

University of Warwick institutional repository: <http://go.warwick.ac.uk/wrap>

**A Thesis Submitted for the Degree of DClInPsych at the University of
Warwick**

<http://go.warwick.ac.uk/wrap/2358>

This thesis is made available online and is protected by original copyright.

Please scroll down to view the document itself.

Please refer to the repository record for this item for information to help you to cite it. Our policy information is available from the repository home page.

Cognitive Function in Posttraumatic Stress Disorder

Claire L Isaac, PhD

A thesis submitted in partial fulfilment of the requirements for the degree of Doctor
of Clinical Psychology

Coventry University, School of Health and Social Sciences and
University of Warwick, Department of Psychology.
September 2002

TABLE OF CONTENTS

LIST OF TABLES.....	6
LIST OF FIGURES.....	7
ACKNOWLEDGEMENTS.....	8
DECLARATION.....	9
SUMMARY.....	10
CHAPTER 1: LITERATURE REVIEW	
Is Posttraumatic Stress Disorder Associated with Specific Deficits in Memory?	
Abstract	11
Introduction	12
Studies of cognitive function in PTSD.....	14
What cognitive deficits would be predicted in PTSD?	21
Deficits related to frontal lobe dysfunction.....	21
Performance on tests of attention	22
Performance on tests of Executive Function.....	24
Evidence for frontal-lobe related memory dysfunction	27
Hypotheses of frontal-lobe related cognitive deficits.....	30
Hippocampal related memory deficits	32
Evidence for hippocampal volume reductions in PTSD	33
Evidence of deficits in episodic memory	37
Amygdala related memory deficits	39

Evidence for abnormalities in amygdala functioning.....	39
Role of the amygdala in emotional memory	40
Evidence for amygdala related memory deficits	41
Summary and Conclusions	41
References	44

CHAPTER 2: MAIN PAPER

Forgetting of Emotional and Non-emotional Stimuli in People with Posttraumatic Stress Disorder.

Abstract	58
Introduction	59
Method.....	64
Participants	64
Materials	67
Design.....	69
Procedure.....	70
Results	73
Free Recall.....	74
Recognition	77
Discussion	79
References	86

CHAPTER 3: BRIEF PAPER

Identification of Negative Facial Expressions in Posttraumatic Stress Disorder.

Abstract	94
Introduction	95
Method.....	98
Participants	98
Materials	101
Procedure.....	101
Results	102
Discussion	107
References	110

CHAPTER 4: RESEARCH REVIEW

Is Neuropsychology Relevant to Clinical Psychology?

Is Neuropsychological Research Clinically Relevant?	114
Is PTSD best understood as a psychological or biological phenomenon?	120
References	124

APPENDICES

Appendix 1: Ethics-related information	126
Appendix 2: Background information on participants	139
Appendix 3: Word stimuli used in Main Paper	140

Appendix 4: Raw data from Main Paper.....	141
Appendix 5: Examples of face stimuli from Ekman-Friesen series.....	143
Appendix 6: Raw data from Brief Paper.....	148
Appendix 7: Instructions for authors.....	150

List of Tables

Chapter 1

Table 1: Studies investigating cognitive function in PTSD.....	14
--	----

Chapter 2

Table 1: Background data for groups of PTSD and control participants	65
--	----

Table 2: Symptom severity (PDS & R-IES) in PTSD group.....	66
--	----

Chapter 3

Table 1: Background data for groups of PTSD and control participants	98
--	----

Table 2: Symptom severity (PDS & R-IES) in PTSD group.....	99
--	----

Table 3: Performance of PTSD and control participants on identification of amygdala related and non-amygdala related negative facial expressions.....	101
---	-----

Table 4: Mean intensity ratings of PTSD and control participants for amygdala and non-amygdala related negative facial expressions.....	102
--	-----

Table 5: Correlation coefficients for identification of amygdala related and non-amygdala related negative facial expressions and PTSD symptomatology	103
---	-----

List of Figures

Chapter 2

- Figure 1: Free recall performance of PTSD and control groups
for emotional and non-emotional word lists 75
- Figure 2: Recognition performance (d') of PTSD and control
groups for emotional and non-emotional word lists 78

Acknowledgements

The work that comprises this thesis was carried out under the supervision of Delia Cushway, my clinical supervisor, and Greg Jones, my academic supervisor. I would like to thank both for their enthusiasm, advice and encouragement throughout the planning and execution of this work. I would also particularly like to thank Trish Gooding for a number of helpful discussions. I am grateful to Dave Quinn, Graham Jones, Graham Bryan, Diana Lorenc and Clare Wright who initially approached potential participants with regard to participating in these studies. I realise that I have benefited from the great goodwill that they engendered in the people that they saw on a therapeutic basis. Many thanks also to Dave for loaning me the standard neuropsychological tests used in the studies. I am also grateful to William Isaac for trouble-shooting the printing process yet again. Finally, and perhaps most of all, I should like to express my sincere gratitude to the anonymous participants, both patients and controls, who generously gave up extended periods of time to participate in these studies. They certainly made data collection into a pleasure rather than a chore for me.

Declaration

The work contained in this thesis was carried out under the supervision of Dr Delia Cushway and Professor Greg Jones.

Design of the studies and all work contained in this thesis is the author's own, carried out in collaboration with the above supervisors. Authorship of any papers arising from this work will be shared with them.

This thesis has not been submitted for a degree at any other university.

The literature review is being prepared for submission to *Clinical Psychology Review* (Isaac, Cushway & Jones, in preparation). The main paper is being prepared for submission to *Neuropsychologia* (Isaac, Cushway & Jones, in preparation) and the brief paper is being prepared for submission to *Cognition and Emotion* (Isaac, Jones & Cushway, in preparation).

Summary

Complaints of poor memory by individuals with posttraumatic stress disorder (PTSD) have engendered research into attention and memory functioning in this disorder. Due to numerous methodological difficulties encountered in research with this group, results have been inconclusive. In Chapter 1 of this thesis the existing literature is reviewed to ascertain whether there is any evidence of a specific pattern of memory disorder associated with PTSD. Studies are reviewed for evidence of cognitive deficits relating to the structures of the limbic system, dysfunction in which has been implicated in PTSD. It is concluded that there is relatively good evidence of deficits related to probable frontal lobe functions. However, there is very little evidence of hippocampal related disorders and no studies have investigated memory functions relating to hypothesised roles of the amygdala in this group.

In chapters 2 and 3 experiments are described that aim to investigate cognitive abilities related to amygdala functioning in PTSD. Chapter 2 investigates an hypothesised role of the amygdala in the consolidation of memory for emotional material. The results confirm the possibility of amygdala dysfunction in PTSD by showing that on a test of free recall participants with PTSD forgot emotional word stimuli at a faster rate than control participants, whereas non-emotional stimuli were forgotten at a more normal rate. Chapter 3 investigated a second hypothesised role for the amygdala in the recognition of facial expressions of fear and anger. Results showed that PTSD participants were somewhat impaired in their recognition of these expressions, which contrasted with an enhanced ability, associated with symptoms of hyperarousal, in identifying other negative facial expressions.

In Chapter 4, the relevance of neuropsychological research to Clinical Psychology is discussed. It is argued that such research is vital if we are to fully understand the difficulties clients could face on a day-to-day basis.

CHAPTER 1 : LITERATURE REVIEW

Is Posttraumatic Stress Disorder Associated with Specific Deficits in Memory?

Abstract

Identifying the cognitive deficits associated with posttraumatic stress disorder (PTSD) has been the focus of a number of recent studies. These have reported deficits in episodic memory, attention and executive processes. There are, however, major difficulties in conducting such studies, which could influence the results reported. The first aim of this paper is to describe these difficulties. The second aim is to review extant studies as they relate to specific areas of cognitive functioning. These are functions relating to the hypothesised role of the limbic system and the rationale for investigating them comes from the results of recent neuroimaging studies, which have indicated dysfunction in this system in people with PTSD. It is argued that future studies should be more theoretically driven and focus on more specific functions, if the likely subtle deficits associated with PTSD are to be identified.

Introduction

PTSD can occur in individuals who have been exposed to traumatic experiences of exceptional severity. The fourth edition of the Diagnostic and Statistical Manual (DSM IV; American Psychiatric Association (APA), 1994) describes three clusters of symptoms that are the hallmark of this disorder. The first are symptoms of re-experiencing the trauma including flashbacks and nightmares. The second are avoidance behaviours including avoiding thinking about the trauma, avoiding situations and reminders associated with the trauma and withdrawal from other activities. The third are persistent symptoms of increased physiological arousal characterised by hypervigilance to any signs of threat in the environment and an exaggerated startle response. The diagnosis of PTSD was first recognised by the APA in 1980 when it was included in the third edition of the DSM. Since then much research has focused on more precisely defining the disorder, both in terms of neurophysiology and cognitive profile, and in developing effective treatments for it.

Long before PTSD was recognised as a specific psychiatric diagnosis, the effects of trauma on the mental wellbeing of individuals had already been well documented. Of particular interest has been the nature of the traumatic memories, experienced as flashbacks. These appear to differ from normal memories in that they are not under conscious control and can be involuntarily elicited by a wide range of internal and external cues. These memories also lack a sense of pastness and during flashbacks individuals feel that they are truly re-experiencing the traumatic episode, rather than remembering it. It has been hypothesised that it is this re-experiencing that leads to the avoidance and hyperarousal symptoms (e.g. Krystal, Bennet, Bremner, Southwick & Charney, 1995).

The vividness of these traumatic memories contrasts with the often poor ability of individuals with PTSD to voluntarily recollect details of the traumatic memory. In addition, many studies have documented subjective complaints about memory and concentration abilities related to everyday situations, not involving memory of the traumatic experience in a great many individuals suffering from PTSD (e.g. Wolfe & Charney, 1991).

The fact that subjective complaints of memory and concentration difficulties in individuals with PTSD are such a consistently reported feature implies that such deficits can have far-reaching consequences in the everyday lives of people with PTSD. They may have severe difficulty in coping with relatively simple tasks such as reading documents, following instructions and keeping track of day to day activities. As a result work may be impossible. It is obviously important, from a clinical perspective, therefore, that cognitive deficits in PTSD should be more precisely defined so that they can be the targets of specific and appropriate rehabilitation strategies.

The aim of this paper is to review the existing literature on episodic memory functioning in individuals with PTSD and to evaluate the evidence that the disorder is associated with a particular pattern of memory dysfunction. The alternative hypothesis is that the memory deficits that have been reported can be more parsimoniously explained in terms of deficits in attention and concentration abilities (e.g. Stein, Hanna, Koverola, Torchia & McClarty, 1997). When conducting research into the cognitive effects of PTSD a number of serious methodological difficulties are encountered. These will be described as it is important to take them into account when considering the findings reported. In recent years, neuroimaging studies

have yielded data concerning structural and functional abnormalities in the brains of individuals with PTSD. These studies, and the abnormalities that they reveal, have provided a rationale for investigating certain specific cognitive functions in PTSD. In reviewing the existing literature on episodic memory deficits these specific hypotheses will be the focus of attention.

To date, there has been some inconsistency in the literature associated with PTSD in the terms used to describe different memory processes. It is first necessary, therefore, to operationally define the terms that will be used in this paper. We will use the terms as described in Baddeley (1990). Thus, 'short-term memory' applies to that memory which persists in the few seconds following presentation of stimuli. 'Long-term memory' will be used to describe new learning that persists beyond the boundaries of STM. The term 'episodic memory' will be used to label the kind of memory that is the focus of this review. This term was introduced by Tulving (1984) and is defined as the retention of knowledge about personally experienced events and their temporal relations in subjective time and the ability to mentally 'travel back' in time.

Studies of cognitive function in PTSD

This review will focus on studies investigating episodic memory functioning in PTSD for non-trauma related material. A literature search was conducted using the Psychinfo database. Appropriate papers were selected from searches using the following terms: 'cognitive and posttraumatic stress disorder', 'memory and posttraumatic stress disorder', 'frontal and posttraumatic stress disorder' and 'hippocampus and posttraumatic stress disorder'. Papers were selected that used an experimental design to compare performance on cognitive tests in a group of patients with PTSD and a control group. Papers were also included that reported the results

of regression analysis on a single group of participants. Papers were excluded if PTSD accompanied exposure to a toxin that could itself have caused neurological damage. Any references in the selected papers that had not been included in the search results were also included. This yielded a total of 25 studies. A list of the studies appears in Table 1 with a description of the patient groups included.

Table 1 . *Studies investigating cognitive function in PTSD*

Study	Patient group
Dalton, Pederson & Ryan (1989)	Vietnam veterans (current PTSD)
Gil, Calev, Greenberg, Kugelmas & Lerer, (1990)	Mixed aetiology (current PTSD)
Sutker Winstead, Galina & Allain, (1991)	Korean POW veterans
Bremner et al. (1993)	Vietnam veterans (current PTSD)
Gurvits et al (1993)	Vietnam veterans (current PTSD)
Uddo, Vasterling, Brailey & Sutker (1993)	Vietnam veterans (current PTSD)
Zalewski, Thompson, & Gottesman (1994)	Vietnam veterans (lifetime PTSD)
Bremner, Randall, Scott, Bronen et al. (1995)	Vietnam veterans (current PTSD)
Bremner, Randall, Scott, Capelli et al. (1995)	Adults with histories of childhood abuse (current PTSD)
Sutker, Vasterling, Brailey & Allain, (1995)	Korean POWs
Yehuda et al. (1995)	Combat veterans (current PTSD)
Barrett, Green, Morris, Giles & Croft (1996)	Vietnam veterans (lifetime PTSD)
Litz et al. (1996)	Vietnam veterans (current PTSD)

Gurvits et al. (1996)	Vietnam veterans (current PTSD)
Jenkins, Langlais, Delis & Cohen (1998)	Rape survivors (current PTSD)
Vasterling, Brailey, Constans & Sutker (1998)	Gulf War veterans (current PTSD)
Moradi, Neshat-Doost, Taghavi, Yule & Dalglish. (1999)	Children (current PTSD)
Stein, Hanna, Vaerum & Koverola (1999)	Women with history of Childhood sexual abuse (77% with current PTSD)
Moradi, Taghavi, Neshat-Doost, Yule & Dalglish (2000)	Children & adolescents (current PTSD)
Sachinvala et al. (2000)	Vietnam veterans (current PTSD)
Gilbertson, Gurvits, Lasko, Orr & Pitman (2001)	Vietnam veterans (current PTSD)
Koenen et al. (2001)	Mixed aetiology (current PTSD)
Diamond, Muller, Rondeau & Rich (2001)	Adults with histories of childhood abuse
Vasterling et al. (2002)	Vietnam veterans (current PTSD)
Beers & De Bellis (2002)	Children (current PTSD)

In reviewing these studies it is clear that investigating cognitive function in PTSD is fraught with severe difficulties. It seems likely that some of the many inconsistencies in the literature can be attributed to this fact. Eight sources of difficulty have been identified and these are described below.

First, PTSD is often associated with co-morbid psychiatric disorders such as depression, anxiety and panic which have themselves been associated with specific information processing and memory deficits (see Williams, Watts, McLeod &

Matthews, 1997). If the cognitive deficits associated with PTSD are to be differentiated from those associated with other emotional disorders a means of controlling for this problem should be identified. To date, studies have differed in the approach they have taken in dealing with co-morbid disorders. Some have excluded patients with co-morbid disorders (e.g. Yehuda et al. 1995); Some have considered co-morbid disorders part of the spectrum of PTSD (e.g. Bremner, Randall, Scott, Bronen et al. 1995; Gil et al. 1990; Jenkins et al. 1998; Sutker et al. 1991; Vasterling et al. 1998) and others have controlled for the effects of co-morbid disorders by partialling these out during data analysis (e.g. Moradi et al. 1999; Sachinvala et al. 2000, Zalewski, Thompson & Gottesman, 1994). This results in some difficulty in comparing the results across different studies.

Second, and related to the first difficulty, some patients with PTSD also have current or past substance abuse issues as alcohol or illicit drugs may be used as a means of coping with distressing symptoms. Although the majority of studies reviewed in this paper control for current substance abuse, past substance abuse is rarely controlled for. This is likely to be particularly important in studies of patients who have a chronic and intractable form of PTSD. This can persist for many years, so if individuals use drugs or alcohol as coping aids, cognitive difficulties could be those relating to substance abuse (see Sullivan, Rosenbloom & Pfefferbaum, 2002) rather than PTSD.

Third, patients with PTSD may also be likely to have head injuries. This may be particularly important in those with combat histories and in those whose PTSD resulted from road traffic accidents. The majority of studies have excluded participants who have suffered loss of consciousness in excess of 10 minutes.

However, evidence suggests that even mild head injuries can lead to subtle cognitive deficits (e.g. Richardson, 1990).

The fourth difficulty relates to the chronicity of PTSD. Studies have included widely varying populations with regard to time since trauma. The majority of studies investigating cognitive deficits in patients with PTSD have recruited combat veterans. Of the 25 studies included in this review 16 focused on this population. Twelve of these were groups of Vietnam War veterans, two Korean War veterans and one Gulf War veterans. It can be assumed that if these individuals meet diagnostic criteria for current PTSD then it is a chronic, and possibly intractable, form of the disorder. Similarly, it could be assumed that in the three studies including women with childhood sexual abuse histories, PTSD would also be long-lasting. In contrast, three studies included children who had experienced trauma in the two years prior to testing. The other four studies included individuals with varying intervals since trauma. Time since trauma is likely to be important for a number of reasons. Yehuda (2000) suggests that biological abnormalities in stress hormones are likely to lead to a progressive course of the disorder. This implies that the pattern of cognitive deficits may change over time. Therefore, it is important to bear in mind the possibility that individuals with PTSD that has persisted over many years may not be representative of all individuals with PTSD. Similarly, studies including individuals with a history of maltreatment may be complicated by the fact that cognitive deficits may be related to neurodevelopmental history and again may not be representative of individuals who develop PTSD in adulthood.

The fifth difficulty may also be more relevant to combat veterans, and is the possibility that individuals may have been, knowingly or unknowingly, exposed to

neurotoxins. A study by Levy (1988) found a greater incidence of PTSD in Vietnam veterans exposed to Agent Orange. It is possible that PTSD associated with neurotoxin exposure and possible neurological damage may be different from that seen in cases with no exposure.

Sixth, inclusion criteria for PTSD diagnosis have varied across studies. The majority of studies have included patients with current PTSD. However, some have used the inclusion criterion of lifetime diagnosis (Barrett et al. 1996; Zalewski, Thompson & Gottesman 1994). Presumably, these studies included participants both with current PTSD and with past PTSD in which the symptoms may have completely resolved or no longer reached full DSM criteria. These groups are likely to be heterogeneous with regard to current symptoms. Whether cognitive deficits would be expected in such groups would depend first on the extent of their current symptomatology but also on whether PTSD can have lasting effects on cognitive function even after symptoms have resolved. It is notable that both these studies found no difference in cognitive functioning of patients and control subjects. However, given the likely heterogeneity of the patient group, the results are difficult to interpret.

Seventh, is the problem of selecting an appropriate control population. The majority of studies have used non-traumatized, non-psychiatric control groups (e.g. Bremner et al. 1993; Bremner, Randall, Scott, Bronen et al. 1995; Moradi et al. 1999; Sachinvala et al. 2000; Yehuda et al. 1995), others have used people exposed to trauma that have not developed PTSD (e.g. Gilbertson et al. 2001; Gurvits et al. 1993; Sutker et al., 1991; Vasterling et al. 1998). Some have included two control groups, for example Jenkins et al. (1998) included both individuals who had been exposed to trauma but who had not developed PTSD and a normal non-

traumatised, non-psychiatric control group. Finally, other studies have included both a psychiatric control group and a non-psychiatric, non-traumatised control group (e.g. Barrett et al. 1996; Gil et al. 1990; Litz et al. 1996; Zalewski, Thompson & Gottesman, 1994). In practice, the control group that researchers select would seem to depend on the aim of the study. Studies that have included a psychiatric control group aim to differentiate the cognitive effects of PTSD from those of other psychiatric disorders. Those that include a traumatised control group aim to differentiate the effects of PTSD from those accompanying exposure to traumatic episodes, whereas those that include a non-traumatised, non-psychiatric control group aim to describe the cognitive effects of PTSD in relation to normal functioning.

The eighth difficulty relates to the design of the studies rather than to the characteristics of the participants. The hypotheses in many studies tend to be very general and these have included a large battery of tests. The resulting large number of statistical comparisons means that there is a substantial risk of a type I error (see also Stein, Hanna et al. 1997). Some studies have tried to correct for this possibility by using a Bonferroni, or other type of correction. With a large number of comparisons and the resulting low alpha, however, these studies could run the risk of a type II error. For example, Gurvits et al. (1993) report no impairment across a range of measures in a group of Vietnam veterans with PTSD. However, the PTSD group performed at a numerically worse level than the control group on 31 out of the 35 scores reported - a result that would not be expected by chance.

The difficulties described above can lead to serious problems in interpreting the

results of studies. It is important to bear these difficulties in mind when considering the results reviewed below.

What cognitive deficits would be predicted in PTSD?

Recent research into the neurobiological basis of PTSD has provided a rationale for investigating certain areas of cognitive functioning. Neurobiological research has focused largely on the limbic system that has long been known to have a major role in both memory and emotion. The limbic system includes structures such as the hippocampus, amygdala and prefrontal cortex. Results from recent neuroimaging studies in PTSD have bolstered evidence for the importance of this system in the disorder. Evidence from cognitive studies of individuals with PTSD will therefore be examined in relation to the hypothesised functions of the regions included in the limbic system.

Deficits related to frontal lobe dysfunction

The motivation for investigating frontal lobe function in individuals with PTSD came largely from clinical impression. Tests of cognitive functioning have generally supported this impression and more recently functional imaging studies have revealed some frontal lobe abnormalities in people with PTSD. Abnormalities have been reported in various regions of the frontal lobes including orbitofrontal cortex (Rauch et al., 1996; Shin et al., 1999), anterior cingulate cortex (Bremner et al. 1999; Lanius et al. 2001; Liberzon et al. 1999; Rauch et al. 1996; Shin et al. 1997; Shin et al. 1999;) medial prefrontal cortex (Bremner et al. 1999; Lanius et al. 2001) and dorsolateral prefrontal cortex (Osuch et al, 2001).

Frontal lobe dysfunction has been associated with a number of cognitive deficits including those in attention, working memory, planning and organisation, perseveration of responses, problem solving and memory. All these functions can be understood as involving the functions of an executive system, however (see Mayes, 1988). Effects on memory are likely to occur because frontal lobe dysfunction disrupts strategic organisation of cognitive processes and patients with frontal lobe damage would be expected to perform poorly on those tasks that rely to a greater extent on these strategies. These include tests of free recall in comparison with item recognition, and tasks in which increased susceptibility to interference could affect performance (see Mayes, 1988). Frontal lobe lesions have also been found to disrupt immediate memory measured by tests of digit-span and spatial-span (see Mayes, 1988)

Attention and working memory processes are obviously vital for the normal functioning of episodic memory. If attention or working memory is impaired information will not be encoded into memory or will be encoded in an impoverished way, leaving the contents susceptible to interference.

Performance on tests of attention

Eleven studies have included specific measures of attention to investigate functioning in patients with PTSD (Dalton, Pederson & Ryan, 1989; Gil et al., 1990; Gilbertson et al., 2001; Gurvits et al. 1996; Sachinvala et al. 2000; Sutker et al. 1991; Sutker et al. 1995; Uddo et al. 1993; Vasterling et al., 1998; Vasterling et al., 2002; Zalewski, Thompson & Gottesman, 1994). Tasks ranged from simple digit-span tests (Gilbertson et al. 2001; Sachinvala et al. 2000; Sutker et al. 1991; Sutker et al. 1995; Uddo et al. 1993), to more complex tasks of attention (Gil et al. 1989; Gurvits et

al. 1996; Sachinvala et al. 2001; Vasterling et al. 1998; Vasterling et al 2002; Zalewski, Thompson & Gottesman 1994).

Three of the four studies investigating performance on digit-span tasks have reported impairment (Gilbertson et al. 2001; Sachinvala et al. 2000; Sutker et al. 1991). In the fourth study, Uddo et al. (1993) found that although performance on a digit span task was unimpaired in a group of patients with PTSD, performance on a spatial span task was impaired. In a regression analysis including only patient participants, Sutker et al. (1995) found that impairment on both digit-span and spatial span tasks was positively correlated with severity of PTSD symptoms. These results showing impairment are supported by those of a study focusing solely on attention processes in PTSD. Thus, Jenkins, Langlais, Delis & Cohen, (2000) reported impaired performance in a group of women with PTSD who were survivors of rape on the digit span task. Further analysis, however, revealed that patients were only impaired in the backward condition. This could indicate that patients are more likely to be impaired when more demands are made on working memory processes.

Studies looking at more complex aspects of attention in PTSD have also tended to report impairments. Gurvits et al. (1996) found that a group of Vietnam veterans with PTSD was impaired relative to veterans without PTSD on the Attention and Concentration index of the Wechsler Memory Scale-Revised (WMS-R). Importantly, the difference in performance between the two groups on the Wechsler Adult Intelligence Scale-Revised (WAIS-R) was not significant indicating that the result could not be accounted for in terms of lower IQ. Sachinvala et al. (2001) reported impairment across a range of three attention tests including a simple reaction time measure and measures of more complex attentional processes. Further evidence

suggests that PTSD may not affect all aspects of attention, however. For example, Vasterling et al. (1998, 2002) investigated four aspects of attention and in both studies found impairments on measures of sustained attention and encoding, but not on measures of focused attention or attentional shifting. Jenkins et al. (2000) also reported sustained attention impairments in women with PTSD who were survivors of rape in comparison with rape survivors without PTSD but no impairment on a measure of visuospatial selective attention. In contrast, Zalewski, Thompson & Gottesman (1994) reported normal performance on the Paced Auditory Serial Addition Task (PASAT), a difficult task of sustained attention in a group of individuals with PTSD. The reason for the discrepancy in results could be that the patients in the latter study had lifetime, rather than current diagnoses of PTSD.

There is good evidence therefore that at least some aspects of attention are impaired in PTSD.

Performance on tests of Executive Function

Twelve studies have included standardised tests of executive functioning. It should be noted, however, that such tests are not specifically sensitive to damage to the frontal lobes (e.g. Anderson, Damasio, Jones & Tranel, 1991). The most commonly employed have been the Wisconsin Card Sorting Test (WCST), the Trail Making Test (TMT) and the Controlled Oral Word Association Test (COWAT) including both letter fluency and category fluency. Six studies investigated performance on the WCST (Barrett et al. 1996; Diamond et al, 2001; Gilbertson et al. 2001; Gurvits et al. 1993; Vasterling et al. 1998; Vasterling et al. 2002). Of these, only one reported individuals with PTSD to be impaired relative to controls (Gilbertson et al. 2001). In this study, however, performance across a number of measures was made, but no

correction for multiple comparisons was used. This result could, therefore represent a type I error. In support of this Gilbertson et al. (2001) found that WCST performance did not discriminate between PTSD and control groups in a discriminant analysis. In addition, in a study using multiple regression analysis, Diamond et al. (2001) found no relationship between performance on the WCST and symptoms of PTSD.

Of six studies including the TMT, two reported individuals with PTSD to be impaired on part B of the task which relies to a greater extent on working memory processes than part A (Dalton, Pederson & Ryan, 1989; Gilbertson et al. 2001). Two others failed to find any impairment (Gurvits et al. 1993; Koenen et al. 2001) although there was a trend for such impairment on part B in the Gurvits (1993) study. Diamond et al (2001) found no relationship between performance on part B of the TMT and severity of PTSD symptomatology although Sutker et al. (1995) found such a relationship with a measure of attention and mental tracking that included the TMT. In a study investigating TMT performance in a group of Vietnam veterans with PTSD, Beckham, Crawford & Feldman, (1998) found that veterans with PTSD were impaired on both parts A and B of the task. However, impairment on part A could be accounted for by the effects of co-morbid diagnoses, medication and compensation seeking status, and so this impairment probably did not reflect the effects of PTSD.

