

Bangor University

PROFESSIONAL DOCTORATES

Effects of traumatic experiences on information processing in patients with mental health problems

Howard, Louise A.

Award date:
2005

Awarding institution:
Bangor University

[Link to publication](#)

General rights

Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal ?

Take down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

**Effects of Traumatic Experiences on Information Processing in
Patients with Mental Health Problems**

Louise A. Howard

Thesis submitted in partial fulfillment of the requirement of the degree of
Doctorate in Clinical Psychology (D. Clin. Psy.)

June 2005



The following material has been excluded from the digitised copy due to 3rd Party Copyright restrictions:

Section 1, Ethics Proposal

Section 2, Appendix I – Notes for Contributors

Section 3, Appendix N – Notes for Contributors

Readers may consult the original thesis if they wish to see this material.

Effects of Traumatic Experiences on Information Processing in Patients with Mental Health Problems

Abstract

All human experiences have an effect on the brain, and in the form of memories and cognitive biases, affect the response of the individual to future events. When people experience traumatic events the brain is sometimes impacted in extreme ways, with major consequences for the individual in the form of psychiatric illness. This study is concerned with examining the way in which the prior experience of traumatic events affects the processing of simple stimuli under experimental conditions, in order to understand better the types of cognitive biases involved in psychiatric illnesses. The study used a well-established computerized test of selective attention, inhibition of return (IOR), and a newer task involving facial expressions and gaze cues. A review of the use of the IOR paradigm in different adult psychiatric disorders was conducted in order to aid interpretation of the findings. The mental health status of the participants was described by use of a number of standardized questionnaires. Participants were people in contact with Adult Mental Health services who had experienced trauma, including those with Posttraumatic Stress Disorder (PTSD), Psychosis and other psychiatric diagnoses. Results were compared with a healthy control group. Despite small sample sizes and heterogeneity within the groups, the study indicated that significant trauma can be associated with different types of mental health problem, and that different mental health problems are associated with different types of cognitive processing difficulty. The Psychosis group showed a lack of inhibitory processing, which is likely to be associated with increased distractibility, and, for example, lack of the ability to follow a train of thought. This is different from the distractibility seen in PTSD, which can be characterized as a tendency for attention to be captured by a salient stimulus without appropriate subsequent disengagement.

Contents

	Page
Title	i
Abstract	ii
Contents	iii
Acknowledgments	v
Declaration	vi
Section 1. Ethics Proposal	
Appendices	
Materials (questionnaires, information sheets, consent form, translations)	A
School of Psychology, University of Wales, Bangor approval	B
North West Wales Trust Local Research Ethics Committee approval	C
North West Wales Trust Research & Development approval	D
Conwy & Denbighshire Local Research Ethics Committee approval	E
Conwy & Denbighshire Research & Development approval	F
School of Psychology approval for change to project	G
Changes to protocol	H
Section 2. A review of the use of the inhibition of return paradigm in clinical research	
Title	1
Abstract	2
Introduction	3
The IOR Procedure	4
Neurological Basis for IOR	5
Variations of the IOR task	7
Abnormal Findings in Clinical Groups	9
Conclusions	16
References	19
Table	24
Appendix	
Notes for Contributors	I
Section 3. Attentional and emotional processing following psychological trauma	
Title	1
Abstract	2
Introduction	3
General Methods	6
Experiment 1	14
Method	14
Results	15
Discussion	17

Contents

Section 3, Continued.

Experiment 2	18
Method	19
Results	20
Discussion	21
General Discussion	22
References	25
Tables	31
Figures	34

Appendices

Total scores for Questionnaires	J
Prescribed Medications	K
Error rate analyses (Experiment 1)	L
Error rate analyses (Experiment 2)	M
Notes for Contributors	N

Section 4. Contributions to theory, practice and learning

Implications for future research and theory development	1
Implications for clinical practice	5
Process and personal issues arising from the conduct of the research	6
References	11

Appendix

Statement of Word Count	O
-------------------------	---

Acknowledgements

I would like to thank the various members of the Tipper lab who have helped with this project: Alex Frischen for agreeing to let me use her 'faces' task, and for getting me started with E-prime, Matt Paul for always having the answers to my questions and time to help, and Amy Hayes for good-naturedly sharing the testing facilities when we both had urgent need!

My supervisors have all had a role and it has been great to know I could call on them when needed. Matt Kimble was important in designing the project at the start, and so was Steve Tipper who helped me decide on appropriate tasks. Paul Gardner was brought in late, in case of clinical crises, and thankfully I did not need to seek him out, but I was glad to know he was there. Dave Daley has been a great source of guidance and advice regarding the writing up and analyses. Finally, my placement supervisor, Mike Jackson, has been supremely tolerant of my distractedness, and supportive throughout the process.

Recruitment of patients for experimental research is notoriously difficult, and so I am also extremely grateful to all the people who were involved in recruitment, and of course, those who generously took part.

Thanks to you all!

Section 1

Ethics Proposal

3rd party copyright material excluded from digitised thesis.

Please refer to the original text to see this material.

Section 2

Review Paper

A review of the use of the Inhibition of Return paradigm in clinical research

Howard, L. A.^a, Kimble, M.^b, & Tipper, S. P.^a

^aUniversity of Wales, Bangor, Wales, UK

^bMiddlebury College, Vermont, USA

Running Head: IOR in clinical research

Please address requests for reprints to: Dr. Louise A. Howard, School of Psychology,
University of Wales, Bangor, Gwynedd, LL57 2DG, UK.

Abstract

A review was conducted of the use of the IOR task in clinical research, specifically, relating to problems found in adult mental health. The articles selected for inclusion dated from 2000. It was found that there are some task manipulations that are known to affect performance in particular ways, while many others have not yet been systematically assessed. Recent task variations included the use of semantic and emotional stimuli, and use of such task variations was increasing. It was found there was considerable variation in the types of abnormal performance shown by different clinical groups on the task, as well as variations within groups. Interpretations involved strategic compensation for deficits, reduced inhibitory processes, automatic response activation, lateralized deficits in disengaging from cues representing an attentional dysfunction, and some results were thought to reflect a biological vulnerability. Clinical implications were usually drawn on the basis of what is known about the disorder. Theoretical advances related to increasing understanding of the relation between the brain and behaviour in attentional processes. It was concluded that the IOR procedure is a useful tool for examining attentional processes, which enables dissociation of many aspects of cognition.

Keywords: Attention; information processing; psychiatric disorders.

A review of the use of the Inhibition of Return paradigm in clinical research

It has long been established that selective attention processes involve a combination of facilitation and inhibition (e.g. Posner & Snyder, 1975). The inhibition of return (IOR) task provides evidence of the change in relative strength of facilitatory and inhibitory attentional processing of a visual stimulus over a brief period of time. It has been used with a variety of psychiatric client groups. The present review seeks to establish whether any abnormal results obtained with the task vary across different client groups, and if similar, whether they are interpreted in the same way, and reflect similar underlying deficits in attentional processes.

The IOR field was reviewed by Klein in 2000, and because the focus of the present review was to illuminate current thinking in the field, articles published before 2000 were excluded. A systematic review was beyond the scope of this paper due to the large number of articles that have been published, which includes neurological and developmental disorders. Research describing IOR in adults with mental health problems, or problems found in adult mental health settings, was selected for inclusion. This comprised use of IOR in normal aging, and in the context of alcohol and alcoholism, anxiety, obsessive compulsive disorder (OCD), and schizophrenia.

With any cognitive task, there is a tension between theoretical advancement in understanding the mechanisms and processes involved in the task, and the use of the task as a tool to explore the differences in cognitive processing of different populations. As the findings with different patient populations and different variants of the task inform progress in that patient field, and in that task version, it is possible to lose an overall view. The present review aims to synthesise the findings from disparate clinical fields to highlight similarities and differences in the way the IOR task is understood, and what it means when used in a clinical context. This will be

done by first explaining the general procedure and value of the task before considering the neurological basis of IOR, then describing the variations of the procedure relevant to this review, and the findings from clinical research using the task.

The IOR Procedure

Posner and Cohen (1984) first demonstrated the existence of inhibitory processing in spatial attention. The basic IOR task involves presentation of visual stimuli on a computer screen in a succession of different displays. A trial usually consists of a screen showing a fixation point in the centre, with two locations outlined, like a box, on either side. A second or so after the onset of the trial, attention is drawn to one location or the other by a small change in the display, typically, a flicker in one of the two peripheral boxes. Participants have been instructed to ignore such events and continue looking at the fixation point while waiting for the target to appear. After a brief interval the target, which is usually a symbol such as an asterisk appears at either the location of the previous cue (cued) or the alternative location (uncued). The participant is to make a manual response on the computer keyboard as soon as they see the target.

The two major manipulations are the comparison between responses to cued and uncued targets, and the comparison of responses at short and long intervals between the onset of the cue and of the target, referred to as stimulus onset asynchrony (SOA). Responses are usually measured as mean reaction time (RT) between the onset of the target and depression of a key in the different conditions. The usual finding is that RTs to cued targets are faster than to uncued ones at SOAs less than about 200 ms. It is thought that at short intervals RTs are speeded because attention has already been drawn to the cued location, and away from the uncued

location. This occurs independently of eye movements. At longer intervals there is a switch, so that RTs to targets appearing in cued locations are slower than those that appear in uncued ones. This is generally thought to reflect inhibitory processes that have been involved in withdrawing attention from the cued location. The inhibition associated with the cued location prevents attention being easily reallocated to the same location when the target subsequently appears in the same place, hence the name IOR, and this means that RTs are slower. An issue that remains unresolved is the time-course of the inhibition. Many accounts assume that the inhibition decays after about 3000 ms, while others argue that inhibition can leave longer-term traces in memory (e.g., Tipper, Grison, & Kessler, 2003). Nevertheless, the popularity of the paradigm lies in part with the ability to separate out components of selective attention. Each trial comprises a chain of cognitive operations: detection, disengagement, movement, engagement, and IOR (e.g. Poy, del Carmen Eixarch & Avila, 2004).

An important aspect of the IOR paradigm is its proposed function and relevance to human evolution. As the inhibition of a previously attended location, IOR has been viewed as a process that facilitates search tasks such as foraging. When searching for food, an organism needs to move on from one location to another, rather than continually returning to the same place. IOR could help to prevent perseveration of this kind (Grison, Kessler, Paul, et al., 2004). It is probable that the fundamental ability to inhibit irrelevant stimuli is an important function for many aspects of cognition, and indeed existence.

Neurological Basis for IOR

Posner, Rafal, Choate & Vaughan (1985) compared five patients with lesions in the frontal lobe, seven with parietal lobe lesions, four patients with Parkinson's disease, and six with progressive supranuclear palsy (PSP). The patients with parietal

and frontal lesions had normal IOR for a cue target SOA of 1000 ms, whereas IOR was eliminated in the group with PSP. PSP affects the superior colliculus, and it was therefore concluded that IOR is mediated by the superior colliculus rather than higher cortical structures.

Initial studies emphasised the importance of oculomotor programs in causing IOR and the possible dependence of this effect on midbrain structures such as the superior colliculus (e.g. Rafal, Calabresi, Brennan, & Sciolto, 1989). More recent studies that will be described in this review suggest that IOR can influence a wide range of cognitive processes as evidenced by IOR effects in a range of discrimination tasks, and such effects may be mediated by high level neural areas including the parietal cortex.

Posner and Petersen (1990), reviewing studies of focal lesion cases, described three different and relatively independent attentional networks: posterior, anterior and vigilance, the latter of which is not relevant to the IOR task. The posterior network is involved in the basic IOR orienting task described above (Posner, 1980). The brain areas involved are portions of the parietal cortex, the pulvinar and parts of the midbrain's superior colliculus. These areas cooperate in orienting to sensory stimuli. Inhibition occurs because the parietal lobe disengages attention from its current focus, the superior colliculus moves attention to the cued location and the pulvinar engages attention in that location.

The anterior network regulates the posterior attentional network, and controls attention to semantic information, maintaining attention when the interval between primes and targets are long. It may enable interaction between motivational and cognitive processes. It has been suggested that the anterior cingulate is involved in tasks in which automatic processes fail, including situations judged to be dangerous.

Mayer, Seidenberg, Dorflinger & Rao (2004) conducted an fMRI study while participants were undertaking IOR. Results provided support for both attentional and oculomotor theories of IOR and suggested that IOR may be mediated by two networks. One may mediate the inhibitory bias following an exogenous cue, whereas a separate network may be activated when a response must be made to stimuli that appear in inhibited locations of space. The former involved anterior cortical areas including the anterior cingulate gyrus, extending into the supplementary motor area and supplementary eye fields (oculomotor areas). The latter involved activation of the posterior parietal, superior temporal, middle temporal, and middle occipital lobes, the anterior cingulate, and dorsal medial thalamus (attention areas).

Variations of the IOR task

Stimuli

One of the initial variations of the task was to compare endogenous with exogenous control of attention (Rafal, et al., 1989). Peripheral cues are believed to capture attention exogenously, without conscious control. That is, even when participants are told to ignore the cue and it does not predict the subsequent target location, people are unable to prevent their attention orienting to the sudden stimulus onset. In contrast, a central cue, such as an arrow pointing towards one location or the other, requires conscious control, and is an example of an endogenous cue. Endogenous control of attention is also affected by task instructions, such as the relative frequency of cued to uncued trials.

Another manipulation relevant to this review is the cue-back method, which means to give a second brief cue at fixation, which ensures that attention disengages from the cued location (Briand, Larrison & Sereno, 2000). IOR onsets earlier because

the cue quickly draws attention away from the peripheral cue, enabling the onset of inhibition to arise earlier (e.g. Larrison-Faucher, Briand & Sereno, 2002).

The double-cue paradigm is also used by one of the reviewed studies. This incorporates a condition in which both boxes are cued simultaneously on a proportion of the trials, and no cue is given on a proportion of trials. The comparison between RTs obtained from these two trial types gives a measure of alertness (Gouzoulis-Mayfrank, Heekeren, Voss, et al., 2004).

