in the comorbidity group were not reported. There may have been more subjects with PTSD and alcohol abuse than subjects with PTSD and GAD, for example, and the findings could reflect this. Further, there was no information on severity or length of onset of each of the comorbid disorders. This clouds data interpretation. Some veterans may have had lifetime alcohol use, and others may just meet the study's criteria of one month of alcohol use, for example. Further, the PTSD groups may differ in their range of PTSD symptoms as well as their severity. The PTSD-only group in this study may represent a subgroup of the PTSD diagnosis which is associated with low severity and intact cognitive functioning. Finally, the groups differed on the baseline measure of cognitive functioning (shortened version of the WAIS-R). The group with PTSD and comorbid disorders had significantly lower baseline IQ scores than the other groups, suggesting that the observed differences in memory and learning may be due to differences in baseline cognitive ability.

Nevertheless, research on depression (e.g. Zakzanis *et al.*, 1998) and anxiety (e.g. Calvo & Ramos, 1989; Eysenck, 1982) suggests that there would be an association between these variables and memory performance. It is possible that with PTSD, the association exists between comorbid diagnoses as opposed to comorbid symptoms. Barrett *et al.* (1996) looked at diagnoses whereas the current study looked at comorbid symptoms. Perhaps if subjects had been assessed for DSM-IV criteria for comorbid conditions, an association may have been revealed. The other possibility is that the finding of poor memory functioning is more strongly associated with PTSD

than the comorbid symptoms of anxiety and depression, many of which are included in the PTSD diagnosis.

The current study also did not find an association between depression and overgeneral memories. This is consistent with previous research in which depression as measured by the BDI was not related to overgeneral memory (e.g. Kuyken & Brewin, 1995).

It appears that overgeneral memories may be related to the number of depressive episodes as opposed to severity of depression. Kuyken & Brewin (1995) found a significant association between the number of depressive episodes and the number of general memories in their sample of depressed women with and without a history of physical and sexual abuse. Furthermore, Brittlebank, Scott, Williams, & Ferrier (1993) found no change in overgeneral memory when depressed subjects recovered from their depressive episode. It is possible that the number of depressive episodes is associated with reduced volume of the hippocampus (e.g. Bremner *et al.*, 2000), a brain region important in the retrieval of episodic memory (e.g. Desgranges, Baron, & Eustache, 1998). This may explain the relationship between overgeneral memories and number of depressive episodes. Future studies of overgeneral memory in depressed subjects that also obtain neuroimaging data may elucidate brain-behaviour relationships between the hippocampus and overgeneral memory in depression. As this has not yet been done, no firm brain-behaviour relationships can be drawn implicating the hippocampus in overgeneral memory.

It should be noted that the current study investigated the relationship between anxiety and depression scores and memory functioning by calculating Pearson's bivariate correlations. There are other statistical methods that could have been employed to assess this relationship. One method would have been to divide the sample into two groups: one group in which subjects had mild to moderate depression and the second group in which subjects had severe depression. Analysis of variance with the memory variables entered as dependent variables could be performed to look at the difference in memory functioning between those with mild to moderate depression versus those with severe depression. The same method could be employed to look at the relationship between anxiety and memory functioning. The advantage of this method over Pearson's correlational analysis is that it does not assume linearity among the variables.

The use of the Spearman correlation is a second alternative method that could have been employed to investigate the relationship between anxiety and depression scores and memory functioning. The Spearman correlation is used when the data are expected to show a consistent, but not necessarily linear relationship (Gravetter & Wallnau, 1988). It measures rank data. Therefore, the anxiety and depression scores would need to be converted to ranks. For example, a BDI score of less than 10 could be assigned '1,' a BDI score between 11 to 18 could be assigned '2,' a BDI score between 19 to 24 could be assigned '3,' a BDI score between 25 to 29 could be assigned '4,' a BDI score between 30 to 44 could be assigned '5,' and a BDI score between 45 and 63 could be assigned 6. The AMIPB percentile range scores were already rank data ranging from 1 to 6. Spearman's correlations could be calculated between the anxiety and depression scores converted to ranks and the AMIPB

percentile range scores to assess the relationship between anxiety and depression and memory functioning.

(5) Is alcohol use measured at intake related to memory functioning?

Alcohol use in units per week ranged from 0 to 54 with a mean of 16.48 units. Although studies of current alcohol use (e.g. Birnbaum *et al.*, 1978; Hastroudi *et al.*, 1984; Lister *et al.*, 1991) suggest that it would affect memory performance, no significant correlation was found in this study. This may reflect problems with the alcohol assessment questionnaire (ADQ; The Alcohol and Drugs Questionnaire) which may be insensitive to screening alcohol problems. The ADQ is a self-report questionnaire which became part of the client's clinical file, and hence property of the National Health Service (NHS). Thus, clients may have minimised their reporting of alcohol use. Studies have shown that clients tend to under-report alcohol and drug use on self-report questionnaires (e.g. Leigh, Gillmore, & Morrison, 1998; Lemmens, Tan & Knibbe, 1992). Nevertheless, the results suggest that current alcohol use is not related to memory functioning in PTSD in this sample. Perhaps an association would exist between chronic alcohol use, PTSD and memory functioning. However, long-term alcohol use was not thoroughly assessed in this study.

4.2.3 Study Hypotheses: Consideration of Results

(1.i) Memory Performance and Outcome

Subjects who met DSM-IV criteria for PTSD at follow-up were found to have poorer pre-treatment verbal memory performance compared to subjects who no longer met the diagnosis at follow-up. Specifically, subjects who did not improve were found to

have significantly poorer immediate verbal recall. That is, they had difficulty recalling a story immediately after it was read to them. Their scores fell in the Low Average (10^{th} – 24.99^{th} percentile) to Abnormal range (1 - 9.99^{th} percentile), while the scores on this task of the subjects who improved fell in the Average range (50^{th} – 74.99^{th} percentile). This particular task was highly predictive of outcome. Subjects who performed poorly on this task were more likely to meet diagnostic criteria for PTSD at follow-up.

Subjects also had difficulty on the List Learning task. That is, learning a list of 15 words over a period of five trials. Although the two groups did not differ significantly in the MANCOVA analysis, in a univariate context, the difference did reach significance. The clients who did not improve fell in the Low Average range (10^{th} – 24.99^{th} percentile) compared to the clients who did improve who fell in the Average range (50^{th} – 74.99^{th} percentile) on this task.

Subjects did not differ on tasks of attention or executive function with almost all scores falling in the Average range, suggesting that the observed memory differences were not due to attention or executive function processes.

Further, subjects did not differ on the WAIS-R Digit Span subtest. Digit Span is thought to measure attention (e.g. Lezak, 1995; Hodges, 1994) and the process of encoding non-meaningful material (Baddeley, 1995).

(1.ii) The Significance of Immediate Story Recall

Immediate Story Recall on the AMIPB requires the subject to recall a story after it has been read to them. Coughlan & Hollows (1985) designed the task to assess immediate registration of verbal information. It therefore measures the ability to encode and immediately recall meaningful verbal material. Subjects who did not do well in therapy had difficulty on this task. They did not have difficulty on Digit Span (consistent with Vasterling *et al.*, 1998), a measure of attention and encoding of non-meaningful numerical material. It is possible that subjects with PTSD have difficulty encoding meaningful verbal material.