Two of three studies looking at verbal fluency have found impairments in individuals with PTSD. Gil et al. (1989) found impairments on both letter and category fluency in comparison with a healthy control group whereas Uddo et al. (1993) found impairments on category but not letter fluency. Koenen et al. (2001) also found no

impairment on a test of letter fluency.

Performance of individuals with PTSD on the Standard Stroop paradigm has also been equivocal. Litz et al, (1996) found patients with PTSD to be impaired on this task in comparison with a healthy control group but not in comparison with a group of individuals with psychiatric diagnoses whereas Vasterling et al. (1998) and Vasterling et al (2002) found no performance decrements in combat veterans with PTSD in comparison to combat veterans without PTSD.

Results using standardised tests of executive functioning reveal equivocal evidence of performance impairments in PTSD. However, a recent study has highlighted the importance of investigating more specific hypotheses related to frontal lobe functions. Koenen et al. (2001) administered a battery of tests developed to tap functions of the frontal lobes, to a group of patients with PTSD and matched control participants. Results showed that individuals with PTSD were impaired on two tests of working memory. These were the Delayed Response task (DR) in which participants were required to remember the position of an object following a variable delay, and the Object Alternation (OA) task in which participants had to select the previously unrewarded position to gain reward. These tasks had previously been shown in animal studies to tap the functions of the dorsolateral prefrontal (DR task) and orbitofrontal (OA task) cortices. Patients were not, however, impaired on standardised tests of frontal lobe dysfunction (TMT & COWAT). Findings of impairments on tests designed to tap very specific functions and lack of difference on the more course-grained standardised tests highlights the importance of administering tasks that will tap subtle deficits in this patient population.

Evidence for frontal-lobe related memory dysfunction

Several studies investigating episodic memory in PTSD have attributed deficits to frontal lobe dysfunction. These studies have tended to investigate memory function with tests such as the California Verbal Learning Test (CVLT) and Rey Auditory Verbal Learning Test (RAVLT). These may be particularly useful for investigating frontal lobe related deficits because they provide measures of learning of a word list over five trials and performance on a second word list which allows investigation of interference effects as well as performance on delayed recall. In addition, the CVLT includes measures of semantic and serial order clustering which give some insight into the efficiency of encoding processes, and also cued recall and recognition components which allow comparison of performance on tests that rely to a lesser extent on organisation and retrieval strategies than does free recall.

Yehuda et al. (1995) administered the CVLT to a group of Vietnam veterans and a group of normal control participants matched for age and IQ. Patients showed normal acquisition of the first list tested by free recall over five trials. They also performed normally on the second list. However, patients were impaired at free recall of the first list at both short and long delays. The authors interpreted this effect as showing that the patients were abnormally susceptible to retroactive interference. Unfortunately they did not report whether patients made a greater number of intrusion errors from List B, a finding that would have supported this interpretation. Neither did they report performance on the cued recall and recognition components of the CVLT nor on the clustering measures that may have indicated whether patients were encoding the material in an abnormal way. Such a deficit could have contributed to the abnormal forgetting found in the study.

A similar pattern of results was reported by Jenkins et al. (1998). They reported that although survivors of rape with PTSD were not significantly impaired on acquisition over five trials, they were significantly impaired at recall of the words at both short and long delays. This was in comparison to both a healthy control group and a group of rape survivors without PTSD. Interestingly, the PTSD group was not impaired at either cued recall or recognition components of this task. They also showed similar levels of serial order and semantic clustering implying normal levels of semantic processing and encoding.

Three further studies using the CVLT have reported normal performance by individuals with PTSD on this task (Barrett et al. 1996; Stein et al. 1999; Zalewski, Thompson & Gottesman, 1994). Two of the studies (Barrett et al. 1996; Zalewski, Thompson & Gottesman, 1994) included patients with a lifetime diagnosis, so the current symptomatology of the patients was not known. Patients with PTSD included in the Stein et al. (1999) study were adult survivors of childhood sexual abuse and so could possibly have developed compensatory memory mechanisms during development.

The RAVLT is similar in many respects to the CVLT. Performance on this test has been investigated in four studies. Vasterling et al. (1998) found that acquisition of the word list from this test over five trials was impaired in a group of Gulf War veterans with PTSD in comparison to those without. Short and long delay recall of this list was also impaired and recall performance of the PTSD group was characterised by an increased number of intrusions (i.e. false positive responses) indicating a greater susceptibility to interference. In addition, on the recognition component, patients made more false positive errors. Vasterling et al. (2002) investigated performance

on this task in a group of Vietnam veterans and found impairment only on initial learning of the list over five trials. Patients showed no evidence of increased interference. Similarly, Uddo et al. (1993) investigating memory in Vietnam veterans with PTSD also found that they were impaired on acquisition of the list over five trials. In addition, they reported that patients showed increased levels of proactive interference, although there was no difference between patient and control groups on delayed recall performance. Dalton, Pederson & Ryan (1989) also included the RAVLT in their test battery. Although patients with PTSD appeared to perform normally on this task, the results are difficult to interpret as no control group was included.

Again, findings from these studies are equivocal. Whereas all studies using the CVLT have reported normal initial acquisition, the majority of those using the RAVLT have reported impairment in initial acquisition. The finding of a different pattern of performance on initial acquisition using these two tasks could be related to an effect of frontal lobe dysfunction. This is because the word list that makes up the CVLT comprises semantically related words drawn from four categories whereas the word-list used in the RAVLT comprises semantically unrelated words. Stuss, Eskes & Foster (1994) have argued that patients with frontal lobe damage are impaired in the planning and organisational abilities needed to direct a memory search and retrieve relevant information and that when material to be free-recalled is already organised this ability may not be so crucial. Therefore, patients with frontal lobe damage may be less impaired when recalling material that is semantically organised than when recalling semantically unrelated material where the structure is not obvious.

The pattern of performance reported in the preceding tests could, therefore, reflect the effects of frontal lobe dysfunction on performance in PTSD.

Hypotheses of frontal-lobe related cognitive deficits

Hypotheses about the cause of these cognitive deficits have focused on all three symptom clusters included in the DSM IV diagnosis.

The first relates to the hyperarousal symptoms experienced in PTSD (see Kolb, 1987; Pitman, Shalev & Orr, 1998). Kolb (1987) writes that excessive emotional stimulation leads to a state of hypersensitivity in which multiple stimuli, both internal and external, lead to arousal. It is well known that states of very low or very high arousal are associated with impaired performance on tests of attention and executive function, giving the characteristic inverted U shaped curve described by Yerkes & Dodson (see Diamond et al. 2001). If people with PTSD are in a chronically aroused state it is possible that they will show similar deficits to those shown by non-traumatised people in a state of high anxiety (e.g. Eysenck, 1979). A number of investigators have put forward this view (e.g. Diamond et al. 2001; Koenen et al. 2001; Kolb, 1987; Sutker et al. 1995; Vasterling et al. 1998).

Hyperarousal and hypervigilance to threat in the environment may divert attentional resources away from relevant aspects of the task (Diamond et al. 2001; Wolfe & Schlesinger, 1995). Support for this hypothesis comes from the information processing literature, which indicates a bias in attention towards stimuli representing threat (see Buckley, Blanchard & Neill (2000). Later studies have shown that patients with PTSD also show biases in explicit memory whereby memory for

trauma related information is superior to that for neutral or non-trauma related information (e.g. Moradi et al. 2000; Zeitlin & McNally, 1991).

A second reason why attentional resources may be depleted in PTSD is related to the symptoms of re-experiencing. Thus, intrusive memories may engage task-irrelevant processes and so leave fewer resources available to complete the task itself (see Wolfe & Schlesinger, 1997). Both Diamond et al. (2001) and Vasterling, et al. (1998), found that cognitive impairment was related to symptoms of re-experiencing rather than to those of hyperarousal. There is also evidence from functional imaging literature that PTSD may be associated with obsessive thinking and/or intrusive imagery. For example (Lucey et al. 1997) found similarities in patients with PTSD and those with Obsessive Compulsive Disorder (OCD) in contrast to patients with panic disorder. They interpreted this effect as indicating a similarity between PTSD and OCD in obsessive thinking, not present in patients with panic disorder.

Finally, Krystal et al. (1995) hypothesise that the frontal lobes have a role in the dissociative states seen in individuals with PTSD and that cognitive deficits may relate to avoidance symptoms. They hypothesise that two aspects of attention are affected. The ability to detach attention from or disrupt the processing of particular stimuli, and the ability to focus attention on other stimuli that may be internal mental functions. Thus, alterations in sensory processing and attention may be linked with dissociative states.

The possibility of a more unifying hypothesis to account for the cognitive deficits in PTSD has recently been suggested by the results of a study by Perlstein, Elbert & Stenger, (2002). Using functional magnetic resonance imaging (fMRI) with

healthy participants they reported that during a working memory task the dorsolateral prefrontal cortex and orbitofrontal cortex were differentially activated according to the valence of the material presented. Thus, during unpleasant affect orbitofrontal cortex activity was increased whereas dorsolateral prefrontal cortex activity was reduced. The dorsolateral prefrontal cortex is known to be important for working memory processes and has been hypothesised to have a specific role in the maintenance of stimulus representations to guide task-relevant behaviour. Thus, it is possible that in PTSD the intrusion of unpleasant memories leads to an increase in orbitofrontal cortex activity which activates the 'defensive' system with a corresponding reduction in dorsolateral prefrontal cortex activity and a resulting inability to maintain task-relevant behaviour.

These hypotheses provide a theoretical basis for the cognitive deficits relating to frontal lobe dysfunction that have been found in PTSD. In the following sections evidence will be considered for the presence of memory deficits relating to the functioning of medial temporal lobe structures, particularly the hippocampus and amygdala.

Hippocampal related memory deficits

Initial interest in the hippocampus and its relationship to memory functioning in PTSD arose largely as the consequence of findings from animal studies showing that exposure to long-term stress resulted in structural damage to the hippocampus (see Bremner, 2001; LeDoux, 1998). Clinical observations of frequent memory difficulties in individuals with PTSD led to volumetric studies of this structure in this population. The first study was published in 1995 (Bremner, Randall, Scott, Bronen

et al. 1995) and since then five further studies have been published.

Evidence for hippocampal volume reductions in PTSD

Bremner, Randall, Scott, Bronen et al. (1995) reported a marginally significant reduction in right hippocampal volume in Vietnam combat veterans in comparison with healthy control participants. No significant difference was found in volume of left hippocampus, the caudate nucleus or the whole temporal lobe implying that the volume reduction was not the result of generalised atrophy. This result was partially replicated in a second study (Bremner, Randall, Scott, Capelli et al. 1995) which reported a reduction in the volume of the left hippocampus in a group of adult survivors of childhood sexual abuse. Subsequently Gurvits et al. (1996) reported selective bilateral hippocampal volume reductions in a group of Vietnam veterans with PTSD in comparison to combat veterans without PTSD. Stein, Koverola, Hanna, Torchia & McClarty,(1997) reported a significant reduction in the volume of the left hippocampus in adult survivors of childhood sexual abuse. Finally, Schuff et al. (1997) failed to find a significant reduction in hippocampal volume in combat veterans.

There are a number of difficulties in the interpretation of these studies. First, all PTSD groups included had a chronic and intractable form of the disorder that had persisted over a number of years. It is unclear, therefore, whether hippocampal volume reduction would be found in individuals with less persistent symptoms of PTSD. Second, there were high levels of co-morbid psychiatric illnesses in these groups which raises the possibility that hippocampal volume reductions are related to co-morbid disorders rather than PTSD (see Pitman, Shin & Rauch, 2001). Third, some of the studies included individuals who had suffered loss of consciousness.

Although this was typically of only short duration, Warden, Reider-Groswasser, Grafman & Salazar, (1995) point out that even short periods of hypoxia can result in hippocampal damage. Fourth, Hippocampal volume reductions may constitute a risk factor for PTSD rather than be a consequence of it (see Pitman, Shin & Rauch, 2001) and so reduced hippocampal volume may have predated PTSD.

A recent paper by Bonne et al. (2001) addresses a number of these alternative possibilities. Using a longitudinal design, they scanned the brains of 37 people, who had experienced a traumatic event, within a few days of the event and again six months later. At six months, ten of the participants met DSM IV criteria for PTSD. Results showed no difference in hippocampal volume between participants with and without PTSD either immediately or six months following trauma. This study provides some evidence that reduced hippocampal volume is a consequence rather than a risk factor for PTSD. However, it does not preclude the possibility that smaller hippocampal volume may render individuals susceptible to particularly severe and chronic forms of the disorder. In addition, a reduction in hippocampal volume did not emerge within six months of the trauma. The authors conclude that either chronic exposure to stress as seen in combat veterans and people with histories of childhood sexual abuse or chronic PTSD may be required to produce changes in hippocampal volume.

Neither is it clear that the reductions in hippocampal volume that have been found can be related to specific memory deficits in PTSD. The role of the hippocampus in memory has long been the focus of research. The results of early studies suggested that hippocampal damage resulted in a global memory deficit affecting all aspects of memory. More recently, however, the precise role of this structure has been the

focus of much debate. The results of several studies have suggested that patients with isolated hippocampal damage perform normally on tests of item recognition (see Aggleton & Brown, 1999; Aggleton & Shaw, 1996; Mayes, Holdstock, Isaac, Hunkin & Roberts, 2001; Vargha-Khadem et al. 1997; but see Zola & Squire, 1999). The crucial difference between performance on tests of item recognition and on tests known to be sensitive to hippocampal damage such as free recall and memory for spatio-temporal information is that the latter are believed to rely to a much greater extent on the formation of associations. Thus, the hippocampus has been hypothesised to play a role in the consolidation of associative material into long-term storage. In contrast, performance on tests of memory that does not include associative information, such as item recognition is hypothesised to rely on cortical structures outside the hippocampus (see Aggleton & Brown, 1999).

Bremner, Randall, Scott, Bronen et al. (1995) administered the Logical Memory and Visual Reproduction subtests of the WMS-R, which should be sensitive to hippocampal damage, to the participants in their study who underwent MRI scanning. They found that the PTSD group was impaired on the Logical Memory subtest but not on the Visual Reproduction subtest. They also found that the volume reduction found in the right hippocampus was related to the impairment on the retention measure of the Logical Memory subtest. This result is counterintuitive as it has been well established that the left hippocampus is involved in verbal memory and the right hippocampus in visual memory. However, evidence that bilateral hippocampal damage has a much greater detrimental effect on memory functioning than the sum of the memory deficits in individuals with unilateral right and unilateral left damage would suggest, indicates that the right hippocampus may have some role

in verbal memory (see Mayes, 1988; Pitman, Shin & Rauch, 2001). In addition, a recent study which investigated the relationship between recognition of new and familiar words using fMRI and performance on the CVLT (Johnson, Saykin, Flashman, McAllister & Sparling, 2001) reported that the presentation of new words was associated with right anterior hippocampal activation.

However, Bremner, Randall, Scott, Capelli et al. (1995) failed to find a relationship between impaired performance on the Logical Memory subtest of the WMS-R and reduction of left hippocampal volume in their study. Similarly, Stein, Koverola, et al (1997) reported no relationship between left hippocampal volume reduction and tests of memory function including the CVLT. Finally, Gurvits et al. (1996) reported no association between hippocampal volume reduction and performance on the WMS-R.

It is not clear why reductions in hippocampal volume should not be associated with deficits in memory. It should be noted, however, that the hippocampal volume reductions found in patients with PTSD are typically much smaller than those reported in the memory literature. Bremner, Randall, Scott, Bronen et al. (1995) report an 8% reduction in right hippocampal volume, Bremner Randall, Scott, Bronen et al. (1995) report a reduction in left hippocampal volume of 12%, and Stein, Koverola, et al. (1997) report a left hippocampal volume reduction of 5%. The only study to report significant bilateral volume reductions in this structure (Gurvits et al., 1996) report reductions in left and right hippocampus of 26% and 22% respectively. In contrast Mayes et al. (2001) report volume reductions of around 50% in their patient with selective hippocampal damage, and a similar reduction is reported by Vargha-Khadem et al. (1997) in their patient. It is possible, therefore,

that small reductions in volume will not lead to a detectable pattern of memory deficits of the kind associated with hippocampal damage.

Evidence of deficits in episodic memory

Other studies in this review were examined to determine whether any evidence exists of memory dysfunction in individuals with PTSD that cannot more parsimoniously be explained in terms of an attention or working memory deficit. Only four studies reported memory deficits that could not be accounted for by an actual or possible attention deficit. Yehuda et al. (1995) reported normal acquisition of the lists from the CVLT on trials one to five. However, patients were impaired in comparison with a normal control group on their recall at the short and long delays. This deficit was interpreted as an abnormal sensitivity to retroactive interference. However, it is also possible that PTSD participants showed abnormally fast forgetting of the stimuli despite normal acquisition. Unfortunately, Yehuda et al. do not report the serial and semantic clustering measures of the CVLT. If these measures were normal it would lend support to the interpretation of abnormally fast forgetting despite normal encoding of the stimuli.

Gilbertson et al. (2001) used a discriminant analysis on the scores of Vietnam veterans with and without PTSD on a range of neuropsychological tests. This revealed that performance on the Digit Span subtest and on the General Memory Index from the WMS-R were the most significant predictors of group membership making independent contributions to the discrimination. Thus, poorer performance on the memory measures could not be accounted for by impaired attention.

Koenen et al. (2001) found that a group of patients with PTSD was significantly impaired on delayed non matching to sample (DNMTS), a paradigm which has been shown to be sensitive to hippocampal dysfunction (e.g. Squire 1992). Koenen et al. (2001) attributed this deficit to a limbic system dysfunction involving the hippocampus and orbitofrontal cortex. However, the condition in which subjects were impaired involved only a very brief (2ms) exposure and so the deficit could be the result of an attention deficit to very brief exposure rather than a true memory deficit. At longer stimulus exposures patients were not impaired on this task.

From the studies reviewed above there appears very little unequivocal evidence that episodic memory is impaired in individuals with PTSD other than those deficits that could be accounted for by attention and working memory deficits related to frontal lobe dysfunction. It has proved difficult to demonstrate the precise relationship between hippocampal volume reductions and impaired episodic memory. Indeed, it is unclear whether these volume reductions are, in fact, related to PTSD. Other possibilities are that they are more closely associated with co-morbid disorders or that they were a premorbid feature of individuals with chronic and intractable forms of PTSD, and therefore, a vulnerability factor.

It is possible that episodic memory impairment in PTSD is more likely to be associated with memory tasks mediated by the amygdala as this has been demonstrated to have a role in emotional memory (LeDoux, 1998). The following section will focus on the memory-related functions of this structure.

Amygdala related memory deficits

The amygdala has long been known to play an important role in emotion and it has been well established that it is important for the acquisition of a conditioned response to fear-evoking stimuli (see LeDoux,1998). More recently the role of the amygdala in emotional memory has also been explored (see Babinsky et al. 1993; Cahill & McGaugh, 1998; McGaugh, Cahill & Roozendaal, 1996).

Evidence for abnormalities in amygdala functioning

Because of its likely association with the re-experiencing phenomena of PTSD, the majority of functional imaging studies have used symptom provocation paradigms in which individuals with PTSD are presented with auditory scripts or visual stimuli related to their trauma in order to elicit the re-experiencing phenomena during the scanning procedure. Studies investigating regional cerebral blood flow in these patients using positron emission tomography (PET; Shin, et al. 1997; Shin, et al. 1999) have found increased activity in the region of the amygdala when subjects recalled or imagined images related to their trauma. Using single photon emission computed tomography, Liberzon, et al. (1999) found similar results in combat veterans with PTSD when presented with combat sounds.

A more recent study by Rauch, et al. (2000) using fMRI found an exaggerated amygdala response to general negative stimuli that were unrelated to the specific trauma of the individuals involved. This study is important because it provides evidence that in individuals with PTSD increased activity in the amygdala may occur to emotional stimuli in general rather than solely to stimuli associated with an individuals own trauma experience.

Role of the amygdala in emotional memory

Findings that individuals with PTSD show an exaggerated amygdala response to arousing stimuli not associated with their own traumatic experience (Rauch et al. 2000) raises the possibility that individuals with PTSD will experience some of the memory-related difficulties experienced by rare patients with structural damage to the amygdala. A number of studies has shown that patients with such damage are impaired on memory tasks that involve emotionally arousing stimuli whereas their performance on tasks involving emotionally neutral stimuli is normal (e.g. Adolphs, Cahill, Schul & Babinsky, 1997; Babinsky et al., 1993; Cahill, Babinsky, Markowitsch & McGaugh, 1995; Markowitsch et al. 1994; Phelps et al. 1998).

Consistent with these findings Cahill et al. (1996) have demonstrated the role of the amygdala in emotional memory in healthy participants. They used PET to investigate amygdala activity while participants viewed emotionally arousing video clips and neutral video clips. Results showed that activity in the right amygdala at encoding was highly correlated with long-term retention over three weeks, of emotionally arousing films, but not of emotionally neutral films.

McGaugh, Cahill & Roozendaal, (1996; see also McGaugh, Roozendaal & Cahill, 2000) hypothesise a specific role for the amygdala in the consolidation of emotional material into long term memory. They hypothesise that enhanced memory for emotional material arises as a consequence of the effect of neurohormones on amygdala functioning which in turn modulates memory consolidation processes occurring in other brain structures including the hippocampus with which it has strong anatomical links.

Evidence for amygdala related memory deficits

As functional imaging studies of individuals with PTSD have found increased activity in the amygdala, it is unclear what predictions would be made regarding memory for general emotional material in this patient group. Whereas patients with structural damage to the amygdala have been found to be impaired in their memory for emotional material, it is possible that people with PTSD could show enhanced memory for emotional material.

There appear to be no published studies of PTSD that have included cognitive tests directed at tapping amygdala dysfunction in this patient group. However, some researchers have examined memory for emotional material in individuals with PTSD and it is memory for this kind of material that has been found to be abnormal in people with structural damage to the amygdala. All the studies have been consistent in finding that memory for trauma-related material was better (and often normal) than memory for neutral, positive or other negative material (Moradi, et al. 2000; Vrana, Roodman & Beckham, 1995; Wolfe, 1994; Zeitlin & McNally, 1991). However, these effects could be accounted for by attentional bias rather than genuine memory effects.

Summary and Conclusions

In spite of the severe difficulties associated with investigating cognitive function in PTSD, progress has been made in identifying areas of dysfunction. The best evidence of memory dysfunction in PTSD is that relating to the functions of the frontal lobes. Evidence regarding episodic memory impairment over and above that which can be attributed to deficits in attentional and executive functions is at best, equivocal.

In relation to possible deficits associated with hippocampal damage, tests specifically designed to tap hippocampal function will be necessary if the small volume reductions found are to be related to memory functioning. Such tests could relate to the hypothesised role of the hippocampus in consolidation of associative information into long term storage and findings that rate of forgetting of information may be accelerated in patients with such damage (see Isaac and Mayes. 1999a, 1999b).

Interest in the amygdala has grown in PTSD due to its well researched role in emotional memory and findings from neuroimaging studies showing hyperactivity in this structure in PTSD. As yet, however, no published studies have looked at amygdala-related memory phenomena in PTSD. This will be important to address if the pattern of cognitive deficits in PTSD is to be fully documented.

In general it would be desirable to address hypotheses that are much more specific to avoid the problem of type I errors. In addition, as it has been well established that cognitive deficits are a feature of PTSD, it would now seem desirable that studies be more theoretically based rather than exploratory. A number of other aspects are also important. First, there has been no systematic investigation of possible difference in cognitive profile across different aetiological subgroups of PTSD. Thus, it remains unclear if the results from studies are applicable beyond the group included in that study. Second, it needs to be systematically investigated whether the cognitive profile of PTSD differs from that of other emotional disorders such as anxiety, depression and obsessive compulsive disorder. At present, there is little evidence to suggest that this is so and with regard to the hypotheses related to frontal lobe dysfunction described earlier, differences may be more likely to relate to the specific

content of intrusive thoughts rather than the underlying deficit in cognitive functioning.

References

- Adolphs, R., Cahill, L., Schul, R. & Babinsky, R. (1997). Impaired declarative memory for emotional material following bilateral amygdala damage in humans. *Learning and Memory*, 4, 291-300.
- Aggleton, J.P. & Brown, M.W. (1999) Episodic memory, amnesia and the hippocampal-anterior thalamic axis. *Behavioural Brain Sciences*, 22, 425-489.
- Aggleton, J.P. & Shaw, C. (1996). Amnesia and recognition memory: A re-analysis of psychometric data. *Neuropsychologia*, 34, 51-62.
- American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorders (4th Edition)*. Washington D.D.: American Psychiatric Association.
- Anderson, S.W., Damasio, H., Jones, R.D. & Tranel, D. (1991). WCST performance as a measure of frontal lobe damage. *Journal of Clinical and Experimental Neuropsychology*, 13, 909-922.
- Babinsky, R., Calabrese, P., Durwen, H.F., Markowitsch, H.J., Brechtelbauer, D., Heuser, L. & Gehlen, W. (1993). The possible contribution of the amygdala to memory. *Behavioural Neurology*, 6, 167-170.
- Baddeley, A. (1990). *Human Memory*. Hove: Erlbaum.

- Barrett, D.H., Green, M.L., Morris, M.A.R., Giles, W.H. & Croft, J.B. (1996). Cognitive functioning and posttraumatic stress disorder. *American Journal of Psychiatry*, *153*, 1492-1494.
- Beckham, J.C., Crawford, A.L. & Feldman, M.E. (1998). Trail making test performance in Vietnam combat veterans with and without posttraumatic stress disorder. *Journal of Traumatic Stress*, *11*, 811-819.
- Beers, S.R. & De Bellis, M.D. (2002). Neuropsychological function in children with maltreatment-related posttraumatic stress disorder. *American Journal of Psychiatry*, *159*, 483-486.
- Bonne, O., Brandes, D., Gilboa, A., Gormi, J.M., Shenton, M.E., Pitman, R.K. & Shalev, A.Y. (2001). Longitudinal MRI study of hippocampal volume in trauma survivors with PTSD. *American Journal of Psychiatry*, *158*, 1248-1251.
- Bremner, J.D. (2001). Hypotheses and controversies related to effects of stress on the hippocampus: An argument for stress-induced damage to the hippocampus in patients with posttraumatic stress disorder. *Hippocampus*, *11*, 75-81.
- Bremner, J.D., Randall, P., Scott, T.M., Bronen, R.A., Seibyl, J.P., Southwick, S.M., Delaney, R.C., McCarthy, G., Charney, D.S. & Innis, R.B. (1995). MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. *American Journal of Psychiatry*, *152*, 973-981.

- Bremner, J.D., Randall, P., Scott, T.W., Capelli, S., Delaney, R., McCarthy, G. & Charney, D.S. (1995). Deficits in short-term memory in adult survivors of childhood abuse. *Psychiatry Research, 59*, 97-107.
- Bremner, J.D., Scott, T.M., Delaney, R.C., Southwick, S.M., Mason, J.W., Johnson, D.R., Innis, R.B., McCarthy, G. & Charney, D.S. (1993). Deficits in short-term memory in posttraumatic stress disorder. *American Journal of Psychiatry, 150*, 1015-1019.
- Bremner, J.D., Staib, L.H., Kaloupek, D., Southwick, S.M., Soufer, R.C. & Dennis, S. (1999). Neural correlates of exposure to traumatic pictures and sound in combat veterans with and without posttraumatic stress disorder: A positron emission tomography study. *Biological Psychiatry, 45*, 806-816.
- Buckley, T.C., Blanchard, E.B. & Neill, W.T. (2000). Information processing and PTSD: A review of the empirical literature. *Clinical Psychology Review, 20*(8), 1041-1065.
- Cahill, L., Babinsky, R., Markowitsch, H.J. & McGaugh, J.L. (1995). The amygdala and emotional memory. *Nature, 377*, 295-296.
- Cahill, L. & McGaugh, J.L. (1998). Mechanisms of emotional arousal and lasting declarative memory. *Trends in Neurosciences, 21*, 294-299.