Other recent variations of IOR have incorporated semantic and emotional stimuli, for example, by use of threat related or neutral words or faces as cues with anxious participants. Threat related stimuli are expected to be processed through connections from the amygdala, hippocampus, and orbito-frontal cortex to the anterior cingulate, and increase the probability of activating the anterior network in anxious participants (Avila & Parcet, 2002). Both fear and attention have the function of alerting individuals to possible danger in the environment, and so an association between the two brain mechanisms might be expected. Many psychological theories have concluded that attentional biases do play an important role in the etiology and maintenance of anxiety disorders (Fox, Russo & Dutton, 2002).

Responses

Variations of response have revealed complexities within the IOR task. Initial understanding of the task was that inhibition was generated by participants preparing an eye movement that was not released (Abrams & Dobkin, 1994). Subsequent studies have shown that inhibition can be seen in saccadic (e.g. Broomfield & Turpin, 2005), reaching (Howard, Lupianez & Tipper, 1999), or physiological responses such as heart rate (Broomfield & Turpin, 2005), as well as the more usually assessed RT.

Klein (2000) noted that to observe IOR two conditions must be met: A combination of methods must be used that elicit or cause inhibition, and the task must be designed so that one or more processes involved in the task are affected by the inhibition. IOR is inferred by poorer performance in one condition than another, but poor performance might be obscured by other processes operating at the same time as the inhibition. Examples given by Klein were increased facilitation, or response-repetition priming.

The time course of spatial cueing effects are affected by factors such as task difficulty, nature of the response, and practice effects (Klein, 2000). Other factors, such as cue and target luminance, eccentricity of cues and target, cue duration, presence or absence of central fixation, number and relation of the chosen SOAs and so forth, while not yet studied systematically, might also affect the time course of IOR (Larrison-Faucher et al., 2002).

Abnormal Findings in Clinical Groups

Summary information about the participants, type of task and main findings of the following studies are shown in Table 2.1. All experiments assessed RT unless stated otherwise.

Table 2.1 about here

Late onset of IOR. Castel, Chasteen, Scialfa & Pratt (2003) compared younger and older adults with a range of SOAs and found a difference between cued and uncued trials in the form of increased facilitation and delayed IOR. This was interpreted as more attention being allocated to cued locations, and for longer, in

compensation for reduced resources. Larrison-Faucher et al., (2002) found delayed onset of IOR in schizophrenia, replicating many previous findings, which may be due to increased facilitation, or delayed inhibition. Facilitation was at normal levels at the short SOA, but rather than an abrupt change to inhibition, there was a gradual change. This suggested delayed inhibition, but a neutral condition would be required to establish this (Larrison-Faucher et al., 2002).

Shorter effect of IOR. In a repeated measures design using the cue-back procedure, social drinkers showed a shorter effect of IOR under a high dose of alcohol (Abroms & Fillmore, 2004). Alcohol slowed performance, and also reduced the delay shown in cued conditions. This resulted in the virtual extinction of IOR at 1200 ms at the high dose of alcohol. It was proposed that inhibitory influences on target detection were reduced by the drug. The implication of this finding is that redundant searching of previously explored locations could occur, and the reacquisition of visual information would slow the rate at which new information could be obtained and processed.

Effects on error rates. In a study examining the effects of nicotine on substance abusers, chronic alcoholics had a tendency to emphasise accuracy at the expense of speed on the IOR task, as in many others, in the absence of any task specific effects. This was taken to be indicative of deficits in cognitive efficiency (Ceballos, Tivis, Lawton-Craddock & Nixon, 2005).

Errors (incorrect responses) were increased in schizophrenics relative to controls whereas anticipations (responses made prior to target onset) were not (Larrison-Faucher et al., 2002). More specifically, comparisons over a range of SOAs showed that errors increased at the transition from facilitation to inhibition in a group of schizophrenics. The authors considered that one function of IOR is to act as a

stabilizing force with respect to response. They proposed that visual information flows automatically into action based representations and that inhibitory mechanisms shape responses to avoid chaotic or overresponding.

Lateralized anomalies. In a sample of people with OCD there was a reduction of IOR in the left visual field, shown by an absence of the relative speeding of RTs at the long SOA when left targets were uncued. This deficit in inhibitory processes was attributed to a selective deficit in shifting attention from the cue in the right visual field, and was related to the perseverative nature of obsessions and compulsions, and probable right hemisphere dysfunction (Rankins, Bradshaw, Moss & Georgiou-Karistianis, 2004). Moritz & von Muhlenen (2005) compared an OCD group with an anxiety group and healthy controls. They found greater IOR for right visual targets than left in the anxiety group but no difference between controls and OCD patients. It is not clear why these researchers obtained different results.

Gouzoulis-Mayfrank et al. (2004) conducted a longitudinal study of schizophrenics when acutely ill and when symptoms were less severe. They found slow RTs to targets in the left visual field regardless of cue condition. They note that previous research has frequently shown slow RTs to targets in the right visual field especially in uncued trials (e.g. Posner, Early, Reinman et al., 1988), and this finding was replicated by a number of other researchers (Potkin, Swanson, Urbaneck et al., 1989; Maruff, Hay, Malone & Currie, 1995; Wigal, Swanson & Potkin, 1997) although not by others (Strauss, Novakovic, Tien, et al., 1991; Strauss, Alphas & Boekamp, 1992; Gold, Randolph, Coppola, et al., 1992). The finding was interpreted as a lateralized deficit in disengaging attention from the previously cued location, and was thought to reflect a left hemispheric dysfunction in attentional networks. Gouzoulis-Mayfrank et al. proposed that the discrepancy between studies of

lateralized deficits in schizophrenia might be due to use of neuroleptic medication, which may restore deficits in disengaging attention.

Absence of IOR. In schizophrenia, IOR is delayed or absent unless the cue-back procedure is used. Using the double-cue procedure, schizophrenics showed normal alerting effects, and general facilitation at the long SOA, but a specific deficit in IOR (Gouzoulis-Mayfrank et al, 2004). This deficit was not related to symptoms, length of illness, or number of previous episodes. They concluded that IOR can develop with long SOAs (e.g. 1 sec) unless the cue-back procedure is used to speed it up. Blunted IOR may be a trait marker of schizophrenia, or may reflect a biological vulnerability. The deficit could be interpreted in terms of motor or attentional processes.

Difficulty disengaging from cues. Poy et al. (2004) asked students to rate themselves as anxious or impulsive on a self-report questionnaire. The task incorporated neutral cues so that benefits and costs of valid and invalid cues could be calculated by comparison. Anxiety was shown to be associated with difficulty disengaging from a peripheral cue at the short SOA. This was shown by correlation of a questionnaire subscale with costs (neutral RTs minus uncued RTs). It was concluded that anxiety facilitates automatic detection of peripheral (potentially dangerous) stimuli. It was attributed to the posterior attentional system, given that it only occurred at the short SOA.

In contrast, at the long SOA, impulsive participants had greater ability to disengage from a centrally cued location, illustrated by the negative correlation of questionnaire subscale with costs at the long SOA. That is, impulsive individuals were better at shifting attention to the target when it appeared in the unexpected location. It was concluded that impulsivity facilitates conscious shifting of attention

when orienting is guided by expectation. On the basis of a model of attention by Corbetta and Shulman (2002), it was proposed that there are parallel dorsal and ventral systems controlling attentional orienting in IOR. The dorsal system controls the version using central cues, whereas the ventral system controls the version using peripheral cues. However, the dorsal system can be modulated by the ventral one if an unexpected stimulus appears.

Variants using semantic stimuli

Increased facilitation for aversive than neutral cues. Avila and Parcet (2002) used word stimuli as cues at peripheral locations in a version of IOR on which they compared participants high in trait anxiety and high in impulsivity. Cues were neutral (e.g., shepherd, paper) or aversive (e.g., war, error). Cue relevance was also manipulated by use of a high ratio of cued to uncued trials, and either informing participants of this or not. Anxious participants showed a greater cuing effect at the short SOA with aversive than neutral cues. The effect was not found when participants were informed of the high probability of a cued trial. The short SOA was taken to indicate that the effect was automatic, and therefore based on the posterior attentional system.

The authors proposed that anxious individuals have greater activation of the anterior system by threat related words, which interferes with the posterior system. Hamner, Lorberbaum and George (1999) proposed a model in which the anterior cingulate functions as a gate for the modulation of conditioned fear responses by determining which stimuli would or would not be attended to. It was suggested that patients with anxiety disorders would find it relatively easy to attend to exogenous emotional stimuli because of activation of the anterior system, including the anterior cingulate, which impacts on the posterior system.

Effects on disengagement from threat cues. Yiend & Mathews (2001) used pictures as peripheral cues in IOR with anxious and non-anxious students. The pictures were either threatening (e.g. corpses, weapons, assaults) or not (e.g. domestic scenes, landscapes). Participants high in anxiety showed a specific deficit in disengaging from threat cues by increased RTs to uncued targets when the cue was a threat-related picture with an SOA of 500 ms. At a longer SOA of 2000 ms, all participants were slower to disengage from a threat cue regardless of anxiety level. This may mean that disengagement from threatening pictures was less complete than that from non-threatening pictures, or that inhibition of return to the threatening location was less marked. The explanation of the effect given was that attentional localization precedes awareness of the threat value of a stimulus. More anxious participants become hypervigilant, and lower the threshold for stimulus localization, even before the threat value of the stimulus is known. Less anxious participants retain a higher threshold.

Fox et al. (2002) used schematic and photographic faces as peripheral cues with angry, happy or neutral expressions in a series of experiments with students who were rated as high or low in trait anxiety. Anxiety level did not affect the amount of IOR obtained except in the third experiment, in which the magnitude of IOR was decreased in the context of a threat related or jumbled schematic face cue in highly anxious participants. This was shown by the lengthening of RTs in the cued condition to that comparable with the uncued condition in the context of angry or jumbled stimuli in this group. The finding was interpreted as increased dwell time, or delayed disengagement, on threat cues in anxious participants.

Broomfield and Turpin (2004) used words as peripheral cues to compare groups of high and low trait anxious participants. Words were either threat-related (e.g. “stab”) or not (e.g. “print”). In contrast to work using pictorial stimuli, in their Experiment 2 Broomfield and Turpin found that high trait anxious participants rapidly disengaged attention from threatening verbal stimuli, as demonstrated by eye movements and faster RTs on uncued threat trials. The finding is consistent with cognitive avoidance. Overall, results support the hypothesis that threat modulates only the disengage component of visual attention. Also, these effects are most prevalent in high trait anxious participants. They related this to the lack of habituation seen in anxious individuals to threat stimuli that automatically capture attention.

Stroop.

Fuentes, Boucart, Vivas et al. (2000) used a complex design incorporating the Stroop task with the IOR procedure. The Stroop task involves identifying the colour of the ink that a word is written in, in this case, by pressing a key marked with the same colour sticker. On each trial the word is a colour name, which may or may not be congruent with the ink. The comparison of RTs between congruent and incongruent words represents the extent to which an incongruent word interferes with naming. In this task, a colour word was used as the target in a basic IOR task with peripheral cues. Unlike the healthy control group, the schizophrenic group did not show a reduction in Stroop effect at cued locations.

An elaborate explanation for the different performance of patient and control participants, involving inhibitory tagging, was provided by the authors. They proposed that control participants showed no Stroop interference at cued locations because the task relevant and irrelevant dimension were separated, so that when an incongruent word and colour were presented, the word had an inhibitory tag, and

therefore did not compete with the colour. Failure of inhibitory tagging in the schizophrenic group may reflect impaired operation of anterior cortical areas (and the anterior cingulate in particular). IOR, but not inhibitory tagging, occur in schizophrenia, so the two processes can be dissociated. It was suggested that they normally interact to regulate stimulus processing. IOR is mediated by oculomotor programming in the superior colliculus. An inhibitory tag generated in the midbrain may need to be transmitted to the parietal cortex through the pulvinar to be encoded in spatiotopic coordinates, as the superior colliculus itself does not maintain a spatiotopic representation. An intact superior colliculus is a necessary but not sufficient condition for IOR.

Conclusions

This review has shown great variability in what constitutes abnormal IOR performance. Using neutral stimuli, absent or delayed IOR has been observed in schizophrenia. This has been attributed to a delay in the onset of inhibition, which is unrelated to disease characteristics. It has been proposed that this may precede the onset of schizophrenia as a biological vulnerability factor. Late onset of inhibition has also been found in older adults, but this was in the context of increased facilitation, which would give the appearance of delayed inhibition. Increased facilitation might be attributed to a strategic compensation for reduced cognitive resources.

Alcohol appeared to have the effect of shortening the duration of inhibition. In chronic alcoholics, accuracy on the task is preserved at the expense of speed. It might be speculated that the reduction of inhibitory processing over a long period of time might reduce the capacity to select stimuli relevant for responding, which necessitates strategic slowing. In schizophrenia, there is an increase in error rates at

the transition from facilitation to IOR, which was interpreted as a demonstration of the direct link from perception to response, and the need for response selection by inhibitory processes. It would be interesting to see whether such effects occur in other client groups by use of a range of SOAs.

Laterality effects are common. In OCD and anxiety a deficit of IOR in the left visual field has been attributed to right hemisphere dysfunction. In schizophrenia a deficit in the right visual field has been attributed to left hemisphere dysfunction, specifically, an inability to disengage from the cue in the opposite visual field, somewhat analogous to the concept of visual extinction. However, results in schizophrenia are mixed. Increased costs of uncued trials may be present during acute stages of the disorder and in drug free patients, but not in chronic patients or in those receiving neuroleptic medication.

Difficulty disengaging from cues was also affected by personality variables such as trait anxiety when using neutral stimuli, and when using threat-related pictures or schematic faces. However, when threat related words were used, there was rapid disengagement from the cues in highly anxious participants. This was related to cognitive avoidance, which is a prominent problem in anxiety disorders, and serves to maintain symptoms. It is interesting that these different classes of stimuli provoked different responses. It may be that the words and images used were not equivalently aversive, or it may relate to the intrinsic nature of the different ways words and images are processed. That is, an aversive image will be immediately associated with a concept, whereas an aversive word needs first to be identified in the mental lexicon (e.g. Johnston & McClelland, 1980). This additional stage of processing might provide an opportunity to evade the full impact of the word.