Ehlers & Clark (2000) note that subjects with persistent PTSD show problematic intentional recall of their trauma memory (i.e. a weak semantic route to retrieval of their trauma memory), and that this is implicated in their poor recovery. It is possible that subjects who do not improve in treatment have poor intentional recall of meaningful material in general. That is, their weak semantic retrieval extends beyond the memory for their trauma, and possibly to meaningful semantic material in general. This would make it difficult to encode and recall relevant verbal material in therapy and would influence outcome.

On an optimistic note, this study found that what subjects did encode they were able to retain. Thus, although their delayed scores fell in the Abnormal to Low Average range, their percent retention scores did not differ significantly from the group who did improve in treatment. Thus, it is possible that if clinicians were aware of the encoding difficulties of particular PTSD clients, then CBT could commence at a

slower pace with more frequent repetition. Also, sessions of therapy could be tape-recorded for these clients thus aiding their learning.

(2) Attention and Executive Function

This study found that performance on tasks of attention and executive function did not contribute to group differences in memory functioning, nor did it account for variance in memory scores in the entire sample. Further, performance on these tasks almost invariably fell in the Average range. This suggests that in this sample, differences in memory functioning were not due to differences in attention and executive function ability.

This is in contrast to Vasterling *et al.* (1998) who found significant differences on tasks of attention, and Beckham *et al.* (1998) who found significant differences on tasks of attention and executive function in their sample of veterans with PTSD.

Vasterling *et al.* (1998) compared 19 Gulf War veterans with PTSD to 24 veterans without PTSD. There were no differences between groups on tasks of executive function (their measure of Shift Attention) as measured by the Wisconsin Card Sorting Test. This is consistent with the current study in which subjects' performance on the Shift Attention tasks of the TEA generally fell in the Average range. However, they found that subjects with PTSD showed relative performance deficiencies compared to veterans without PTSD on tasks of sustained attention, mental manipulation, initial acquisition of information, and retroactive interference. Close inspection of the results, however, showed that the measure of sustained attention consisted of an omission and commission score. That is, a false negative

score, and a false positive score. The groups did not differ on the false negative score, meaning that they both had equal ability in identifying correct hits. It was only on the false positive score that the PTSD subjects performed more poorly. It is difficult to conclude from this that the sample had true difficulties with sustaining attention. It appears that they were able to sustain their attention to identify correct hits despite making false positive errors.

The other difficulty with the Vasterling *et al.* (1998) study was that their measure of mental manipulation was the Arithmetic subtest of the WAIS-R. This could equally reflect differences in baseline cognitive ability. The two groups did differ significantly on the Vocabulary subtest which was used as an approximation of Full-Scale IQ. Although Vocabulary was not a significant covariate in their MANCOVA analysis, a more thorough baseline measure of Full-Scale IQ may have revealed a significant association, particularly as the two groups differed significantly on the two WAIS-R subtests that were administered.

The third difficulty with the Vasterling *et al.* (1998) study was that their inclusion criteria allowed for up to 30 minutes of loss of consciousness. Even relatively brief periods of unconsciousness can result in diffuse axonal injury (Gennarilli *et al.*, 1996). Other studies exclude subjects when loss of consciousness exceeds 10 minutes (e.g. Bremner *et al.*, 1993) or even a few minutes (e.g. Beckham *et al.*, 1998). It is therefore possible, that some of the sample included participants with mild head injury which would adversely influence neuropsychological performance.

Finally, the study compared 19 PTSD subjects to a control group of 24 subjects. This is a small sample and with greater numbers, a more thorough IQ screener, and more stringent head injury criteria these differences would possibly be minimised.

In the current study, attention and executive function and memory performance was investigated in the whole sample of 27 subjects. Inclusion criteria for head injury were stringent, with subjects excluded for any period of loss of consciousness or hospitalisation for head injury. Thus, this study may reflect a more accurate picture of attention and executive function performance in subjects with PTSD.

However, Beckham *et al.* (1998) found differences on measures of attention and executive function in their sample of Vietnam veterans with and without PTSD. They compared 45 Vietnam veterans with PTSD to 45 Vietnam veterans without PTSD on measures of simple attention, motor function, manual dexterity, sustained attention, concentration and complex processing. Subjects with PTSD did significantly worse on measures of sustained attention, concentration and complex processing.

Beckham *et al.* (1998) conducted subgroup analyses to look at the effect of comorbid anxiety, depression, alcohol/drug use, medication and compensation-seeking status on neuropsychological performance. They divided the PTSD and no-PTSD groups into smaller groups that excluded one variable, but included all others. It is therefore difficult to conclude the independent effect of these variables on performance. For example, an analysis to consider the effect of anxiolytic medication on neuropsychological performance compared veterans with PTSD without anxiolytic

medication to veterans without PTSD and without anxiolytic medication. However, the groups included subjects with antidepressant medication, alcohol use, and heavy combat exposure. Although, the authors conclude that the observed results were not due to anxiolytic medication, they cannot conclude that they were due to PTSD alone. The observed results may have been attributable to alcohol use, history of depression, and so on.

Further, although Beckham *et al.* (1998) found relative differences on tasks of attention and executive function, it is not possible to generalise these results to a group of adults with PTSD with different precipitating traumas presenting for clinical treatment. Vietnam veterans may represent a unique population of adults with PTSD whose precipitating trauma is qualitatively different to adults with PTSD who attend treatment centres in the U.K. Further, the post-trauma period is much longer in Vietnam veterans than clients who typically present at NHS centres.

Given the difficulties in drawing firm conclusions in the research to date on studies of attention and executive function in PTSD, it is possible that the results of this study hold true. That is, measures of attention and executive function are intact in subjects with PTSD who have experienced trauma other than combat exposure, and that these abilities do not account for the observed differences in memory functioning.

(3) Re-experiencing Symptomatology and Intrusions on Memory Tasks

The current study looked at the relationship between intrusive errors on the verbal learning task of the AMIPB and scores of re-experiencing symptoms on the CAPS

and the IES-R. No significant correlations were found, suggesting that in this sample there was no relationship between intrusive symptomatology and intrusive errors on memory tasks.

This is in contrast to the Vasterling *et al.* (1998) study which found that symptoms of re-experiencing were correlated with degree of intrusions on memory tasks in their sample of Gulf War veterans with PTSD. However, for reasons discussed in 4.2.3.(2) (Attention and Executive Function), it is possible that the Vasterling *et al.* (1998) findings are inflated due to the inclusion of subjects with possible head injury, as well as the small sample size. It is also possible that another variable accounts for the observed relationship in the Vasterling *et al.* (1998) study, a variable that they did not fully consider, such as IQ, for example. Perhaps veterans with lower IQ and PTSD are more likely to commit these kinds of errors.

(4) Autobiographical Memory

Specificity of autobiographical memories did not distinguish those who improved from those who did not. The mean specificity score in response to positive and negative cue words was 54 and 45.2 percent respectively. This is consistent with previous studies of PTSD and autobiographical memory (e.g. McNally *et al.*, 1994) in which subjects with PTSD obtained mean scores of 45 and 42 percent for positive and negative cue words respectively.