- Cahill, L., Haier, R.J., Fallon, J., Alkire, M.T., Tang, C., Keator, D., Wu, J. & McGaugh, J.L. (1996). Amygdala activity at encoding correlated with long-term free recall of emotional information. *Proceedings of the National Academy of Sciences of the USA*, *93*, 8016-8021.
- Dalton, J.E., Pederson, S.L. & Ryan, J.J. (1989). Effects of post-traumatic stress disorder on neuropsychological test performance. *International Journal of Clinical Neuropsychology*, *11*, 121-124.
- Diamond, T., Muller, R., Rondeau, L.A. & Rich, J.G. (2001). The relationships among PTSD symptomatology and cognitive functioning in adult survivors of child maltreatment. In: Columbus, F. (Ed.). *Advances in psychology research Vol. V*. (pp. 253-279). Huntington, New York: Nova Science Publishers.
- Eysenck, M.W. (1979). Anxiety, learning and memory: A reconceptualization. *Journal of research in Personality*, *13*, 363-385.
- Gil, T., Calev, A., Greenberg, D., Kugelmass, S. & Lerer, B. (1990). Cognitive functioning in post-traumatic stress disorder. *Journal of Traumatic Stress*, *3(1)*, 29-45.
- Gilbertson, M.W., Gurvits, T.V., Lasko, N.B., Orr, S.P. & Pitman, R.K. (2001). Multivariate assessment of explicit memory function in combat veterans with posttraumatic stress disorder. *Journal of Traumatic Stress*, *14(2)*, 413-432.

- Gurvits, T.V., Lasko, N.B., Schacter, S.C., Kuhne, A.A., Orr, S.P. & Pitman, R.K. (1993). Neurological status of Vietnam veterans with chronic posttraumatic stress disorder. *Journal of Neuropsychiatry*, 5, 183-188.
- Gurvits, T.V., Shenton, M.E., Hokama, H., Ohta, H., Lasko, N.B., Gilbertson, M.W., Orr, S.P., Kikinis, R., Jolesz, F.A., McCarley, R.W. & Pitman, R.K. (1996). Magnetic resonance imaging study of hippocampal volume in chronic combat related posttraumatic stress disorder. *Biological Psychiatry*, 40, 1091-1099.
- Isaac, C.L. & Mayes, A.R. (1999a). Rate of forgetting in amnesia I: Recall and recognition of prose. *Journal of Experimental Psychology: Learning, Memory and Cognition*, 25, 942-962.
- Isaac, C.L. & Mayes, A.R. (1999b). Rate of forgetting in amnesia II: Recall and recognition of word lists at different levels of organisation. *Journal of Experimental Psychology: Learning, Memory and Cognition*, 25, 963-977.
- Jenkins, M.A., Langlais, P.J., Delis, D. & Cohen, R. (1998). Learning and memory in rape victims with posttraumatic stress disorder. *American Journal of Psychiatry*, 155, 278-279.
- Jenkins, M.A., Langlais, P.J., Delis, D. & Cohen, R. (2000). Attentional dysfunction associated with posttraumatic stress disorder among rape survivors. *The Clinical Neuropsychologist*, 14, 7-12.

- Johnson, S.C, Saykin, A.J., Flashman, L.A., McAllister, T.W. & Sparling, M.B. (2001). Brain activation on fMRI and verbal memory ability: Functional neuroanatomic correlates of CVLT performance. *Journal of the International Neuropsychological Society*, 7, 55-62.
- Koenen, K.C., Driver, K.L., Oscar-Berman, M., Wolfe, J., Folsom, S., Huang, M.T. & Schlesinger, L. (2001). Measures of prefrontal system dysfunction in posttraumatic stress disorder. *Brain and Cognition*, 45, 64-78.
- Kolb, L.C. (1987). A Neuropsychological hypothesis explaining posttraumatic stress disorder. *American Journal of Psychiatry*, 144, 989-995.
- Krystal, J.H., Bennett, A.L., Bremner, J.D., Southwick, S.M. & Charney, D.S. (1995). Toward a cognitive neuroscience of altered memory functions in posttraumatic stress disorder. In M.J. Friedman, D.S. Charney & A.Y. Deutsch (Eds.) *Neurobiological and clinical consequences of stress: From normal adaptation to PTSD*. Philadelphia: Lippincott-Raven.
- Lanius, R.A., Williamson, P.C., Densmore, M., Boksman, K., Neufeld, R.W., Gati, J.S. & Menon, R.S. (2001). Neural correlates of traumatic memories in posttraumatic stress disorder: A functional MRI investigation. *American Journal of Psychiatry*, 158, 1920-1922.
- LeDoux, J. (1998). *The Emotional Brain*. London: Phoenix.

- Levy, C.J. (1988). Agent Orange exposure and posttraumatic stress disorder. *The Journal of Nervous and Mental Disease*, 176, 242-245.
- Liberzon, I., Taylor, S.F., Amdur, R., Jung, T.D., Chamberlain, K.R., Minoshima, S. & Koeppe, R.A. (1999). Brain activation in PTSD in response to trauma-related stimuli. *Biological Psychiatry*, 45, 817-826.
- Litz, B.T., Weathers, F.W., Monaco, V. Herman, D., Wulfsohn, M., Marx, B. & Keane, T.M. (1996). Attention, arousal and memory in posttraumatic stress disorder. *Journal of Traumatic Stress*, 9, 497-520.
- Lucey, J.V., Costa, D.C., Adshead, G., Deahl, M., Busatto, G., Gacinovic, S., Travis, M., Pilowsky, L., Ell, P.J., Marks, I.M. & Kerwin, R.W. (1997). Brain blood flow in anxiety disorders. *British Journal of Psychiatry*, 171, 346-350.
- Markowitsch, H.J., Calabrese, P., Wurker, M., Durwen, H.F., Kessler, J., Babinsky, R., Brechtelsbauer, D., Heuser, L. & Gehlen, W. (1994). The amygdala's contribution to memory – a study of two patients with Urbach-Wiethe disease. *Neuroreport*, 5, 1349-1352.
- Mayes, A.R. (1988). *Human Organic Memory Disorders*. Cambridge: Cambridge University Press.
- Mayes, A.R., Holdstock, J.S., Isaac, C.L., Hunkin, N.M. & Roberts, N. (in press). Relative sparing of item recognition memory in a patient with adult-onset damage limited to the hippocampus. *Hippocampus*.

- McGaugh, J.L., Cahill, L. & Roozendaal, B. (1996). Involvement of the amygdala in memory storage: Interaction with other brain systems. *Proceedings of the National Academy of Sciences*, 93, 13508-13514.
- McGaugh, J.L., Roozendaal, B. & Cahill, L. (2000). Modulation of memory storage by stress hormones and the amygdaloid complex. In Gazzaniga, M.S. (Ed). *The New Cognitive Neurosciences (2nd ed.)*. Cambridge, US: The MIT Press.
- Moradi, A.R., Neshat-Doost, H.T., Taghavi, M.R., Yule, W. & Dalgleish, T. (1999). Everyday memory deficits in children and adolescents with PTSD: Performance on the Rivermead Behavioural Memory Test. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 40, 357-361.
- Moradi, A.R., Taghavi, R., Neshat-Doost, H.T., Yule, W. & Dalgleish, T. (2000). Memory bias for emotional information in children and adolescents with posttraumatic stress disorder: A preliminary study. *Journal of Anxiety Disorders*, 14, 521-534.
- Osuch, E.A., Benson, B., Geraci, M., Podell, D., Herscovitch, P., McCann, U.D. & Post, R.M. (2001). Regional cerebral blood flow correlated with flashback intensity in patients with posttraumatic stress disorder. *Biological Psychiatry*, 50, 246-253.

- Perlstein, W.M., Elbert, T. & Stenger, A. (2002). Dissociation in human prefrontal cortex of affective influences on working memory-related activity. *Proceedings of the National Academy of Sciences (USA)*, 99, 1736-1741.
- Phelps, E.A., LaBar, K.S., Anderson, A.K., O'Connor, K.J., Fulbright, R.K. & Spencer, D.D. (1998). Specifying the contributions of the human amygdala to emotional memory: A case study. *Neurocase*, 4, 527-540.
- Pitman, R.K., Shalev, A.Y. & Orr, S.P. (2000). Posttraumatic stress disorder: Emotion, conditioning and memory. In Gazzaniga, M.S. (Ed). *The New Cognitive Neurosciences (2nd ed.)*. Cambridge, US: The MIT Press.
- Pitman, R.K., Shin, L.M. & Rauch, L. (2001). Investigating the pathogenesis of posttraumatic stress disorder with neuroimaging. *Journal of Clinical Psychiatry*, 62 (suppl.17) 47-54.
- Rauch, S.L., van der Kolk, B.A., Fisler, R.E., Alpert, N.M., Orr, S.P., Savage, C.R., Fischman, A.J., Jenike, M.A. & Pitman, R.K. (1996). A symptom provocation study of posttraumatic stress disorder using positron emission tomography and script-driven imagery. *Archives of General Psychiatry*, 53, 380-387.
- Rauch, S.L., Whalen, P.J., Shin, L.M., McInerney, S.C., Macklin, M.L., Lasko, N.B., Orr, S.P. & Pitman, R.K. (2000). Exaggerated amygdala response to masked facial stimuli in posttraumatic stress disorder: a functional MRI study. *Biological Psychiatry*, 47, 769-776.

- Richardson, J.T.E. (1990). *Clinical and Neuropsychological Aspects of Closed Head Injury*. London: Taylor & Francis.
- Sachinvala, N., von-Scotti, H., McGuire, M., Fairbanks, L., Bakst, K., McGuire, M. & Brown, N. (2000). Memory, attention, function and mood among patients with chronic posttraumatic stress disorder. *Journal of Nervous and Mental Disease*, 188, 818-823.
- Schuff, N., Marmar, C.R., Weiss, D.S., Neylan, T.C., Schoenfeld, G., Fein, G. & Weiner, M.W. (1997). Reduced hippocampal volume and n-acetyl aspartate in posttraumatic stress disorder. *Annals of the New York Academy of Sciences*, 821, 516-520.
- Shin, L.M., Kosslyn, S.M., McNally, R.J., Alpert, N.M., Thompson, W.L., Rauch, S.L., Macklin, M.L., Pitman, R.K. (1997). Visual imagery and perception in posttraumatic stress disorder. A positron emission tomographic investigation. *Archives of General Psychiatry*, 54, 233-241.
- Shin, L.M., McNally, R.J., Kosslyn, S.M., Thompson, W.L., Rauch, S.L., Alpert, N.M., Metzger, L.J., Lasko, N.B., Orr, S.P. & Pitman, R.K. (1999). Regional cerebral blood flow during script-driven imagery in childhood sexual abuse-related PTSD: A PET investigation. *American Journal of Psychiatry*, 156, 575-584.
- Squire, L.R. (1992). Memory and the hippocampus: A synthesis from findings with rats, monkeys and humans. *Psychological Review*, 99, 195-231.

- Stein, M.B., Hanna, C., Koverola, C., Torchia, M. & McClarty, B. (1997). Structural brain changes in PTSD. Does trauma alter neuroanatomy? In R. Yehuda & A.C. McFarlane (Eds.) *Psychobiology of Posttraumatic Stress Disorder*. New York: Annals of the New York Academy of Sciences.
- Stein, M.B., Hanna, C., Vaerum, V. & Koverola, C. (1999). Memory function in adult women traumatised by childhood sexual abuse. *Journal of Traumatic Stress, 12*, 527-534.
- Stein, M.B., Koverola, C., Hanna, C., Torchia, M.G. & McClarty, B. (1997). Hippocampal volume in women victimized by childhood sexual abuse. *Psychological Medicine, 27*, 951-959.
- Stuss, D.T., Eskes, G.A. & Foster, J.K. (1994). Experimental neuropsychological studies of frontal lobe functions. In F. Boller & J. Grafman (Eds.) *Handbook of Neuropsychology, Vol 8*. Amsterdam: Elsevier.
- Sullivan, E.V., F, R., Rosenbloom, M.J. & Pfefferbaum, A. (2002). A profile of neuropsychological deficits in alcoholic women. *Neuropsychology, 16*, 74-83.
- Sutker, P.B., Winstead, D.K., Galina, Z.H. & Allain, A.N. (1991). Cognitive deficits and psychopathology among former prisoners of war and combat veterans of the Korean conflict. *American Journal of Psychiatry, 148*, 67-72.

- Sutker, P.B., Vasterling, J.J., Brailey, K. & Allain, A.N. (1995). Memory, attention and executive deficits in POW survivors: Contributing biological and psychological factors. *Neuropsychology*, 9, 118-125.
- Tulving, E. (1984). How many memory systems are there? *American Psychologist*, 40, 385-398.
- Uddo, M., Vasterling, J.J., Brailey, K. & Sutker, P.B. (1993). Memory and attention in combat-related post-traumatic stress disorder (PTSD). *Journal of Psychopathology and Behavioral Assessment*, 15, 43-52.
- Vargha-Khadem, F., Gadian, D.G., Watkins, K.E., Connelly, A, van Paesschen, W. & Mishkin, M. (1997). Differential effects of early hippocampal pathology on episodic and semantic memory. *Science*, 277, 376-380.
- Vasterling, J.J., Brailey, K., Constans, J.I. & Sutker, P.B. (1998). Attention and memory dysfunction in posttraumatic stress disorder. *Neuropsychology*, 12, 125-133.
- Vasterling, J.J., Duke, L.M., Brailey, K., Constans, J.I., Allain, A.N. & Sutker, P.B. (2002). Attention, learning and memory performances and intellectual resources in Vietnam veterans: PTSD and no disorder comparisons. *Neuropsychology*, 16, 5-14.

- Vrana, S.R., Roodman, A. & Beckham, J.C. (1995). Selective processing of trauma-relevant words in posttraumatic stress disorder. *Journal of Anxiety Disorders*, 9, 515-530.
- Warden, D., Reider-Groswasser, I., Grafman, J. & Salazar, A. (1995). PTSD and hippocampal volume. *American Journal of Psychiatry*, 153, 1657.
- Williams, J.M.G., Watts, F.N., McLeod, C. & Mathews, A. (1997). *Cognitive Psychology and Emotional Disorders*. Second Edition. England: Wiley.
- Wolfe, J. & Charney, D.S. (1991). Use of neuropsychological assessment in posttraumatic stress disorder. *Psychological Assessment: A Journal of Consulting and Clinical Psychology*, 3, 573-580.
- Wolfe, J. & Schlesinger, L.K. (1997) Performance of PTSD patients on standard tests of memory. In R. Yehuda & A.C. McFarlane (Eds.) *Psychobiology of Posttraumatic Stress Disorder*. New York: Annals of the New York Academy of Sciences.
- Yehuda, R., Keefe, R.S.E., Harvey, P.D., Levengood, R.A., Gerber, D.K., Geni, J. & Siever, L.J. (1995). Learning and memory in combat veterans with posttraumatic stress disorder. *American Journal of Psychiatry*, 152, 137-139.
- Yehuda, R. (2000). Biology of posttraumatic stress disorder. *Journal of Clinical Psychiatry*, 61(supp 17), 14-21.

Zalewski, C., Thompson, W. & Gottesman, I. (1994). Comparison of neuropsychological test performance in PTSD, generalized anxiety disorder and control Vietnam veterans. *Assessment* 1, 133-142.

Zeitlin, S.B. & McNally, R.J. (1991). Implicit and explicit memory biases for threat in post-traumatic stress disorder. *Behavior Research and Therapy*, 29, 451-457.

Zola, S.M. & Squire, L.R. (1999). Remembering the hippocampus. *Behavioral and Brain Sciences*, 22, 469-470.

CHAPTER 2 : MAIN PAPER

Forgetting of Emotional and Non-Emotional Stimuli in People with Posttraumatic Stress Disorder

Abstract

Research into the neuropathological basis of posttraumatic stress disorder (PTSD) has focused on the structures of the limbic system. Despite evidence for functional abnormalities in the amygdala in PTSD, no studies have investigated the possibility that people with this disorder may suffer cognitive deficits associated with dysfunction in this structure. Evidence suggests a role for the amygdala in the consolidation of emotional material into long-term memory via its links with the hippocampus. Consistent with this, the results of this study showed that despite being matched for initial levels of recall at a delay of 20 seconds, individuals with PTSD forgot emotional stimuli at an accelerated rate over a delay of one hour in comparison with healthy controls. Forgetting of non-emotional stimuli proceeded at a more normal rate over this delay. Forgetting assessed by recognition proceeded at a normal rate regardless of the emotionality of the stimuli.

Introduction

PTSD can occur in individuals exposed to traumatic experiences of exceptional severity. It is characterised by symptoms of re-experiencing of the trauma, avoidance behaviours and increased physiological arousal [see 49]. The disorder was first recognised by the American Psychiatric Association in 1980. Since then much research has been carried out to more closely define it, both in physiological and psychological terms and to developing effective treatments for it.

Research into the neurobiological basis of PTSD has focused on the limbic system, comprising the hippocampus, amygdala, thalamus and pre frontal cortex [e.g. 24, 44]. This system has long been known to have a major role in both emotion and memory. Recent theories of PTSD have particularly emphasised the roles of the amygdala in the symptoms of re-experiencing and of the hippocampus in clinical observations of poor episodic memory for the traumatic episode [e.g. 11].

In response to frequent complaints of poor episodic memory from people with PTSD [see 48] current cognitive functioning in this disorder has been the focus of a number of studies. There is relatively good evidence from these studies that some cognitive functions thought to be mediated by the frontal lobes are impaired. Thus, deficits have been reported in some aspects of attention [e.g. 21], working memory [e.g. 17], executive function [e.g. 8] and in aspects of memory thought to depend on frontal lobe function [e.g. 46].

The memory functions thought to be subserved by the hippocampus and amygdala have been the focus of very little systematic investigation in this patient group. There

is evidence however of abnormality in these brain regions associated with PTSD. Thus, PTSD may be associated with volume reductions in the hippocampus [see 10]. There is also evidence from functional imaging studies of abnormal functioning of the amygdala. This has been demonstrated in studies using symptom provocation paradigms in which participants are presented with reminders of their traumatic experience [25, 39, 43]. More recently, a study has been published [40] in which increased amygdala activity in PTSD was found in response to more general threat-related stimuli. This study is important because it provides evidence of an abnormal response to general emotional stimuli in PTSD rather than solely to stimuli associated with an individual's own trauma experience.

Recent studies have highlighted the importance of the amygdala in emotional memory. In a study with healthy volunteers [13] it was reported that retention of emotionally arousing films was superior to that of neutral films following a three-week delay. In addition, activity in the right amygdala at encoding revealed by Positron Emission Tomography (PET) was highly correlated with retention of the emotionally arousing films, but not of emotionally neutral films. Similar results have been reported in another study [18].

Further evidence of a role for the amygdala in emotional memory comes from studies of rare patients who have selective damage to this structure. Attention and intellectual functioning appears normal in these patients [e.g. 3, 7] as does general memory functioning [e.g. 28] although there have been reports that visual memory may be impaired [see 5]. The most consistent finding in this small group of patients is that they show deficits in memory for emotional material whereas memory for

neutral material appears normal [e.g. 2, 7, 12].

Based on evidence from both the human and animal literature, a selective role for the amygdala has been postulated in the consolidation of emotional material into long term memory [31, 32]. Consolidation is the process that is widely believed to stabilise representations into long-term memory [e.g. 1]. McGaugh et al. hypothesise that emotional arousal associated with the release of neurohormones such as adrenaline and corticosterone enhances memory for the stressful event by activation of the amygdala. The amygdala, in turn, is hypothesised to modulate memory consolidation processes occurring in other brain regions including the hippocampus with which it has strong anatomical links. If the amygdala mediates consolidation processes the disruption of these processes would be expected to result in abnormal forgetting. This prediction is based on the belief that the process of consolidation occurs during a limited time following encoding [e.g. 1]. If this process were impaired, therefore, forgetting would be expected to proceed at an abnormal rate over the time period when consolidation would normally be occurring.

If the amygdala modulates memory in other brain structures, stimulation of the amygdala should influence the formation of the type of memory thought to involve these other structures [e.g. 36]. So, if the amygdala plays a role in the consolidation of emotional memory via its modulatory effects on the hippocampus, it would be expected that patients with damage to the amygdala would show abnormal rates of forgetting for emotional material on tests dependent on hippocampal functioning. There has been much recent debate about the precise role of the hippocampus [see 30]. It is thought to play a role in the consolidation of associative material into long-term storage. This associative material almost certainly includes information

tapped by tests of free recall. The question of whether the hippocampus is also important for performance on tests of item recognition, which is thought not to depend on the formation of associations, is the focus of much current debate [see 6, 30, 51].

There is some evidence that rate of forgetting of emotional material may indeed be abnormal in people with amygdala damage [37]. This study reported rapid forgetting of emotionally arousing, but not neutral stimuli using a test of free recall in a patient with structural damage to the amygdala. This occurred despite the fact that the emotionality ratings she assigned to stimuli during the presentation phase of the experiment did not differ from those of the control participants.

No studies have been conducted in which rate of forgetting has been systematically investigated in people with PTSD. The overall aim of this study, therefore is to investigate rate of forgetting in this patient group. If the amygdala is involved in consolidation of emotional memory and patients with PTSD show brain abnormalities in this region it could be hypothesised that their rate of forgetting of free recall of emotional material (reflecting the consolidation process) will be abnormal.

As people with PTSD have been found to have deficits in attention [e.g. 46] it will be necessary to use a matching procedure whereby patients are given increased exposure times to stimuli in the presentation phase in order to match their performance to that of the control group at a short delay [20]. This is necessary because unless groups are initially performing at similar levels, forgetting cannot

reliably be compared. This is because it cannot be assumed that the forgetting function across different levels of performance is linear [26].

The prediction concerning rate of forgetting on tests of free recall of neutral material in these patients is unclear. This would depend on the structural and functional integrity of the hippocampus. At present there appear to be no published studies specifically investigating hippocampal function in people with PTSD, although both [35] and [42] detected abnormalities in this region. In addition, several studies have reported reductions in hippocampal volume [10]. However, it is not clear from these studies that such reductions are specifically associated with PTSD or whether they are associated with co-morbid conditions. In addition, there are no reports of hippocampal damage in patients in whom the condition is not chronic and intractable [e.g. 9]. If people with PTSD do not have hippocampal abnormalities then their rate of forgetting for neutral material should be normal. If there are abnormalities in hippocampal functioning, however, rate of forgetting of free recall of neutral material would be expected to be accelerated.

The first aim of this study, therefore, is to compare the rate of forgetting of free recall of emotional and neutral words in a group of patients with PTSD with that of a group of healthy controls matched for age, sex and IQ. In relation to this aim the hypothesis is that people with PTSD will forget emotional words at an abnormal rate in comparison with the control participants. The hypothesis concerning neutral words is less clear and will depend on the normality of hippocampal function as discussed above.

The second aim of this study is to compare performance on the above tasks using a recognition paradigm, which could further help to define the precise pattern of memory deficit associated with PTSD. Rate of forgetting on tests of recognition may be expected to proceed at a normal rate regardless of whether hippocampal abnormalities are present in PTSD. This is because recent studies have suggested that item recognition does not depend on hippocampal function [e.g. 30, 45]. Thus, the second hypothesis is that people with PTSD will forget at a normal rate when tested by recognition regardless of the emotionality of the stimuli.

Method

Participants

Participants with PTSD were recruited via the regional clinical psychology services of Worcestershire and Herefordshire and gave their informed consent to take part in the study (see Appendix 1). All had either received therapy, were undergoing therapy or were awaiting therapy for PTSD. The group comprised seven individuals (five males, two females). Patient participants were administered the Posttraumatic Stress Disorder Diagnostic Scale (PDS)[15] to determine current PTSD status. Five fulfilled full DSM IV criteria for PTSD as assessed by this scale and two fulfilled all criteria except for cluster C which assesses symptoms of avoidance. As the focus of this study was on symptoms relating to re-experiencing phenomena, these two participants were nevertheless included. Four participants had PTSD as the result of road traffic accidents (RTA), one as the result of a farming accident, one as the result of a hotel fire and one as the result of a fall while heavily pregnant. Time since trauma varied from one year and three months to three years. No patient had experienced any period

of unconsciousness either as a result of the trauma that caused PTSD or on any other occasion, apart from one who had suffered a fractured skull as a five-year old and had been rendered unconscious for several hours. The patient reported no effects on memory or cognition as a result of this incident, however. Current alcohol consumption in the group varied from less than one unit to 16 units per week. No patient reported alcohol related problems in the past. Only one participant was on current medication (Seroxat (30mg) and Zopiclone (7.5mg) per day). Three others reported taking anti-depressant or anxiolytic medication following their trauma but had been off medication for at least four months prior to this study.

Control participants were recruited from the non-academic staff of the Universities of Coventry and Warwick. The participants included in the analysis were selected from a group of 13 who originally participated in the study. From these 13 participants the seven that were best matched in terms of age, sex and IQ were selected. This group also comprised seven individuals, therefore, (five males, two females). No individual had experienced any period of unconsciousness, and none were currently, or ever had been, on medication for a psychiatric or neurological condition. Current alcohol consumption varied from approximately one unit to 21 units per week.

Background data for the two groups are presented in Table 1. Participants were administered the National Adult Reading Test-Revised (NART-R) [34] to provide an estimate of premorbid IQ. To allow comparison with current IQ assessed by the Wechsler Abbreviated Scale of Intelligence (WASI) three points were subtracted from NART-R Full Scale IQ estimates (see Technical Manual). Participants were

also administered the Hospital Anxiety and Depression Scale (HADS) [49] to provide brief measures of anxiety and depression. Based on normative data, scores on this assessment are classed as non-cases, doubtful cases or cases. For the measure of anxiety three patients were classed as cases, two control participants were classed as doubtful cases and one as a case. For the depression measure two patients were classed as cases, but none of the controls were.

	Age	NART	WASI IQ			HADS	
			FS	V	P	Anxiety	Depression
PTSD	39.4	106.1	105.9	104.1	107.6	10.0	6.6
	(10.9)	(11.4)	(66.8)	(7.6)	(13.5)	(5.4)	(5.4)
Control	37.9	109.1	111.9	112.7	108.7	6.9	2.1
	(12.7)	(11.7)	(9.5)	(10.8)	(11.5)	(3.4)	(1.3)

Table 1. *Background data for the groups of PTSD and control participants. Group mean values are given for each with standard deviations in parentheses. See text for details. [FS=Full Scale; V=Verbal; P=Performance.]*

A series of independent t tests carried out on the background data from the two groups indicated that the groups did not differ significantly in terms of age, NART FS IQ, WASI FS IQ, WASI V IQ, WASI P IQ, HADS anxiety, or HADS depression (all $p > 0.05$; see Appendix 2 for details). There was, however a strong trend for a group difference on the depression measure.

The participants with PTSD completed both the PDS and the Revised Impact of Events Scale (R-IES) [19]. The scores for each individual participant for the severity of the intrusion, avoidance and hyperarousal symptoms as measured by these instruments are shown in Table 2. Also shown is the classification of the severity of

the symptoms from the PDS

	R-IES			PDS			
	I	A	H	I	A	H	Severity
NS	18	15	12	9	8	10	Moderate-severe
WS	10	12	1	7	8	4	Moderate
RN	13	4	4	3	2	3	Mild
KS	19	22	18	14	20	13	Severe
DH	10	0	1	6	4	8	Moderate
AW	12	1	14	5	1	4	Mild
AY	12	9	4	3	3	4	Mild

Table 2. *Scores on PDS and IES for symptoms of intrusion (I), avoidance (A) and hyperarousal (H) and severity measure from PDS.*

Materials

The emotional words were taken from [22]. Two categories were included; those associated with ‘anger’ and ‘anxiety’. The words appeared either as stimulus words or in the free association responses to those words. These categories were selected because these are the emotions that have been most closely associated with amygdala functioning [4]. Thirty five words from each category were selected for piloting.

The non-emotional words were selected to be as similar to the emotional words as possible. Thus, it was necessary to select neutral words from categories that were somewhat related as well as selecting related words within categories. Two words from the neutral word list from [22] were selected. These were ‘active’ and ‘gigantic’. The associates of each of these words were also included. This did not

yield a sufficient number of words so a pilot study was conducted in which five individuals were requested to think of as many associates of these words as possible. Individuals were asked to think of words similar in meaning to the targets rather than opposites to ensure that the stimuli in these categories would be similarly related to those in the emotional categories.