The findings from the patient studies reviewed reveal an intricate mixture of results and explanatory viewpoints. There are many variables and many unknowns. It may be that greater clarity might be obtained with a standard version of the IOR task, and obtaining norms. However, there is at present a richness of information available from the different versions of the IOR task. Greater clarity will probably arise as knowledge of the neural processes involved in IOR become more precisely linked to their psychological correlates, and knowledge of disease processes progresses. The most constructive approach is probably to combine the results of an IOR study with what is known about the disorder, e.g. that OCD is associated with right hemisphere dysfunction, and allow that to guide interpretation.

The review has also shown great variability within a disorder, e.g. schizophrenia and OCD. The variations in results from schizophrenia research have previously been explained as differences in the tasks used, heterogeneity of the disorder, different medications, and aspects of the age of onset and long-term course (Larrison-Faucher et al., 2002). Factors affecting interpretations are not only the type of deficit shown, but also the model of IOR used to interpret the findings. It has long been established that the anterior attentional system can exert control over the posterior attentional system, as in the model used by Avila & Parcet (2002), but a recent development is that the ventral system can also override the expectations generated and held by the dorsal system, as in the model used by Poy et al. (2004). Explanatory frameworks tend to focus on neurological functioning and this appears to be the future for the field. This work will need to be based on a thorough understanding of the task components.

References

Abrams, R. A., & Dobkin, R. S. (1994). Inhibition of return: effects of attentional cuing on eye movement latencies. *Journal of Experimental Psychology: Human Perception and Performance*, 20, 467-477.

Abroms, B. D., & Fillmore, M. T. (2004). Alcohol-induced impairment of inhibitory mechanisms involved in visual search. *Experimental and Clinical Psychopharmacology*, 12, 243-250.

Avila, C., & Parcet, M. A. (2002). The role of attentional anterior network on threat-related attentional biases in anxiety. *Personality and Individual Differences*, 32, 715-728.

Briand, K. A., Larrison, A. L., & Sereno, A. B. (2000). Inhibition of return in manual and saccadic response systems. *Perception and Psychophysics*, 62, 1512-1524.

Broomfield, N. M., & Turpin, G. (2005). Covert and overt attention in trait anxiety: a cognitive psychophysiological analysis. *Biological Psychology*, 68, 179-200.

Castel, A. D., Chasteen, A. L., Scialfa, C. T., & Pratt, J. (2003). Adult age differences in the time course of inhibition of return. *Journal of Gerontology*, 58B, 256-259.

Ceballos, N. A., Tivis, R., Lawton-Craddock, A., & Nixon, S. J. (2005). Visual-spatial attention in alcoholics and illicit stimulant abusers: effects of nicotine replacement. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, 29, 97-107.

Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Review Neuroscience*, 3, 215-229.

Fox, E., Russo, R., & Dutton, K. (2002). Attentional bias for threat: Evidence for delayed disengagement from emotional faces. *Cognition and Emotion*, *16*, 355-379.

Fuentes, L. J., Boucart, M., Vivas, A. B., Alvarez, R., & Zimmerman, M. A. (2000). Inhibitory tagging in inhibition of return is affected in schizophrenia: Evidence from the Stroop task. *Neuropsychology*, *14*, 134-140.

Gold, J. M., Randolph, C., Coppola, R., Carpenter, C. J., Goldberg, T. E., & Weinberger, D. R. (1992). Visual orienting in schizophrenia. *Schizophrenia Research*, *7*, 203-209.

Gouzoulis-Mayfrank, E., Heekeren, K., Voss, T., Moerth, D., Thelen, B., & Meincke, U. (2004). Blunted inhibition of return in schizophrenia – evidence from a longitudinal study. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *28*, 389-396.

Grisson, S., Kessler, K., Paul, M., Jordan, H., & Tipper, S. P. (2004). Object- and location-based inhibition in goal-directed action: Inhibition of return reveals behavioural and anatomical dissociations and interactions with memory processes. In G. W. Humphreys & J. Riddoch (Eds). *Attention in action: Advances from Cognitive Neuroscience* (pp. 171-208). Psychology Press: Hove.

Hamner, M. B., Lorberbaum, J. P., & George, M. S. (1999). Potential role of the anterior cingulate cortex in PTSD: Review and hypothesis. *Depression and Anxiety*, *9*, 1-14.

Howard, L. A., Lupianez, J., & Tipper, S. P. (1999). Inhibition of return in a selective reaching task: An investigation of reference frames. *Journal of General Psychology: Special Issue on Visual Attention*, *126*, 421-442.

Johnston, J. C., & McClelland, J. L. (1980). Experimental tests of a hierarchical model of word identification. *Journal of Verbal Learning and Verbal Behavior*, *19*, 503-524.

Klein, R. M. (2000). Inhibition of return. *Trends in Cognitive Sciences*, *4*, 138-147.

Larrison-Faucher, A., Briand, K. A., & Sereno, A. B. (2002). Delayed onset of inhibition of return in schizophrenia. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *26*, 505-512.

Maruff, P., Hay, D., Malone, V., & Currie, J. (1995). Asymmetries in the covert orienting of visual spatial attention in schizophrenia. *Neuropsychologia*, *33*, 1205-1223.

Mayer, A. R., Seidenberg, M., Dorflinger, J. M., & Rao, S. M. (2004). An event-related fMRI study of exogenous orienting: supporting evidence for the cortical basis of inhibition of return? *Journal of Cognitive Neuroscience*, *16*, 1262-1271.

Moritz, S., & von Muhlenen, A. (2005). Inhibition of return in patients with obsessive-compulsive disorder. *Anxiety Disorders*, *19*, 117-126.

Posner, M. I. (1980). Orienting of attention. The VIIth Sir Frederick Bartlett lecture. *Quarterly Journal of Experimental Psychology*, *32*, 3-25.

Posner, M. I., & Cohen Y. (1984). Components of visual orienting. In H. Bouma, & D. Bouwhuis (Eds.), *Attention and Performance X* (pp. 55-66). Hillsdale, NJ: Erlbaum.

Posner, M. I., Early, T. S., Reinman, E., Pardo, P. J., & Dhawan, M. (1988). Asymmetries in hemispheric control of attention in schizophrenia. *Archives of General Psychiatry*, *45*, 814-821.

Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience, 13*, 25-42.

Posner, M. I., Rafal, R., Choate, L. S., & Vaughan, J. (1985). Inhibition of return: Neural basis and function. *Cognitive Neuropsychology, 2*, 211-228.

Posner, M. I. & Snyder, C. R. R. (1975). Facilitation and inhibition in the processing of signals. In P. M. A. Rabbitt & S. Dornic (Eds.) *Attention and Performance V*. NY: Academic Press.

Potkin, S. G., Swanson, J. M., Urbanek, M., Carreon, D., & Bravo, G. (1989). Lateralized deficits in covert shifts of visual attention in chronic and never-medicated schizophrenics compared to normal controls. *Schizophrenia Research, 2*, 95.

Poy, R., del Carmen Eixarch, M., Avila, C. (2004). On the relationship between attention and personality: Covert visual orienting of attention in anxiety and impulsivity. *Personality and Individual Differences, 36*, 1471-1481.

Rafal, R. D., Calabresi, P. A., Brennan, C. W., & Sciolto, T. K. (1989). Saccade preparation inhibits reorienting to recently attended locations. *Journal of Experimental Psychology: Human Perception and Performance, 15*, 673-685.

Rankins, D., Bradshaw, J., Moss, S., & Georgiou-Karistianis, N. (2004). Inhibition of return in obsessive-compulsive disorder. *Journal of the International Neuropsychological Society, 10*, 54-59.

Sapir, A., Henik, A., Dobrusin, M., & Hochman, E. Y. (2001). Attentional asymmetry in schizophrenia: Disengagement and inhibition of return deficits. *Neuropsychology, 15*, 361-370.

Strauss, M. E., Alphas, L., & Boekamp, J. (1992). Disengagement of attention in chronic schizophrenia. *Psychiatric Research, 43*, 87-92.

Strauss, M. E., Novakovic, T., Tien, A. Y., Bylsma, F., Pearlson, G. D.

(1991). Disengagement of attention in schizophrenia. *Schizophrenia Research*, 37, 394-401.

Tipper, S.P., Grison, S., & Kessler, K. (2003). Long-term inhibition of return of attention. *Psychological Science*, 14, 19-25.

Wigal, S. B., Swanson, J. M., & Potkin, S. G. (1997). Lateralized attentional deficits in drug-free and medicated schizophrenic patients. *Neuropsychologia*, 35, 1519-1525.

Yiend, J., & Mathews, A. (2001). Anxiety and attention to threatening pictures. *Quarterly Journal of Experimental Psychology*, 54A, 665-681.

Table 2.1. List of articles published since 2000 using IOR in clinical groups

Client group	Authors	Participants	Task	Abnormal finding in patient group
Normal aging	Castel et al. (2003)	20 Younger (21 yrs) 20 Older (68 yrs)	Basic IOR. Range of SOAs.	IOR later in older adults
Alcohol	Abroms & Fillmore (2004)	10 young adult social drinkers	Manipulated alcohol level. Cue back.	Slowed RTs. IOR extinguished by 1200 ms
	Ceballos et al. (2004)	Treatment seeking alcoholics, controls, illicit stimulant abusers	Nicotine v no stimulant. Central cue, 4 locations, high ratio of valid trials.	Amelioration of deficit
Schizophrenia	Fuentes et al. (2000)	13 schizophrenics, 13 controls	Basic IOR combined with Stroop	No alteration of Stroop effect at cued locations
	Gouzoulis-Mayfrank et al. (2003)	40 schizophrenics (completed) & 34 controls	Longitudinal, double cue	Slow RTs to targets in lvf
	Larrison-Faucher et al. (2002)	14 schizophrenic or schizoaffective disorder, 14 controls	Cue-back, eye movement responses. Range of SOAs	Delayed onset, increased error rate
	Sapir et al. (2001)	18 schizophrenic, 17 controls	Basic IOR & cue back	Absence of IOR in basic
OCD	Moritz & von Muhlenen (2005)	30 OCD, 14 psychiatric & 14 healthy controls	3 SOAs, exogenous	Greater IOR in rvf than lvf for psychiatric group
	Rankins et al. (2004)	10 OCD, 10 controls	Exogenous	Reduced IOR in lvf, normal IOR on rvf
Anxiety	Avila & Parcet (2002)	76 anxious & non-anxious students	Verbal cues (neutral & aversive) Informed v uninformed	Greater facilitation effect for aversive than neutral cues
	Broomfield & Turpin (2004)	Subclinical high & low trait anxious & repressors (60 & 40 in 2 expts)	Threat word stimuli. Eye-movements and heart rate responses	Absence of facilitation at short SOA. Bias towards threat.
	Fox et al. (2002)	Nonclinical state & trait anxious (34+ students in each of 3 expts)	Face stimuli Probe classification	Threat related invalid cues increase IOR in state anxious
	Poy et al. (2004)	96 trait anxious or impulsive students	Peripheral v central informative cues	Failure to disengage related to group
	Yiend & Mathews (2001)	40 high v low trait anxious students in each of 3 expts.	Threatening pictorial stimuli	Effect related to trait anxiety

Lvf = left visual field, rvf = right visual field.

Appendix I

3rd party copyright material excluded from digitised thesis.

Please refer to the original text to see this material.

Section 3

Empirical Paper

Attentional and Emotional Processing following Psychological Trauma

Louise A. Howard^a, Steven P. Tipper^a, Matthew Kimble^b, Alexandra Frischen^c

^aSchool of Psychology, University of Wales, Bangor, Wales, UK.

^bMiddlebury College, Vermont, USA

^cYork University, Toronto, Canada

Running Head: Attentional and Emotional Processing following Psychological
Trauma

Mailing address:

Dr Louise Howard
School of Psychology
University of Wales
Bangor
LL57 2DG
Wales
UK

e-mail: l.a.howard@bangor.ac.uk

Word count: 7622

Abstract

Psychological trauma is a frequent precursor to psychiatric illness and one mechanism by which this effect might occur is by changes in basic attention processes. Two experiments are reported in which 19 traumatized psychiatric patients were subgrouped and compared with 17 healthy control participants, assessing attention by using directional cues and reaction time responses. The first experiment used Posner's (1980) inhibition of return paradigm. The groups did not differ at the short interval between cue and target, whereas at the long interval the Psychosis subgroup showed little cueing effect. The second experiment incorporated facial stimuli, used eye-gaze to direct attention, and manipulated facial expression. The traumatized patients showed greater effects of facial expression than the control group, and there was some evidence of hypervigilance in the form of facilitation for threat in the PTSD group. The results are discussed in terms of theories of attention and emotional processing.

Key words: psychological-trauma, cueing, attention, emotion.

Introduction

Traumatic experiences often feature prominently in the life stories of individuals with psychiatric disorders. There is the obvious case of posttraumatic stress disorder (PTSD), which is precipitated by an excessive ordeal (e.g., Ehlers & Clark, 2000). But traumatic experiences such as childhood sexual abuse are also often associated with subsequent vulnerability to serious mental illness in adulthood (e.g., Bryer, Nelson, Baker-Miller, & Krol, 1987). The study of the effects of extreme stress on mental processes might potentially facilitate the development of therapeutic techniques that will help to ameliorate the long-term consequences of psychological trauma.

PTSD has been studied extensively over the past 20 years. It comprises a number of symptom clusters, usually identified clinically as hyperarousal, re-experiencing, avoidance, and emotional numbing (e.g. Ehlers & Clark, 2000). Theories of PTSD provide explanations as to how these symptom clusters emerge and are maintained. PTSD is regarded as an anxiety disorder (e.g. DSM-IV-TR, APA, 2000) in which the coping strategies adopted by the individual unintentionally prevents full information processing and adaptation to the stressful event, which serves to maintain a state of overwhelming and debilitating anxiety (e.g., Ehlers & Clark, 2000).

A review of the evidence on information processing in PTSD by Buckley, Blanchard and Neill (2000) concluded that there are deficits in memory function, particularly verbal memory, that may be associated with hippocampal changes, and that there are changes in autobiographical memory similar to those found in depression. Experimental evidence has accumulated to indicate that attentional processes are also disrupted in PTSD, and that disturbed arousal is of central

importance. Attention, and in particular vigilance, is disrupted by arousal dysregulation (Southwick, Bremner, Rasmussen et al., 1999). Felmingham, Bryant, Kendall & Gordon (2002) ran an event-related potential study finding disturbed parietal n200 and p300 components that represented impairments in stimulus discrimination and attention. Schell, Marshall, & Jaycox (2004) found hyperarousal was the best single predictor of subsequent PTSD symptomatology in a cross-lagged design. Other authors have reviewed the evidence and found more specifically an attentional bias for emotional information in PTSD (e.g. Thrasher & Dalgleish, 1999), and psychophysiological reactions to trauma related cues (Southwick et al., 1999).