Hierarchical memory theory proposes that access to a specific memory depends on (a) adequate encoding of the material and (b) not aborting retrieval before a specific memory is accessed (Williams, 1996). Research suggests that both of these

processes can be affected by a traumatic event (e.g. Kuyken & Brewin, 1995; Maas & Kohnken, 1989). This may explain the finding of overgeneral memories in this sample.

Harvey *et al.* (1998) found that overgeneral memories in a sample of 22 subjects (10 with Acute Stress Disorder (ASD), 12 without ASD) following a road traffic accident predicted the development of PTSD six months later. One would expect that in PTSD overgeneral memories would predict outcome. However, there were no group differences in outcome on this variable. Perhaps this finding would have been obtained if, as in the Harvey *et al.* (1998) study, participants were not attending treatment. That is, overgeneral memory may predict chronic PTSD in participants not attending therapy. However, in this sample, participants were attending CBT treatment, a structured therapy in which the therapist was likely to persist with questioning until the necessary level of detail had been obtained. It is more likely that overgeneral memories would predict a greater number of treatment sessions as the therapist would require more time to elicit specific details necessary to therapy.

(5) Avoidance and Overgeneral Memory

Kuyken & Brewin (1995) found that the total score on the Avoidance subscale of the Impact of Event Scale (IES) was significantly correlated with overgeneral memories in their sample of 35 depressed women with a history of child physical or sexual abuse. The Avoidance subscale of the IES is a measure of previous week symptoms of cognitive, behavioural and emotional avoidance. Kuyken & Brewin (1995) reported that avoidance of memories of childhood sexual or physical abuse was associated with retrieval of more overgeneral memories.

It was therefore thought that previous week cognitive avoidance of the index trauma would be significantly correlated with overgeneral memory in the current study, and that the total score on the Avoidance subscale of the IES-R would be related to overgeneral memory. This was not found to be the case. There was no significant relationship between the IES-R cognitive avoidance items and overgeneral memory, and there was no relationship between the total score on the Avoidance subscale of the IES-R and the retrieval of overgeneral memories.

On closer examination of the Kuyken & Brewin (1995) study, the value of Pearson's *r* for the correlation between the IES Avoidance total score and overgeneral memory was .30. Such a value reflects a weak relationship (Cohen & Holliday, 1982). The relationship could become even weaker with a larger sample size. It could, of course, become stronger.

It is difficult to generalise from the Kuyken & Brewin (1995) study to subjects with PTSD because they looked at a sample of depressed women with and without a history of physical or sexual abuse. Diagnosis of PTSD was not considered, and in this respect, cannot be generalised to subjects with a current diagnosis of PTSD. Further, there are a number of interpretative difficulties with their study.

Kuyken & Brewin (1995) suggest that the effort to avoid intrusive memories may interfere with patients' ability to carry out the task at hand because of constraints on working memory. However, there was no relationship in their sample between intrusive symptoms on the IES and overgeneral memory. If avoidance symptoms

were occurring because subjects were trying to avoid intrusive memories, one may expect the Intrusive subscale of the IES to be correlated with overgeneral memories as well. However, this was not found to be the case.

Further, there were only two items on the IES which could measure avoidance of memories. They were: 'I tried not to think about it' and 'I tried to remove it from my memory.' The other avoidance items refer to behavioural and emotional avoidance. Kuyken & Brewin (1995) report that avoidance of memories is correlated with overgeneral memory. However, they report the correlation between the total score on the IES Avoidance subscale and overgeneral memories without commenting on the specific items of cognitive avoidance on this scale and how they may correlate with overgeneral memory. It is therefore problematic to conclude that cognitive avoidance is associated with overgeneral memory because the total IES Avoidance subscale score reflects cognitive, emotional and behavioural avoidance.

Finally, Kuyken & Brewin (1995) found that the number of depressive episodes was moderately correlated with overgeneral memories. It is possible that subjects with overgeneral memories were not avoiding intrusive memories, but rather they had a greater number of depressive episodes, and it is this relationship that explains the association between avoidance and overgeneral memory.

4.3 STUDY RESULTS: THEORETICAL IMPLICATIONS

The present study results have theoretical implications for understanding the nature and development of memory difficulties in PTSD, as well as psychological models of PTSD and treatment outcome.

4.3.1 Memory Problems and PTSD

This study found that within the subject sample, there was a group of adults with PTSD who showed memory difficulties and who also did not improve with treatment. This suggests that the memory problems may not characterise all clients who develop PTSD, but rather, a group of clients who may be more vulnerable to developing them.

It is also possible that the memory difficulties are not related to PTSD per se, but rather characterise those individuals who are likely to have difficulty responding to verbally oriented treatment. However, if this were the case, then one would expect clients with PTSD and memory problems to continue to have memory difficulties even when their PTSD improves. Current research does not support this expectation. Nishith, Weaver, Resick, & Uhlmansiek (1999) compared the memory functioning of female rape victims with PTSD before and after cognitive-behavioural treatment to a wait-list control of female rape victims with PTSD. Both groups had similar levels of memory difficulties before treatment as measured on the Wechsler Memory Scale – Revised (WMS-R; Wechlser, 1987). Eighty-six percent of the treatment group no longer met diagnostic criteria for PTSD at follow-up. These clients showed significant improvement in memory functioning compared to the wait-list control

group. This suggests that memory difficulties are related to PTSD, and that they can improve as symptoms improve.

4.3.2 Mechanism of Memory Difficulties

As reviewed in Chapter 1, Section 1.2.2.5, a number of reasons have been put forth to explain the observed memory difficulties in PTSD. The results of this study suggest that in this sample, they were not due to symptoms of comorbid anxiety, depression and/or alcohol and substance use.

Severity of PTSD symptomatology was not found to be related to memory functioning. Given that there were no significant differences on Full-Scale IQ, and no differences on tasks of attention and executive function, it is believed that the results were not due to global impairment.

It is possible that the subjects who did not improve had poor premorbid memory status and the condition of PTSD exacerbated their memory functioning. This is difficult to rule out. However, subjects consistently reported that they had not experienced memory difficulties prior to the onset of their symptoms.

It is possible that there is a physiological explanation for the observed memory difficulties that precedes or follows the onset of the disorder.

(i) The Hippocampus

In this study it was found that subjects with poor memory had particular difficulty encoding a meaningful story. They also had difficulty learning a list of words over a period of five trials.

The hippocampus has been implicated in encoding and retrieval processes of new learning and memory (Dolan & Fletcher, 1997; Kapur, Tulving, Cabeza, McIntosh, Houle & Craik, 1996; Nyberg, McIntosh, Cabeza, Habib, Houle & Tulving, 1996). The prefrontal cortex also has a role in encoding, particularly in guiding retrieval strategies (e.g. Schacter, Alpert, Savage, Rauch & Albert, 1996). It is possible that the observed memory difficulties in subjects with PTSD reflect inefficient prefrontal cortex and hippocampal function.

Some authors have argued the role of the frontal lobes over the medial temporal lobes in the observed memory difficulties in PTSD (e.g. Vasterling *et al.*, 1998). However, subjects did not show encoding difficulties on the Digit Span subtest. This is consistent with Vasterling *et al.* (1998) who also found intact performance on the Digit Span subtest in their sample of PTSD Gulf War veterans. Digit Span represents an ability that is associated with the frontal lobes, and remains intact even after damage to the hippocampus (e.g. Cave & Squire, 1992). One would expect, therefore, to observe difficulties on the Digit Span subtest if there was extensive frontal lobe involvement. One would also expect to observe difficulties with attention and executive function if this were the case. However, in this study, subjects did not demonstrate difficulties on tasks of attention or executive function.