The words from the four categories were compiled into a pilot questionnaire comprising 140 words randomly arranged with a blank column in which subjects made their rating. This was completed by 23 respondents who were asked to provide a rating for each word on a six-point scale according to how emotional it was to them. The mean rating for each word was calculated. From each of the emotional categories the three words with the lowest mean emotionality ratings were excluded and from each of the non-emotional categories the three words with the highest emotionality ratings were excluded. This resulted in two sets of words, emotional and non-emotional in which emotionality ratings were non-overlapping (Emotional words, mean rating=4.0, range=2.8-5.6; Non-emotional, mean=1.8, range=1.1-2.6).

Words from each emotional and non-emotional list were randomly sorted into four lists comprising eight words from the two categories. Thus, there were four lists of emotional words each containing eight words related to 'anger' and eight words related to 'anxiety' and four lists of non-emotional words each comprising eight words relating to 'active' and eight words relating to 'gigantic'. Statistical comparison ensured that the lists comprising emotional words and those comprising non-emotional words were closely matched for emotionality rating. In addition, each emotional list differed significantly from each non-emotional list in

terms of emotionality rating. Across emotional and non-emotional lists words were matched for number of letters and number of syllables and where data were available, for frequency, concreteness and imageability [33]. Copies of the stimulus lists can be found in Appendix 3.

The four lists of emotional words and four lists of non-emotional words were used in the following way. One list was for piloting, one list served as the target list in the rate of forgetting test, one list served as the foil list for the recognition test at the short delay and the final list served as the foil list for the recognition test at the long delay.

Design

The experiment comprised a two groups (PTSD, control participants) by two list types (emotional, non-emotional) by two delays (20 seconds, one hour) design. Presentation of the lists was counterbalanced across participants such that they were equally likely to be presented with list one, two, three or four during the pilot session and so on. Within lists words were blocked by category but for half the participants in the emotional condition words from the ‘anger’ category were presented first whereas the other half were presented first with the ‘anxiety’ category. A similar procedure was followed for the non-emotional word lists. Order of presentation of the emotional and non-emotional lists was also counterbalanced so that half the participants were presented and tested first on the emotional list and half on the non-emotional list.

Procedure

Participants were seen individually for three sessions. The first session was a pilot session during which free recall of one emotional and one non-emotional word list was tested at the short delay of 20 seconds only. This was to determine performance levels so that presentation times could be manipulated to achieve a match between groups at the short delay. For the pilot session participants were seated in front of an Apple Macintosh Ibook. First, participants were given a practise session of the filler task that they would perform during the 20 second delay between list presentation and recall of that list. The purpose of this task was to eliminate, as far as possible, any contribution of short-term memory to the recall task. Participants were told that they would see a series of two or three-digit numbers appear on the screen one at a time and that they should read aloud these numbers and say whether they were odd or even as quickly as possible. Participants were given a brief practise , following which, a series of numbers appeared on the screen at a rate controlled by the experimenter via a mouse-click. The rate at which participants could perform the task was stored and the numbers appeared at this predetermined rate during all 20 second delays for that participant.

Participants were then asked to read the instructions for the word-list task which appeared on the computer screen. These informed them that a list of words would be presented one at a time in a box in the centre of the screen. They were told that these words would be related to each other in meaning and that noticing this may help them remember the words. Participants were asked to read the words aloud as they appeared and to try to remember them. They were informed that after

reading each word they would be asked to rate it according to how emotional the word was to *them*. The rating was to be made on a scale of 1 to 4 using the labelled keys (where 1=very unemotional and 4=very emotional). Participants were asked to make their rating as soon as possible after the word disappeared, and were told that after they had made their rating the next word in the list would appear. They were told that the purpose of the rating was to help them remember the words and that their priority was, therefore, was to remember the words rather than provide accurate ratings. Participants were informed that when they had seen all the words they would be asked to perform the odd/even task following which they would be asked to think back to this list and say as many of the words as they could remember. Before the first list appeared there was a short practise session so that participants would become familiar with making the emotionality rating.

Following this, the first pilot list was presented. For the pilot session all participants were shown each word in the list for 4 seconds. As participants were required to make their rating after the word had disappeared, presentation times for the list varied slightly between participants, a record was kept of the exact presentation times, however. Following presentation of the words and the 20 second delay participants were asked to say as many words from the list as they were able. They were given two minutes in which to do this and responses were recorded by the experimenter.

During the pilot session, one list was presented and tested at the beginning of the session and the other at the end of the session. Between list presentations, each participant also completed the NART-R and the two verbal subtests of the WASI.

In addition, the participants with PTSD completed the R-IES.

The recall results from the pilot session were used to determine presentation times for the stimuli during the presentation phase of the experimental lists. If participants demonstrated low levels of recall, presentation times during the subsequent two sessions were increased and if they showed exceptionally high levels of recall, presentation times were reduced. All participants received the same presentation times across emotional and non-emotional lists, however. In practise, the majority of participants in both groups received one presentation of four seconds per word. However, in the PTSD group two participants received two presentations of three seconds per word (during the second presentation words were shown in the same order as the first) and in the control group one participant received one presentation of three seconds per word and another participant one presentation of two seconds per word.

During the second session participants were presented with and tested for free recall and recognition of one list at a short delay of 20 seconds and a long delay of one hour. The list comprised either emotional or non-emotional words depending on the counterbalance condition. The presentation procedure for this was identical to that described above except that following the two-minute recall test, participants were administered the test of recognition. For this, participants were presented with a list of 32 words one at a time on the computer screen (16 targets, 16 foils) and for each were required to say whether it had appeared in the original list, by making a key-press. There was no time limit set on this test. During the one-hour delay, participants completed the performance subtests from the WASI, the HAD scale and other cognitive tests not involving verbal material. The third

session was similar to the second and involved presentation of the final list. During the hour delay, participants completed other cognitive tests not involving verbal material. During the second two sessions, any time that was not taken up with cognitive testing was spent in conversation with the experimenter. At the end of the final session, participants with PTSD were asked to complete the PDS.

Results

Throughout this paper an alpha level of 0.05 has been adopted, unless otherwise stated. Where mean scores are presented, standard deviations are given in parentheses. Raw data for each condition can be found in Appendix 4.

The data from the presentation phase of the experiment were analysed first to investigate possible differences between groups in duration of stimulus presentation and in emotionality ratings assigned to the stimuli.

To determine the difference in presentation times across groups and conditions a two-way analysis of variance (ANOVA) with a between subjects factor of group and a within subjects factor of condition (emotional, non-emotional) was carried out. This revealed a trend for a significant main effect of group [$F(1,12)=3.5$, $p=0.09$] indicating that PTSD participants tended to have longer presentation times than control participants.

A similar mixed ANOVA carried out on the mean emotionality ratings for each participant in the two conditions revealed a highly significant effect of condition [$F(1,12)=66.53$, $p<0.01$]. However, the main effect of group and the interaction between group and condition were not significant indicating that groups performed

similarly in assigning higher emotionality ratings to words in the emotional condition.

Free Recall

The data from this condition were then analysed to ensure that a match had been achieved between the two groups at the short delay in both emotional and non-emotional conditions. In the emotional condition the PTSD group obtained a mean score of 8.7 (2.4) and the control group a mean score of 8.6 (1.9). In the non-emotional condition the PTSD group obtained a mean score of 7.4 (1.8) and the control group a mean score of 8.1 (1.5). These data were submitted to a one way mixed ANOVA. This revealed a trend for participants to recall more words in the emotional condition than in the non-emotional condition [$F(1,12)=3.00$, $p=0.11$]. However, there was no main effect of group and no group by condition interaction indicating that the two groups were performing similarly at the short delay across the two conditions.

Although the groups were performing at a numerically similar level, three further tests were carried out on the recall data from the short delay to ensure, as far as possible, that the performance of the groups was also qualitatively similar. This is important as evidence suggests that recall of material in a disorganised manner is associated with a faster forgetting rate [38]. Recall of the two groups at the short delay was therefore analysed for evidence of semantic and serial order clustering. The semantic clustering measure was based on the 'C' measure [16] in which a cluster is defined as the successive recall of two items from the same semantic category, corrected for chance clustering. In the emotional condition the PTSD group obtained a mean clustering score of 0.72 (0.16) and the control group a mean

clustering score of 0.74 (0.10). In the non-emotional condition the PTSD group obtained a mean clustering score of 0.56 (0.21) and the control group a mean clustering score of 0.74 (0.21). A two way mixed ANOVA revealed no significant main effect of group [$F(1,12)=4.08, p=0.07$], although there was a trend towards fewer semantic clusters in the PTSD group. Although the group by condition interaction failed to achieve significance, it is clear from the mean scores that semantic clustering is closely matched between groups for the emotional stimuli, whereas there is a non-significant trend in the non-emotional condition for poorer clustering in the PTSD group.

For the serial order clustering measure a cluster was defined as the occurrence of two words successively in recall that had also occupied successive positions in the list at presentation. In the emotional condition the PTSD group obtained a mean serial order score of 0.15 (0.01) and the control group a mean score of 0.18 (0.09). In the non-emotional condition the PTSD group obtained a mean serial order score of 0.19 (0.17) and the control group a mean score of 0.17 (0.06). A two way mixed ANOVA revealed no significant effects, indicating no evidence of any difference in serial order clustering across groups or conditions.

Finally, the number of intrusion errors occurring in the recall of the two groups was analysed as an increased intrusion rate could also indicate qualitatively poorer recall. Overall number of intrusions in both groups was very low and the data were not statistically viable. Control participants made a total of 8 intrusions in the emotional condition and 7 in the non-emotional condition. PTSD participants made a total of 0 intrusions in the emotional condition and 6 in the non-emotional condition. Thus

there was no evidence of an increased intrusion rate in the PTSD group.

The results of these analyses indicated that the recall of the PTSD and control participants was both quantitatively and qualitatively similar. Following this, the rate of forgetting of the two groups across the two conditions was examined. The data from the emotional and non-emotional conditions are shown in Figure 1.

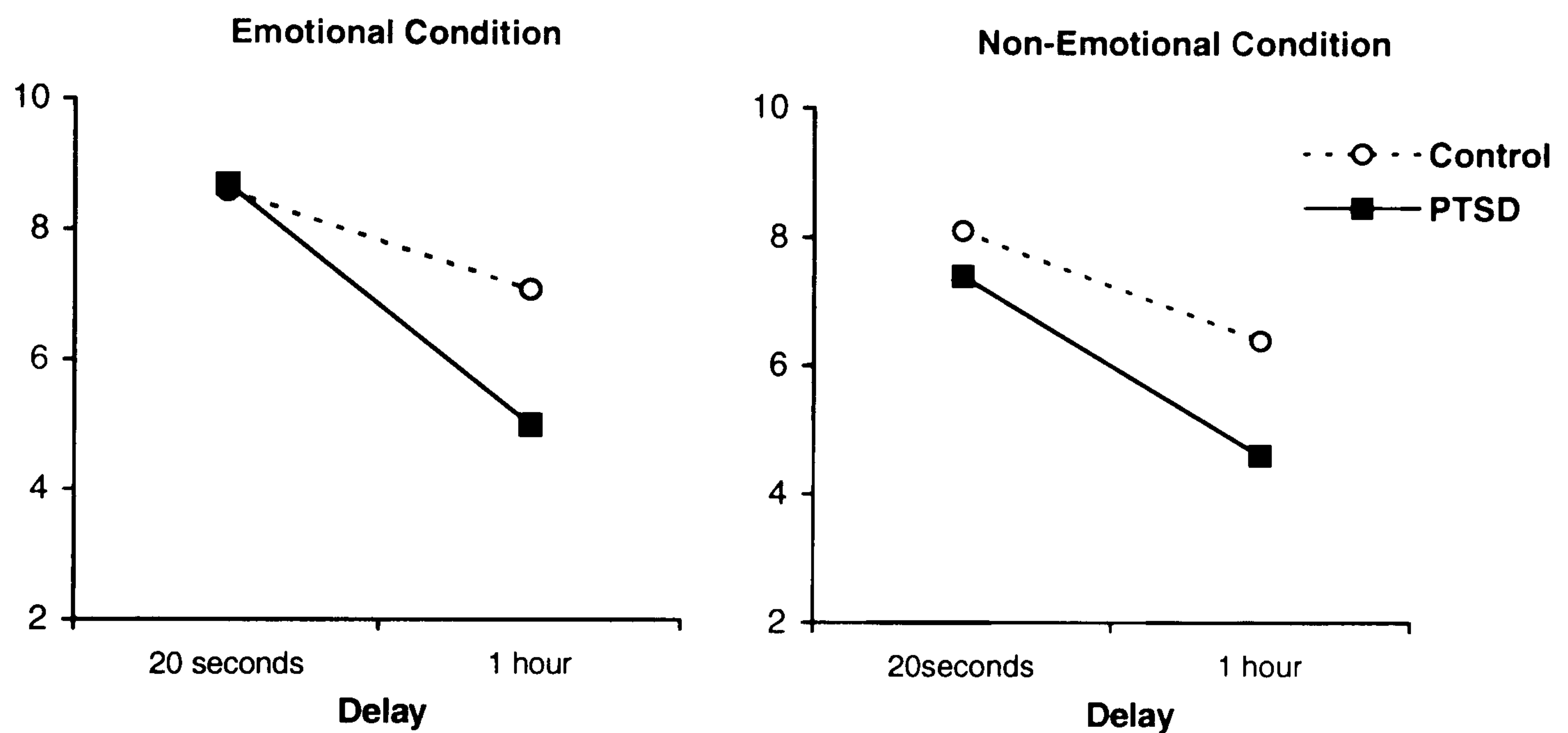


Figure 1 *Free recall performance of PTSD and control groups for emotional and non-emotional word lists.*

The data were submitted to a three way ANOVA with a between subjects factor of group (PTSD, control) and within subjects factors of condition (emotional, non-emotional) and delay (20 seconds, one hour). This revealed a significant main effect of delay [$F(1,12)=65.43, p<0.01$] and a significant group by delay interaction [$F(1,12)=8.15, p=0.01$] indicating that the PTSD group showed a faster rate of forgetting than the control group. However, there was no condition by delay interaction and no three way interaction between group, condition and delay

indicating no significant difference in forgetting of the groups across the two conditions.

To determine whether there was a trend for performance across conditions to differ separate two way mixed ANOVAs were carried out on the data from the emotional and non-emotional conditions. The analysis of the data from the emotional condition revealed a significant main effect of delay [$F(1,12)=84.52, p<0.01$] and a significant group by delay interaction [$F(1,12)=16.70, p<0.01$] indicating that the PTSD group was forgetting at a significantly faster rate than the control group. The data from the non-emotional condition were analysed in a similar way. The two-way ANOVA revealed a significant main effect of delay [$F(1,12)=17.46, p<0.01$], but no group by delay interaction, indicating no difference in forgetting rates across groups.

Correlational analyses were carried out to investigate a possible relationship between rate of forgetting and severity of PTSD symptoms, measured on the revised IES. However, correlations were low and no relationship could be demonstrated (all $ps>0.05$). The relationship between depression and rate of forgetting was also investigated across both groups of participants, however again no relationship could be demonstrated.

Recognition

The performance of the participants in the recognition condition was scored for number of hits and number of false alarms. The probabilities of hits and false alarms for each participant were then analysed using signal detection theory [see 27] which yields independent measures of memory sensitivity (d') and bias (c).

The recognition data from the short delay were first analysed to determine whether the groups were performing at a similar level. In the emotional condition the PTSD group obtained a mean d' score of 3.1 (0.7) and a mean c score of -0.1 (0.4), the control group a mean d' score of 3.1 (0.6) and a mean c score of -0.2 (0.2). In the non-emotional condition the PTSD group obtained a mean d' score of 2.4 (0.6) and a mean c score of -0.3 (0.4), and the control group a mean d' score of 2.4 (0.6) and a mean c score of -0.2 (0.3). Measures of d' and c were submitted to separate two way mixed ANOVAs. Analysis of the d' data revealed a significant main effect of condition [$F(1,12)=15.88$, $p<0.01$] indicating that performance was superior in the emotional condition. However, there was no group by condition interaction indicating that there was no difference in the pattern of performance of the two groups at the short delay. Analysis of the c data revealed no significant effects.

To investigate rate of forgetting the d' data were submitted to a three way mixed ANOVA with one between subjects factor of group and two within subjects factors of condition and delay. These data are shown in Figure 2. This revealed a significant main effect of condition [$F(1,12)=6.48$, $p=0.03$] indicating superior performance in the emotional condition, but no group by condition interaction indicating no difference in the performance of the two groups across the conditions. There was also a significant condition by delay interaction [$F(1,12)=10.20$, $p<0.01$] indicating more forgetting in the emotional than in the non-emotional condition. However, the three way interaction between group, condition and delay was not significant indicating no difference in the pattern of performance across groups.

As in the free recall condition the pattern of performance in each condition was examined using two-way mixed ANOVAs. The analysis of the data from the

emotional condition revealed a significant main effect of delay [$F(1,12)=6.60$, $p=0.02$], but no group by delay interaction indicating no difference in forgetting rates between the groups. The analysis of the data from the non-emotional condition revealed a marginally significant group by delay interaction [$F(1,12)=4.72$, $p=0.05$]. From Figure 2 it can be seen that whereas the PTSD group forgot at a similar rate across conditions, the control group actually showed improved performance at the long delay in the non-emotional condition.

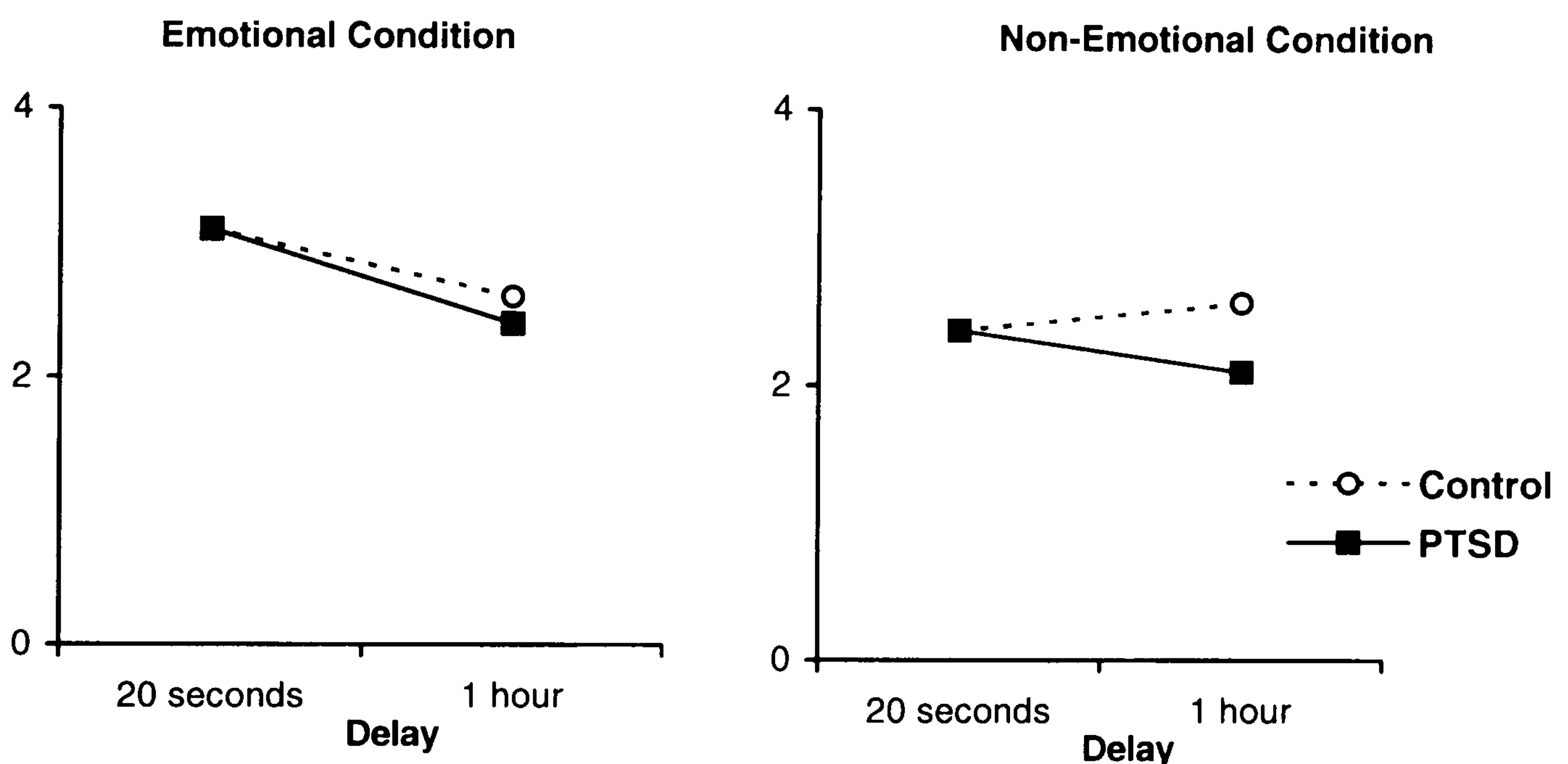


Figure 2. *Recognition performance (d') of PTSD and control groups for emotional and non-emotional word lists.*

A similar analysis carried out on the bias data revealed no significant main effects and no interactions (all $p>0.05$) indicating that the two groups adopted similar criteria for responding across the conditions.

Discussion

The results of this study showed that free recall performance of the patients with

PTSD and the control participants was successfully matched at a short delay in both emotional and non-emotional conditions. Despite this, the PTSD patients forgot emotional material at an accelerated rate, whereas non-emotional material was forgotten at a more normal rate. In the recognition paradigm forgetting in the PTSD group proceeded at a normal rate for emotional words. Forgetting of non-emotional words, tested by recognition was difficult to interpret as control performance improved over the delay. Also in the recognition condition, there appeared to be no difference in the response criteria adopted by the patient and control groups, assessed by a measure of bias.

It should be noted that the PTSD group contained individuals who had PTSD as a result of being involved in RTA. It is known that memory function is particularly vulnerable to the damage caused by closed head injury [see 41]. It is possible, therefore, that the faster forgetting in this group could be attributable to the incidence of closed head injury rather than to the effects of PTSD. This explanation is unlikely, however, because the effects of head injury would be expected to affect forgetting of emotional and non-emotional stimuli similarly and this was not the case in this study. In addition, although a statistical comparison of the groups of PTSD patients who had been involved in RTAs with those not involved in RTAs was not viable, inspection of the mean scores provided no evidence that forgetting proceeded at a different rate in these groups.

There was no evidence that the fast forgetting for emotional words seen in the patient group was associated with increased levels of depression. Neither did there appear to be a relationship, however, with severity of PTSD symptomatology measured with the Revised IES. The results are consistent with the hypothesis outlined in the

introduction that people with PTSD suffer a consolidation deficit for emotional material. However, there are a number of alternative explanations of these results, which will be discussed in turn below.

First, the fact that patients required more exposure to the material during the presentation phase in order to match their level of recall to that of the control participants implies that they also suffer an encoding deficit possibly related to an attention or concentration problem. Such a deficit has been well-documented in previous studies [e.g. 21]. It is likely that encoding deficits that result in material being encoded in an impoverished way could also lead to the appearance of faster forgetting because memory is known to decline at a slower rate for material encoded in an organised manner [e.g. 38]. Because of this possibility, care was taken in the present study to investigate the qualitative nature of recall at the short delay. Participants in the patient and control groups recalled similar numbers of words, with similar numbers of serial order clusters. There was some evidence of reduced semantic clustering in the recall of non-emotional stimuli in the PTSD group. However, this did not achieve significance and levels of semantic clustering in the emotional condition were closely matched across groups. Neither group made large numbers of intrusion errors. In addition, patient participants showed a similar enhancement to the control participants in recalling words from the emotional list at the short delay, providing further evidence that they were processing the material in a normal way.

The second means by which an encoding deficit could account for the results is related to the fact that the test of recognition followed the test of free recall. This makes it possible that the control participants used the presentation of the targets

in the recognition test as a further encoding opportunity, which may have attenuated their rate of forgetting. The opportunity may not have been available to the patient participants to the same extent because of their impaired encoding ability. Although it is very difficult to discount this possibility entirely there are two reasons why it is unlikely that this could have accounted for the pattern of results reported. First, the patients in this study required a non-significantly longer presentation time than the controls to match performance at the short delay. It seems unlikely that the mild encoding deficit could account for the full magnitude of the difference in forgetting rate found. In addition, such an explanation could not account for the difference in pattern of performance across emotional and non-emotional conditions.

The third alternative explanation of the results is that the increased presentation times given to the patients during the presentation phase could also have affected their rate of forgetting. The matching procedure [20] whereby performance at a short delay is matched between groups by giving one group increased exposure to material at presentation was designed to investigate rate of forgetting in people with organic amnesia. Since then, the procedure has been extensively used to compare the performance of memory impaired and healthy individuals. There are methodological difficulties associated with this procedure [see 29 for a discussion]. However, these would tend to bias against finding faster forgetting in the group with increased exposure. In addition, the difference in presentation times between patient and control groups in the current study is likely to be much less than those in studies comparing rate of forgetting across healthy individuals and those with profound memory impairment associated with organic amnesia. It is unlikely therefore that the results reported in this study arise as an artefact of the matching procedure.

The results of this study are consistent with studies showing abnormal amygdala function in people with PTSD. The PTSD participants in this study showed a similar pattern of performance to that of a patient with structural damage to the amygdala [37]. Their patient, like the PTSD patients in the study reported here, assigned similar emotionality ratings in comparison with control participants to the words during the presentation phase of the experiment. She also showed a similar advantage to the control participants in showing superior recall of emotional words in comparison with neutral words at the short delay. However, across a delay of one hour, she showed fast forgetting of emotionally arousing stimuli but normal forgetting of neutral stimuli.

The results of the study reported here are consistent with the hypothesis described in the introduction [31], that the amygdala mediates consolidation into long term memory of emotional information via its links with the hippocampus. It is not clear why increased activity in the amygdala, which has been found in patients with PTSD, results in a similar pattern of performance to that seen in people with structural damage in this region. However, the results are consistent with another study (see Chapter 3 this volume) which showed, an abnormal pattern of identification of facial expressions in PTSD, consistent with than shown in patients with structural damage to the amygdala.

Although the results showing accelerated forgetting in the PTSD group in the emotional condition are clear-cut, results from the non-emotional condition are more difficult to interpret. There was a trend towards faster forgetting in the PTSD group in this condition, although there was also a trend for participants with PTSD to produce fewer semantic clusters in their recall, which could indicate that encoding

was abnormal in this condition. Consistent with a more general consolidation deficit are the results of a study [14] employing regression techniques to show that delayed, but not immediate, verbal memory is associated with symptoms of re-experiencing in PTSD. As discussed in the introduction, there is no evidence at present for hippocampal volume reductions in people with PTSD in whom the condition is not chronic and intractable. However the possibility has not been extensively investigated and the additional possibility that there are functional abnormalities in the hippocampus of patients with PTSD has received some support [35, 42]. Although this hypothesis has not been fully investigated, such a deficit is postulated by a number of PTSD theorists [e.g. 23, 24].

Findings in the recognition condition of this study are also difficult to interpret. There was no evidence of a difference in forgetting across groups for emotional material. However, performance of the control group in the non-emotional condition was characterised by improved performance over the delay. Inspection of the raw data indicated that the effect was mainly due to the performance on one control participant. If this participant was removed from the data, the resulting interaction became non-significant. It seems likely, therefore, that this represents an anomalous result. Generally, the data from the recognition condition support the contention that the amygdala does not support consolidation into long-term memory of information assessed by item recognition via its links with the hippocampus.

The results of this study provide preliminary evidence of a consolidation deficit, possibly associated with dysfunction of the amygdala, in people with PTSD. It will be important to replicate these results using a larger group of participants. Whether

people with PTSD also have a consolidation deficit related to possible hippocampal dysfunction remains unclear.