Much of the early research on attention in PTSD used relatively gross assessments such as digit span (e.g. Bremner, Scott, Delaney et al., 1993). Jacoby (1991) recommended the use of process dissociation procedures instead of task comparisons, in order to pinpoint more specifically subtle changes in information processing. Buckley et al. (2000) distinguished automatic versus strategic processes, as initially defined by Posner and Snyder (1975). Automatic processes were seen as being involuntary, not available to conscious recognition, but not necessarily capacity free, whereas strategic processes were voluntary, available to conscious recognition, and capacity limited. They concluded that there was insufficient evidence to be certain of the effects of PTSD on automatic or strategic stimulus processing.

There is some evidence that extent of PTSD affects attention task performance, for example, Cassiday, McNally & Zeitlin (1992) found Stroop interference correlated with the intrusion subscale of the Impact of Event Scale (IES; Horowitz, Wilner & Alvarez, 1979). PTSD severity, narrowed down to combat exposure severity, was highly correlated with attention as measured by digit span (Gilbertson, Gurvits, Lasko, et al., 2001). Felmingham et al. (2000) found that

amplitude of parietal p300, a measure of attention, was negatively correlated with emotional numbing symptoms.

An experimental paradigm that might shed light on attentional processing in PTSD is inhibition of return (IOR), first described by Posner (1980). In this task, visual cues and targets are presented on a computer screen. In each trial, the cue indicates the potential location of the subsequent target, but may or may not be valid. If the cue is valid, response time to the target is initially speeded, and then slowed compared to trials in which the cue is invalid. It is believed that this is because attention is drawn to the correct location by the cue, so a target appearing at this time is processed quickly. If no target appears, then attention is withdrawn by inhibitory processes. If a target then appears in the inhibited location, processing resources take longer to be reallocated, and so response times are slowed. The IOR task has been used to examine attention in other clinical groups, but no published studies have been identified that use IOR in PTSD.

IOR would be a useful task to investigate PTSD because, unlike much of the previous research that has used verbal material (Buckley et al., 2002), it is non-verbal and can therefore tap other aspects of attentional processing, which may be both more fundamental and more generalisable. It can provide evidence for both facilitatory and inhibitory processing of non-semantic information using a basic manual response. It is also a task that would appear to simulate the difficulties reported by people with PTSD, in that IOR is believed to benefit efficient search of the environment, as inhibition prevents perseveration, and enables scanning to continue. The hypervigilance described in PTSD may therefore be evident in disrupted IOR performance. Evidence from functional neuroimaging techniques reviewed by Horner and Hamner (2002) suggests that the function of the anterior cingulate is

compromised in PTSD, and this area is also believed to be involved in IOR processing (Mayer, Seidenberg, Dorflinger, & Rao, 2004). Furthermore, unlike some other attention tasks, IOR is very simple for participants and is not unduly arduous.

The present study recruited individuals in contact with mental health services who had experienced significant trauma. The study was inclusive in order to try to establish general effects of psychological trauma on the experimental tasks, compared with a healthy control group. Psychiatric difficulties such as psychotic symptoms can be traumatic in themselves, as can their management by the medical system (e.g. Shaw, McFarlane, Bookless & Air, 2002). The traumatized patient group was subdivided into those with diagnoses of PTSD, those with diagnoses of Psychosis, and those with other diagnoses, on the grounds that individuals in these subgroups would have managed the symptoms and aftermath of their trauma in different ways, and this ongoing cognitive style would be reflected in their response to the experimental procedure.

Experiment 1 used a basic IOR task with neutral cues. It was predicted that participants with psychoses would show abnormal inhibitory processes on this task that would not be shown by the other traumatized participants. Experiment 2 incorporated a photograph of face, which morphed into different expressions, and incorporated gaze as a cue. It was predicted that participants with PTSD would show problems disengaging from aversive stimuli (Hamner, Lorberbaum & George, 1999).

General Method

Participants

Nineteen service users were recruited from NHS mental health teams. Participants were selected as those who were known to have experienced a significant trauma, and were initially identified and invited to participate in the study by their

clinical psychologist or key worker. PTSD was assessed (details below) but a full diagnostic interview was beyond the scope of this study due to time constraints, so the traumatized participants were subdivided into three groups depending on their previous diagnosis. The groups comprised those diagnosed with PTSD (PTSD group, $n = 6$), psychotic disorders (Psychosis group, $n = 7$), and those with other diagnoses, including anxiety, depression and anorexia nervosa (Depressed group, $n = 7$). If anyone had more than one diagnosis, they were put into the more specific group, e.g. if a participant had diagnoses of PTSD and depression, they were put into the PTSD group. Nobody had a diagnosis of both PTSD and psychosis.

A healthy control group of 17 people was selected from the School of Psychology Community Participant Panel at the University of Wales, Bangor, to match the patients on age and sex. One participant recruited in this way was on the waiting list for a clinical psychologist, had a trauma history comparable of that with the trauma group, and comparable scores on the questionnaires. The participant was retrospectively included in the Depressed group in all analyses. The groups therefore comprised 20 traumatised participants and 16 normal controls. Participation was voluntary and participants were free to withdraw at any time. They were paid a small fee to cover expenses.

Measures

Presence of PTSD. It was necessary to establish which of the traumatised participants had PTSD and which did not. The PTSD Symptom Scale-Interview Version (PSS-I) was selected as a short semi-structured interview of 17 items based on DSM-IV (APA, 1994) criteria. Participants are asked to report on symptoms, and severity is rated based on frequency or severity/intensity over the previous 2 weeks on a scale of 0 (not at all) to 3 (5 or more times per week/very much). The scale yields an

overall severity score ranging from 0-51, and three subscales (Re-experiencing, Avoidance, and Arousal) (Foa, Riggs, Dancu, & Rothbaum, 1993). PTSD is considered present if there are one or more symptoms on each subscale. Internal consistency of the subscales range from .65 to .71 in a sample of female assault victims. One-month test-retest reliability ranges from .93 to .95. The PSS-I has good concurrent validity as indicated by good correlations with measures of PTSD symptoms, depression, and general anxiety (Foa et al., 1993). In the present study the reliability of the total scale was .95, for the Re-experiencing subscale it was .87, for the Avoidance subscale it was .90, and for the Arousal subscale it was .88.

Self-reported Symptoms of PTSD. The Impact of Event Scale (IES; Horowitz et al., 1979) is a short scale of 15 items comprising separate subscales for Avoidance and Intrusion symptoms of PTSD, originally designed to assess reactions to bereavement. The Avoidance subscale measures the extent to which the individual tries to exclude unpleasant memories from consciousness and deliberately tries to avoid getting upset, and reminders of the event. The Intrusion subscale measures the extent to which memories of the traumatic event continue to impinge on the mind. Items are rated from 0 (not at all) to 5 (often). There is a large amount of research on the psychometric properties of the scale (e.g. Joseph, 2000). Internal consistency for the Intrusion subscale ranges from .72-.92, and for IES Avoidance it ranges from .65-.90. Test retest reliability shows that the scale is stable over a period of less than 6 months (around .87), but at longer intervals is less so (around .51-.57). Content validity is shown by relative independence of the two factors (mean correlation of .63). Convergent validity is shown by moderate correlations with other measures of PTSD, indicating that the scale captures information that is not obtained in other symptom inventories. It can be used as a brief screening instrument for PTSD, but is

not diagnostic and there are no cut-offs (Sundin & Horowitz, 2002). In the present study, Cronbach's alpha for the total scale was .91, for the Intrusion subscale was .84, and for the Avoidance subscale was .84.

Depression. Depression is often comorbid with PTSD, and can affect attentional processes. This was done using Beck's Depression Inventory II (BDI; Beck, Steer & Brown, 1996), which is widely used in clinical settings and research. The scale is based on DSM-IV criteria and consists of 21 statements focusing on experiences such as sadness, pessimism, and loss of pleasure. Severity of depression in the two weeks prior to and including the day of completion of the measure is rated on a scale of 0-3. A cut-off score of 23 is recommended by Martinsen, Friis and Hoffart (1995). The scale has high internal consistency (.89) and construct validity in an undergraduate population (Steer & Clark, 1997). Cronbach's alpha for the present study was .97.

Anxiety. Anxiety is associated with arousal, and so Beck's Anxiety Inventory (BAI; Beck & Steer, 1990) was used. This scale is also widely used in clinical settings and research. It measures general anxiety and panic attack symptoms during the week prior to completion of the inventory (Cox, Cohen, Dorenfeld & Swinson, 1996). The scale consists of 21 items rated from 0 to 3 depending on severity, with a cut-off of 20 for moderate anxiety. Items include symptoms such as numbness or tingling, heart pounding or racing, feeling terrified, shaky, and fear of losing control. The scale possesses good reliability (Cronbach's alpha = .92) and validity (Beck, Epstein, Brown and Steer, 1988). Cronbach's alpha in the present study was .95.

Dissociation. The Dissociative Experiences Scale II (DES; Carlson & Putnam, 1993) is a self-report scale of dissociative experiences consisting of 28 statements such as "Some people have the experience of finding themselves in a place and

having no idea how they got there,” that are endorsed from 0% (never) to 100% (always occurs) according to the frequency with which they occur in everyday life. The average of the scale is calculated. A cut-off score of 30 for the scale is indicative of Dissociative Identity Disorder, whereas one of 20 or so indicates PTSD or other dissociative disorders. Scores above 20 indicate significant dissociation. Zingrone and Alvarado (2001) obtained a Cronbach’s alpha of .92, showing good internal consistency. Cronbach’s alpha in the present study was .96.

Visual Acuity. Visual acuity was assessed using the Vocational Near Vision Test Type (Clement Clark International Ltd.), which is set in Times Roman Series 327. It provides a value in percent for near visual acuity when read at a distance of 38 cm.

Materials

The experiments were run on an Intel PC with Windows (98SE) using E-Prime 1.0 software (Psychology Software Tools, Inc.). Stimuli were presented on a 17-inch Iiyama VisionMaster 450 monitor set to high colour and screen resolution of 640 x 480. The participant’s head position was stabilised using a chinrest approximately 63 cm from the screen on which the stimuli appeared, where circumstances permitted.

General Procedure

Participants were tested at their usual clinic base, at their own home, or in a laboratory at the University of Wales, Bangor. The session began with obtaining informed consent, collection of demographic information and questionnaires, and ended with Experiment 2, after which participants were debriefed. Patients opted to complete the whole procedure in a single session of about 90 minutes, with breaks as required, or in two sessions spaced about 1 week apart. Control participants

completed a single session usually lasting just over an hour. Each participant completed demographic information, the PSS-I, BDI and BAI and the two experiments. Patient participants also completed the IES and DES. In order to answer the PSS-I, control participants were asked to identify a traumatic event they had experienced and to report on how that still influenced them. Clarification and assistance were given where required. If a participant had literacy problems all written information was read out to them by the experimenter. Total questionnaire scores can be found in Appendix J.

During the experiments, the room was dimly lit where possible to maximize the visual display. Participants were given time to read the instructions from the screen which were then clarified by the experimenter as required. The experiments were self-paced, each trial being initiated by the participant pressing the space bar with their thumb. Trials were terminated by indicating the side on which the target had appeared by pressing the 'x' key with the left index finger on the computer keyboard for a left response or the 'm' key with the right index finger for a right response. The response keys were marked with textured stickers. There was auditory feedback for correct (high beep) or incorrect (low beep) responses. Trials were presented in a new random order for each participant. Each experiment began with a block of practice trials. Although the experiments were conducted at the same time, they will be reported consecutively.

Data Processing and Analysis

Reaction time (RT) between onset of the target and the response was collected for each trial for each participant, excluding practice trials. There were three types of error: anticipations were RTs less than 200 ms, late responses were over 800 ms, and incorrect responses were those that were between 200 and 800 ms but in which the

wrong key was pressed. The mean RT for correct responses was produced for each of the experimental conditions. A large number of comparisons were made so only those that were significant, or otherwise of interest will be reported. Analyses were conducted using SPSS 11.0.2 (SPSS Inc., 2003) for Macintosh OS X. Bonferroni corrections were used for multiple comparisons, and the Greenhouse-Geisser correction was used for repeated measures (Field, 2000).

Participant Characteristics

Demographic information. IQ is negatively correlated with PTSD symptoms (McNally & Shin, 1995), and premorbid IQ predicts PTSD (Macklin, Metzger, Litz, et al., 1988). Assessment of IQ was beyond the scope of the present study, but number of years of education can be used as an approximation (Herrnstein & Murray, 1994) and was noted for each participant. Age has a strong bearing on RT and speed of information processing (e.g. Hasher & Zacks, 1988). Age was also noted and the groups were compared on education and age using a one-way ANOVA (Table 3.1).

Neurological and medical conditions. It was not possible to exclude those with a history of substance abuse or dependence, severe head injury, neurological or medical conditions that might interfere with attention processes, or uncorrected visual impairment, and so the presence of these extraneous variables was noted.

Depressed group: One participant had epilepsy, dyslexia, and a 30-point discrepancy between verbal and performance IQ, one was 8 months pregnant at the time of testing, one reported regularly using cannabis, and one reported a significant left-frontal head injury (no coma).

PTSD: One was dyslexic, one reported a temporal lobe head injury, and one had spinabifida.

Psychosis: One reported nerve damage resulting from chemotherapy, one had Chronic Fatigue Syndrome (CFS), one had epilepsy, and one reported regular substance use.

Healthy controls: One of the healthy control participants described himself as a former drug addict, and one had CFS.

Other factors that might influence performance

Vision. One of the PTSD group was blind at the fovea in one eye (24% on acuity test), and one of the Psychosis group reported a haemorrhage in one eye many years previously (64%). All remaining participants scored 48% or more on the test of visual acuity.

Handedness. Four of the patient group and one of the control group described themselves as left-handed, but handedness was not formally assessed.