Thus, the explanation of the results best fits current neurobiological models of memory which highlight the prefrontal cortex in the guiding of retrieval strategies, and the hippocampus in the encoding and retrieval of episodic memory. It is possible that in subjects who did not improve, these brain regions were not working to an optimum level. This may reflect HPA axis alterations which can lead to hippocampal atrophy (e.g. Golier & Yehuda, 1998) and hence, potential poor functioning. Inefficient functioning of the hippocampus may affect functioning of the prefrontal cortex and vice versa. However, no firm conclusions can be drawn because no neuroimaging data were collected, and no data relating to the HPA axis were collected, such as levels of cortisol or number of lymphocyte glucocorticoid receptors.

(ii) A Disrupted System of Arousal

Vasterling *et al.* (1998) found that PTSD-related memory difficulties closely resembled those typically associated with frontal system dysfunction, that is inefficient acquisition and errors of intrusion. They concluded that in PTSD, memory difficulties represent a disrupted arousal system associated with the frontal lobes.

If PTSD-related memory problems do indeed reflect a disrupted arousal system, then one would expect hyperarousal symptoms to be correlated with memory performance. Consistent with Vasterling *et al.* (1998), this study found that symptoms of hyperarousal were not related to memory functioning. It is possible that perhaps only some symptoms of hyperarousal (e.g., feeling watchful and on guard) are correlated with memory functioning as opposed to the total hyperarousal

scores as measured in assessment questionnaires. Future research is necessary to look at specific symptoms of hyperarousal and memory functioning.

One would also expect symptoms of intrusion to be correlated with memory performance, as they could represent reduced capacity of the frontal lobes to inhibit unwanted information. Vasterling *et al.* (1998) did find that re-experiencing symptoms were correlated with memory functioning. However, this finding was not replicated in this study.

Although the authors stress the involvement of the frontal lobes over the medial temporal lobes in memory-related impairments in PTSD, it is possible that the two interact. That is, a disrupted arousal system could interfere with the functioning of the medial temporal lobes in the process of memory consolidation. This would suggest involvement of both the prefrontal cortex and the hippocampus, as described above.

4.3.3 Development of Memory Difficulties

The question remains as to why some individuals with PTSD exhibit memory difficulties and others do not.

Golier & Yehuda (1998) suggest that memory symptoms fluctuate in PTSD, and that they are related to neuropathologic processes reflecting neuronal atrophy (which is reversible) rather than neuronal loss (which is irreversible). It is possible that in this study, the subjects with poor memory were tested at a point in time reflecting hippocampal neuronal atrophy.

It is also possible that prior trauma is a crucial variable. This study measured whether or not subjects had a history of prior trauma. This was measured dichotomously. That is, subjects either did or did not have a history of prior trauma. The number of traumatic events was not recorded. Further, perception of whether the event was traumatic was also not recorded. Thus, some subjects reported prior events that were perceived as traumatic to the examiner, but which they did not themselves perceive to be traumatic. For example, one subject reported being involved in a bombing in Northern Ireland, but he did not view this as a traumatic event. Nevertheless, the event was considered to reflect a history of prior trauma. It is possible that the key component in considering prior trauma is whether or not the subject identified the previous traumatic event as being traumatic. Thus, a relationship may be found between prior trauma and poor memory if measured in this way. Prior trauma is thought to lead to neurobiological changes that could be associated with the later development of memory problems (Golier & Yehuda,1998).

This study also raises the potential importance of negative appraisals in memory problems. Negative appraisals of the traumatic event and its sequelae are associated with persistent PTSD (Ehlers & Clark, 2000). It is possible that the negative appraisals of some subjects with PTSD extended to their neuropsychological assessment. Thus, they may have experienced more negative self-talk during neuropsychological testing which affected their performance or they may have perceived themselves to be failing at the task at hand which would also have affected their performance. Weiner (1966) found that anxious subjects did worse on memory tasks when they perceived themselves to be failing. Identifying the self-talk of

subjects with PTSD and their perception of success-failure during neuropsychological tasks is an area of future research.

4.3.4 Models of PTSD and Treatment Outcome

The results have implications for psychological models of PTSD that emphasise the role of maintenance factors. These would be information processing and cognitive models (e.g. Foa & Kozak, 1986; Janoff-Bulman, 1985, 1989, 1992; Brewin *et al.*, 1996). These models suggest that PTSD persists in some individuals because of negative appraisals of the traumatic event and/or its sequelae which can induce a sense of current threat and lead to dysfunctional coping styles that maintain or even worse, enhance PTSD symptoms (Ehlers & Clark, 2000). Such appraisals inhibit the trauma memory from being consolidated as clients retrieve biased information which confirms their appraisals. Failure to consolidate the memory prolongs the course of the disorder (Foa & Kozak, 1986).

The finding of poor verbal memory ties in with these models in terms of an additional maintenance factor. Poor verbal memory may make it difficult to recall the trauma memory in a coherent framework. Encoding difficulties would make it difficult to register verbal material. Thus, clients may have difficulty registering the rationale behind various CBT techniques, and thus, may respond less well to them. This is likely going to inhibit consolidation of the trauma memory and prolong the course of the disorder.

Again, it is possible that the hippocampus is implicated in this process. Further, there may be a functional effect of inefficient hippocampal function (described below).

Thus, the results suggest a cognitive neurobiological model in the maintenance of PTSD. Poor verbal memory may make it difficult to benefit from CBT and thus, consolidate the trauma memory. Further, poor verbal memory may reflect inefficient hippocampal function which has a functional effect in the manifestation of PTSD symptoms.

(i) The functional effect of atrophy

A number of models suggest that the hippocampus acts as a temporary store or link for new memories before they are integrated into neocortical networks (Alvarez & Squire, 1994; McClelland *et al.*, 1995; Murre, 1997). Neuroendocrine alterations that affect the HPA axis and ultimately reduce the size of the hippocampus may affect hippocampal function. Further, emotional activation of the amygdala interferes with hippocampal functioning (Van der Kolk *et al.*, 1997). These factors may prevent the hippocampus from fully consolidating the trauma memory. In PTSD, it is possible that the trauma memory is under-consolidated and re-presented in the form of intrusions, in a continual attempt to consolidate it and place it within the long-term memory store. Intrusions may represent a failure of the hippocampal complex to consolidate the memory and transfer it to the neocortex. Future research utilising neuroimaging techniques is required to identify whether or not this is the case.

4.4 STUDY RESULTS: CLINICAL IMPLICATIONS

Clients with chronic conditions present a long-term burden to health services. Financial and staffing limitations often mean lengthy waiting lists and/or limited

number of sessions in which to offer treatment, potentially reducing treatment efficacy and adversely affecting the course of the disorder.

The results of this study have identified a group of patients who are unlikely to respond to standard intervention for PTSD. These patients have poor verbal memory skills, particularly the ability to encode and retrieve meaningful material. This may translate into therapy, making it difficult for them to encode, retain and utilise therapeutic models and meanings.