References

- [1] Abel, T., Alberini, C., Ghirardi, M., Huang, Y-Y., Nguyen, P. & Kandel, E.R. Steps toward a molecular definition of memory consolidation. In: D. Schacter (Ed.) *Memory Distortion*. Cambridge: Harvard University Press, 1995.
- [2] Adolphs, R., Cahill, L., Schul, R. & Babinsky, R. Impaired declarative memory for emotional material following bilateral amygdala damage in humans. *Learning and Memory* 1997; 4: 291-300.
- [3] Adolphs, R., Tranel, D., Damasio, H. & Damasio, A. Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. *Nature* 1994; 372: 669-672.
- [4] Adolphs, R., Tranel, D., Hamann, S., Young, A.W., Calder, A.J., Phelps, E.A., Anderson, A., Lee, G.P. & Damasio, A.R. Recognition of facial emotion in nine individuals with bilateral amygdala damage. *Neuropsychologia* 1999; 37: 111-117.
- [5] Aggleton, J.P. (Ed) *The Amygdala*. Oxford: Oxford University Press, 2000.
- [6] Aggleton, J.P. & Brown, M.W. Episodic memory, amnesia and the hippocampal-anterior thalamic axis. *Behavioural Brain Sciences* 1999; 22: 425-489.

- [7] Babinsky, R., Calabrese, P., Durwen, H.F., Markowitsch, H.J., Brechtelbauer, D., Heuser, L. & Gehlen, W. The possible contribution of the amygdala to memory. *Behavioural Neurology* 1993; 6: 167-170.
- [8] Beckham, J.C., Crawford, A.L. & Feldman, M.E. Trail making test performance in Vietnam combat veterans with and without posttraumatic stress disorder. *Journal of Traumatic Stress* 1998; 11: 811-819.
- [9] Bonne, O., Brandes, D., Gilboa, A., Gormi, J.M., Shenton, M.E., Pitman, R.K. & Shalev, A.Y. Longitudinal MRI study of hippocampal volume in trauma survivors with PTSD. *American Journal of Psychiatry* 2001; 158: 1248-1251.
- [10] Bremner, J.D. Hypotheses and controversies related to effects of stress on the hippocampus: An argument for stress-induced damage to the hippocampus in patients with posttraumatic stress disorder. *Hippocampus* 2001; 11: 75-81.
- [11] Brewin, C.R. (2001). A cognitive neuroscience account of posttraumatic stress disorder and its treatment. *Behaviour Research and Therapy* 2001; 39: 373-393.
- [12] Cahill, L., Babinsky, R., Markowitsch, H.J. & McGaugh, J.L. The amygdala and emotional memory. *Nature* 1995; 377: 295-296.

- [13] Cahill, L., Haier, R.J., Fallon, J., Alkire, M.T., Tang, C., Keator, D., Wu, J. & McGaugh, J.L. Amygdala activity at encoding correlated with long-term free recall of emotional information. *Proceedings of the National Academy of Sciences of the USA* 1996; 93: 8016-8021.
- [14] Diamond, T., Muller, R., Rondeau, L.A. & Rich, J.G. The relationships among PTSD symptomatology and cognitive functioning in adult survivors of child maltreatment. In: Columbus, F. (Ed.). *Advances in psychology research* Vol. V. (pp. 253-279). Huntington, New York: Nova Science Publishers, 2001.
- [15] Foa, E.B. *Posttraumatic Stress Diagnostic Scale*. National Computer Systems Inc. Minneapolis, 1995.
- [16] Folarin, B.A. A critical evaluation of clustering measures with worked examples. *The psychological Record* 1983; 33: 117-129.
- [17] Galletly, C., Clark, C.R., McFarlane, A.C. & Weber, D.L. Working memory in posttraumatic stress disorder: An event related potential study. *Journal of Traumatic Stress* 2001; 14: 295-309.
- [18] Hamann, S., Elt, T., Grafton, S. & Kilts, C. Amygdala activity related to enhanced memory for pleasant and unpleasant aversive stimuli. *Nature Neuroscience* 1999; 2: 289-293.

- [19] Horowitz, M.J., Wilner, N. & Alvarez, M.A. Impact of Event Scale: A measure of subjective stress. *Psychosomatic Medicine* 1979; 41: 209-218.
- [20] Huppert, F.A. & Piercy, M. Dissociation between learning and remembering in organic amnesia. *Nature* 1978; 275: 317-318.
- [21] Jenkins, M.A., Langlais, P.J., Delis, D. & Cohen, R. Attentional dysfunction associated with posttraumatic stress disorder among rape survivors. *The Clinical Neuropsychologist* 2000; 14: 7-12.
- [22] John, C. Emotionality ratings and free-association norms of 240 emotional and non-emotional words. *Cognition and Emotion* 1988; 2: 49-70.
- [23] Koenen, K.C., Driver, K.L., Oscar-Berman, M., Wolfe, J., Folsom, S., Huang, M.T. & Schlesinger, L. Measures of prefrontal system dysfunction in posttraumatic stress disorder. *Brain and Cognition* 2001; 45: 64-78.
- [24] Krystal, J.H., Bennett, A.L., Bremner, J.D., Southwick, S.M. & Charney, D.S. Toward a cognitive neuroscience of altered memory functions in posttraumatic stress disorder. In M.J. Friedman, D.S. Charney & A.Y. Deutsch (Eds.) *Neurobiological and clinical consequences of stress: From normal adaptation to PTSD*. Philadelphia: Lippincott-Raven, 1995.
- [25] Liberzon, I., Taylor, S.F., Amdur, R., Jung, T.D., Chamberlain, K.R., Minoshima, S. & Koeppe, R.A. Brain activation in PTSD in response to trauma-related stimuli. *Biological Psychiatry* 1999; 45: 817-826.

- [26] Loftus, G.R. Evaluating forgetting curves. *Journal of Experimental Psychology. Learning, Memory and Cognition* 1985; 2: 397-406.
- [27] Macmillan, N.A. & Creelman, C.D. *Detection theory: A users guide*. Cambridge: Cambridge University Press, 1991.
- [28] Markowitsch, H.J., Calabrese, P., Wurker, M., Durwen, H.F., Kessler, J., Babinsky, R., Brechtelsbauer, D., Heuser, L. & Gehlen, W. The amygdala's contribution to memory – a study of two patients with Urbach-Wiethe disease. *Neuroreport* 1994; 5: 1349-1352.
- [29] Mayes, A.R. *Learning and memory disorders and their assessment*. *Neuropsychologia* 1986; 24: 25-39.
- [30] Mayes, A.R., Holdstock, J.S., Isaac, C.L., Hunkin, N.M. & Roberts, N. Relative sparing of item recognition memory in a patient with adult-onset damage limited to the hippocampus. *Hippocampus* 2001; in press.
- [31] McGaugh, J.L., Cahill, L. & Roozendaal, B. Involvement of the amygdala in memory storage: Interaction with other brain systems. *Proceedings of the National Academy of Sciences, USA* 1996; 93: 13508-13514.
- [32] McGaugh, J.L., Roozendaal, B. & Cahill, L. Modulation of memory storage by stress hormones and the amygdaloid complex. In M.S. Gazzaniga, (Ed) *The new cognitive neurosciences (2nd ed.)*. Cambridge, US: The MIT Press, 2000.

- [33] MRC Psycholinguistic Database, University of Western Australia.
www.psy.uwa.edu.au/MRCDataBase.
- [34] Nelson, H.E. *The National Adult Reading Test.*, Windsor, U.K: NFER-Nelson, 1982.
- [35] Osuch, E.A., Benson, B., Geraci, M., Podell, D., Herscovitch, P., McCann, U.D. & Post, R.M. Regional cerebral blood flow correlated with flashback intensity in patients with posttraumatic stress disorder. *Biological Psychiatry* 2001; 50: 246-253.
- [36] Packard, M.G., Cahill, L. & McGaugh, J.L. Amygdala modulation of hippocampal-dependent and caudate nucleus-dependent memory processes. *Proceedings of the National Academy of Sciences USA* 1994; 91: 8477-8481.
- [37] Phelps, E.A., LaBar, K.S., Anderson, A.K., O'Connor, K.J., Fulbright, R.K. & Spencer, D.D. Specifying the contributions of the human amygdala to emotional memory: A case study. *Neurocase* 1998; 4: 527-540.
- [38] Postman, L., Fraser, J. & Burns, S. Unit-sequence facilitation in recall. *Journal of Verbal Learning and Verbal Behavior* 1968; 7: 217-224.
- [39] Rauch, S.L., van der Kolk, B.A., Fisler, R.E., Alpert, N.M., Orr, S.P., Savage, C.R., Fischman, A.J., Jenike, M.A. & Pitman, R.K. A symptom provocation study of posttraumatic stress disorder using positron emission tomography and script-driven imagery. *Archives of General Psychiatry* 1996; 53: 380-387.

- [40] Rauch, S.L., Whalen, P.J., Shin, L.M., McNemer, S.C., Macklin, M.L., Lasko, N.B., Orr, S.P. & Pitman, R.K. Exaggerated amygdala response to masked facial stimuli in posttraumatic stress disorder: a functional MRI study. *Biological Psychiatry* 2000; 47: 769-776.
- [41] Richardson, J.T.E. (1990) *Clinical and Neuropsychological Aspects of Closed Head Injury*. London: Taylor & Francis.
- [42] Semple, W.E., Goyer, P., McCormick, R., Compton-Toth, B., Morris, E., Donovan, B. Muswick, G., Nelson, D., Garnett, M.L., Sharkoff, J., Leisure, G., Miraldi, F. & Schultz, S.C. Attention and regional cerebral blood flow in posttraumatic stress disorder patients with substance abuse histories. *Psychiatry Research Neuroimaging* 1996; 67: 17-28.
- [43] Shin, L.M., Kosslyn, S.M., McNally, R.J., Alpert, N.M., Thompson, W.L., Rauch, S.L., Macklin, M.L., Pitman, R.K. Visual imagery and perception in posttraumatic stress disorder. A positron emission tomographic investigation. *Archives of General Psychiatry* 1997; 54: 233-241.
- [44] van der Kolk, B.A. The psychobiology and psychopharmacology of PTSD. *Human Psychopharmacology Clinical and Experimental* 2001; 16: S49-S64.
- [45] Vargha-Khadem, F., Gadian, D.G., Watkins, K.E., Connelly, A. van Paesschen, W. & Mishkin, M. Differential effects of early hippocampal pathology on episodic and semantic memory. *Science* 1997; 277: 376-380.

- [46] Vasterling, J.J., Brailey, K., Constans, J.I. & Sutker, P.B. Attention and memory dysfunction in posttraumatic stress disorder. *Neuropsychology* 1998; 12: 125-133.
- [47] Wechsler Abbreviated Scale of Intelligence. The Psychological Corporation. Harcourt, Brace & Company: San Antonio, 1999.
- [48] Wolfe, J. & Charney, D.S. Use of neuropsychological assessment in posttraumatic stress disorder. *Psychological Assessment: A Journal of Consulting and Clinical Psychology* 1991; 3: 573-580.
- [49] Yule, W., Williams, R. & Joseph, S. Post-traumatic stress disorder in adults. In W. Yule (Ed.). *Post-Traumatic Stress Disorders: Concepts and Therapy*. England: Wiley, 1999.
- [50] Zigmond, A.S. & Snaith, R.P. The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavica* 1983; 67: 361-370.
- [51] Zola, S.M., Squire, L.R., Teng, E., Stefanacci, L. Buffalo, E. & Clark, E. Impaired recognition memory in monkeys after damage limited to the hippocampal region. *Journal of Neuroscience* 2000; 20: 451-463.

CHAPTER 3 : BRIEF PAPER

Identification of Negative Facial Expressions in Posttraumatic Stress Disorder

Abstract

Evidence suggests that social cognition is dependent on a number of brain structures including the amygdala and orbitofrontal cortex (OFC). A role for the amygdala in the identification of certain facial expressions, particularly those of fear and possibly anger, has been identified. The role of the OFC has been less well defined but recent evidence suggests that it is important in processing emotionally relevant stimuli, particularly those with negative valence. Functional imaging studies of posttraumatic stress disorder (PTSD) provide evidence of abnormal functioning in both amygdala and OFC. The aim of this study was to investigate identification of negative facial expressions, those related to amygdala functioning and those not closely related to amygdala functioning. Results showed an abnormal pattern of performance in the PTSD group characterised by a relative enhancement in the identification of non-amygdala related negative expressions and increased intensity ratings for these expressions.

Introduction

Social cognition is the ability to recognise, manipulate and behave in response to socially relevant information (Adolphs, 2001). The most well-studied aspect of social perception has involved the perception of faces and facial expressions. The ability to correctly perceive and interpret facial expressions is known to be vital for appropriate social interaction (see Cole, 1998).

Social cognition has been shown to be dependent on a number of interconnected neural structures including the amygdala, orbitofrontal cortex (OFC), cingulate cortex and right somatosensory cortex (see Adolphs, 2001). These structures are hypothesised to play a role in linking perception of socially relevant stimuli to motivation, emotion and cognition and thereby, social behaviour.

Evidence is accumulating from studies of people with posttraumatic stress disorder (PTSD) of functional abnormalities in structures linked with social perception. Most studies have used symptom provocation paradigms, in which trauma-related information is presented to participants. Under these conditions studies have reported increased function in the amygdala (e.g. Liberzon, et al. 1999; Rauch et al. 1996; Shin, et al. 1997) and in OFC (e.g. Rauch et al., 1996; Shin et al., 1999). Functional abnormalities in these regions have also been found in response to more general stimuli. For example Rauch et al. (2000) found an exaggerated amygdala response in PTSD to fearful versus happy faces and Semple et al. (1996) reported increased activity in OFC in PTSD patients, in comparison with controls, while they performed a cognitive challenge task. These studies are important because they provide evidence in PTSD of abnormal functioning in amygdala and OFC not related to

information concerning the individual's own traumatic memories.

Hypotheses about the kind of difficulties that abnormal amygdala functioning could cause can be formed by studying the literature focusing on rare patients with selective damage to this structure. Such studies have found that although these patients are generally unimpaired in their ability to recognise the unique identity of faces, they are impaired in identifying facial expressions of fear and to a lesser extent, anger (e.g. Adolphs, Tranel, Damasio & Damasio, 1994; Adolphs et al. 1999; Calder et al. 1996). Further support for these findings comes from a study by Scott et al (1997) who report a selective impairment in the identification of fear and anger from stimuli in the auditory modality in a patient with selective amygdala damage.

As well as being impaired in the identification of these expressions patients with amygdala damage have also been reported to rate levels of arousal depicted in expressions of fear and anger as less intense than did control participants (e.g. Adolphs et al. 1999; Weniger, Irle, Exner & Ruther, 1997). Consistent with these findings, Adolphs, Tranel & Damasio, (1998) reported that patients with amygdala damage consistently rated faces as more approachable and trustworthy than did control participants.

The precise role of the OFC in social cognition has not been as widely researched as that of the amygdala, although damage in this region has been found to disrupt social behaviour (see Damasio, 1994). More recently, in a study using functional magnetic resonance imaging, Perlstein, Elbert and Stenger (2002) found activation in the OFC while healthy participants processed emotionally arousing material. In a task that

including a working memory component this role was particularly prominent for stimuli with negative valence.

The possibility that impairments in social cognition are a feature of PTSD, related to abnormalities in structures important for these functions has not been the focus of any published studies. It is however a possibility that could have important implications for the adjustment and therapy of people with this disorder.

The purpose of the present study was to investigate the performance of individuals with PTSD on the identification of negative facial expressions from the Ekman-Friesen (1976) series. The predictions of the current paper are based on studies of people with structural amygdala damage showing impairments particularly related to identifying facial expressions of fear and anger. It is hypothesised that people with PTSD will show an abnormal pattern of performance on these expressions.

Findings of abnormal OFC functioning in PTSD imply that more general negative stimuli may also be processed abnormally. Findings that people with PTSD are hypervigilant for signs of threat in the environment and show an attentional bias for these signs (e.g. Buckley, Blanchard & Neill, 2000) support this possibility. To investigate this, identification of other negative emotions, disgust and sadness, was also investigated.

No impairment was predicted on identification of happiness. Predictions regarding the expression of surprise are uncertain, as it is not clear whether this should be classed as a positive or a negative emotion. In this study, therefore, surprise was not included as a negative emotion. Consequently the identification of expressions of

happiness and surprise were not included in the main analysis.

The first aim of the study, therefore, was to investigate performance of people with PTSD at identifying negative emotions associated with amygdala dysfunction (fear and anger) and to compare this with identification of negative emotions that have not been closely related to amygdala dysfunction (sadness and disgust). The second aim was to investigate ratings of intensity assigned to correctly identified expressions in people with PTSD and compare them with those assigned by healthy control participants. In order to accurately interpret the results of the study it was also necessary to ascertain whether face perception ability and the ability to recognise the unique identity of faces was intact in this patient group.

Method

Participants

Participants with PTSD were recruited via the regional clinical psychology services of Worcestershire and Herefordshire and gave their informed consent to take part in the study (see Appendix 1). All had either received therapy, were undergoing therapy or were awaiting therapy for PTSD. The group comprised seven individuals (five males and two females). Patient participants were administered the Posttraumatic Stress Disorder Diagnostic Scale (PDS; Foa, 1995) to determine current PTSD status. Five fulfilled full DSM IV criteria for PTSD as assessed by this scale and two fulfilled all criteria except for cluster C which assesses symptoms of avoidance. As the focus of this study was on symptoms relating to re-experiencing phenomena, these two participants were nevertheless included. Four participants had PTSD as the result of road traffic accidents, one as

the result of a farming accident, one as the result of a hotel fire and one as the result of a fall while heavily pregnant. Time since trauma varied from one year and three months to three years. No patient had experienced any period of unconsciousness either as a result of the trauma that caused PTSD or on any other occasion, apart from one who had suffered a fractured skull as a five-year old and had been rendered unconscious for several hours. The patient reported no effects on memory or cognition as a result of this incident, however. Current alcohol consumption varied from less than one unit to 16 units per week. No patient reported alcohol related problems in the past. Only one participant was on current medication (Seroxat (30mg) and Zopiclone (7.5mg)). Three others reported taking anti-depressant or anxiolytic medication following their trauma but had been off medication for at least four months prior to this study.

Control participants were recruited from the non-academic staff of the Universities of Coventry and Warwick. The participants included in the analysis were selected from a group of 13 who originally participated in the study. From these 13 participants the seven that were best matched in terms of age, sex and IQ were selected. This group also comprised seven individuals (five males and two females). No individual had experienced any period of unconsciousness, and none were currently, or ever had been, on medication for a psychiatric or neurological condition. Current alcohol consumption varied from approximately one unit to 21 units per week.

Background data for the two groups are presented in Table 1. Participants were administered the National Adult Reading Test-Revised (NART-R; Nelson, 1982) to provide an estimate of premorbid IQ. To allow comparison with current IQ

assessed by the Wechsler Abbreviated Scale of Intelligence (WASI) three point were subtracted from NART-R Full Scale IQ estimates (see Technical Manual). Participants were also administered the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) to provide brief measures of anxiety and depression. Based on normative data, scores on this assessment are classed as non-cases, doubtful cases or cases. For the measure of anxiety three patients were classed as cases, two control participants were classed as doubtful cases and one as a case. For the depression measure two patients were classed as cases, but none of the controls were.

	Age	NART	WASI IQ			HADS	
			FS	V	P	Anxiety	Depression
PTSD	39	106.1	105.9	104.1	107.6	10.0	6.6
	(10.9)	(11.4)	(66.8)	(7.6)	(13.5)	(5.4)	(5.4)
Control	37	109.1	111.9	112.7	108.7	6.9	2.1
	(12.7)	(11.7)	(9.5)	(10.8)	(11.5)	(3.4)	(1.3)

Table 1. *Background data for the groups of PTSD and control participants. Group mean values are given for each with standard deviations in parentheses. See text for details. [FS=Full Scale; V=Verbal; P=Performance.]*

A series of independent t tests carried out on the background data from the two groups indicated that the groups did not differ significantly in terms of age, NART FS IQ, WASI FS IQ, WASI V IQ, WASI P IQ, HADS anxiety, or HADS depression (all $p > 0.05$; see Appendix 2 for details). There was, however a strong trend for a group difference on the depression measure.

The participants with PTSD completed both the PDS and the Revised Impact of

Events Scale (R-IES; Horowitz, Wilner & Alvarez, 1979). The scores for each individual participant for the severity of the intrusion, avoidance and hyperarousal symptoms as measured by these instruments are shown in Table 2. Also shown is the classification of the severity of the symptoms from the PDS

	R-IES			PDS			
	I	A	H	I	A	H	Severity
NS	18	15	12	9	8	10	Moderate-severe
WS	10	12	1	7	8	4	Moderate
RN	13	4	4	3	2	3	Mild
KS	19	22	18	14	20	13	Severe
DH	10	0	1	6	4	8	Moderate
AW	12	1	14	5	1	4	Mild
AY	12	9	4	3	3	4	Mild

Table 2. *Scores on PDS and IES for symptoms of intrusion (I), avoidance (A) and hyperarousal (H) and severity measure from PDS.*

Materials

Participants were administered two standardised tests, one of face recognition memory, the faces subtest of the Recognition Memory Test (Warrington, 1984) and one of face processing, the Benton Test of Facial Recognition (Benton, Hamsher, Varney & Spreen, 1983). Recognition of facial expressions was assessed using the faces of the Ekman-Friesen series (Ekman & Friesen, 1976). This comprised a set of 60 face photographs. In the set were photographs of ten individuals and each individual was depicted portraying all six basic emotions of happiness, sadness, anger, surprise, fear and .

Procedure

Testing took place over two sessions. The tests used for this study were administered

in the context of a larger study, investigating rate of forgetting, the results of which are reported elsewhere. The tests of face recognition, face processing and recognition of facial expressions were presented during an hours delay in two separate sessions. In the first session participants were administered the faces subtest of the RMT and following this they completed the Benton Facial Recognition Test. In the second session participants identified the facial expressions of the Ekman-Friesen series. Examples of the negative expressions can be found in Appendix 5.

Participants were told that they would see a series of facial photographs, one at a time, and that each face was depicting one of six basic emotions: happiness, sadness, anger, disgust, fear and surprise. The labels of the six emotions appeared beneath each photograph. Participants were first asked to identify which of the six emotions the person in the photograph was expressing. Following this they were required to assign a rating according to how intense they thought the expressed emotion was. They were required to make their rating on a ten-point scale where one was equivalent to a neutral expression and ten was a equivalent to very intense expression. This rating was performed whether or not participants had correctly identified the expression being shown. Before the main test, six practise items were presented, one of each facial expression. During this phase participants were given feedback about the correctness of their expression identification, feedback was not given during the main test, however.

Results

Throughout this paper an alpha level of 0.05 has been adopted, unless otherwise stated. Where mean scores are presented, standard deviations are given in

parentheses. Raw data from all conditions can be found in Appendix 6.

The results were first analysed to determine whether there was a difference between groups in their face processing ability (assessed by the Benton Facial Recognition Test) or in their face recognition memory (assessed by the faces subtest of the RMT). The results from these tests are shown in Table 3.

	RMT	Benton
PTSD	43.9 (3.0)	49.6 (1.7)
Control	46.3 (4.0)	48.7 (2.1)

Table 3. *Performance of PTSD and Control groups on the RMT and Benton Face Recognition Test.*

Independent t tests carried out on these data revealed no group differences in performance on these tasks.

Next the data from the Ekman-Friesen faces were analysed. Data for the expressions of happiness and surprise were not included in the analysis. Performance of the groups on their identification of the two negative emotions (fear & anger) that have been related to amygdala function and the two negative emotions (sadness & disgust) not related to amygdala function were analysed. The performance of the two groups is shown in Table 4.

	Amygdala related		Non-amygdala related	
	Fear	Anger	Sadness	Disgust
PTSD	7.9 (2.3)	8.3 (0.8)	9.6 (1.1)	8.7 (1.3)
Control	8.6 (1.6)	8.6 (1.4)	8.9 (1.5)	7.7 (2.6)

Table 4. *Performance of PTSD and control participants on identification of amygdala and non-amygdala related negative facial expressions.*

The analysis was performed on the total score for each participant for amygdala and non-amygdala related facial expressions. The data were submitted to a two way analysis of variance (ANOVA) with a between subjects factor of group and a within subjects factor of condition (amygdala related, non-amygdala related). This revealed a significant group by condition interaction [$F(1,12)=4.79$, $p<0.05$] indicating that the groups performed differently across the two conditions. Post hoc independent t-tests were carried out to compare the performance of the PTSD group with that of the control group separately for amygdala-related and non-amygdala related expressions. In neither condition was the group difference significant, however, indicating that the difference was relative rather than absolute.

A similar analysis was carried out on the ratings of intensity of correctly identified facial expressions made by the two groups in the two conditions. Data from this analysis are presented in Table 5.

	Amygdala related		Non-amygdala related	
	Fear	Anger	Sadness	Disgust
PTSD	7.2 (1.4)	7.2 (1.2)	6.6 (1.5)	7.4 (1.6)
Control	7.2 (1.1)	7.1 (0.8)	6.2 (1.1)	6.6 (1.0)

Table 5. *Mean intensity ratings of PTSD and control participants for amygdala and non-amygdala related negative facial expressions.*

The analysis was performed on the total intensity rating for each participant for amygdala and non-amygdala related facial expressions. A two way mixed ANOVA revealed a significant main effect of condition [$F(1,12)=15.40$, $p<0.01$] and a significant group by condition interaction [$F(1,12)=4.73$, $p<0.05$] indicating that the groups performed differently across the two conditions. From the means it can be seen that whereas the intensity ratings of the PTSD group are similar to those of the control group for the amygdala related facial expressions, they tend to rate the non-amygdala related facial expressions as more intense than the control group.

Correlational analyses were carried out to investigate the relationship between identification of facial expressions and PTSD symptomatology measured by the revised IES and also between intensity ratings and current PTSD symptomatology. First correlations were carried out between amygdala-related facial expressions and symptoms of intrusion, avoidance and hyperarousal. The resulting coefficients are shown in Table 6.

	Intrusion	Avoidance	Hyperarousal
Amygdala related	- 0.4	-0.7	-0.2
Non-amygdala related	0.3	0.3	0.7

Table 6. *Pearson correlation coefficients for identification of amygdala related and non-amygdala related negative facial expressions and PTSD symptomatology (from R-IES).*

As can be seen from the table, all coefficients between amygdala-related expression identification and PTSD symptomatology were negative, indicating that a higher level of symptomatology was associated with poorer identification of these emotions. The correlation between identification of amygdala related expressions and symptoms of avoidance achieved significance at the 0.10 level.

Correlations between identification of non-amygdala related facial expressions and PTSD symptomatology can be seen in the second row of Table 6. All coefficients were positive indicating that a higher level of symptomatology was associated with enhanced identification of these facial expressions. In this analysis, the correlation between identification of non-amygdala related facial expressions and symptoms of hyperarousal achieved significance at the 0.10 level.

Correlations were also carried out between ratings of intensity assigned to amygdala related and non-amygdala related facial expressions. These were all in the positive direction indicating that higher intensity ratings were associated with higher levels of symptomatology. The highest coefficients were with symptoms of hyperarousal.

which were 0.4 and 0.5 for amygdala related and non-amygdala related expressions respectively, although these values were not significant.

Discussion

The results of this study showed that individuals with PTSD performed normally on tests of facial identification and recognition memory for faces indicating that face perception and memory for identity is intact in this group. However, they showed an abnormal pattern of performance in comparison with the control participants in their identification of facial expressions and in the ratings of intensity that they assigned to their correct identifications. Specifically, identification of amygdala-related expressions of fear and anger tended to be worse in the PTSD group than in the control group whereas their identification of non-amygdala related negative expressions of sadness and disgust tended to be superior. In relation to the ratings of intensity, individuals with PTSD and controls assigned similar ratings to amygdala-related expressions, whereas the PTSD group tended to assign higher intensity ratings than the control group to non-amygdala related facial expressions.