Medication. None of the healthy control group was prescribed psychoactive medication. All of the patients were prescribed at least one medication. The PTSD patients were prescribed analgesics, anti-inflammatories, anti-depressants, selective serotonin reuptake inhibitors (SSRIs), and sleeping tablets, and one was prescribed an atypical antipsychotic. The Psychosis group was prescribed antipsychotics, atypical antipsychotics, hypnotics, analgesics, antidepressants, antimaniacs, anxiolytics, thyroid hormone and medication for the control of epilepsy. The Depressed group was prescribed antidepressants, SSRIs, atypical antipsychotics, hypnotics, anxiolytics, antibiotics and analgesics (Appendix K).

Trauma Information

The patient group reported traumas of childhood sexual abuse, rape, parental mental illness in childhood and family breakdown, witnessing violence, suicide bombing, combat, and psychotic breakdown. The healthy control group reported

stressors of bereavement, road traffic accident, childbirth, onset of parent's dementia, non-sexual assault, a colleague's suicide attempt, and a partner's stroke. All of the traumas experienced by the patient group met DSM-IV-TR Criterion A (APA, 2000), none of the control group stressors did. The years since the onset of the trauma or the initial trauma were noted and compared (Table 3.1).

The groups were compared using ANOVA and pairwise comparisons on demographic information and questionnaire scores. Means and standard deviations for the three patient groups and control participants are shown in Table 3.1, together with significance levels. Although the control group had been individually matched as closely as possible to participants in the patient groups on age, they were older than the Psychosis group, and the PTSD group was older than the other two patient groups. The control group was better educated than the patients, and the PTSD group had less education. The Depressed and PTSD groups scored higher than the other groups on the BDI, BAI, and PSS scales. The depressed group scored highest on the DES. The PTSD group scored highest on IES intrusions.

Table 3.1 about here

Experiment 1: Basic IOR

Method

Procedure. There was a practice block of 16 trials before the start of the experiment, which consisted of 80 trials. A warning screen notified participants at the start of the data collection trials. A central cross subtending $.8^\circ$ served as a fixation point for 1000 ms. The display onset with a square 8.6° to left and right of fixation for

500 ms. One of the squares then darkened, which constituted a cue, and then an asterisk subtending $.8^\circ$, which was the target, appeared in either the left or right square after a further 200 or 1200 ms (Figure 3.1). The display was terminated with the response, or after 800 ms had elapsed.

Figure 3.1 about here

Design. Experiment 1 involved three within subject factors. These were the stimulus onset asynchrony (SOA) between cue and subsequent target (200 or 1200 ms), Cuing (cued or uncued) and target side (left or right). Each condition was equiprobable within each of 10 blocks and trials were presented in a new random order for each participant.

Results

Errors. Full analysis of errors can be found in Appendix L. One participant in the PTSD group made a high number of incorrect responses (17.5%) and was excluded from further analyses of this task. The PTSD group made more late and incorrect responses than the other three groups, who did not differ.

RTs. RTs were analysed using a 4x2x2x2 ANOVA with Group as a between subject factor, SOA, Target Side and Cuing as within subject factors. The overall effects were as usually found in research using this paradigm, which is that RTs at the short SOA are longer than at the long SOA, responses to targets in the left visual field are faster than those on the right, and the IOR effect which is represented by an interaction of Cuing with SOA (Table 3.2). In addition, there was a significant Group x Target Side x Cuing interaction, $F(3,31) = 3.470$ ($p < .05$). The interaction was explored using a 2x2x2 ANOVA on each subgroup.

Table 3.2 about here

The Control group showed the usual effects as expected. The Depressed group did not show the usual effect of SOA, $F(1,6) = 2.137$ (ns) but an additional effect was that the Target Side x Cuing interaction was borderline, $F(1,6) = 4.730$ ($p = .07$). The Cuing effect was bigger for targets in the right visual field (26 ms) than targets in the left visual field (6 ms) across both SOAs. The Psychosis group also showed no main effect of SOA, the usual effect of Target Side and the SOA x Cuing interaction. In addition, there was a main effect of Cuing, $F(1,6) = 18.916$ ($p < .005$). This indicated that cued targets were responded to faster (415 ms) than uncued targets (441 ms), regardless of SOA.

The PTSD subgroup showed none of the usual effects. When the original RTs of the five PTSD participants were examined it was noted that at the long SOA one of the participants showed a large facilitation effect for targets and cues on the left (166 ms) and a smaller one on the right (34 ms) rather than inhibition, which might account for the discrepant results in this group. When this participant was excluded from

analyses, the remaining PTSD group showed a borderline effect of SOA, no Target Side effect, but a substantial interaction indicating the presence of IOR. There was also a borderline interaction of Target Side x Cuing, $F(1,3) = 6.126$ ($p = .09$). Means are plotted in Figure 3.2.

Figure 3.2 about here

Discussion

The groups all performed similarly on the IOR task at the short SOA, showing normal facilitation following cues on the same side as the subsequent target. At the long SOA, both the PTSD and Psychosis groups initially showed different patterns of responding. However, the PTSD group performed normally when one participant was excluded from analyses. The pattern of responding of this participant showed facilitation at both locations, which might indicate a failure of the inhibitory system. This participant was only 2 years post-trauma, but although questionnaire scores were high, they were not outlying. It is possible that in early stages of PTSD basic attention processes are particularly affected, and that this ameliorates over time, a change that is not reflected in subjective questionnaire scores. It is also interesting to note that the participant excluded on the basis of a high error rate showed the normal pattern of RTs for the remaining responses. This indicates perhaps a different type of difficulty, in which attention processes work normally when they work, but with an occasional failure to notice the target.

The increased number of incorrect responses found in the PTSD group has a precedent. Semple, Goyer, McCormick, et al. (1996) found an increased false alarm

rate in a continuous auditory test in a PTSD group. The increased rate of incorrect responses suggests a lowered response threshold.

The performance of the Psychosis group was more variable. It is noteworthy that inspection of the data showed that only one of the depressed group showed facilitation in any condition at the long SOA, whereas all but one of the psychosis group showed some degree of facilitation at the long SOA to targets on one side or the other, or both. The Psychosis group as a whole showed facilitation at the long SOA to targets in the left visual field. The absence of a cueing effect at the long SOA for the Psychosis group has been found before (e.g., Sapir, Henik, Dobrusin & Hochman, 2001) and probably reflects the variability within the group. The control participants were also variable, and the reason for that is unclear, but may mean that some participants made eye movements.

Targets on the left were responded to faster than those on the right. This does not reflect a speed-accuracy trade off, as errors were also lower for targets on the left. The findings may represent increased sensitivity to targets on the left and has often been found before (Klein, 2000). The minimal amount of IOR in the left visual field for the control participants seen in Figure 3.2 is an unusual finding, possibly related to aspects of the task, and was not statistically significant.

Experiment 2: Face and Gaze Cues

Experiment 2 was identical to Experiment 1 except for the cueing procedure. In recent years, there has been increasing interest in ecological aspects of attentional processing. For example, attention plays an important role in social development, and social interactions. Joint attention develops in infants when they learn to look where someone else is looking (Baron-Cohen, 1995). Studies using facial stimuli and gaze direction as cues have shown robust facilitation effects (e.g. Frischen & Tipper,

2004). A further adaptation has been to morph faces into expressions of fear, anger, or happiness prior to shifting gaze. It is thought that processing expressions of fear and happiness occurs in a separate neural circuit from anger (Adams & Kleck, 2003).

The areas of the brain that are affected by PTSD, in particular the anterior cingulate and amygdala are likely to be involved in the emotional cueing task. Using an fMRI study, Rauch, Whalen, Shin, et al. (2000) found increased responses in the amygdala to fearful faces in combat veterans with PTSD compared to those without. The emotional cueing task may therefore be a sensitive measure of the difficulties faced in managing real world interactions for people with PTSD.

Method

Procedure. In each trial a central cross subtending 0.7° served as a fixation point for 1000 ms. A photograph of a face was presented at fixation looking straight ahead and with a neutral expression for a further 1000 ms, during which it morphed into an expression (happiness, anger or fear) and oriented to left or right, or remained looking straight ahead, using 3 photographic images, each presented for 20 ms. The orientation of the face constituted the cue. The target was an asterisk subtending 0.7° presented 8.6° to the left or right side of the centre of the screen approximately in line with the eyes (Figure 3.3). Participants were informed that target presentation was random and were encouraged to ignore the face. The interval between the face beginning to orientate and a target appearing was 200 or 1200 ms. The display was terminated with the response, or after 800 ms had elapsed.

Each participant completed a block of 36 practice trials before beginning the experiment, which consisted of 288 trials, with a break in the middle. Participants were told that they could pause the experiment at any time if they wished by not pressing the spacebar.

Figure 3.3 about here

Design. There were 36 conditions comprised of two SOAs (200 and 1200 ms), three facial expressions (happy, angry and fearful), cue type (cued and uncued) and two target sides (left and right). The 36 conditions were equiprobable within each of 8 blocks and were presented in a new random order for each participant.

Results

One participant in the Psychosis group did not wish to take part in this experiment and was not tested.

Errors. One participant from each of the Depressed and PTSD groups were excluded from analyses due to a high rate of incorrect responses (> 11%). A full analysis of errors can be found in Appendix M.

RTs. RTs were analysed with a 2x3x2x3x2 repeated measures ANOVA with Group as a between subject factor, Cuing, SOA, Expression, and Target Side as within subject factors (Table 3.3). As in Experiment 1, RTs were faster at the longer SOA, and for targets in the left visual field. There was also a main effect of Cuing, with RTs to cued targets quicker than RTs to uncued ones. There was also an interaction of SOA with Cuing, and borderline interactions of Cuing x SOA x Group, and Cuing x Expression, and SOA x Target Side.

To explore these interactions further, separate ANOVAs were conducted on each subgroup. Regarding Cuing x SOA, the Control group showed a bigger effect of Cuing at the short SOA than at the long. The PTSD group also showed a borderline

interaction of SOA x Cuing, but with a greater effect at the long SOA. The other two groups did not show this interaction.

The control group showed no significant effects of Expression, whereas the patient groups did: The Depressed group showed a Cuing x Expression interaction. Post hoc comparisons were not available from SPSS, but means indicated that the effect of Expression was least in the cued condition (8 ms difference) and biggest in the neutral condition (30 ms difference) (means are plotted in Figure 3.4). The Psychosis group showed a borderline main effect of Expression, being slower to respond to happy faces (400 ms) than to either fearful (385 ms) or threatening ones (391 ms, $p = .07$). The PTSD group showed a borderline Cuing x Expression x SOA interaction. There was no effect of Cuing at the short SOA for the angry face. At the long SOA effects of expression were more pronounced, especially for the fearful face.

Table 3.3 about here

Figure 3.4 about here

Discussion

The Control participants showed effects of cuing that faded over time, which replicated previous findings (Frischen & Tipper, 2004). This pattern was reversed in the PTSD group, who showed greater cueing effects at the long SOA in this task. A possible reason for this is that the PTSD group was slower to begin to encode the cue.

This might be due to the use of facial stimuli that captured processing capacity, so that preparing for the target became like doing a dual task at the short SOA.

Scrutiny of errors showed that most occurred following the fearful face and at the short SOA, and least occurred after the fearful face at the long SOA. This suggests that the fearful face had an alerting effect that initially damaged task performance until it faded at the long SOA. The effect of Expression on RTs among the patient groups distinguished them from the control group. The Depressed group was affected most by expression when the face was looking straight ahead. The emotional impact of the face smiling, looking fearful, or turning angry is very likely to be increased with a direct gaze (Adams & Kleck, 2003), but it is interesting that only the Depressed group were affected in this way. The Psychosis group was quicker to respond to the negative expressions than to the positive one, indicating increased sensitivity to this class of stimuli than the Control group. The PTSD group showed marked effects of expression, and in particular, fast RTs to uncued targets in the context of a fearful face at the long SOA. This does not represent IOR as it appears to be facilitation in the uncued condition rather than inhibition in the cued condition, relative to the neutral condition. This does therefore look potentially like hypervigilance.

The small sample sizes in these groups mean that any conclusions about group differences must be very tentative, but it is possible that the response of the patients to the emotional expressions in this task is more acute than that of healthy control participants.

General Discussion

The findings of the study have been mixed. The PTSD group showed a normal pattern of response on the IOR task, with some participants producing aberrant

performances, such as high error rates, or facilitation at the long SOA. On the emotional cueing task, the PTSD group did not show the normal pattern of facilitation that fades, as facilitation for the fearful face increased. This suggests that the PTSD group responded differently from control participants to the cues used in Experiment 2 even though the time course of the two experiments was the same. The most likely explanation is that the emotional information contained in the face cue required the use of a different processing network. It is possible that the face cue provoked emotional memories that activated the amygdala and could not be damped down by the inhibitory processes from the prefrontal cortex (Brewin & Holmes, 2003).

The depressed group showed normal IOR, in fact a bigger effect than that of the control group, indicating that their selective attention processes are intact. The Psychosis group did not show normal IOR, indicating that inhibitory processes are impaired, although there was variability within the group. On the emotional cueing task, both these patient groups were more affected than the control group by the facial expression in the cues. This suggests a heightened sensitivity to emotional expression, which is encoded in the anterior attentional neural circuit, perhaps via a lowered threshold (e.g., Avila & Parcet, 2002).

The impairment of inhibitory processing in the Psychosis group could be expected to have clinical implications. Such individuals are likely to find it difficult to direct their attention, and to be easily distracted by irrelevant stimuli in the environment. The PTSD group showed a pattern of performance suggesting that attention can be captured by irrelevant aspects of a stimulus, causing a decrement on the task. Disengagement of attention appears to be a problem. Such individuals are likely to need increased time to process information, especially if it has emotional significance.

The PTSD group was older than the other groups. It is possible that cognitive aging increased vulnerability to PTSD (Floyd, Rice & Black, 2002), or this may reflect a chance difference arising because of the small sample sizes. The age difference might have affected the study, as older participants can show increased variance and shifts in attention, especially as they are less used to computers than younger participants (Elsesser, Sartory & Tackenberg, 2004). However, in this study the PTSD group did not have the slowest RTs.