It is possible that these clients progress in therapy at a slower pace than clients with PTSD who have intact verbal memory, or that they genuinely make little progress because of their memory difficulties.

This has both assessment and treatment implications. A verbal memory screening task could be introduced during assessment that could identify clients with poor verbal memory. Therapy could then progress at a slower pace with more repetition of CBT material, or other treatment protocols could be used that do not rely so heavily on verbal skills and memory, such as neurofeedback or Eye Movement Desensitisation and Reprocessing Therapy (EMDR). Neurofeedback is a biofeedback-type procedure and has been reported to be effective with PTSD sufferers (e.g. Peniston & Kulkosky, 1991). EMDR has also been reported to be effective with PTSD sufferers (e.g. Vaughan, Wiese, Gold & Tarrier, 1994).

Identifying clients who are unlikely to respond to CBT for PTSD at the outset will potentially reduce the percentage of individuals who fail to recover from PTSD,

thereby alleviating some of the long-term costs and suffering associated with the condition.

4.5 LIMITATIONS OF THE STUDY

4.5.1 Sample

The sample consisted of subjects who had different precipitating traumas. This is in contrast to other studies of PTSD and memory that have investigated subjects according to their traumatic stressor. Thus, the results of this study may not generalise well to specific populations of adults with PTSD, such as survivors of RTAs or combat exposure. However, the results may generalise well to clients with PTSD who present for treatment.

History of physical and sexual abuse was not thoroughly assessed. There may have been subjects included in the sample who had previous trauma of this nature. Childhood trauma has been associated with memory difficulties in adulthood. For example, Kuyken & Brewin (1995) found that subjects with a history of childhood sexual or physical abuse had more overgeneral memories than subjects without a history of abuse. However, Stein *et al.*, (1999) did not find evidence of memory difficulties in adult survivors of childhood sexual abuse on their neuropsychological measures of memory functioning.

A further limitation of the current study was the small sample size. It is possible that the relationship between verbal memory and poor outcome would become weaker with a greater sample size. It is also possible that it would become stronger.

4.5.2 Design

Outcome studies of PTSD have measured outcome at various intervals. For example, Ehlers (1998) measured outcome at session eight while Nisith *et al.* (1999) measured outcome at session six. In this study, outcome was measured at session eight irrespective of severity or type of PTSD symptomatolgy at intake.

This may not have been the most appropriate time to measure outcome, particularly as clients with different types of PTSD reactions were given different lengths of treatment. For example, a client with PTSD complicated by problems of shame and guilt would likely have been offered between 16 to 20 sessions of treatment, whereas a client with PTSD characterised by intense fear would likely have been offered between eight to ten sessions. Thus, subjects were offered different lengths of treatment that may have progressed at different rates. Some subjects may have experienced more active ingredients of recovery (e.g. imaginal exposure) in one length of treatment versus another, and this may have affected the rate of recovery.

This could be addressed in a future study by assessing outcome at a similar time depending on the overall number of sessions offered. Thus, for clients streamlined into a CBT protocol of 10 sessions, outcome could be measured at session eight. For clients streamlined into a protocol of 16 sessions, outcome could be measured at session 14.

The study did not control for therapist ability or treatment protocol. Therefore, some subjects may have received more behavioural versus cognitive treatment and vice

versa. Some therapists may have been more skilled than other therapists. In order to correct this sort of shortcoming, an outcome study in which therapists are all trained to the same level and all trained in the same protocol would be necessary. Further, it would be necessary to monitor the protocol adherence of therapists. This could be done by taping sessions and assessing treatment adherence.

4.5.3 Measures

(i) Sensitivity of the Diagnostic Questionnaires

At intake, the CAPS (either clinician administered or self-report version), identified similar numbers of clients with DSM-IV criteria for PTSD as the SRS-PTSD. However, at outcome, the CAPS identified 30 percent of clients as meeting a diagnosis of PTSD, whereas the SRS-PSTD identified 42 percent. This discrepancy suggests that perhaps the CAPS was sensitive to treatment changes or that the SRS-PTSD was insensitive.

The SRS-PTSD is a self-report questionnaire and Carlier *et al.* (1998) reported that clients tended to overreport PTSD symptoms on the SRS-PTSD compared to the Structured Clinical Interview for PTSD (SI-PTSD).

It is possible that the clinician-administered version of the CAPS is an under-estimation of the true prevalence of PTSD in clients who have attended treatment. The CAPS was administered by the client's therapist at outcome and this may reflect experimenter bias. That is, the therapists may have been biased in observing improvement in their clients, particularly if they perceived clients' outcome to reflect on their skills as a therapist.

These reasons may explain the discrepancies between the CAPS and the SRS-PTSD at outcome. In retrospect, it would have been helpful to have had a blind assessor administer the CAPS at outcome.

(ii) Convergent Validity of the CAPS Self-Report Version

Ideally, in order to provide a more accurate picture of the convergent validity of the CAPS self-report version, it would have been best to administer the CAPS clinician version to a group of subjects who also had completed the self-report version. Correlations could then have been calculated between the self-report CAPS scores and the clinician-administered CAPS scores to assess the strength of association.

(iii) The Adult Memory and Information Processing Battery (AMIPB)

Although the AMIPB is becoming a more widely recognised measure of memory, learning and information processing in British samples, it only provides age-related percentile range scores as opposed to age-related standardised scores. Therefore statistical analyses must use range scores, which are not as accurate as standardised scores. Further, the delay period on the verbal and non-verbal task is just thirty minutes, whereas on other measures of memory, the delay period is 45 minutes or more, and often for verbal material 90 minutes (e.g. Wechsler Memory Scale).

(iv) The Alcohol and Drugs Questionnaire (ADQ)

The Alcohol and Drugs Questionnaire used in this study is a self-report questionnaire. Research has suggested that clients under-report alcohol and drug-related behaviour on self-report questionnaires (e.g. Leigh *et al.*, 1998; Lemmens *et*

al., 1992). It is possible that clients attending treatment are even more likely to under-report this behaviour, as the information becomes part of their clinical file which then becomes property of the NHS.

It is difficult to assess alcohol and drug related behaviour and perhaps in retrospect, administering a separate alcohol and drug questionnaire as part of the research protocol may have reduced under-reporting, if there was indeed under-reporting. If an alcohol questionnaire was administered during the research session, and clients could be ensured of confidentiality, this may have yielded more frequent and severe alcohol and drug use. However, as the treatment centre screened for alcohol and drug use before commencing treatment, it is also possible that the measure of alcohol and drug use is accurate in this sample.

4.6 FUTURE RESEARCH

The results of this study hold exciting opportunities for future research. They fall into the following categories: treatment outcome, memory and PTSD, and models of PTSD.

4.6.1 Treatment Outcome: Improving Treatment

The results of this study suggest that verbal memory is important to the recovery of clients with PTSD attending CBT treatment. It is possible that introducing a verbal memory screening task prior to therapy could identify clients who are likely to meet the diagnosis eight sessions later. Future research is needed to look at improving treatment through repetition and conducting CBT at a slower pace for those clients

who are identified as having poor verbal memory. A randomised study that assigns clients with PTSD and poor verbal memory to CBT with repetition as one group, and clients with PTSD and poor verbal memory to CBT without repetition as another group could look at the rate of improvement in these clients.