Correlational analyses indicated that higher levels of PTSD symptomatology were associated with poorer identification of amygdala related facial expressions. There was a significant association between identification of these expressions and symptoms of avoidance. In contrast, higher levels of symptomatology tended to be associated with superior identification of non-amygdala related facial expressions with the relationship with symptoms of hyperarousal achieving significance. Although correlations between ratings of intensity and symptoms of PTSD were lower and non-significant, the relationship between intensity ratings and symptoms

of hyperarousal was the strongest indicating that higher intensity ratings were associated with greater symptoms of hyperarousal.

The pattern of performance in the PTSD group is consistent with evidence reviewed in the introduction of the effects of amygdala damage on the identification and rating of expressions of fear and anger. Thus, identification of amygdala-related expressions and ratings of intensity were reduced in the PTSD group in comparison with those for non-amygdala related expressions. It is possible that patients with more severe PTSD symptomatology would have shown an absolute, rather than a relative impairment on this task. Findings that symptom severity was negatively associated with performance in relation to these expressions supports this contention.

The finding that identification non-amygdala related emotions was generally positively associated with PTSD symptomatology and in particular with symptoms of hyperarousal is consistent with symptoms of hypervigilance for signs of threat in the environment in this patient group. Recent findings indicate that the OFC may play a particular role in processing affectively negative arousing stimuli in tasks involving working memory (Perlstein, Elbert & Stenger, 2002). This is also consistent with findings of abnormal OFC activity in PTSD (Rauch et al., 1996; Shin et al., 1999).

By this account, increased activity in OFC associated with hyperarousal in PTSD could cause a focus of attention onto negative stimuli in general which could thereby enhance identification of negative emotions. The greater sensitivity to expressions of sadness and disgust may therefore be a general consequence of increased arousal/attention. It is possible that this attentional effect does not result in a

similar enhancement in identification of fear and anger because these are particularly dependent on normal functioning of the amygdala. Relative deficits in the identification of those expressions are consistent with other evidence suggesting that PTSD may be associated with cognitive deficits similar to those seen in people with structural damage to the amygdala (Isaac, chapter 2, this volume).

This account is speculative at present and the findings of this study need to be replicated with a greater number of participants. However, the results provide preliminary evidence of an abnormality in social cognition in this patient group.

References

- Adolphs, R. (2001). The neurobiology of social cognition. *Current Opinion in Neurobiology*, *11*, 231-239.
- Adolphs, R., Tranel, D. & Damasio, A.R. (1998). The human amygdala in social judgement. *Nature*, *393*, 470-474.
- Adolphs, R., Tranel, D., Damasio, H. & Damasio, A. (1994). Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. *Nature*, *372*, 669-672.
- Adolphs, R., Tranel, D., Hamann, S., Young, A.W., Calder, A.J., Phelps, E.A., Anderson, A., Lee, G.P. & Damasio, A.R. (1999). Recognition of facial emotion in nine individuals with bilateral amygdala damage. *Neuropsychologia*, *37*, 111-117.
- Benton, A.L., Hamsher, K., Varney, N.R. & Spreen, O. (1983). *Facial Recognition Test. Contributions to Neuropsychological Assessment*. Oxford:Oxford University Press.
- Buckley, T.C., Blanchard, E.B. & Neill, W.T. (2000). Information processing and PTSD: A review of the empirical literature. *Clinical Psychology Review*, *28*, 1041-1065.
- Cole, J. (1998). *About Face*. Massachusetts:MIT Press.

- Calder, A.J., Young, A.W., Rowland, D., Perret, D.I., Hodges, J.R. & Etcoff, N.L. (1996). Facial emotion recognition after bilateral amygdala damage: differentially severe impairment of fear. *Cognitive Neuropsychology*, *13*, 699-745.
- Damasio, A.R. (1994). *Descartes' Error*. New York : Putnam's Sons.
- Ekman, P. & Friesen, W.V. (1976). *Pictures of facial affect*. Consulting Psychologists Press, Palo Alto, California.
- Foa, E.B. (1995). *Posttraumatic Stress Diagnostic Scale*. National Computer Systems Inc. Minneapolis.
- Horowitz, M.J., Wilner, N. & Alvarez, M.A. (1979). Impact of Event Scale: A measure of subjective stress. *Psychosomatic Medicine*, *41*, 209-218.
- Liberzon, I., Taylor, S.F., Amdur, R., Jung, T.D., Chamberlain, K.R., Minoshima, S. & Koeppe, R.A. (1999). Brain activation in PTSD in response to trauma-related stimuli. *Biological Psychiatry*, *45*, 817-826.
- Nelson, H.E. (1982). *The National Adult Reading Test*. NFER-Nelson, Windsor, U.K.
- Perlstein, W.M., Elbert, T. & Stenger, A. (2002). Dissociation in human prefrontal cortex of affective influences on working memory-related activity. *Proceedings of the National Academy of Sciences (USA)*, *99*, 1736-1741.

- Rauch, S.L., van der Kolk, B.A., Fisler, R.E., Alpert, N.M., Orr, S.P., Savage, C.R., Fischman, A.J., Jenike, M.A. & Pitman, R.K. (1996). A symptom provocation study of posttraumatic stress disorder using positron emission tomography and script-driven imagery. *Archives of General Psychiatry*, *53*, 380-387.
- Rauch, S.L., Whalen, P.J., Shin, L.M., McInerney, S.C., Macklin, M.L., Lasko, N.B., Orr, S.P. & Pitman, R.K. (2000). Exaggerated amygdala response to masked facial stimuli in posttraumatic stress disorder: A functional MRI study. *Biological Psychiatry*, *47*, 769-776.
- Scott, S.K., Young, A.W., Calder, A.J., Hellawell, D.J., Aggleton, J.P. & Johnson, M. (1997). Impaired auditory recognition of fear and anger following bilateral amygdala lesions. *Nature*, *385*, 254-257.
- Semple, W.E., Goyer, P., McCormick, R., Compton-Toth, B., Morris, E., Donovan, B., Muswick, G., Nelson, D., Garnett, M.L., Sharkoff, J., Leisure, G., Miraldi, F. & Schultz, S.C. (1996). Attention and regional cerebral blood flow in posttraumatic stress disorder patients with substance abuse histories. *Psychiatry Research Neuroimaging*, *67*, 17-28.
- Shin, L.M., Kosslyn, S.M., McNally, R.J., Alpert, N.M., Thompson, W.L., Rauch, S.L., Macklin, M.L., Pitman, R.K. (1997). Visual imagery and perception in posttraumatic stress disorder. A positron emission tomographic investigation. *Archives of General Psychiatry*, *54*, 233-241.

Shin, L.M., McNally, R.J., Kosslyn, S.M., Thompson, W.L., Rauch, S.L., Alpert, N.M., Metzger, L.J., Lasko, N.B., Orr, S.P. & Pitman, R.K. (1999). Regional cerebral blood flow during script-driven imagery in childhood sexual abuse-related PTSD: A PET investigation. *American Journal of Psychiatry*, 156, 575-584.

Warrington, E.K. (1984). *Recognition Memory Test*. NFER-Nelson, Windsor, U.K.

Wechsler Abbreviated Scale of Intelligence (1999). The Psychological Corporation. Harcourt, Brace & Company: San Antonio.

Weniger, G., Irl, E., Exner, C. & R  ther, E. (1997). Defective conceptualization of emotional facial expressions during T2 signal enhancement of the right amygdala. *Neurocase*, 3, 259-266.

Zigmond, A.S. & Snaith, R.P. (1983). *The Hospital Anxiety and Depression Scale*. *Acta Psychiatrica Scandinavica*, 67, 361-370.

CHAPTER 4 : RESEARCH REVIEW

Is Neuropsychology Relevant to Clinical Psychology?

The research reported in the preceding papers has depended very much on the experimental method, the aim of which is to identify patterns of cognitive functioning in people with PTSD. In doing this work as part of a doctorate in clinical psychology I am aware that I have made at least two important assumptions. The first is that the results of this kind of research are actually useful and relevant to the field of clinical psychology. The second is that PTSD, traditionally characterised as a truly psychological phenomenon, can be understood in terms of how biology affects cognitive functioning. I will focus this review on examining further these two assumptions.

Is Neuropsychological Research Clinically Relevant?

Memory is fundamental to who we are and how we perceive ourselves. In both clinical psychology and neuropsychology it is clear that memory deficits, such as those seen in organic amnesia, and memory excesses, such as those seen in PTSD can have profound consequences on the lives of individuals. It is difficult to conceptualise a life in which we have no memory of our past, or of our ongoing day to day experiences, although it is clear from reading the neuropsychological literature, that such experience is not exceptionally rare. In my previous research work with people with profound memory problems, the devastating effect of living without a fully functioning memory was clear. Difficulties were not only centred around practical everyday functioning but also on quality of emotional life. I recall a conversation with a woman who wondered whether there was any point in doing

things that she enjoyed when she knew she would not subsequently remember them.

Memory also features prominently in many psychiatric disorders. In PTSD traumatic memories are thought to trigger a chain of events resulting in a cascade of biological changes (see van der Kolk, 1996). Biases and distortions, which are a feature of normal memory (e.g. Schacter, 1996) can become exaggerated in other emotional disorders. For example, both depressed people and anxious people tend to remember negative events from their autobiographical memories (e.g. Williams, Watts, MacLeod & Matthews, 1997). People with panic disorder are more likely to remember memories associated with threat (Williams, et al. 1997) and people with social phobia are more likely to recall negative information that is presented to them (Clark & McManus, 2002).

Findings regarding memory function in individuals with acquired brain injury and with emotional disorders have emerged from careful study, they are not always obvious to the individuals who suffer the difficulties, to the people who interact with them or to the clinicians who treat them. In his book *The Emotional Brain* LeDoux (1998) writes 'Things that are obvious are not necessarily true, and many things that are true are not at all obvious'. He is referring to scientific exploration in general and in the striving of science to uncover facts that may not be intuitively obvious. This statement also sums up part of the value of neuropsychological investigation, however. Some difficulties experienced by people are not amenable to being tapped by even the most sensitive questionnaires or to being described by even the most insightful and articulate client.

In relation to some of the work reviewed earlier in this thesis is the difficulty of differentiating between problems of attention and concentration and those of memory. Difficulties in attention and concentration will necessarily lead to memory problems. However, pure memory problems can only be defined as occurring in the absence of an attention or concentration problem. The implications for the patient of these two kinds of difficulties and the rehabilitation strategies used to address them could vary considerably so it is important that they be precisely defined. For example, in people with an attention rather than a memory deficit any rehabilitation strategy will focus on the acquisition of information. This could include strategies to more deeply process information so that its meaning is more relevant, to organise information in a structured way, or to use imagery. Once the information has been successfully acquired it could be assumed that it will be retained normally.

In the main paper of this volume, it is reported that people with PTSD not only have difficulty in acquiring information, but once acquired, have difficulty retaining it, so it is lost from memory at a faster rate than normal. This finding could have implications both for the everyday functioning of people with PTSD and also for their therapeutic treatment. In people with this kind of memory deficit it is not enough to ensure that information is acquired efficiently. Although the above strategies will also be helpful to people with such a memory impairment to acquire information, it cannot be assumed that once information has been learned, it will be retained. In people with PTSD in whom memory deficits are relatively mild, just being aware that they may forget information when it has been acquired and suggesting strategies to attenuate forgetting such as rehearsal, or keeping a diary could be helpful.

With regard to treatment, a feature of the majority of psychotherapeutic approaches at present is to assist the patient in constructing a narrative account of the traumatic event that can then be incorporated into episodic memory. Despite the presence of flashbacks, patients are often impaired in their ability to provide a narrative account of the trauma from episodic memory. It is obviously important to be aware of the possibility that even when such an account has been constructed, details of it may well be lost and it may impair the effectiveness of therapy, not to reinstate these details.

A more specific example of the likely importance in therapy of accelerated forgetting relates to the 'installation' phase of Eye Movement Desensitization and Reprocessing (EMDR) therapy (Shapiro, 1995). During this phase of therapy the client is encouraged to replace a negative cognition about their trauma with a positive cognition. The aim is to install this new positive cognition and to increase its strength. It would be important for the clinician to be aware that the installation could be affected by fast forgetting and that even when a positive cognition seems to have been successfully installed, it is possible that aspects of it will be subsequently forgotten.

The brief paper reports findings of an abnormality in the way people with PTSD identify facial expressions and in the ratings of intensity that they assign to correctly identified expressions. This is characterised mainly by an enhanced ability to identify negative expressions relating to sadness and disgust and by the tendency to assign increased intensity ratings to these expressions. These characteristics of the performance of the PTSD group were related to measures of hyperarousal. It appears.

therefore, that hyperarousal renders these individuals particularly sensitive to detecting negative expressions.

Such a tendency to interpret facial expressions differently has obvious implications for social interaction in people with PTSD. Accurately reading the facial expressions of other people appears vital for normal social interaction. The inability to do so can have devastating effects on an individual's capacity to interact in socially appropriate ways and on their subsequent mental wellbeing (see Cole, 1998). Even subtle deficits in this ability will be likely to increase social distance (see Cole, 1998). If people with PTSD are particularly sensitive to the detection of negative emotions in others, this could well affect how they respond to these people. Two of the participants with PTSD who participated in the study described a certain paranoia in which they felt that other people were looking at them and appraising them in a negative way. This in itself is likely to have a negative impact on well being in these individuals. Although it may not be possible to address the difficulty directly in therapy, it could be very important to educate clients about the possibility that this is a feature of PTSD. If the difficulty can be made explicit it could be amenable to cognitive therapy, thereby being treated in a similar way to the way feelings of negative appraisal in other psychiatric disorders.

Despite their utility in defining precise cognitive deficits, an obvious criticism of the scientific/empirical frameworks is that they leave little room for the vital human dimensions affected by trauma. This kind of work is perhaps exemplified by the psychosocial approach, which aims to explain the impact of trauma on both psychological and social systems. It would seem, however, that these approaches are complementary rather than in conflict. Both are obviously important and just as

the discussion above focuses on the necessity of scientific/empirical work to identify underlying cognitive difficulties, the human dimensions can only be understood by therapeutic work which explores the whole person within the system in which they live. Recent papers indicate some success in attempting to bridge the divide between neuroscientific and psychosocial accounts of the disorder (e.g. Brewin, 2001) and it would seem that partnership which focuses on the strengths of the different approaches, rather than competition is likely to lead to the greatest success.

Although the aim of neuropsychological research largely ignores the role of the individual, in conducting my own research I have become aware of other benefits over and above those included as specific aims of the study. Some of these benefits I was aware of in carrying out the research I did before clinical training. These are basic aspects such as social contact for people who are otherwise socially isolated, the opportunity to talk with someone who has some grasp of the difficulties that they encounter and perhaps most important to them, the feeling that they are doing something positive to help people in the future who may have similar problems to their own. In addition to these benefits, however, I have also come to realise the importance of making the research a collaborative venture. To regard participants as experts who can inform the research over and above providing data via testing. The way participants described their experiences gave me greater insight into what it's like to live with PTSD and from there to generate ideas for future research. Thus, basic principles in clinical psychology, such as collaboration and transparency can also contribute to the research process.

The preceding discussion links very much with the issue of whether PTSD is best

conceptualised as relating to the mind or to the body.

Is PTSD best understood as a psychological or biological phenomenon?

PTSD has traditionally been thought of as the archetypal psychological disorder, a severe psychological reaction to a severe environmental stressor (e.g. Andreason, 1995). However, since the advent of modern neuroimaging techniques, psychiatric disorders previously described as being 'functional' or lacking in identified organic structural correlates are being tied down to abnormalities in brain structure, function and histochemistry. In addition, the cognitive deficits associated with these disorders are coming under increasing scrutiny. This raises the question as to whether it is still appropriate to regard PTSD as a psychological disorder (a disorder of the mind) or whether it is best understood as a biological disorder (a disease of the body). Indeed, this question could be asked regarding any psychiatric diagnosis.

First, it seems necessary to operationally define how the terms 'mind' and 'body' will be used in this discussion. The mind/body debate is one that dates back to the time of the philosophers of ancient Greece. However, dualism as the term is used today is mainly associated with Descartes (see Slezak, 2000). Descartes believed that the mind which included the higher functions of reasoning, consciousness, moral feelings and emotions, was separate from the body (and therefore the brain) and as such studying the brain would tell us little about the functioning of the mind. Dualists can be contrasted with materialists who believe that the mind and the brain are essentially the same thing and that by understanding how the brain works we can also understand the mind. Today the debate is referred to both as the mind body and the mind/brain problem but as the brain is generally considered part of the body, the

argument is the same.

The mind-brain/mind-body question is therefore essentially a question of reduction and is this: Can mental states and processes be reduced to brain states and processes? At the apex of the argument is the fundamental question of consciousness: Can consciousness be attributed to brain states and processes? It is generally accepted that at the present time it is not possible to explain consciousness in these terms (see Churchland, 1986). So the real question is: Will it ever be possible to do so? The alternative is that the mind and therefore consciousness is an entity in its own right, or somehow an 'emergent' process that is more than the sum of its parts and so can never be tied down to the brain (See Churchland, 1986 for a discussion.) This is an important question for psychology, because the increasing emphasis on structural and functional neuroimaging techniques and links being formed across disciplines such as neuroscience, psychiatry, cognitive psychology and neuropsychology, assumes that the answer to it is 'yes'.

The emerging ascendancy of neurobiology and neuroimaging techniques in identifying the underlying biological markers of psychiatric disorders has also been reflected in the dropping from DSM IV of the dichotomy between 'functional' and 'organic'. This has been seen as a very positive move by some. For example, van der Kolk, (1996) contrasts society's response to psychological disorders with that of its response to biological disorders. He sees the former as being associated with issues of blame and people who develop psychological disorders seen as having a failure of will or moral fibre. In contrast, biological disorders are seen as being beyond individual control as people cannot be held responsible for their biological makeup. Van der Kolk, goes further and claims that the Cartesian split between mind and

body is one of the issues that has negatively influenced the capacity of mainstream psychiatry to grasp the significance of trauma in people's lives.

This then, can be seen as one advantage of diagnosis and of thereby emphasising the biological (brain/body) over the psychological (mind). Another could be that diagnosis facilitates ease of communication about disorders. In addition, it can often be a relief for a client to receive a label for the devastating symptoms they are experiencing. One participant in my own study described such a reaction when at her first appointment with a clinical psychologist he showed her a list of the DSM IV criteria for PTSD and she realised for herself that these were exactly the symptoms she was experiencing. Prior to this she had feared she was going mad.

There are also many disadvantages to psychiatric diagnosis. First, diagnosis is arbitrary. A person could in theory suffer all the symptoms of PTSD but be unable to receive a diagnosis because the trauma they suffered does not fit the description offered in DSM IV. Similarly a person could receive a diagnosis of PTSD based on one edition of DSM, but not on a later edition. Second, diagnosis is necessarily a reductionist activity and as such cannot do justice to the complexity of human life (e.g. Smith, 2000). Andreason (1995) decries the high demand to 'validate' psychiatric diagnoses by identifying biological markers or neurobiological substrates, because of the danger of losing the subtlety and complexity of the disorder. Third, to understand the effect of overwhelming experiences on the individual we also need to understand the interaction between that individual and the social systems in which they operate. Thus, individual differences in the way people perceive trauma, how society as a whole reacts to victims of trauma and the role played by aspects such as

social support are also important.

It seems unsatisfactory however to equate diagnosis, and the problems with which it is associated, with the materialist position and arguments against diagnosis with the dualist position. Such dichotomising could lead clinical psychologists into dismissing the findings relating to biological bases of disorders, and findings relating to cognitive functioning associated with them, as irrelevant. At present, the materialist approach cannot do justice to the complexity of human feelings and emotions and so diagnosing and identifying the underlying biology of disorders cannot hope to be adequate for individual treatment. However, this does not mean that feelings, emotions, cognitions and all the other components of mental life that make us individuals are not dependent on brain functioning. There is overwhelming evidence that this is indeed the case (see Damasio, 1994).

There is an implication related to this argument that by accepting that feelings and emotions are dependent on the brain/body, rather than on the mind, they are somehow devalued or made less real and individual (e.g. Damasio, 1994). Yet thoughts, emotions and feelings are the same regardless of their underlying substrate. In this sense, then, perhaps the mind/body problem is superfluous to clinical psychologists, working with individuals on a therapeutic basis. However, as discussed earlier, some aspects of psychological difficulties relating to cognitive function are not available to be described by the individual client. Ignoring or dismissing these aspects could risk not fully understanding the difficulties that clients are experiencing.

References

- Andreason, N.C. (1995). Posttraumatic stress disorder: Psychology, biology and the manichaeon warfare between false dichotomies. *American Journal of Psychiatry*, 152, 963-965.
- Brewin, C.R. (2001). A cognitive neuroscience account of posttraumatic stress disorder and its treatment. *Behaviour Research and Therapy*, 39, 373-393.
- Churchland, P.S. (1986). *Neurophilosophy*. Massachusetts:MIT Press.
- Clark, D.M& McManus, F. (2002). Information processing in social phobia. *Biological-Psychiatry*, 51, 92-100.
- Cole, J. (1998). *About Face*. Massachusetts:MIT Press.
- Johnson, G. (1992). *In the Palaces of Memory. How We Build Worlds Inside Our Heads*. London:HarperCollins.
- Damasio, A.R. (1994). *Descartes' Error*. New York : Putnam's Sons.
- LeDoux, J. (1998). *The Emotional Brain*. London: Phoenix.
- Schacter, D.L. (1996). *Searching for Memory: the Brain, the Mind and the Past*. New York: Basic Books.
- Shapiro, F. (1995). *Eye Movement Desensitization and Reprocessing: Basic*

Principles, Protocols and Procedures. New York: Guilford Press.

Slezak, P. (2000). The mind-brain problem. In E. Gordon (Ed.) *Integrative Neuroscience.* The Netherlands: Harwood.

Smith, G.C. (2000). Models of the brain in Psychiatry. In E. Gordon (Ed.) *Integrative Neuroscience.* The Netherlands: Harwood.

van der Kolk, B.A., McFarlane, A.C. & Weisaeth, L. (1996). *Traumatic Stress. The effects of overwhelming experience on mind, body and society.* New York: Guilford Press.

Williams, J.M.G., Watts, F.N., McLeod, C. & Mathews, A. (1997). *Cognitive Psychology and Emotional Disorders.* Second Edition. Chichester: Wiley.

Appendix 1

Herefordshire Ethics

Letter confirming ethics committee approval	127
Participant Information Sheet	128
Consent form	131

Worcestershire Ethics

Letter confirming ethics committee approval	132
Participant Information Sheet	134
Consent form	138

AS/JPD/C7b(i)

Victoria House
Eign Street
Hereford
HR4 0AN

30th April, 2001.

Tel: 01432 262000
Fax: 01432 341958
Direct Line: 01432 262000

Dr. C. Isaac,
The Old Stables,
Pensax,
Abberley,
Worcs.,
WR6 6AH.

Dear Dr. Isaac,

**RATE OF FORGETTING OF EMOTIONAL & NON-EMOTIONAL MATERIAL IN
POST-TRAUMATIC STRESS DISORDER – LREC.455:**

Please accept my apologies for the delay in writing formally to give you the Herefordshire District Ethics Committee's decision in respect of the above study.

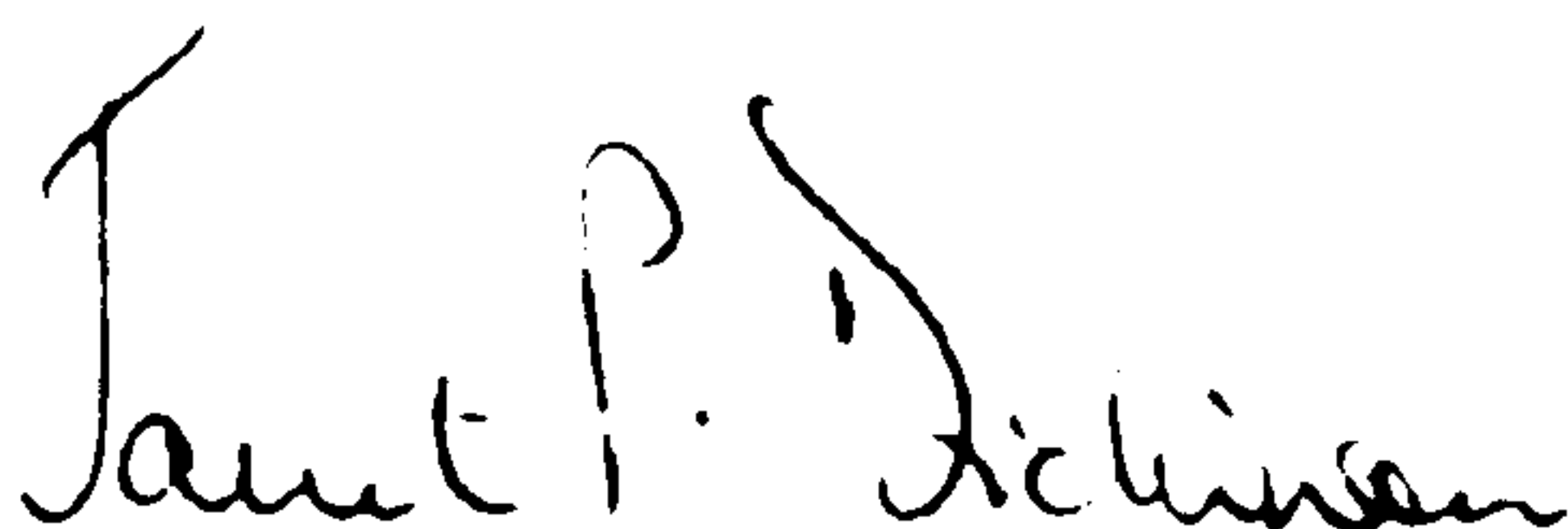
May I first of all thank you for making the time available to attend the meeting and answer members' questions.

I confirm that, following discussion, the Committee gave its approval to the study with the following suggestions:

- The reference to disorder should be removed throughout, as opening the study to those suffering from post-traumatic stress would enable you to increase the sample size
- The reference in the Information Sheet to "slow" as a non-emotional word should be changed, as it could be construed as insulting by some participants.

The Committee would welcome a report on your study's outcome in due course.

Yours sincerely,



Mrs. J. P. Dickinson,
Administrative Services Manager.

Programme Director
Doctorate Course in Clinical Psychology
Dr Delia Cushway
BA (Hons) MSc PhD AFBPS CPsychol
School of Health and Social Sciences
Coventry University
Priory Street Coventry CV1 5FB
Telephone 01203 838328
Fax 01203 838784



C O V E N T R Y
U N I V E R S I T Y

May 2001 version 2

TAKING PART IN RESEARCH

INFORMATION FOR PARTICIPANTS ABOUT THE STUDY

Study Title:

Rate of forgetting of emotional and non-emotional words in people with Post-Traumatic Stress (PTS)

We are inviting you to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please consider the following information carefully and take time to discuss it with other people if you wish. Please ask us if there is anything that is not clear or if you would like further information. Do take time to decide whether or not you wish to take part in this study, you will not be asked for a decision for at least 24 hours.

What is the purpose of the study?

The purpose of this study is to investigate different kinds of memory in people suffering from PTS. One of the main symptoms of PTS is the experience of vivid memories, which are very difficult to forget. Previous research has found that these traumatic memories are different to everyday memories and are dealt with by a different part of the brain. This part of the brain, called the amygdala, has been found to be more active in people with PTS than in people without PTS. This greater activity may be linked with the very vivid traumatic memories that these people experience and may also affect the way in which people with PTS remember other events in their everyday lives.

People with PTS sometimes complain that their memory and concentration are generally not as good as they were previously. There is evidence that long-term stress can also have effects on memory and that this is what causes difficulties with memory and concentration in people with PTS.

We are interested in finding out more about everyday memory in people with PTS. To do this we will give people different lists of words to remember. One list will contain emotional words (e.g. disgusting) and the other will contain non-emotional words (e.g. move). We will then see how quickly these two kinds of words are lost from memory over a delay of one hour. We will compare the forgetting found in people with PTS to that of people who do not have PTS on these tests to see if there is a difference.

The amygdala has also been found to be very important in identifying certain facial expressions from photographs. We are also interested, therefore, in finding out how well people with PTS identify expressions of happiness, sadness, fear, anger, disgust and surprise from photographs of faces.