It is difficult to draw firm conclusions about the behavioural assessment of hyperarousal on the basis of this study. Hyperarousal is likely to have many different components such as exaggerated startle, sleep difficulties, difficulty concentrating, and it is possible that different symptom clusters will need to be assessed in different ways (Schell et al., 2004).

In terms of treatment, hyperarousal needs to be targeted to help prevent onset of other symptoms (Schell et al., 2004). Hyperarousal is usually assessed by questionnaire, and is one of the factors in the PSS-I. The present study looked at cognitive processes by using RT tasks, which are more sensitive to changes in attentional processing than paper and pencil tests. The facial cuing task did appear to differentiate between PTSD and the other disorders and may have potential as a measure of hypervigilance. It would be interesting to follow-up individuals who took part in the study as they progress in therapy to see whether their response patterns change over time. Such measures might be used to assess risk of relapse.

References

- Adams, R. B., & Kleck, R. E. (2003). Perceived gaze direction and the processing of facial displays of emotion. *Psychological Science, 14*, 644-647.
- American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders, 4th Ed.* APA: Washington, DC.
- American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders, 4th Ed (revised).* APA: Washington, DC.
- Avila, C., & Parcet, M. A. (2002). The role of attentional anterior network on threat-related attentional biases in anxiety. *Personality and Individual Differences, 32*, 715-728.
- Baron-Cohen, S. (1995). *Mindblindness: An essay on autism and theory of mind.* Cambridge, MA: MIT Press.
- Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology, 56*, 893-897.
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck Depression Inventory-II.* San Antonio, TX: Psychological Corporation.
- Beck, A. T., & Steer, R. A. (1987). *Beck Depression Inventory Manual.* San Antonio. The Psychological Corporation.
- Beck, A. T., & Steer, R. A. (1990). *Beck Anxiety Inventory Manual.* San Antonio: The Psychological Corporation.
- 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.

Brewin, C. R., & Holmes, E. A. (2003). Psychological theories of posttraumatic stress disorder. *Clinical Psychology Review, 23*, 339-376.

Bryer, J. B., Nelson, B. A., Baker-Miller, J., & Krol, P. A. (1987). Childhood sexual and physical abuse as factors in adult psychiatric illness. *American Journal of Psychiatry, 144*, 1426-1430.

Buckley, T. C., Blanchard, E. B., & Neil, W. T. (2000). Information processing and PTSD: A review of the empirical literature. *Clinical Psychology Review, 20*, 1041-1065.

Carlson, E. B., & Putnam, F. W. (1993). An update on the Dissociative Experiences Scale. *Dissociation, 6*, 16-27.

Cassiday, K. L., McNally, R. J., & Zeitlin, S. B. (1992). Cognitive processing of trauma cues in rape victims with Post-Traumatic Stress Disorder. *Cognitive Therapy and Research, 16*, 283-295.

Cox, B. J., Cohen, E., Dorenfeld, D. M., & Swinson, R. P. (1996). Does the Beck Anxiety Inventory measure anything beyond panic attack symptoms? *Behaviour Research and Therapy, 34*, 949-954.

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy, 38*, 319-345.

Elsesser, K., Sartory, G., & Tackenberg, A. (2004). Attention, heart rate, and startle response during exposure to trauma-relevant pictures: A comparison of recent trauma victims and patients with posttraumatic stress disorder. *Journal of Abnormal Psychology, 113*, 289-301.

Felmingham, K. L., Bryant, R. A., Kendall, C., & Gordon, E. (2002). Event-related potential dysfunction in posttraumatic stress disorder: the role of numbing. *Psychiatry Research, 109*, 171-179.

Field, A. (2000). *Discovering Statistics using SPSS for Windows*. London: Sage.

Floyd, M., Rice, J., & Black, S. R. (2002). Recurrence of posttraumatic stress disorder in late life: A cognitive aging perspective. *Journal of Clinical Gerontology*, 8, 303-311.

Foa, E. B., Riggs, D. S., Dancu, C. V., & Rothbaum, B. O. (1993). Reliability and validity of a brief instrument for assessing posttraumatic stress disorder. *Journal of Traumatic Stress*, 6, 459-473.

Frischen A., & Tipper, S. P. (2004). Orienting attention via observed gaze shift evokes longer-term inhibitory effects: Implications for social interactions, attention and memory. *Journal of Experimental Psychology: General*, 133, 516-533.

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, 413-432.

Hamner, M. B., Lorberbaum, J. P., & George, M. S. (1999). Potential role of the anterior cingulate cortex in PTSD: Review and hypothesis. *Depression and Anxiety*, 9, 1-14.

Hasher, L., & Zacks, R. T. (1988). Working memory, comprehension and aging: A review and a new view. *Psychology of Learning and Motivation*, 22, 193-255.

Herrnstein, R. J., & Murray, C. (1994). *The bell curve*. Free Press: NY.

Horner, M. D., & Hamner, M. B. (2002). Neurocognitive functioning in posttraumatic stress disorder. *Neuropsychology Review*, 12, 15-30.

Horowitz, M. J., Wilner, N. & Alvarez, W. (1979). Impact of Event Scale: A measure of subjective stress. *Psychosomatic Medicine*, 41, 209-218.

Jacoby, L. L. (1991). A process dissociation framework: Separating automatic from intentional uses of memory. *Journal of Memory and Language*, 30, 513-541.

Joseph, S. (2000). Psychometric evaluation of Horowitz's Impact of Event Scale: A review. *Journal of Traumatic Stress*, 13, 101-113.

Klein, R. M. (2000). Inhibition of return. *Trends in Cognitive Sciences*, 4, 138-147.

Macklin, M. L., Metzger, L. J., Litz, B. T., McNally, R. J., Lasko, N. B., Orr, S. P., & Pitman, P. K. (1988). Lower precombat intelligence is a risk factor for posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, 66, 323-326.

McNally, R. J., & Shin, L. M. (1995). Association of intelligence with severity of posttraumatic stress disorder symptoms in Vietnam combat veterans. *American Journal of Psychiatry*, 152, 936-938.

Martinsen, E. W., Friis, S., & Hoffart, A. (1995). Assessment of depression: Comparison between Beck Depression Inventory and subscales of Comprehensive Psychopathological Rating Scale. *Acta Psychiatrica Scandinavia*, 92, 460-463.

Mayer, A. R., Seidenberg, M., Dorflinger, J. M., & Rao, S. M. (2004). An event-related fMRI study of exogenous orienting: supporting evidence for the cortical basis of inhibition of return? *Journal of Cognitive Neuroscience*, 16, 1262-1271.

Posner, M. I. (1980). Orienting of attention. *Quarterly Journal of Experimental Psychology*, 32A, 3-25.

Posner, M. I. & Snyder, C. R. R. (1975). Facilitation and inhibition in the processing of signals. In P. M. A. Rabbitt & S. Dornic (Eds.) *Attention and Performance V*. NY: Academic Press.

Rauch, S. L., Whalen, P. J., Shin, M. C., McInerney, S. C., Macklin, M. L., Lasko, N. B., Orr, S. P., & Pitman, R. K. (2000). Exaggerated startle response to masked facial stimuli in posttraumatic stress disorder: A functional MRI study. *Biological Psychiatry, 47*, 769-776.

Sapir, A., Henik, A., Dobrusin, M., & Hochman, E. Y. (2001). Attentional asymmetry in schizophrenia: Disengagement and inhibition of return deficits. *Neuropsychology, 15*, 361-370.

Schell, T. L., Marshall, G. N., & Jaycox, L. H. (2004). All symptoms are not created equal: The prominent role of hyperarousal in the natural course of posttraumatic psychological distress. *Journal of Abnormal Psychology, 113*, 189-197.

Semple, W. E., Goyer, P. F., McCormick, R., Compton-Toth, B., Morris, E., Donovan, B., Muswick, G., Nelson, D., Garnett, M. L., Sharkoff, J., Leisure, G., Miraldi, F., & Schulz, S. C. (1996). Attention and regional cerebral blood flow in posttraumatic stress disorder patients with substance abuse histories. *Psychiatry Research: Neuroimaging, 67*, 17-28.

Shaw, K., McFarlane, A. C., Bookless, C., & Air, T. (2002). The aetiology of post psychotic posttraumatic stress disorder following a psychotic episode. *Journal of Traumatic Stress, 15*, 39-47.

Southwick, S. M., Bremner, J. D., Rasmusson, A., Morgan, C. A., Arnsten, A., & Charney, D. S. (1999). Role of norepinephrine in the pathophysiology and treatment of posttraumatic stress disorder. *Biological Psychiatry, 46*, 1192-1204.

Sundin, E. C., & Horowitz, M. J. (2002). Impact of Event Scale: Psychometric properties. *British Journal of Psychiatry, 180*, 203-209.

Steer R. A., & Clark, D. A. (1997). Psychometric characteristics of the Beck Depression Inventory-II with college students. *Measurement and Evaluation in Counseling and Development, 30*, 128-136.

Thrasher, S. & Dalglish, T. (1999). Information processing research in PTSD. In W. Yule (Ed.) *Posttraumatic Stress Disorders*. Wiley: Chichester.

Zingrone, N.L., & Alvarado, C. S. (2001). The Dissociative Experiences Scale-II: Descriptive statistics, factor analysis, and frequency of experiences. *Imagination, Cognition and Personality, 21*, 145-157.

Tables

Table 3.1. Mean, standard deviation and significance levels for the four groups on demographic and questionnaire variables.

	Healthy Control (n = 16)	Psychosis (n = 7)	Depressed (n = 7*)	PTSD (n = 6)	F	p <
Age (yrs)	43.7 (12.0)	32.0 (5.3)a	43.6 (5.3)	53.2 (8.7)bc	5.848	.005
Education (yrs)	16.4 (1.5)	14.0 (2.4)a	14.1 (3.0)a	11.0 (1.1)abc	10.522	.001
Time since trauma (yrs)	8.9 (6.5)	14.4 (9.6)	26.8 (14.7)a	27.3 (18.5)a	3.292	.05
BDI	5.7 (5.4)	18.4 (12.4)	35.3 (15.6)a	33.0 (14.1)ab	8.183	.001
BAI	6.1 (6.3)	14.1 (11.1)	32.8 (14.3)ab	31.2 (9.6) ab	10.519	.001
PSS total	4.7 (4.7)	15.7 (7.0)	30.8 (14.0)a	18.2 (7.9)abc	14.308	.001
PSS re-experiencing	1.2 (1.4)	3.0 (2.7)	8.1 (5.1)ab	11.3 (3.4)abc	18.268	.001
PSS avoidance	1.4 (2.0)	7.8 (4.6)a	12.1 (6.9)a	14.9 (5.6)ac	8.220	.001
PSS hyperarousal	2.1 (2.9)	4.8 (3.2)	10.6 (5.2)a	12.0 (2.3)ac	7.754	.001
DES	-	17.3 (15.0)	40.2 (27.2)b	20.3 (11.6)	2.688	ns
IES total	-	36.6 (17.3)	39.7 (25.5)	51.8 (11.2)	1.151	ns
IES intrusions	-	17.0 (7.9)	16.5 (11.0)	27.5 (2.8)bc	3.703	.05
IES avoidance	-	19.6 (9.8)	23.2 (15.0)	24.3 (8.8)	.311	ns

Notes

Df = 3 and 32.

a = significantly different from healthy control; b = significantly different from Psychosis; c = significantly different from Depressed.

*N = 6, df = 2 and 16 for DES and IES.

Table 3.2. Results of ANOVA on Experiment 1

ALL	df	F	p <
SOA	1,31	14.947	.001
Target Side	1,31	17.873	.001
SOA x Cuing	1,31	72.986	.001
Target Side x Cuing	1,31	9.415	.005
Target Side x Cuing x Group	3,31	3.470	.05
CONTROL			
SOA	1,15	10.407	.01
Target Side	1,15	10.311	.01
SOA x Cuing	1,15	36.554	.001
DEPRESSED			
SOA	1,6	2.137	ns
Target Side	1,6	6.287	.05
SOA x Cuing	1,6	69.494	.001
PSYCHOSIS			
SOA	1,6	3.354	ns
Target Side	1,6	11.612	.01
SOA x Cuing	1,6	47.050	.001
PTSD			
SOA	1,3	7.583	.07
Target Side	1,3	.547	ns
SOA x Cuing	1,3	22.518	.05

Table 3.3. Results of ANOVA on Experiment 2

ALL	df	F	p <
Cuing	2,48	11.994	.001
SOA	1,29	55.083	.001
Target Side	1,29	8.647	.01
Cuing x SOA	2,56	10.588	.001
Cuing x SOA x Group	6,56	2.655	.05
Cuing x Expression	3,92	2.277	.08
SOA x Target Side	1,29	3.259	.08
CONTROL			
Expression	1,21	.590	ns
Cuing x Expression	2,39	.205	ns
SOA x Cuing	2,29	9.820	.001
DEPRESSED			
Expression	2,8	.399	ns
Cuing x Expression	2,12	3.943	.05
SOA x Cuing	2,9	1.916	ns
PSYCHOSIS			
Expression	2,8	4.136	.06
Cuing x Expression	2,8	2.483	ns
SOA x Cuing	2,8	47.050	ns
PTSD			
Expression	1,5	.606	ns
Cuing x Expression	1,6	.740	ns
SOA x Cuing	1,6	3.813	.09
Cuing x Expression x SOA	1,5	2.484	.17

Figure 3.1. Procedure used in Experiment 1. Example of a validly cued trial in which the target appears in the left visual field.