Having identified clients with poor verbal memory prior to therapy, outcome studies could also compare CBT with repetition to non-verbal therapies, such as EMDR or neurofeedback for these clients.

Finally, outcome studies could look at CBT with memory improvement strategies versus CBT without memory improvement strategies to see if improving memory has an effect on treatment outcome.

4.6.2 Memory and PTSD

(i) Memory, Attention, and Executive Function

This study did not find attention and executive function difficulties in clients with PTSD. It is possible that in a larger sample or with a more comprehensive assessment of executive function, these difficulties would become apparent. Thus, a future study could look more specifically at measures of attention and executive function, administering the entire TEA or the Trail Making Test of the Halstead-Reitan neuropsychological test battery (Reitan & Wolfson, 1993), for example.

Vasterling *et al.* (1998) found that high intrusions and low avoidance were associated with poor functioning on measures of attention. Studies are needed to look at PTSD symptomatology and specific measures of memory and attention to determine if

PTSD sufferers with high levels of avoidance and low levels of intrusion also exhibit difficulties.

(ii) Overgeneral Memory

This study did not find an association between overgeneral memory and avoidance of intrusive memories. Possibly a larger sample size would be needed to investigate this more fully, and possibly the inclusion of more questionnaire items that accurately assess avoidance of intrusive memories.

4.6.2.1 Mechanism of Memory Difficulty

(i) The HPA Axis

It is difficult to determine the mechanism of poor verbal memory in the present study. It is possible that the HPA axis is implicated and these clients have an attenuated HPA axis. Further research is needed to compare cortisol levels in clients with poor memory and PTSD and cortisol levels in clients with intact memory and PTSD to determine if the HPA axis is implicated as a mechanism of memory difficulty.

(ii) Negative Appraisals

One possible way of researching the role of perceived success-failure on the performance of adults with PTSD would be to carry out a memory study that gives false feedback. This could be done using a paired-associate paradigm. Subjects' performance could be investigated in two conditions, one in which they are given false feedback, and the other in which they are given accurate feedback. If performance differs significantly between the two conditions, then it is possible that

subjects with PTSD are sensitive to failure and this affects their memory performance. In order to determine whether such sensitivity has an explanatory role in the memory performance of subjects with poor memory, it would be necessary to conduct this sort of study for two sets of samples: subjects with PTSD and intact memory, and subjects with PTSD and poor memory. One could then compare the change in performance in the false feedback condition between the two groups of subjects.

Another important study in this area would be to identify subjects with PTSD and poor memory and to assess their self-talk qualitatively during administration of a memory task.

4.6.3 Neurobiological Models of PTSD

In PTSD, it is possible that the trauma memory is under-consolidated and re-presented in the form of intrusions in a continual attempt to consolidate it and place it within the long-term memory store. Intrusions may represent a failure of the hippocampal complex to consolidate the memory and transfer it to the neocortex.

Neuroimaging studies are needed to look at intrusions and the hippocampal complex. Subjects with high levels of intrusion may also have hippocampal atrophy which may impede consolidation of the trauma memory. MRI studies could further investigate brain regions associated with specific PTSD symptom profiles.

However, as it would be difficult and unethical to scan a client while they are experiencing high levels of intrusions, functional neuroimaging studies of related processes may be a more accessible method of investigating this area. For example, functional MRI could be used to look at brain activity during thought suppression experiments. Thought suppression is associated with intrusions (Ehlers & Clark, 2000), and thus, this sort of study may shed light on the brain regions associated with intrusive thoughts. Such information would further our understanding of the neurobiological underpinnings of PTSD.

4.7 CONCLUSION

This study investigated the memory, attention and learning profiles of 27 adults diagnosed with PTSD who presented at a specialist treatment centre prior to commencing cognitive-behavioural therapy. The contribution of these variables to therapeutic outcome was investigated in a follow-up sample of 23 participants. Verbal memory was found to significantly predict outcome at session eight, and the theoretical, clinical and research implications of this were discussed.

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APPENDIX 1

DSM-IV Criteria for PTSD

A. The person has been exposed to a traumatic event in which both the following were present:
   (1) The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others
   (2) The person's response involved fear, helplessness, or horror. *Note:* In children, this may be expressed instead by disorganised or agitated behaviour.

B. The traumatic event is persistently re-experienced in one (or more) of the following ways:
   (1) Recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. *Note:* In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.
   (2) Recurrent distressing dreams of the event. *Note:* In children, there may be frightening dreams without recognisable content.
   (3) Acting or feeling as if the traumatic event were recurring (includes a sense of episodes, including those that occur on awakening or when intoxicated). *Note:* In young children, trauma-specific re-enactment may occur.
   (4) Intense psychological distress at exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.
   (5) Physiological reactivity on exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:
   (1) Efforts to avoid thoughts, feelings, or conversations associated with the trauma.
   (2) Efforts to avoid activities, places, or people that arouse recollections of the trauma.
   (3) Inability to recall an important aspect of the trauma.
   (4) Markedly diminished interest or participation in significant activities.
   (5) Feeling of detachment or estrangement from others.
   (6) Restricted range of affect (e.g. unable to have loving feelings).
   (7) Sense of foreshortened future (e.g. does not expect to have a career, marriage, children, or a normal life span).

D. Persistent symptoms of increased arousal (not present before the trauma) as indicated by two (or more) of the following:
   (1) Difficulty falling or staying asleep.
   (2) Irritability or outbursts of anger.
   (3) Difficulty concentrating.
   (4) Hypervigilance
   (5) Exaggerated startle response.

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

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

Specify if:

Acute: if duration of symptoms is less than three months.

Chronic: if duration of symptoms is three months or more.

Specify if:

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

APPENDIX 2

CAMDEN & ISLINGTON
Community Health Services NHS Trust
Your Partner for Health
LOCAL RESEARCH ETHICS COMMITTEE
Research Office, 3rd Floor, West Wing,
St Pancras Hospital Conference Centre
St Pancras Hospital
Tel: 0171 530 3376 Fax: 0171 530 3235
e-mail: research.office@dial.pipex.com
Chair: *Stephanie Ellis* Administrator: *Michael Peat*

Ms. Jennifer Wild
Sub-Department of Clinical Health Psychology
University College London
Gower Street
London
WC1E 6BT

Dear Ms. Wild

Application No: **99/37** (please quote in all further correspondence)
Title: **Memory impairment as a predictor of treatment failure in Chronic Post-Traumatic Stress Disorder *(memory functioning before therapy)***

The Local Research Ethics Committee considered the above application at its meeting on 29th March 1999. I am pleased to inform you that it approved this research project, subject to the following point being addressed:

(i) The Patient Information Sheet should state that this project is part of a D.Clin.Psych. degree.

Please could you write and inform **Angela Williams** of the start date of your project, at the above address. Please note that the following general conditions of approval apply:

- Investigators must ensure that all associated staff, including nursing staff, are informed of research projects and are told that they have the approval of the Local Research Ethics Committee.

- If data are to be stored on a computer in such a way as to make it possible to identify individuals then the project must be registered under the Data Protection Act 1984. Please consult your department data protection officer for advice.

- The Committee *must* receive immediate notification of any adverse event or unforeseen circumstances arising out of the trial.