Why have I been chosen?

This study will include participants from Warwickshire, Herefordshire and Worcestershire. You have been asked to participate in the study because the clinician you are seeing or have seen in the past has contacted you. People are being asked to take part in the study who are suffering from symptoms of PTS and are between the ages of 18 and 70.

Do I have to take part?

It is entirely up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet and you will be asked to sign a consent form. If you decide to take part you are still free to withdraw from the study at any time and without giving a reason. Whether or not you take part will not affect the standard of care you receive.

Who is organising the study?

The study is being conducted by Claire Isaac as part of a training course for the Doctorate in Clinical Psychology. The course is run by the Universities of Coventry and Warwick and this project is being supervised by two members of staff from the Department of Clinical Psychology at Coventry University and one member of staff from the Psychology Department at the University of Warwick. Four clinical psychologists are also involved in the study who work within the National Health Service in the Warwickshire, Herefordshire and Worcestershire regions.

What will happen to me if I take part?

If you decide to take part in the study we will first ask you to sign a consent form. After this I would need to see you on three separate occasions. Each of these appointments would last for about an hour and would be arranged at a time and a place convenient for you. At each appointment I will see you individually, although if you would prefer it, you could have a friend or a member of your family with you. During each of the three appointments you would be shown some words on a computer screen that you will be asked to remember. Some of the words would be 'emotional' words (for example, "disgust") and some would be 'non-emotional' words (for example, "slow"). Your memory for these words would then be tested.

During each appointment you will also be asked to complete some other tests to give us some more information about the symptoms that you experience. You will be asked to complete two short questionnaires asking about some of the symptoms that you experience. You will also be shown some photographs of faces that you will be asked to remember, and you will be asked to look at other photographs of faces and identify some facial expressions from them. Finally, there will be a short task involving reading. All the tests are quite short and most people find them enjoyable. If you wish you can be given feedback about how you did in the tests. When the project is completed you could be informed about the results.

What are the possible disadvantages and risks of taking part?

There are no obvious disadvantages or risks of taking part in this study. However, we cannot guarantee that you will not find some of the emotional words upsetting. We have tried to minimise this risk by including only general words in common everyday usage that are less likely to remind you of the traumatic incident you experienced.

What are the possible benefits of taking part?

As far as your treatment is concerned there are no obvious benefits to you from taking part in this study. The aim of the study is to find out more about memory in people with PTS, and the information that we find should help treat people with PTS in the future.

Will my taking part in this study be kept confidential?

All information, which is collected, about you during the course of this research will be kept strictly confidential. Any information you give us will not be linked with your name and address so that you cannot be recognised from it.

GP notification

If you wish, we can inform your GP that you are taking part in this study.

Medical Records

If you decide to participate in this study, we will **not** need to look at your medical records.

What will happen to the results of the research study?

The results of this study will be written up and submitted in May 2002 to the Universities of Coventry and Warwick to fulfil part of the requirement for the Doctorate in Clinical Psychology qualification. Following this, the results will also be submitted to academic journals for possible publication. You will not be identified in any of the research reports that may be submitted as a result of this study.

Who has reviewed the study?

This study has been reviewed by the Herefordshire, Worcestershire and Warwickshire Research Ethics committees.

Contact for further information

For further information you can contact Claire Isaac at the Department of Clinical Psychology, School of Health and Social Sciences, Coventry University, Priory Street, Coventry, CV1 5FB, telephone number 01299 832545. Alternatively you could contact the clinician who referred you.

Programme Director
Doctorate Course in Clinical Psychology
 Dr Delia Cushway
 BA (Hons) MSc PhD AFBPS CPsychol
School of Health and Social Sciences
 Coventry University
 Priory Street Coventry CV1 5FB
 Telephone 01203 838328
 Fax 01203 838784



CONSENT FORM

RATE OF FORGETTING OF EMOTIONAL AND NON-EMOTIONAL WORDS IN POST TRAUMATIC STRESS

RESEARCHER: Claire Isaac

Please initial box

- 1 I confirm that I have read and understand the information sheet dated.....(version.....) for the above study and have had the opportunity to ask questions.
- 2 I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected.
- 3 I agree to take part in the above study

 Name of patient

 Date

 Signature

 Name of person taking consent
 (if different from researcher)

 Date

 Signature

 Researcher

 Date

 Signature

**WORCESTERSHIRE
LOCAL RESEARCH ETHICS COMMITTEE**

Isaac Maddox House
Shrub Hill Road
Worcester
WR4 9BJ

Tel: 01905 760000
Fax: 01905 26159
DX 709431 Worcester 7

Chairman:
Mrs Carol Thompson B.Sc.

Ms Claire Isaac
The Old Stables
Pensax
Abberley
Worcestershire WR6 6AH

Our ref: KG/
Your ref:
19 June 2001

Direct Line: (01905) 760091
Fax Line: (01905) 617051

E-mail Number:
Kath.Garrad@wha.worcester-ha.wmids.nhs.uk

Dear Ms Isaac

Re: LREC: 01/29 (please use in all correspondence)

Rate of forgetting of emotional and non-emotional material in post traumatic stress disorder

(Local Researchers: Ms Claire Isaac, Dr Delia Cushway, Professor Greg Jones)

Papers reviewed:

- **LREC application form, received 3rd April 2001**
- **Protocol**
- **Information Sheet, dated May 2001, version 2**
- **Consent Form, dated May 2001, version 2**
- **List of words to be used in the forgetting rate study**
- **Impact of event scale (IES)**
- **HAD scale**
- **Letter to Dr**
- **Letter to Whom it may Concern, dated 2nd March 2001**
- **CV for Ms Claire Isaac**

❖ THIS APPLICATION HAS BEEN GIVEN A UNIQUE REFERENCE NUMBER. PLEASE QUOTE THIS ON ALL CORRESPONDENCE.

Chairman: Mrs Jacqueline M Cravos
Chief Executive: Mrs Pat Archer-Jones

Following the meeting of the Local Research Ethics Committee (LREC) on 26th April 2001, we write to confirm that, with the additional information which you have now kindly provided with your letters of 15th June and 10th May 2001, the Committee have no objection to the above research proceeding.

Conditions of approval

- Satisfactory Indemnity arrangements being in place.
- You will no doubt realise that, whilst The Committee has no objection to the study on ethical grounds, it is still necessary for you to obtain approval from the relevant Clinical Directors and/or bodies in which the work will be carried out.
- In keeping with the Committee's protocol and in line with the Good Clinical Practice guidelines, would you please inform us of the results of the study when it is completed. If this is not within twelve months, please inform us of progress on an annual basis.
- Active approval is required until the study has been completed.
- The Committee would wish to be kept informed of serious adverse events, amendments and any other modifications to patient information sheets and patient consent forms.

ICH GCP Compliance

Worcestershire LREC is fully compliant with the International Committee on Harmonisation/Good Clinical Practice (ICH) Guidelines for the Conduct of Trials Involving the Participation of Human Subjects as they relate to the responsibilities, composition, function, operations and records of an Independent Ethics Committee/Independent Review Board. To this end it undertakes to adhere as far as is consistent with its Terms of Reference, to the relevant clauses of the ICH Harmonised Tripartite Guideline for Good Clinical Practice, adopted by the Commission of the European Union on 17th January 1997.

LREC Membership

Please find attached, for information, a list of members of the LREC.

If the project continues after THREE YEARS from the date of this letter Worcestershire Local Research Ethics Committee will wish to re-examine it.

Would you please communicate this approval immediately to all members of the investigating team and, where appropriate, the sponsoring commercial company.

Yours sincerely



Kath Garrad

Administrator, Worcestershire Local Research Ethics Committee

Programme Director
Doctorate Course in Clinical Psychology
Dr Delia Cushway
BA (Hons) MSc PhD AFBPS CPsychol
School of Health and Social Sciences
Coventry University
Priory Street Coventry CV1 5FB
Telephone 01203 838328
Fax 01203 838784



COVENTRY
UNIVERSITY

May 2001 version 2

TAKING PART IN RESEARCH

INFORMATION FOR PARTICIPANTS ABOUT THE STUDY

Study Title:

Rate of forgetting of emotional and non-emotional material in people with Post-Traumatic Stress Disorder (PTSD)

We are inviting you to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please consider the following information carefully and take time to discuss it with other people if you wish. Please ask us if there is anything that is not clear or if you would like further information. Do take time to decide whether or not you wish to take part in this study, you will not be asked for a decision for at least 24 hours.

What is the purpose of the study?

The purpose of this study is to investigate different kinds of memory in people suffering from PTSD. One of the main symptoms of PTSD is the experience of vivid memories, which are very difficult to forget. Previous research into memory has found that a part of the brain called the amygdala is very important in remembering emotional events. Some studies have used specialised scanners that detect activity in the brain to look at this structure. They have found that in people with PTSD, the amygdala may show an increased amount of activity in comparison with the amygdalas of people without PTSD. This increased activity may be linked with the very vivid traumatic memories that these people experience. This overactivity may also affect the way in which people with PTSD remember other emotional events in their everyday lives.

As well as these symptoms, however, people with PTSD sometimes complain that their memory and concentration are generally not as good as they were previously. There is evidence that long-term stress can have other effects on memory generally and that this is what causes difficulties with memory and concentration in people with PTSD.

We are interested in finding out more about emotional memories and more general memories in people with PTSD. To do this we will give people different lists of words to remember. One list will contain emotional words (e.g. disgusting) and the other will contain non-emotional words (e.g. slow). We will then see how quickly these two kinds of words are lost from memory over a delay of one hour. We will compare the forgetting found in people with PTSD to that of people who do not have PTSD on these tests to see if there is a difference.

The amygdala has also been found to be very important in identifying certain facial expressions from photographs. We are also interested, therefore, in finding out how well people with PTSD identify expressions of happiness, sadness, fear, anger, disgust and surprise from photographs of faces.

Why have I been chosen?

This study will include participants from Warwickshire, Herefordshire and Worcestershire. You have been asked to participate in the study because your consultant gave us permission to approach you and with your consent, passed on your name and contact details to us. People are being asked to take part in the study who are suffering from symptoms of PTSD and are between the ages of 18 and 70. We are hoping to see a minimum of 15 people and a maximum of 30 people during the course of the project.

Do I have to take part?

No, it is entirely up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet and you will be asked to sign a consent form. If you decide to take part you are still free to withdraw from the study at any time and without giving a reason. Whether or not you take part will not affect the standard of care you receive.

Who is organising the study?

The study is being conducted by Claire Isaac as part of a training course for the Doctorate in Clinical Psychology. The course is run by the Universities of Coventry and Warwick and this project is being supervised by two members of staff from the Department of Clinical Psychology at Coventry University and one member of staff from the Psychology Department at the University of Warwick. Four clinical psychologists are also involved in the study who work within the National Health Service in the Warwickshire, Herefordshire and Worcestershire regions.

What will happen to me if I take part?

If you decide to take part in the study we will first ask you to sign a consent form. After this I would need to see you on three separate occasions. Each of these appointments would last for about one and a half hours and would be arranged at a time and a place convenient for you. At each appointment I will see you individually, although if you would prefer it, you could have a friend or a member of your family with you.

During each of the three appointments you would be shown some words on a computer screen that you will be asked to remember. Some of the words would be 'emotional' words (for example, "disgust") and some would be 'non-emotional' words (for example, "slow"). Your memory for these words would then be tested. During each appointment you will also be asked to complete some other tests to give us some more information about the symptoms that you experience. These will be two short written questionnaires asking about some of the symptoms that you experience. The other tests will require a verbal response; you will be shown some photographs of faces that you will be asked to remember, and you will be asked to look at other photographs of faces and identify some facial expressions from them. Finally, there will be a short task involving reading. All the tests are quite short and most people find them enjoyable.

If you wish you can be given feedback about how you did in the tests. When the project is completed you could, if you wish, be informed about the results.

What do I have to do?

I would need to arrange an appointment with you on three separate occasions. You will **not** be asked to take any medication and you will **not** be asked to stop taking any medication that you are currently taking.

What are the possible disadvantages and risks of taking part?

There are no obvious disadvantages or risks of taking part in this study. However, we cannot guarantee that you will not find some of the emotional words upsetting. We have tried to minimise this risk by including only general words in common everyday usage that are less likely to remind you of the traumatic incident you experienced.

What are the possible benefits of taking part?

As far as your treatment is concerned there are no benefits to you from taking part in this study. The aim of the study is to find out more about memory in people with PTSD, and the information that we find should help treat people with PTSD in the future.

What if something goes wrong?

If you are harmed by taking part in this research project, there are no special compensation arrangements. If you are harmed due to someone's negligence, then you may have grounds for a legal action but you may have to pay for it. Regardless of this, if you wish to complain about any aspect of the way you have been approached or treated during the course of this study, the normal National Health Service complaints mechanisms may be available to you.

Will my taking part in this study be kept confidential?

All information, which is collected, about you during the course of this research will be kept strictly confidential. Any information you give us will not be linked with your name and address so that you cannot be recognised from it. You will not be identified in any publications arising from this study.

GP notification

With your agreement, we will inform your GP and any other clinician that you are currently seeing, that you are taking part in this study.

Medical Records

If you decide to participate in this study, we will **not** need to look at your medical records.

What will happen to the results of the research study?

The results of this study will be written up and submitted in May 2002 to the Universities of Coventry and Warwick to fulfil part of the requirement for the Doctorate in Clinical Psychology qualification. Following this, the results will also be submitted to academic journals for possible publication. You will not be identified in any of the research reports that may be submitted as a result of this study.

Who has reviewed the study?

This study has been reviewed by the Ethics Committees of Worcestershire, Warwickshire and Herefordshire.

Contact for further information

The researcher carrying out this study is Claire Isaac who is a Trainee Clinical Psychologist. For further information you can contact Claire at the Department of Clinical Psychology, School of Health and Social Sciences, Coventry University, Priory Street, Coventry, CV1 5FB, telephone number 024 76838328. Alternatively you could contact the clinician who referred you.

Independent Advice

If you would like independent advice about taking part in the study, you can contact the Community Health Council at:

- Burgage Lodge, 184 Franche Road, Kidderminster, Worcs., DY11 5DA. Tel: 01562 69243; or
- Red House, Church Green West, Redditch, B97 4BG – Tel: 01527 61375; or
- Severn House, 10 The Moors, Worcester, WR1 3EE – Tel: 01905 2271

Programme Director
Doctorate Course in Clinical Psychology
 Dr Delia Cushway
 BA (Hons) MSc PhD AFBPS CPsychol
School of Health and Social Sciences
 Coventry University
 Priory Street Coventry CV1 5FB
 Telephone 01203 838328
 Fax 01203 838784



COVENTRY
UNIVERSITY

May 2001 version 2

CONSENT FORM

RATE OF FORGETTING OF EMOTIONAL AND NON-EMOTIONAL STIMULI IN POST TRAUMATIC STRESS DISORDER

RESEARCHER: **Claire Isaac**

Please initial box

- | | | |
|---|--|--------------------------|
| 1 | I confirm that I have read and understand the information sheet dated.....(version.....) for the above study and have had the opportunity to ask questions. | <input type="checkbox"/> |
| 2 | I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. | <input type="checkbox"/> |
| 3 | I agree to take part in the above study. | <input type="checkbox"/> |
| 4 | I understand that any information obtained about me during the course of this research will be kept strictly confidential. | <input type="checkbox"/> |

Name of patient

Date

Signature

Name of person taking consent
(if different from researcher)

Date

Signature

Researcher

Date

Signature

Independent Advice

If you would like independent advice about taking part in the study, you can contact the Community Health Council at:

- Burgage Lodge, 184 Franche Road, Kidderminster, Wores., DY11 5DA. Tel: 01562 69243, or
- Red House, Church Green West, Redditch, B97 4BG – Tel: 01527 61375; or
- Severn House, 10 The Moors, Worcester, WR1 3EE – Tel: 01905 22715

If you decide not to participate in this study, the treatment that you receive will not be affected

1 copy for participant, 1 for researcher, 1 for file

Appendix 2

Background details of PTSD and control participants.

ID	Group	Age	NART-R FS IQ	WASI IQ			HADS	
				FS	V	P	ANX	DEP
NS	PTSD	34	95	91	103	79	11	12
WS	PTSD	34	100	100	107	105	14	4
RN	PTSD	54	91	105	92	119	6	6
KS	PTSD	54	120	114	113	111	20	16
DH	PTSD	29	115	107	96	118	6	2
AW	PTSD	42	117	113	109	112	7	2
AY	PTSD	29	105	111	109	109	6	4
Group Means & SD		39.4 (10.9)	106.1 (11.4)	105.9 (66.8)	104.1 (7.6)	107.6 (13.5)	10.0 (5.4)	6.6 (5.4)
NN	Control	29	121	114	119	106	7	1
NR	Control	26	107	118	123	109	6	4
NP	Control	50	123	120	109	128	0	2
WS	Control	22	88	95	99	90	9	1
EN	Control	56	104	113	108	115	8	1
EG	Control	39	109	103	103	104	11	4
LS	Control	43	112	120	128	109	7	2
Group Means & SD		37.9 (12.7)	109.1 (11.7)	111.9 (9.5)	112.7 (10.8)	108.7 (11.5)	6.9 (3.4)	2.1 (1.3)

Key: NART-R=National Adult Reading Test Revised; WASI=Wechsler Abbreviated Scale of Intelligence, FS=Full Scale IQ, V=Verbal IQ, P=Performance IQ; HADS=Hospital Anxiety and Depression Scale, ANX=Anxiety, DEP=Depression.

A series of independent t tests carried out on the background data from the two groups indicated that the groups did not differ significantly in terms of age ($t(12)=-0.25$, $p=0.81$); NART FS IQ ($t(12)=0.49$, $p=0.64$); WASI FS IQ ($t(12)=1.27$, $p=0.23$); WASI V IQ ($t(12)=1.72$, $p=0.11$); WASI P IQ ($t(12)=0.17$, $p=0.87$); HADS anxiety ($t(12)=-1.30$, $p=0.22$) or HADS depression ($t(12)=-2.11$, $p=0.06$), although it is clear that there is a strong trend for a group difference on the depression measure.

Appendix 3

Experimental Stimuli Described in Chapter 2: Main Paper.

EMOTIONAL WORD LISTS					
	LIST 1	LIST 2	LIST 3	LIST 4	Category
1	enemy	bitter	killing	assault	Anger
2	ranting	stern	hostile	cruel	
3	awkward	angry	evil	rage	
4	violent	hate	mean	critical	
5	spite	livid	attack	quarrel	
6	vicious	mad	revenge	fury	
7	outraged	damage	annoy	curse	
8	inflict	cross	provoke	retribution	
9	urgent	tense	uneasy	anxiety	Anxiety
10	fright	harm	panic	scared	
11	suffer	pain	victim	terror	
12	risk	afraid	nervous	injury	
13	intense	horrific	torture	warning	
14	shocked	conflict	fearful	brooding	
15	danger	shy	ominous	threat	
16	tremble	crisis	trap	troubled	
NON-EMOTIONAL WORD LISTS					
	LIST 1	LIST 2	LIST 3	LIST 4	Category
1	motion	rapid	scramble	leap	Active
2	jog	lithe	fast	keen	
3	scurry	stamina	busy	quick	
4	nimble	energetic	hurry	active	
5	relax	haste	athletic	agile	
6	brisk	rush	sport	vigour	
7	dash	venture	swift	bustle	
8	alert	prompt	scamper	occupied	
9	voluminous	heavy	immense	compact	Gigantic
10	big	wide	broad	expanse	
11	profuse	mass	enormous	abundant	
12	bulky	copious	sizable	giant	
13	vast	hefty	spacious	numerous	
14	massive	colossal	tall	full	
15	sturdy	solid	substantial	huge	
16	ample	gigantic	extensive	grand	

Appendix 4

Data from main paper

Raw data from free recall condition

			EMOTIONAL			NON-EMOTIONAL		
Group	No. pres.		mean rating	20 sec	1 hour	mean rating	20 sec	1 hour
NN	Control	1*4	3.0	10	8	1.9	8	7
NR	Control	1*4	2.8	9	6	1.6	8	6
NP	Control	1*2	2.3	5	4	1.3	7	3
WS	Control	1*4	2.8	7	6	2.6	6	3
EN	Control	1*4	2.8	10	8	1.4	10	11
EG	Control	1*4	2.8	10	10	1.3	10	10
LS	Control	1*3	3.2	9	8	2.9	8	5
Mean & SD			2.8 (0.3)	8.6 (1.9)	7.1 (2.0)	1.9 (0.1)	8.1 (1.5)	6.4 (3.2)
NS	PTSD	2*3	2.6	9	6	1.5	6	5
WS	PTSD	1*4	2.4	8	5	1.1	6	3
RN	PTSD	2*3	3.1	7	2	2.8	9	5
KS	PTSD	1*4	3.0	5	3	1.8	5	2
DH	PTSD	1*4	2.4	11	7	1.8	8	6
AW	PTSD	1*4	3.2	12	7	1.8	8	8
AY	PTSD	1*4	2.4	8	5	1.5	9	3
Mean & SD			2.7 (0.4)	8.7 (2.4)	5.0 (1.9)	1.8 (0.5)	7.4 (1.8)	4.6 (2.1)

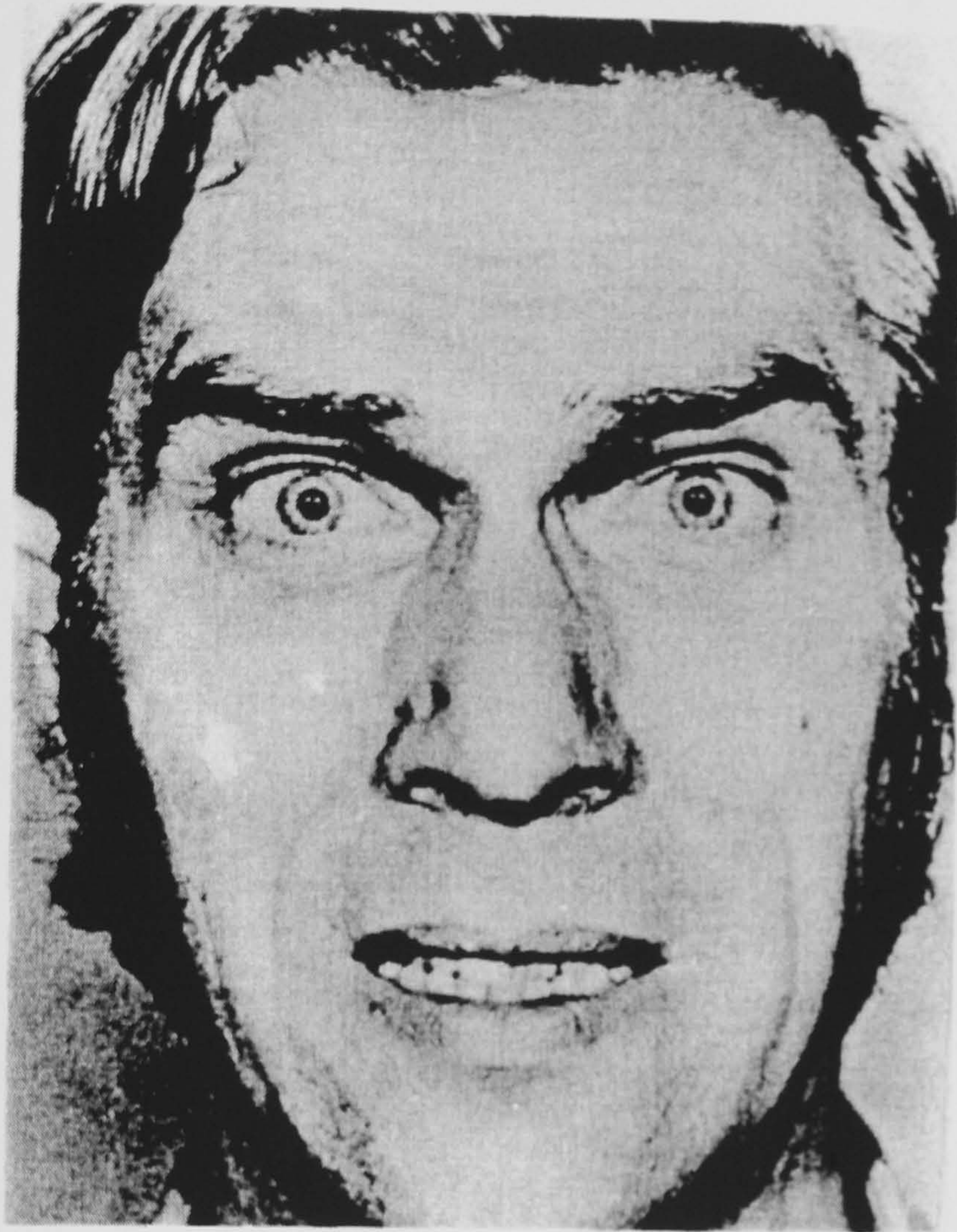
Raw data from recognition condition

		d'				c			
		Emotional		Non-emotional		Emotional		Non-emotional	
		20 sec	1 hour	20 sec	1 hour	20 sec	1 hour	20 sec	1 hour
NN	Control	3.4	1.9	2.8	2.5	-0.17	-0.93	-0.49	-0.60
NR	Control	3.4	2.7	3.1	3.0	0.16	-0.19	-0.04	0.36
NP	Control	3.4	2.7	1.8	2.0	-0.17	0.19	-0.24	0.520
WS	Control	3.0	1.6	2.4	2.4	-0.36	-0.33	0.32	-0.36
EN	Control	3.4	2.8	1.6	3.1	-0.17	-0.49	-0.11	-0.04
EG	Control	3.4	3.7	3.0	3.7	-0.17	0.00	-0.36	0.00
LS	Control	1.8	3.1	2.0	2.7	-0.24	-0.04	-0.50	-0.19
Mean & SD		3.1 (0.6)	2.6 (0.7)	2.4 (0.6)	2.9 (0.6)	-0.16 (0.2)	-0.25 (0.4)	-0.12 (0.4)	-0.22 (0.5)
NS	PTSD	3.7	2.4	3.1	1.8	0.00	-0.33	-0.02	0.00
WS	PTSD	3.7	3.7	2.7	2.4	0.00	0.00	-0.19	-0.33
RN	PTSD	3.0	1.4	2.7	2.0	0.36	0.20	0.19	0.52
KS	PTSD	3.1	1.7	2.0	2.2	0.00	-0.69	-0.86	-0.77
DH	PTSD	2.7	2.7	1.2	1.2	-0.19	0.19	-0.09	0.28
AW	PTSD	3.7	3.4	2.8	3.1	0.00	0.16	-0.49	-0.04
AY	PTSD	1.7	1.5	2.2	2.0	-1.01	-1.09	-0.43	-0.13
Mean & SD		3.1 (0.7)	2.4 (0.9)	2.4 (0.6)	2.1 (0.6)	-0.12 (0.4)	-0.22 (0.5)	-0.27 (0.4)	-0.06 (0.4)