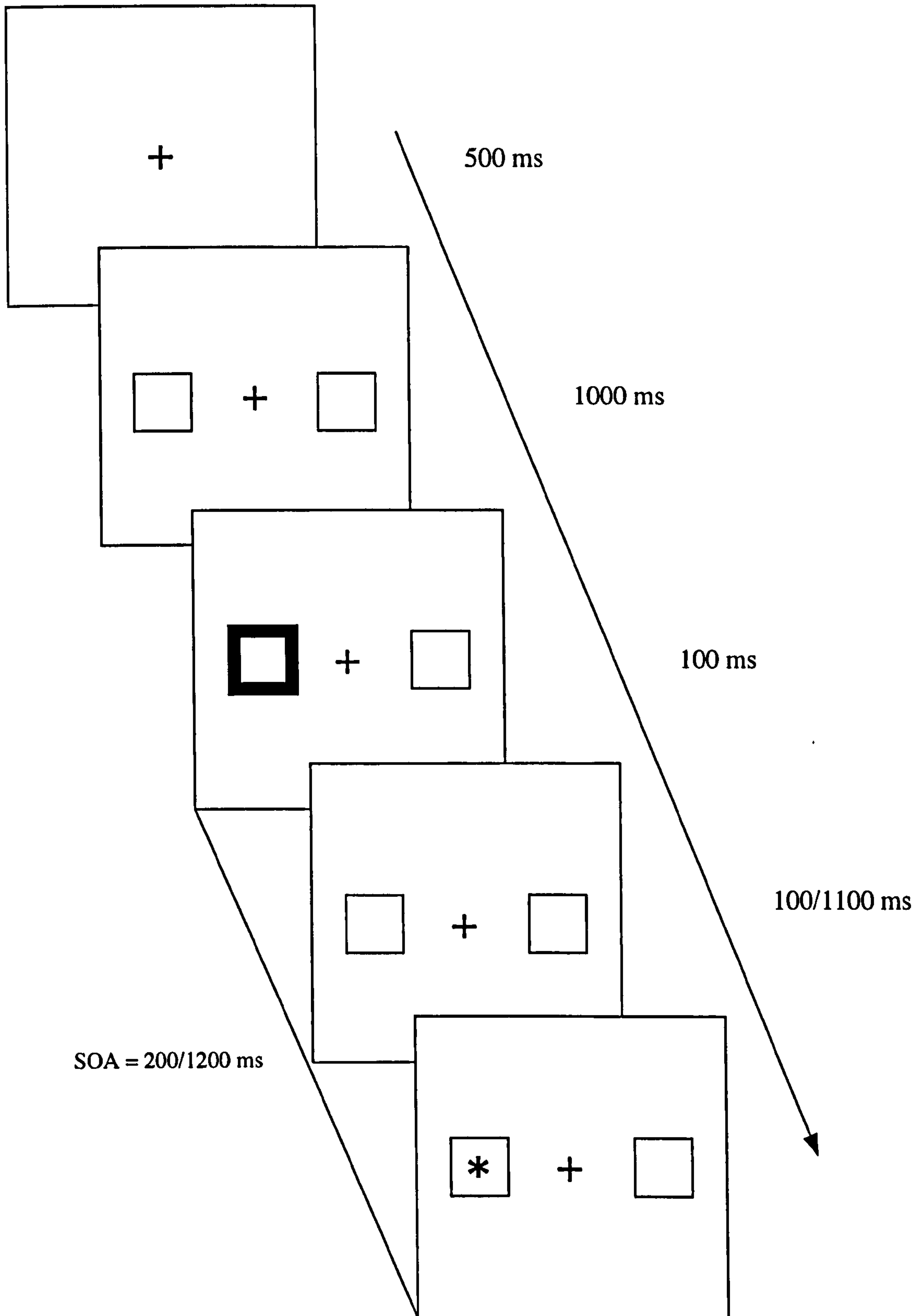
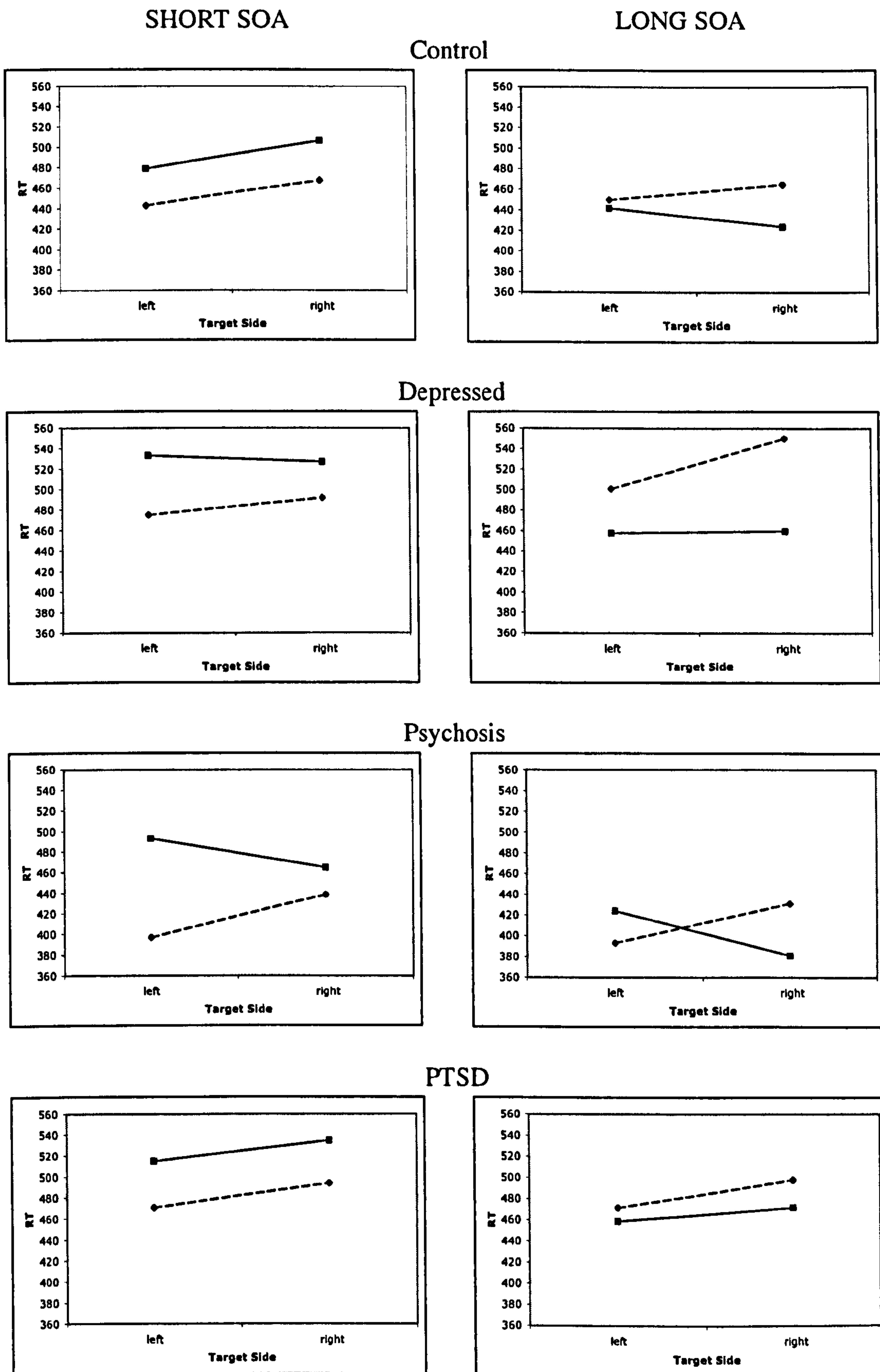


Figure 3.2. Mean overall RT (ms) for Experiment 1 at the short (200 ms) and long (1200 ms) SOA in the four subgroups



Legend: _____ Uncued, - - - - - Cued

Figure 3.3. Procedure used in Experiment 2. Example of a validly cued trial with the angry expression in which the target appears in the left visual field.

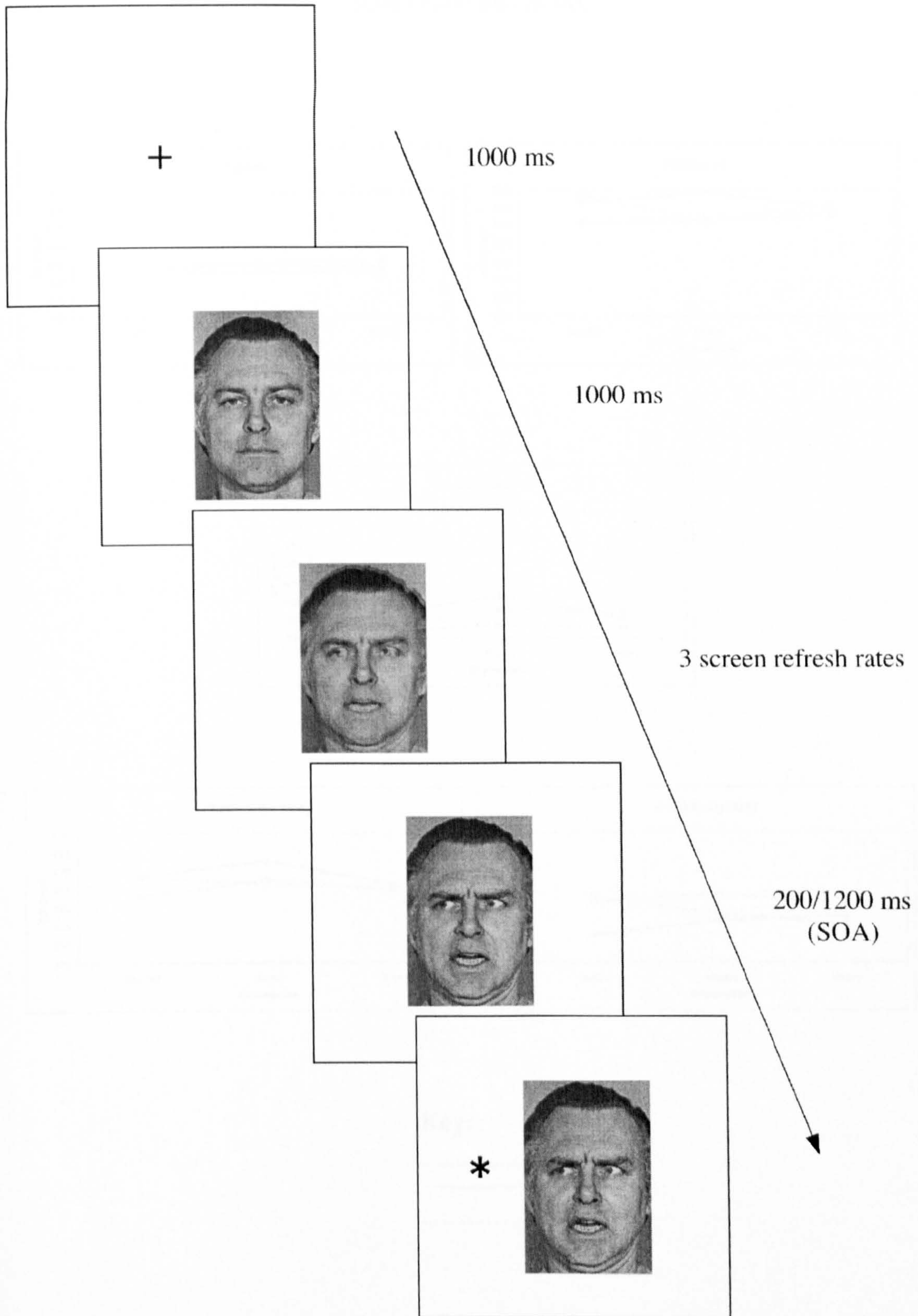
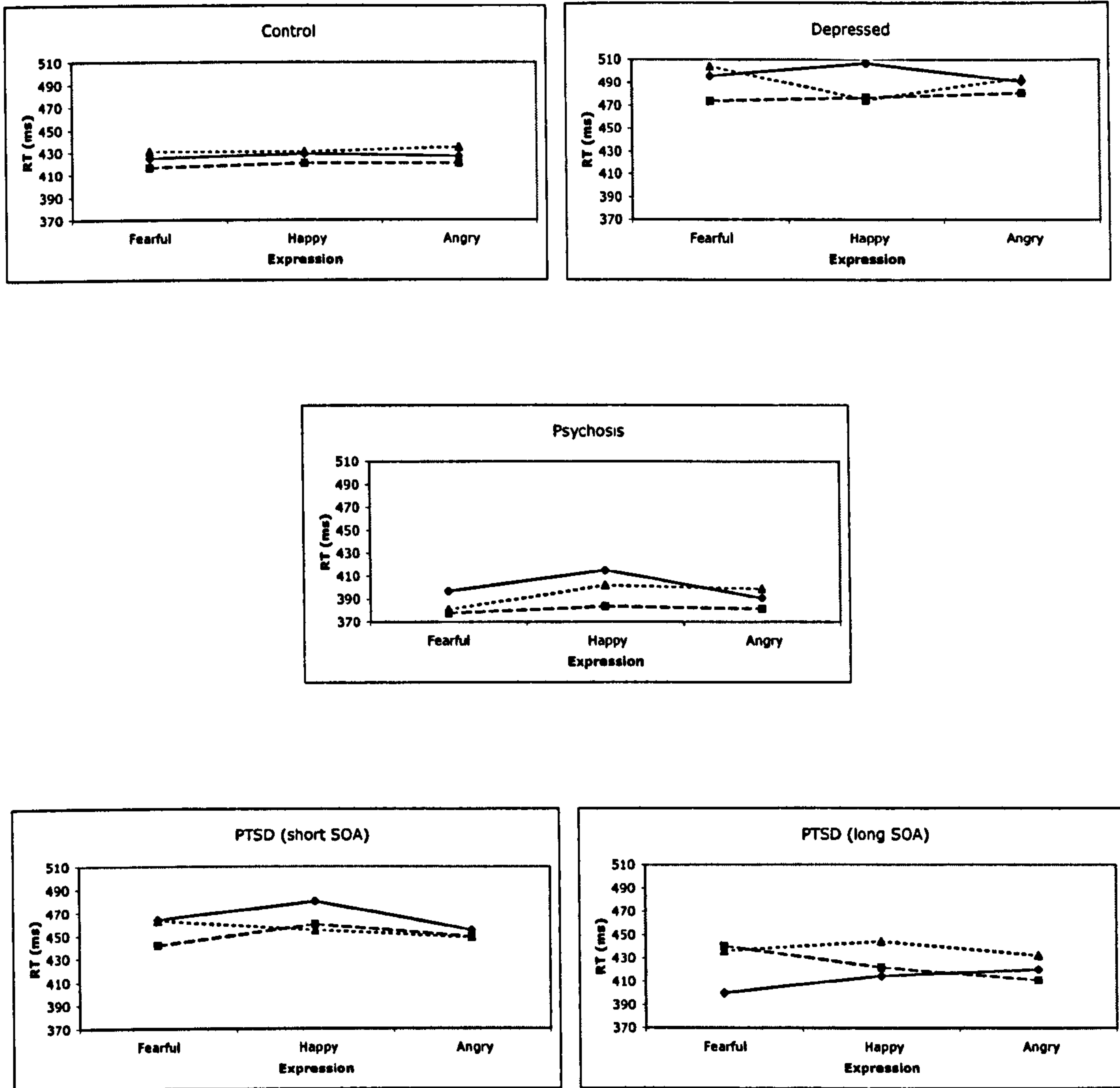