- The Committee *must* receive notification: (a) when the study is complete; (b) if it fails to start or is abandoned; (c) if the investigator/s change; and (d) if any amendments to the study are proposed or made.

- The Committee will request details of the progress of the research project periodically (i.e. annually) and require a copy of the report on completion of the project.

Please forward any other requested additional information and/or amendments regarding your study to the Administrator, Michael Peat, at the above address. If you have any queries, please do not hesitate to contact him on tel: 0171 530 3376.

Yours sincerely

Stephanie Ellis
CHAIR

CAMDEN & ISLINGTON
Community Health Services NHS Trust
Your Partner for Health

LOCAL RESEARCH ETHICS COMMITTEE

Research Office, 3rd Floor, West Wing,
St Pancras Hospital Conference Centre
St Pancras Hospital
Tel: 0171 530 3376 Fax: 0171 530 3235
e-mail: research.office@dial.pipex.com
Chair: *Stephanie Ellis* Administrator: *Michael Peat*

27th April 1999

Ms. Jennifer Wild
Sub-Department of Clinical Health Psychology
University College London
Gower Street
London
WC1E 6BT

Dear Ms. Wild

Application No: 99/37 (please quote in all further correspondence)
Title: **Memory impairment as a predictor of treatment failure in Chronic Post-Traumatic Stress Disorder** ***(memory functioning before therapy)***

Thank you for your fax dated 20th April 1999. I am pleased to inform you that the revised Patient Information Sheet for this project has been approved. Please could you write and inform **Angela Williams** of the start date of your project, at the above address.

Please note that the following general conditions of approval apply:

- Investigators must ensure that all associated staff, including nursing staff, are informed of research projects and are told that they have the approval of the Local Research Ethics Committee.

- If data are to be stored on a computer in such a way as to make it possible to identify individuals then the project must be registered under the Data Protection Act 1984. Please consult your department data protection officer for advice.

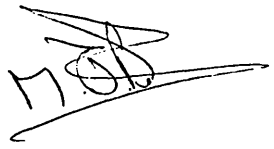
- The Committee *must* receive immediate notification of any adverse event or unforeseen circumstances arising out of the trial.

- The Committee *must* receive notification: (a) when the study is complete; (b) if it fails to start or is abandoned; (c) if the investigator/s change; and (d) if any amendments to the study are proposed or made.

- The Committee will request details of the progress of the research project periodically (i.e. annually) and require a copy of the report on completion of the project.

Please forward any other requested additional information/amendments regarding your study to the Ethics Committee Administrator, at the above address. If you have any queries, please do not hesitate to contact Michael Peat or myself at the above address.

Yours sincerely



pp. Stephanie Ellis
Committee Chair

APPENDIX 3

INFORMATION SHEET
The Traumatic Stress Clinic
73 Charlotte Street, London W1P 1LB
Tel: 0171-530-3666

STUDY: MEMORY FUNCTIONING BEFORE THERAPY
INVESTIGATORS: JENNIFER WILD, Clinical Psychologist in Training
DR. PETER SCRAGG, Clinical Psychologist

Dear Client,

I would like to invite you to take part in this study which is part of my Doctorate in Clinical Psychology. The study is looking at memory functioning before receiving therapy. The aim is to understand whether the trauma we've endured in the past affects the way our memory works today. An understanding of how past trauma affects memory will help us to develop new treatment strategies. This will enable us to provide maximum benefit to all clients.

Your participation will help to clarify the effect of trauma on memory.

You will be asked to attend the clinic at a time that is convenient for you. The testing will take no more than 3 hours during which time you will be given breaks for refreshments. The data collected from your participation will be completely confidential and destroyed once the study is completed.

You do not have to take part in this study if you do not want to. If you decide to take part you may withdraw at any time without having to give a reason. Your decision whether or not to take part will not affect your care and management in any way.

If you require further information, please feel free to ask me directly or to contact me by phone at the above-noted telephone number.

Thank you for considering this research.

.....
Jennifer Wild, B.Sc. (Hons), M.Ed.

Title of Study: Memory Functioning Before Therapy
Investigator's Name: Jennifer Wild, Clinical Psychologist in Training

To be completed by the client. Delete as necessary

1. I have read the information sheet about this study. YES/NO

2. I have had an opportunity to ask questions and discuss this study. YES/NO

3. I have received satisfactory answers to all my questions. YES/NO

4. I have received sufficient information about this study. YES/NO

5. Which health professional have you spoken to about this study?

6. I understand that I am free to withdraw from this study : -

   *at any time
   *without giving a reason for withdrawing
   *without affecting my future medical care YES/NO

7. Do you agree to take part in this study? YES/NO

Signed .....Date.....

Name in Block Letters.....

Signature of Investigator.....

APPENDIX 4

NAME:..... *DATE*.....

THINKING OF THE PAST MONTH, PLEASE ANSWER THE FOLLOWING BY CIRCLING THE ANSWER THAT BEST FITS YOU:

1	**In the past month, have you ever had unwanted memories of the traumatic event?** *No* *Once or Twice* *Once or Twice a Week* *Several Times a Week* *Daily or Almost Every Day*	**How much distress did the memories cause you?** *None* *Mild – mild disruption of activities* *Moderate – some disruption of activities* *Severe – marked disruption of activities* *Extreme – extreme disruption of activities*
2	**In the past month, have you ever had unpleasant dreams about the event?** *No* *Once or Twice* *Once or Twice a Week* *Several Times a Week* *Daily or Almost Every Day*	**How much distress did the dreams cause you?** *None* *Mild – mild disruption of sleep* *Moderate – some disruption of sleep* *Severe – marked disruption of sleep* *Extreme – extreme disruption of sleep*
3	**In the past month, have you ever suddenly acted or felt as if the event were happening again?** *No* *Once or Twice* *Once or Twice a Week* *Several Times a Week* *Daily or Almost Every Day*	**How much did it seem like the event was happening again?** *Not at all* *A little bit* *It was quite real* *It was very real* *It was extremely real*
4	**In the past month, have you gotten emotionally upset when something reminded you of the event?** *No* *Once or Twice* *Once or Twice a Week* *Several Times a Week* *Daily or Almost Every Day*	**How much distress did the reminders cause you?** *None* *Mild – mild disruption of activities* *Moderate – some disruption of activities* *Severe – marked disruption of activities* *Extreme – extreme disruption of activities*
5	**In the past month, have you ever had any physical reactions (i.e. heart racing, sweating or feeling shaky) when something reminded you of the event?** *No* *Once or Twice* *Once or Twice a Week* *Several Times a Week* *Daily or Almost Every Day*	**How strong were the reactions?** *No physical reactions* *Mild physical reactions* *Moderate physical reactions* *Severe physical reactions* *Extreme physical reactions*
6	**In the past month, have you ever tried to avoid thoughts or feelings about the event?** *No* *Once or Twice* *Once or Twice a Week* *Several Times a Week* *Daily or Almost Every Day*	**How much effort did you make to avoid thoughts or feelings about the event?** *No effort* *A little bit of effort* *Quite a bit of effort* *Lots of effort* *Huge amounts of effort*