Appendix 5

Examples of negative facial expressions from the Ekman-Friesen series

Fear	144
Anger	145
Sadness	146
Disgust	147



HAPPINESS

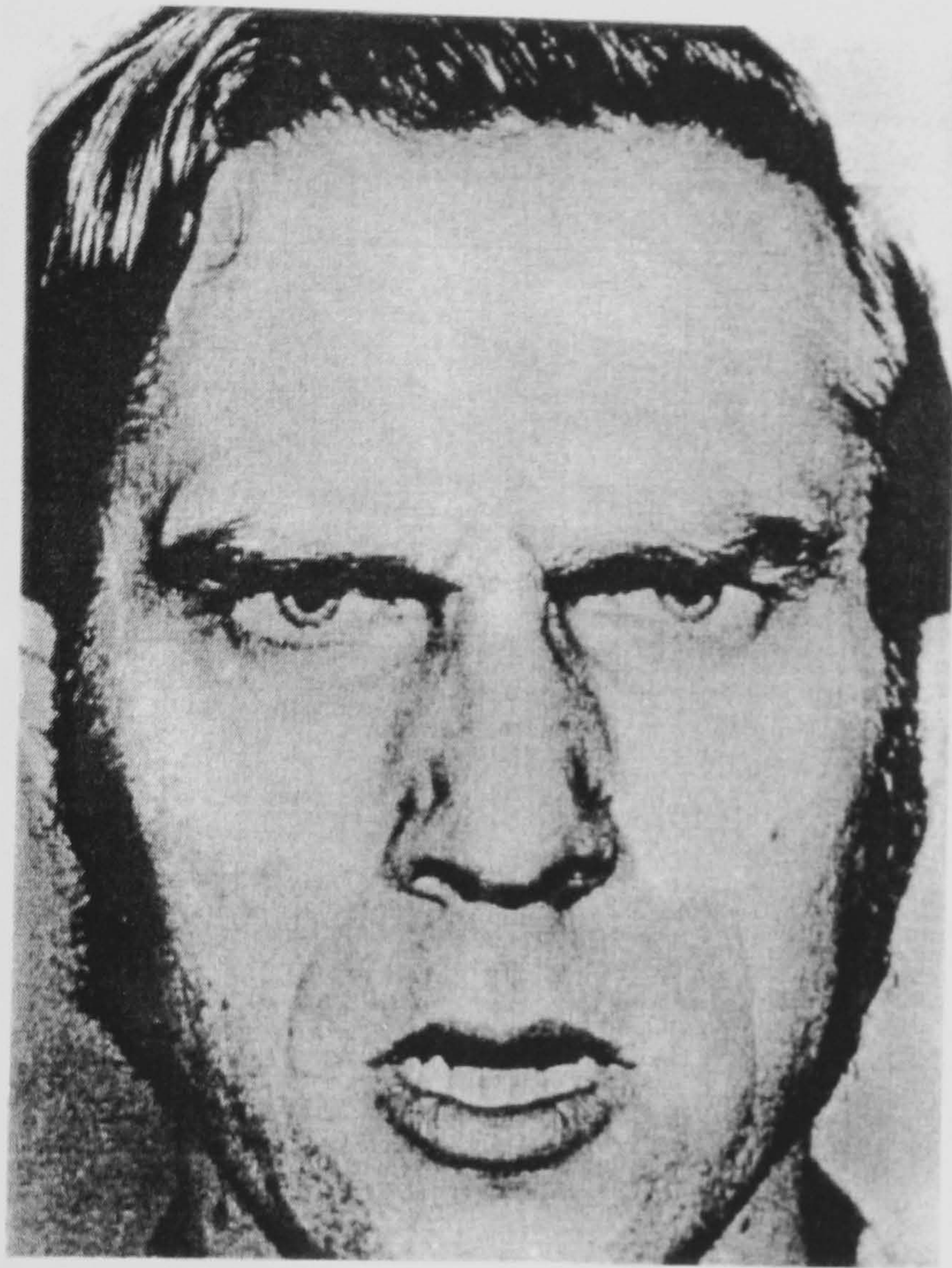
FEAR

SURPRISE

SADNESS

DISGUST

ANGER



HAPPINESS

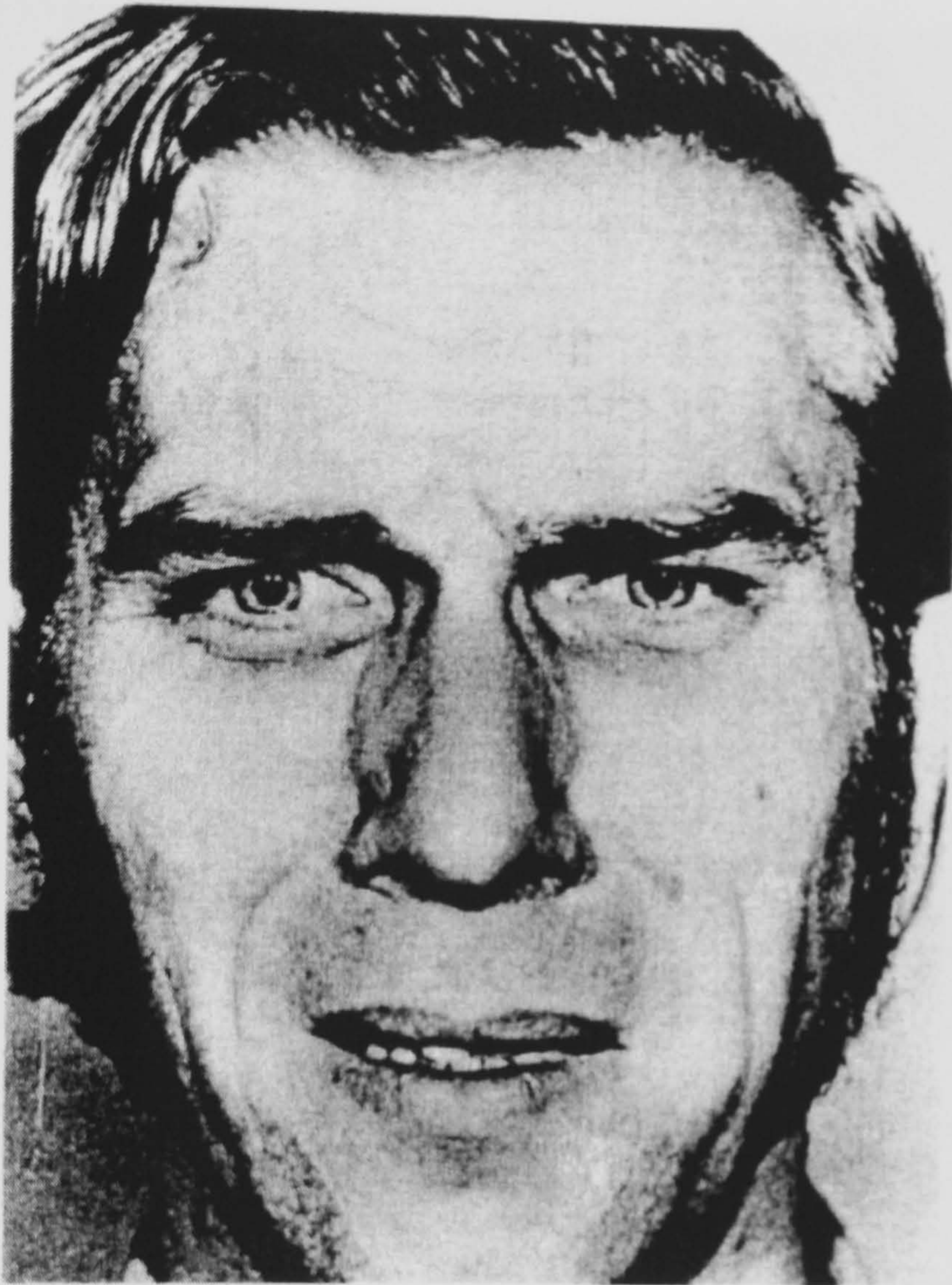
FEAR

SURPRISE

SADNESS

DISGUST

ANGER



HAPPINESS

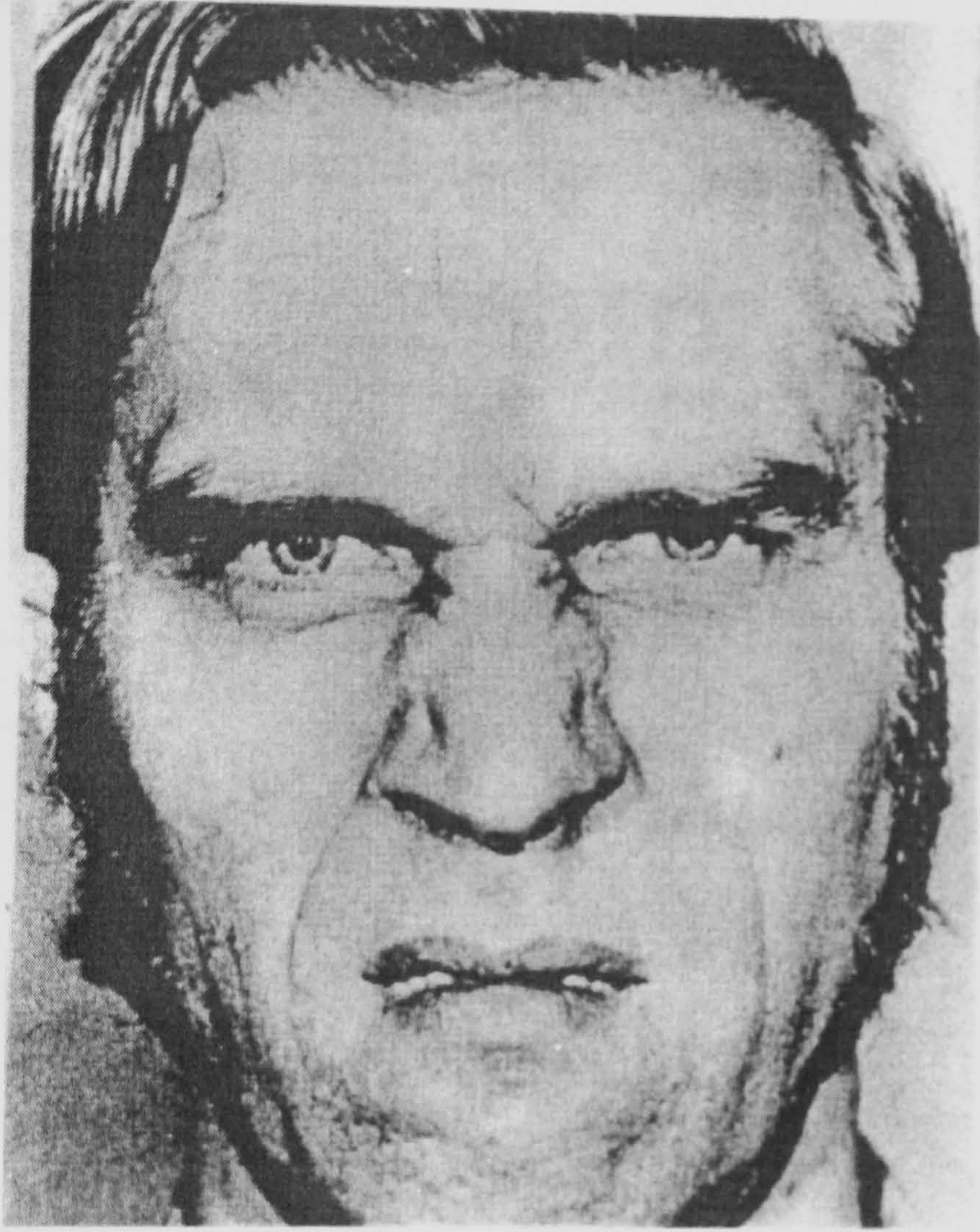
FEAR

SURPRISE

SADNESS

DISGUST

ANGER



HAPPINESS

FEAR

SURPRISE

SADNESS

DISGUST

ANGER

Appendix 6

Raw data from Chapter 3: Brief Paper

Expression Identification from Ekman-Friesen faces

	Group	Amygdala related		Non-amygdala related	
		Fear	Anger	Sadness	Disgust
NN	Control	10	9	10	9
NR	Control	10	8	9	7
NP	Control	10	9	10	10
WS	Control	6	6	8	6
EN	Control	7	10	6	9
EG	Control	9	10	10	10
LS	Control	8	8	9	3
Mean & SD		8.6 (1.6)	8.6 (1.4)	8.9 (1.5)	7.7 (2.6)
NS	PTSD	9	9	10	9
WS	PTSD	9	7	10	9
RN	PTSD	7	8	7	10
KS	PTSD	4	8	10	9
DH	PTSD	10	8	10	7
AW	PTSD	10	9	10	10
AY	PTSD	6	9	10	7
Mean & SD		7.9 (2.3)	8.3 (0.8)	9.6 (1.1)	8.7 (1.3)

Intensity ratings for correctly identified expressions

	Group	Amygdala related		Non-amygdala related	
		Fear	Anger	Sadness	Disgust
NN	Control	7.8	7.6	6.8	7.0
NR	Control	7.5	7.9	6.9	5.6
NP	Control	7.3	7.3	6.2	6.8
WS	Control	6.5	7.0	4.9	6.7
EN	Control	8.3	7.5	7.0	7.0
EG	Control	8.0	7.3	7.4	7.9
LS	Control	5.0	5.4	4.4	5.0
Mean & SD		7.2 (1.1)	7.1 (0.8)	6.2 (1.1)	6.6 (1.0)
NS	PTSD	7.8	7.8	8.2	8.9
WS	PTSD	8.3	8.1	6.7	8.4
RN	PTSD	7.1	6.4	5.4	7.6
KS	PTSD	7.3	7.1	7.0	7.0
DH	PTSD	6.4	7.0	6.6	6.7
AW	PTSD	9.0	8.8	8.1	9.0
AY	PTSD	4.8	5.3	4.0	4.5
Mean & SD		7.2 (1.4)	7.2 (1.2)	6.6 (1.5)	7.4 (1.6)

Appendix 7

Instructions to authors

Clinical Psychology Review.....	151
Neuropsychologia.....	152
Cognition and Emotion.....	153

CLINICAL PSYCHOLOGY REVIEW

INSTRUCTIONS TO AUTHORS

AIMS AND SCOPE: *Clinical Psychology Review* publishes substantive reviews of topics germane to clinical psychology. Its purpose is to help clinical psychologists keep up-to-date on relevant issues outside of their immediate areas of expertise by publishing scholarly but readable reviews. Papers cover diverse issues, including: psychopathology, psychotherapy, behavior therapy, behavioral medicine, community mental health, assessment, and child development.

Reviews on other topics, such as psychophysiology, learning therapy, and social psychology, often appear if they have a clear relationship to research or practice in clinical psychology. Integrative literature reviews and summary reports of innovative ongoing clinical research programs are also sometimes published. Reports on individual research studies are not appropriate.

SUBMISSION REQUIREMENTS: All manuscripts should be submitted to Alan S. Bellack, The University of Maryland at Baltimore, Department of Psychiatry, 737 W. Lombard St., Suite 551, Baltimore, MD 21201, USA. Submit three (3) high-quality copies of the entire manuscript; the original is not required. Allow ample margins and type double-space throughout. Papers should not exceed 50 pages (including references). One of the paper's authors should enclose a letter to the Editor, requesting review and possible publication; the letter must also state that the manuscript has not been previously published and has not been submitted elsewhere. One author's address (as well as any upcoming address change), telephone and FAX numbers, and E-mail address (if available) should be included; this individual will receive all correspondence from the Editor and Publisher.

Papers accepted for *Clinical Psychology Review* may not be published elsewhere in any language without written permission from the author(s) and publishers. Upon acceptance for publication, the author(s) must complete a Transfer of Copyright Agreement form.

COMPUTER DISKS: Authors are encouraged to submit a 3.5" HD/DD computer disk to the editorial office. Please observe the following criteria: (1) Send only hard copy when first submitting your paper. (2) When your paper has been refereed, revised if necessary, and accepted, send a disk containing the final version with the final hard copy. If the disk cannot be converted, the hard copy will be used. (3) Specify what software was used, including which release, e.g., WordPerfect 6.0a. (4) Specify what computer was used (IBM compatible PC, Apple Macintosh, etc.). (5) The article file should include all textual material (text, references, tables, figure captions, etc.) and separate illustration files, if available. (6) The file should follow the general instructions on style/arrangement and, in particular, the reference style of this journal as given in the Instructions to Contributors. (7) The file should be single-spaced and should use the wrap-around end-of-line feature, i.e., returns at the end of paragraphs only. Place two returns after every element such as title, headings, paragraphs, figure and table call-outs. (8) Keep a back-up disk for reference and safety.

TITLE PAGE: The title page should list (1) the article; (2) the authors' names and affiliations at the time the work was conducted; (3) a concise running title; and (4) an unnumbered footnote giving an address for reprint requests and acknowledgments.

ABSTRACT: An abstract should be submitted that does not exceed 200 words in length. This should be typed on a separate page following the title page.

KEYWORDS: Authors should include up to six keywords with their article. Keywords should be selected from the APA list of index descriptors, unless otherwise agreed with the Editor.

STYLE AND REFERENCES: Manuscripts should be carefully prepared using the *Publication Manual of the American Psychological Association*, 4th ed., 1994, for style. The reference section must be double spaced, and all works cited must be listed. Avoid abbreviations of journal titles and incomplete information.

Reference Style for Journals:

Raymond, M. J. (1964). The treatment of addiction by aversion conditioning with apomorphine. *Behavior Research and Therapy*, 3, 287-290.

For Books:

Barlow, D. H., Hayes, S. C., & Nelson, R. O. (1984). *The scientist practitioner: Research and accountability in clinical and educational settings*. Elmsford, NY: Pergamon.

TABLES AND FIGURES: Do not send glossy prints, photographs or original artwork until acceptance. Copies of all tables and figures should be included with each copy of the manuscript. Upon acceptance of a manuscript for publication, original, camera-ready photographs and artwork must be submitted, unmounted and on glossy paper. Photocopies, blue ink or pencil are not acceptable. Use black india ink and type figure legends on a separate sheet. Write the article title and figure number lightly in pencil on the back of each.

PAGE PROOFS AND OFFPRINTS: Page proofs of the article will be sent to the corresponding author. These should be carefully proofread. Except for typographical errors, corrections should be minimal, and rewriting the text is not permitted. Corrected page proofs must be returned within 48 hours of receipt. Along with the page proofs, the corresponding author will receive a form for ordering offprints and full copies of the issue in which the article appears. Twenty-five (25) free offprints are provided; orders for additional offprints must be received before printing in order to qualify for lower publication rates. All coauthor offprint requirements should be included on the offprint order form.

COPYRIGHT: Publications are copyrighted for the protection of the authors and the publisher. A Transfer of Copyright Agreement will be sent to the author whose manuscript is accepted. The form must be completed and returned to the publisher before the article can be published.

NEUROPSYCHOLOGIA

An international journal in behavioural and cognitive neuroscience

Information for contributors

- Papers submitted for publication should be sent to the Editorial Office in Oxford (Editor-in-Chief, Professor S. D. Iversen).
- Neuropsychologia* considers for publication papers dealing with the neural bases of cognition and behaviour and having the following types of format: (a) Research Reports (up to 20 printed pages) and Notes (up to 10 printed pages). These should describe new and original observations in various fields of the behavioural and cognitive neurosciences, whether in humans (normal or brain damaged) or in animals, and should afford significant contributions to neuropsychological theorization; (b) Reviews and Perspectives (up to 30 printed pages). These notes and purely theoretical papers will be considered for publication only exceptionally.
- (a) Submission of a paper to *Neuropsychologia* is understood to imply that it is an original paper which has not previously been published (except in the form of an abstract or preliminary report), and that it is not being considered for publication elsewhere.
(b) Papers will be published as quickly as possible after acceptance and, subject to space being available, should appear in the issue following the receipt of the author's corrected proofs.
(c) All authors must sign the 'Transfer of Copyright' agreement before the article can be published. This transfer agreement enables Elsevier Science Ltd to protect the copyrighted material for the authors, but does not relinquish the author's proprietary rights. The copyright transfer covers the exclusive rights to reproduce and distribute the article, including reprints, photographic reproductions, microform or any other reproductions of similar nature and translations, and includes the right to adapt the article for use in conjunction with computer systems and programs, including reproduction or publication in machine-readable form and incorporation in retrieval systems. Authors are responsible for obtaining from the copyright holder permission to reproduce any figures for which copyright exists.
- 50 reprints will be sent free of charge to the corresponding author. Additional reprints may be ordered on the reprint order form which will accompany the proofs sent to the author. Reprints may also be obtained after publication of the paper at a somewhat higher cost.
- (a) Papers will be accepted in English only.
(b) The title page should include: the name(s) of the author(s); the name of the department and institution where the study was carried out; the institutional affiliation of each author; the name, the postal and e-mail address and the telephone and fax number of the corresponding author; a running head of not more than 50 letters. A shortened title (a caption of no more than 5 words) to appear on the front cover of *Neuropsychologia* should it be chosen
(c) Abstracts should be less than 250 words, and should be followed by a list of up to six keywords (which do not appear in the title) to be used for indexing purposes.
(d) Research Reports and Notes as a rule should include an Introduction, a section on Methods, a section on Results and a Discussion. The description of methods and results should be sufficiently detailed so as to allow a critical assessment of their appropriateness and validity.
(e) The section on Methods of research reports, footnotes and the whole text of notes will be printed in small type. Authors may mark other passages which may also be printed in small type.
(f) Three double-spaced typewritten copies of the manuscript should be submitted in all cases but No disks at this stage please. A manuscript must be in the correct format, i.e. double-spaced, references in correct format, and top-quality figures or it will be returned to the author. Only one side of each page should be used, allowing generous margins. Manuscripts of rejected papers will not be returned to the authors.
(g) In order to expedite publication, authors are required to submit the final version of their paper on disk. The instructions for preparing electronic documents will be communicated to the authors in the final letter of acceptance of their paper. Two final paper copies plus disks and a complete set of original figures must be returned to the Editor and not directly to the Publishers.
(h) If a manuscript is to be revised, the revised version must be received within 6 months. If not, the file will be destroyed, unless the Editorial Office has been notified that a revision is in hand.
- (a) Illustrations should accompany the typescript, but should not be inserted in the text. All figures, charts and diagrams are to be referred to as "Figures" (abbreviated to "Fig.") and should be numbered consecutively in the order they are referred to in the text. All figures should be submitted in a form suitable for direct reproduction, therefore, original figures or glossy prints should be provided. It is not possible to reproduce from prints with weak lines. Illustrations for reproduction should normally be about twice the final size required. The following standard symbols should be used on line drawings, since they are easily available to the printers:

○ ● △ ▲ □ ■ + × ▽ ▼

(b) Tables should if possible be so constructed as to be intelligible without reference to the text, every table and column being provided with a heading, and should be suitable for direct reproduction. Units of measurement must always be clearly indicated. Unless it is essential to the argument, tables should summarize results by an accepted method of expression, e.g. standard deviation (S.D.). The same information should not be reproduced in both tables and figures. In the interests of economy and in order to avoid the introduction of errors, tables will be reproduced by photographic means directly from the authors' typed manuscripts. In cases of difficulty please contact the Photoreprographic Unit of your institution.

(c) References should be indicated in the text by numbers which correspond to a strictly alphabetical list at the end of the paper.

References must be in the following form:

Jeeves M A. Agenesis of the Corpus callosum. In: Boller F, Grafman J, editors. *Handbook of Neuropsychology*, vol. 4. Amsterdam: Elsevier, 1990: 99-114.

Miller E. Verbal fluency as a function of a measure of verbal intelligence and in relation to different types of cerebral pathology. *British Journal of Clinical Psychology* 1984; 23: 53-7.

References should contain the names of all authors with their initials, the title of the paper, the name of the Journal in full, the year of publication, the volume number, and the inclusive page numbers.

(d) Footnotes, as distinct from literature references, should be indicated by a consistent series of symbols starting a new one on each page; they should not be included in the numbered reference system.

(e) Page proofs will be sent to the corresponding author for correction when the paper has been set in type. Authors should notify the publisher of any change of address which occurs whilst their paper is in the process of publication.

(f) After a paper has been published the file will only be kept by the Editor for a further three months, after which it will be destroyed.

Disclaimer. Whilst every effort is made by the publishers and editorial board to see that no inaccurate or misleading data, opinion or statement appears in this journal, they wish to make it clear that the data and opinions appearing in the articles and advertisements herein are the sole responsibility of the contributor or advertiser concerned. Accordingly, the publishers, the editorial board and editors and their respective employees, officers and agents accept no responsibility or liability whatsoever for the consequences of any such inaccurate or misleading data, opinion or statement.

COGNITION AND EMOTION

Editor

Craig A. Smith, Department of Psychology & Human Development, Vanderbilt University, Box 512, Peabody College, Nashville, TN 37203, USA (please send all books for review to Craig Smith at the address above) *email:* craig.a.smith@vanderbilt.edu

Associate Editors

Joseph P. Forgas, *University of New South Wales, Sydney, Australia*

Elaine Fox, *University of Essex, Colchester, UK*

Ursula Hess, *University of Quebec at Montreal, Canada*

Ann Kring, *University of California, Berkeley, USA*

Karin Mogg, *University of Cambridge, Cambridge, UK*

Brian Parkinson, *Brunel University, Uxbridge, UK*

Gerhard Stemmler, *Philipps-Universität, Marburg, Germany*

Board of Advisors

James R. Averill

Bruno Bara

Gerald Clore

Richard Davidson

Judith F. Dunn

Nico H. Frijda

Ian Gotlib

Alice M. Isen

Arvid Kappas

Dacher Keltner

Peter J. Lang

Catherine Lutz

Antony Manstead

Andrew Mathews

Keith Oatley

Arne Öhman

W.G. Parrott

Rainer Reisenzein

James A. Russell

Klaus R. Scherer

John D. Teasdale

Masanao Toda

Fraser N. Watts

(Founding Editor)

Bernard Weiner

Anna Wierzbicka

Aims and Scope of *Cognition and Emotion*

Cognition and Emotion is devoted to the study of emotion, especially to those aspects of emotion related to mental processes. The Journal aims to bring together work on emotion undertaken by researchers in cognitive, social, clinical, and developmental psychology, neuropsychology, and cognitive science; it also welcomes psychologically orientated submissions from those in philosophy, the humanities, anthropology, sociology, and other social sciences. Examples of topics appropriate for the Journal include emotional appraisals; effects of emotion or mood on cognition and motivation; the nature of emotional experience; self-regulation of emotion or mood; social, historical, or cultural aspects of emotion; and the nature of particular emotions or emotionality in general. *Cognition and Emotion* publishes theoretical papers, original research reports, literature reviews, and extended reviews of selected books. Submissions can be considered for expedited publication as brief reports. The Journal also publishes critical abstracts of recent books.

Submission of manuscripts. Manuscripts should be prepared in APA format. Five copies should be submitted to the Editor Dr C. Smith, Department of Psychology & Human Development, Vanderbilt University, Box 512, Peabody College, Nashville TN 37203, USA.

Cognition and Emotion is published by Psychology Press Ltd, a member of the Taylor & Francis Group. Correspondence for the publisher should be addressed to the Head Office, 27 Church Road, Hove, East Sussex BN3 2FA, UK.

New subscriptions and changes of address should be sent to Psychology Press, c/o Taylor & Francis, Rankine Road, Basingstoke, Hants, RG24 8PR, UK. Please send change of address notices at least six weeks in advance, and include both old and new addresses.

Advertising enquiries should be sent to Philip Law, Advertising Assistant, Taylor & Francis Ltd, P.O. Box 25, Abingdon, OX14 3UE, UK. Tel: +44 (0) 1235 401000; Fax: +44 (0) 1235 401550; Email: philip.law@CARFAX.CO.UK

Information about Psychology Press journals and other publications is available from <http://www.psypress.co.uk>. Go to <http://www.tandf.co.uk/journals/pp/02699931.html> for current information about this journal, including how to access the online version or register for the free table of contents alerting service.

Subscription rates to Volume 14, 2000 (6 issues) are as follows:

To individuals: UK: £118.00 • Rest of world: \$195.00 To institutions: UK: £310.00 • Rest of world: \$512.00. These prices are inclusive of postage and packing. Application to mail at second class postage rate is paid at Jamaica, NY 11431, USA. US Postmasters: send address changes to *Cognition & Emotion*, Publications Expediting Inc, 200 Meacham Avenue, Elmont, NY 11003, USA. Air freight and mailing in the USA by Publications Expediting Inc.

Cognition and Emotion is covered by the following abstracting, indexing and citation services: ASSIA, Current Contents (ISI); EMBASE, Elsevier BIOBASE, Executive Digest, LLBA, Mental Health Abstracts, PsycINFO, Research Alert (ISI); Sociological Abstracts; Social Sciences Citation Index (ISI); Social SciSearch (ISI); Social Services Abstracts.

Copyright: The material published in this journal is copyright. No part of this journal may be reproduced in any form, by photostat, microform, retrieval system, or any other means, without the prior written permission of the publisher. Typeset by J&L Composition Ltd., Filey, North Yorkshire. Printed in the U. K. by Hobbs The Printers Ltd., Totton, Southampton.