7	**In the past month, have you tried to avoid certain activities, places or people that reminded you of the event?** *No* *Once or Twice* *Once or Twice a Week* *Several Times a Week* *Daily or Almost Every Day*	**How much effort did you make to avoid activities, places or people?** *No effort* *A little bit of effort* *Quite a bit of effort* *Lots of effort* *Huge amounts of effort*
8	**In the past month, have you had difficulty remembering some important part of the event?** *No* *Once or Twice* *Once or Twice a Week* *Several Times a Week* *Daily or Almost Every Day*	**How much difficulty did you have recalling important parts of the event?** *No difficulty* *A little bit of difficulty* *Quite a bit of difficulty* *Lots of difficulty* *Huge amounts of difficulty*
9	**In the past month, have you been less interested in activities that you used to enjoy?** *No* *Once or Twice* *Once or Twice a Week* *Several Times a Week* *Daily or Almost Every Day*	**How strong was your loss of interest?** *No loss of interest* *Mild loss of interest* *Quite a bit of loss of interest* *Marked loss of interest* *Complete loss of interest*
10	**In the past month, have you felt distant or cut off from other people?** *No* *Very little of the time* *Some of the time* *Much of the time* *Most or all the time*	**How strong were your feelings of being distant or cut off from others?** *No feelings of being cut off* *Mild feelings of being cut off* *Definite feelings of being cut off* *Severe feelings of being cut off* *Extremely strong feelings of being cut off*
11	**In the past month, have there been times when you felt emotionally numb or had trouble experiencing feelings like love or happiness?** *No* *Very little of the time* *Some of the time* *Much of the time* *Most or all the time*	**How much difficulty did you have experiencing emotions like love or happiness?** *No difficulty* *A little bit of difficulty* *Quite difficult to experience* *Severe difficulty* *Extreme difficulty*
12	**In the past month, have there been times when you felt there is not need to plan for the future, that somehow it will be cut short?** *No* *Very little of the time* *Some of the time* *Much of the time* *Most or all the time*	**How strong was this feeling that your future would be cut short?** *No feelings that it would be cut short* *Mild feelings that it would be cut short* *Strong feelings that it would be cut short* *Severe feelings of that it would be cut short* *Extremely strong feelings that it would be short*

13	**In the past month, have you had any problems falling or staying asleep?** *No* *Once or Twice* *Once or Twice a Week* *Several Times a Week* *Daily or Almost Every Day*	**How much of a problem did you have with your sleep?** *No problem* *A little bit of a problem* *Quite a bit of a problem* *A severe problem* *An extreme problem*
14	**In the past month, have you felt especially irritable or showed strong feelings of anger?** *No* *Once or Twice* *Once or Twice a Week* *Several Times a Week* *Daily or Almost Every Day*	**How strong was your anger?** *No anger* *Mild anger* *Quite a bit of anger* *Severe anger* *Extreme anger*
15	**In the past month, have you found it difficult to concentrate on what you were doing or on things going on around you?** *No* *Very little of the time* *Some of the time* *Much of the time* *Most or all the time*	**How difficult was it for you to concentrate?** *No difficulties* *A little difficulty* *Quite a bit of difficulty* *Severe difficulties* *Extreme difficulties*
16	**In the past month, have you been especially alert or watchful even when there was no real need to be?** *No* *Very little of the time* *Some of the time* *Much of the time* *Most or all the time*	**How hard did you try to be watchful of things going on around you?** *I didn't try* *I tried a little bit* *I tried hard to be watchful* *I tried really hard* *I tried extremely hard*
17	**In the past month, have you had any strong startle reactions?** *No* *Once or Twice* *Once or Twice a Week* *Several Times a Week* *Daily or Almost Every Day*	**How strong were these startle reactions?** *No startle reactions* *Mild startle reactions* *Strong startle reactions* *Severe startle reactions* *Extreme startle reactions*
18	**Overall, how much distress have the symptoms mentioned in this questionnaire caused you in the past month?** *No distress* *Mild distress* *Moderate distress* *Severe distress* *Extreme distress*	**Overall, how much have the symptoms mentioned in this questionnaire affected your relationships?** *No impact* *Mild impact* *Moderate impact* *Severe impact* *Extreme impact*

19	**Overall, how much have the symptoms affected your ability to work?** *No impact* *Mild impact* *Moderate impact* *Severe impact* *Extreme impact*	
20	**In the past month, have you felt guilty about anything you did or didn't do during the event?** *No* *Very little of the time* *Some of the time* *Much of the time* *Most or all the time*	**How strong were these feelings of guilt?** *No feelings of guilt* *Mild feelings of guilt* *Strong feelings of guilt* *Severe feelings of guilt* *Extreme feelings of guilt*
21	**In the past month, have there been times when you felt out of touch with things going on around you?** *No* *Once or Twice* *Once or Twice a Week* *Several Times a Week* *Daily or Almost Every Day*	**How strong was this feeling of being out of touch?** *No feelings of being out of touch* *Mild feelings of being out of touch* *Strong feelings of being out of touch* *Severe feelings of being out of touch* *Extreme feelings of being out of touch*
22	**In the past month, have there been times when things going on around you have seemed unreal or very unfamiliar?** *No* *Once or Twice* *Once or Twice a Week* *Several Times a Week* *Daily or Almost Every Day*	**How strong was this feeling?** *Not strong* *Mild* *Quite strong* *Very strong* *Extremely strong*
23	**In the past month, have there been times when you felt as if you were outside of your body, watching yourself as if you were another person?** *No* *Once or Twice* *Once or Twice a Week* *Several Times a Week* *Daily or Almost Every Day*	**How strong was this feeling?** *Not strong* *Mild* *Quite strong* *Very strong* *Extremely strong*

THANK YOU VERY MUCH FOR COMPLETING THE QUESTIONS

APPENDIX 5

ALCOHOL & DRUGS QUESTIONNAIRE (ADQ)

Have you ever felt you should cut down on your drinking?
Have people annoyed you by criticizing your drinking?
Have you every felt bad or guilty about your drinking?
Have you every had a drink first thing in the morning to steady your nerves or to get rid of a hangover (Eye-opener)?

Fill in the amounts and type of drinks that you have drunk in the last seven days putting the units at the bottom.

MON	TUES	WED	THURS	FRI	SAT	SUN

1 Unit = half pint of ordinary beer, single measure of spirits, 1 glass of wine, 1 small glass sherry
1+ Units = 1 standard can of lager/bitter
2+ Units = 1 strong can of lager
4 Units = 1 extra strong can of lager

Has your drinking INCREASED/DECREASED/STAYED THE SAME since the incident?

Look at the medicines in Section A below. What tablets is your doctor giving you at the moment.

.....

.....

How long have you been taking these medicines?

.....

Look at the drugs at the bottom of the page in Section B. Do you use any of them?

.....

How much do you spend on drugs a week?

.....

How do you take the drugs? Injection/Smoking/By Mouth

SECTION A
PRESCRIBED DRUGS
Sleeping pills
Anti-depressants, pain killers
Tranquillizers
Others

SECTION B
NON-PRESCRIBED DRUGS
Cannabis, Glue/Solvents, Heroin/other opiates,
Cocaine/crack, Ecstasy/LSD/Magic Mushrooms
Amphetamines/Speed, Poppers, Benzodiazepines,
Tranquillizers/Sleeping pills, any other pills