Figure 3.4. Mean RT (ms) for Cues across three Expressions in Control, Depressed and Psychosis groups, and in the PTSD group at the short (200 ms) and long (1200 ms) SOAs



Key:

- Uncued
- - - - - Cued
- Neutral

Appendix J

Appendix J: Total scores for questionnaires

S. No.	BDI	BAI	DES	PSS Tot	PSS R	PSS A	PSS H	IES Tot	IES I	IES A
1	24	27	7	37	8	17	12	43	25	18
2	31	37	75	30	12	9	9	30	9	21
3	40	24	10	40	13	18	9	60	27	33
4	34	26	48	13	3	5	5	37	19	18
5	10	14	15	14	3	5	6	42	17	25
6	12	4	8	11	0	9	2	43	20	23
7	20	25	15	43	15	16	12	47	27	20
8	54	42	23	45	9	21	15	71	33	38
9	24	31	16	19	7	3	9	34	19	15
10	39	32	21	35	8	12	15	27	15	12
11	57	42	69	41	11	20	10	69	31	38
12	41	46	32	41	15	12	14	46	27	19
13	37	39	23	35	7	17	11	60	24	36
14	13	10	8	23	6	13	4	42	19	23
15	19	27	35	23	8	5	10	44	26	18
16	34	14	23	25	2	15	8	57	25	32
17	6	6	9	0	0	0	0	0	0	0
18	2	0	3	5	0	5	0	1	0	1
19	45	50	44	39	15	9	15	52	20	32
36	32	24		36	4	18	14			
20	4	3		0	0	0	0			
21	2	8		2	2	0	0			
22	3	4		8	2	0	6			
23	6	6		0	0	0	0			
24	12	10		8	1	4	3			
25	7	1		8	0	5	3			
26	12	7		6	5	0	1			
27	4	1		2	1	0	1			
28	9	17		9	0	5	4			
29	20	7		15	3	2	10			
30	0	6		0	0	0	0			
31	0	0		4	1	3	0			
32	3	1		0	0	0	0			
33	1	0		0	0	0	0			
34	8	23		11	2	4	5			
35	1	3		2	2	0	0			

Appendix K

Appendix K (prescribed medication)

PTSD
<p>Analgesics: Co-codamol, aspirin, Fortral, Pentazocine, & anti-inflammatories Antidepressants (Mirtazepine) and sleepers Selective serotonin and noradrenaline reuptake inhibitor: Venlafaxine, SSRI: Cipralex, escitalopram Atypical antipsychotic: Risperidone,</p>
Psychosis
<p>Antipsychotic: Trifluoperazine, Atypical Antipsychotics: Risperidone, Olanzapine, Clozapine, Zuclopenthixol decanoate, Amisulpride Sleeping tablets: Analgesics: Tramedol Antidepressants: Venlafaxine Antimanic: Lithium, Carbamazepine Epilepsy: Sodium valproate, Thyroid hormone (thyroxine sodium) Anxiolytic: Diazepam</p>
Depressed
<p>SSRIs: Paroxetine, Fluoxetine Antidepressants (tricyclic): Amitryptiline, Dothiepin hydrochloride Atypical Antipsychotic; Seroquel Sleeper: Nitrazepam Anxiolytic: Diazepam Antibiotics & Analgesics: Aspirin, Zolmitriptan</p>

Appendix L

Appendix L. Percentage error rates and significant differences in Experiment 1 for the four groups.

	Healthy Control (n = 16)	Psychosis (n = 7)	Depressed (n = 7)	PTSD (n = 5)	F	p
Anticipations	.2%	0	.5%	.5%	1.613	ns
Late	3%	2.5%	6.1%	10.2%abc	2.806	.056
Incorrect	.3%	.2%	.3%	1.7%abc	2.496	.078
Total	3%	2.5%	6.4%	12.5%	2.130	<.05

Df = 3 and 31 (except total df = 9 and 73 using Wilks' Lambda)

a = significantly different from healthy control; b = significantly different from Psychosis; c = significantly different from Depressed.

Experiment 1. Error Rate Analyses

Error rates were calculated by summing across conditions according to error type (anticipations, late responses, and incorrect responses) for each participant¹. The scores were analysed using a 4x3 ANOVA, with 4 groups (Control, PTSD, Psychosis and Depressed) as a between subject factor and 3 error types as a within-subject factor. There was no difference between the groups on rate of anticipations, $F(3,32) = 1.613$ (ns). The PTSD group made more incorrect responses and more late responses than the other groups. Late responses indicated that the PTSD group was slower than the other groups. One PTSD participant in particular made a high number of incorrect responses (17.5%) and was excluded from further analyses on this task. Once having

¹ The overall rate of late responses was higher than usual in this type of task. This is probably because of a rather tight cut-off for late responses of 800 ms, which is more suitable for healthy control participants. Follow up work should use a later cut-off.

excluded the participant the multivariate comparison of the overall error rate was significant using the Wilks' Lambda correction, $F(9,73) = 2.130$ ($p < .05$) although univariate analyses showed that the groups did not significantly differ on Late Responses, $F(3,31) = 2.496$ ($p = .078$) or Incorrect Responses, $F(3,31) = 2.806$ ($p = .056$). Pairwise comparisons showed that the PTSD group made more late responses and more incorrect responses than the other three groups, who did not differ.

The pattern of errors was also analysed using a 4x2x2x2 ANOVA with group as a between subject factor and SOA, target side and cue side as within subject factors on total errors (summing across the three error types). Errors were more common at the short SOA than the long, $F(1,31) = 8.843$ ($p < .01$), and when targets appeared on the right rather than the left, $F(1,31) = 5.613$ ($p < .05$). The SOA x cue side x group interaction was significant, $F(3,31) = 4.666$ ($p < .01$), as were the SOA x target side, $F(1,31) = 4.142$ ($p < .05$), target side x cue side, $F(1,31) = 4.736$ ($p < .05$), cue side x target side x group, $F(3,31) = 3.874$ ($p < .05$), and the SOA x cue side x target side x group interactions, $F(3,31) = 4.780$ ($p < .01$). A series of ANOVAs were conducted on the subgroups which showed no significant effects in the Healthy control, Psychosis or PTSD groups, and a main effect of target side in the Depressed group ($p < .05$), who also showed a SOA x cue side interaction ($p < .05$), in which at the short SOA there were more errors following cues to the right and more errors following cues to the left at the long SOA.

Appendix M

Appendix M. Percentage error rates and significant differences in Experiment 2 for the four groups.

	Healthy Control (n = 16)	Depressed (n = 6)	Psychosis (n = 6)	PTSD (n = 5)	F	p
Anticipations	.1%	.05%	0	.4%	2.731	ns
Late	1.7%	3.1%	2.2%	6.4%	1.654	ns
Incorrect	.4%	.5%	.4%	.5%	.160	ns
Total	2.2%	3.5%	2.6%	7.4%	2.133	ns

Df = 3 and 29

Experiment 2. Error Rate Analysis

There were two clear outliers who scored highly on overall errors (over 36%), but especially incorrect responses (over 11%), which were otherwise rare. Both participants were excluded from analysis of this task, one from the Depressed group and one from the PTSD group. The remaining error rates were analysed using a multivariate 4 x 3 ANOVA with group as a between subject factor and error type (anticipations, incorrect and late responses) as a within subject factor. Mean percentages are shown in Table 3. There was a main effect of error type, $F(2,28) = 14.910$ ($p < .001$). Pairwise comparisons showed that the most frequent error type was late responses, and the least frequent was anticipations, and all differences were significantly different at $p < .01$ using the Bonferroni correction for multiple comparisons. However, the rate of errors did not differ between groups, and there were no significant interactions. One participant in the PTSD group made a high rate of slow responses, which had the effect of increasing the mean and SD, but in other respects this participant performed well. As in Experiment 1, it is likely that the cut-off point was too stringent for the older participants in the PTSD group. The

participant was not excluded from analyses of RTs at this stage, on the grounds that there were sufficient scores retained to produce reliable RTs.

The pattern of errors was also analysed using a 4x3x2x3x2 ANOVA with group as a between subject factor, validity, SOA, expression and target side as within subject factors. There were no main effects, but there was an SOA x expression interaction, $F(2,49) = 5.541$ ($p < .01$), and comparison of means indicated that most errors occurred at the short SOA with the fearful expression and least occurred at the long SOA with this expression, whereas the other expressions were less affected by SOA. There were interactions of validity x SOA x group, $F(6,58) = 2.795$ ($p < .05$), validity x expression x group, $F(10,98) = 2.113$ ($p < .05$), and validity x SOA x expression x group, $F(11,104) = 2.312$ ($p < .05$). The control group showed no significant differences in error rate across condition except in validity x SOA, $F(2,30) = 3.699$ ($p < .05$) and validity x SOA x expression x target side interactions, $F(2,37) = 3.921$ ($p < .05$). Neither the depressed nor PTSD groups showed a significant pattern of errors. The psychosis group showed an interaction of validity and expression, $F(2,11) = 5.152$ ($p < .05$). Scrutiny of the means indicated that errors in this group were lowest when the fearful face was an invalid cue (0), and highest when it was a valid cue (.1%)

The increased level of errors in the Psychosis group when the fearful face was a valid rather than an invalid cue is an odd result, and probably spurious due to the very small numbers involved. This does not affect the results from RTs, which were based on larger samples.

Appendix N

3rd party copyright material excluded from digitised thesis.

Please refer to the original text to see this material.

Section 4

Contributions to Theory, Practice and Learning

Contributions to Theory, Clinical Practice, and Learning

Implications for future research and theory development

The initial aim of this research project was to find a behavioural measure of hypervigilance in posttraumatic stress disorder (PTSD). Previous research had established that people with PTSD show particular sensitivity to threat words in experimental paradigms such as the modified Stroop (e.g. Buckley, Blanchard & Neill, 2000). However, in such tasks, the threat words used tended to be matched to the nature of the trauma. It seemed to be a worthwhile goal to try to find a task that differentiated participants with PTSD from other traumatized participants on the basis of hypervigilance. This is because unlike most other disorders, for a number of reasons, some traumatized individuals are motivated to over-report symptoms (Kimbrell & Freeman, 2003). The behavioural assessment of hypervigilance is more reliable than a self-report measure (Buckley et al., 2000), and could be used both to objectively assess the extent of the difficulty, and possibly also as an outcome measure as symptoms ameliorated.

The inclusion of psychosis as a traumatized group is possibly controversial, although there is a growing recognition of the relevance of trauma to this group (e.g., Mueser, Rosenberg, Goodman & Trumbetta, 2002). Trauma can be a precursor to psychosis, and can also result from being seriously mentally ill. Structurally, the brains of people with schizophrenia, PTSD or depression all look similar, with decreased hippocampal volume. This could reflect a biological vulnerability to mental illness, or result from the action of stress hormones, glucocorticoids (Horner & Hamner, 2002). This similarity would suggest that there is little difference in brain processes available to patients with these different disorders.

The fact that there are different psychiatric syndromes, albeit overlapping, implies a difference in brain function that is not dependent on structure. It is possible that the form of the disorder might relate to underlying metacognitive and metaphysical beliefs and assumptions. Someone high in schizotypy might tend to attribute hallucinatory symptoms externally, and therefore does not link them to their trauma history, so they develop delusions about their cause. Schizophrenia can then develop as they try to manage their symptoms and fears. On the other hand, someone with a strong belief that they need to be strong and in control of themselves at all times might well recognize that flashbacks relate to a previous trauma, but believe they should be able to forget it, and so develop PTSD in a similar way, as they attempt to manage their symptoms inappropriately¹.

In both schizophrenia and PTSD there is a sense of current threat, although someone with PTSD will know consciously that the threat has passed, the content of the schizophrenic's delusions usually mean the threat is ongoing, unless they are doing well in therapy. Taking this viewpoint, a more appropriate measure for research purposes than the commonly used 'time since initial trauma' might be 'time since last trauma', as this, along with measures of symptom severity, is more likely to indicate the extent of recovery.

Unfortunately, a main limitation of the present study is that the findings cannot point to the cause of any differences between clinical groups. Differences may reflect biological vulnerabilities, or acquired changes in functioning. It is not possible to relate the findings to trauma. This is particularly the case with the Psychosis group, in which, in this group of patients, the disorder preceded the trauma.

¹ These ideas are based on a conference presentation by Craig Steele (2005)

If there were enough participants, in future, it would be interesting to develop norms on the basic IOR, and an emotional cue version for clinical use. An improvement to consider might be to use faces as peripheral cues, as in the design by Poy, del Carmen Eixarch, & Avila (2004). According to their theory, people who are trait anxious process the face cues with an anterior attentional system, whereas healthy adults use the posterior system (Posner & Cohen, 1984). However, this theory is based on developmental motivational theories in which differential sensitivity to reward and punishment affects the path of early development. It is not clear at present how somebody with PTSD would respond to peripheral face cues. The evidence from the present study suggests that they would also use the anterior system. This would not in itself differentiate people with PTSD from those who are high in trait anxiety, but a longitudinal study would do so, as the trait anxious would not be expected to change.

The small sample sizes make any conclusions from the present research very tentative. However, it does appear that the three patient groups did differ from each other and from the control participants. Future research would need to improve the study by extending the RT cut-off from 800 ms. The number of trials in the face version of the task could be reduced, perhaps by excluding the angry expression, as the fearful expression showed more effects.

Research on PTSD is difficult to conduct for a number of reasons. Horner and Hamner (2002) reviewed neurocognitive functioning in PTSD and noted that it is very hard to recruit participants with pure PTSD, as most have psychiatric comorbidity. They suggested PTSD may be associated with pre-morbid ADHD, and studies would improve if they were to assess for this. Also, the prevalence of comorbid alcoholism is between 24-84%, and depression 28-84% (Keane & Kaloupek, 1997, cited by

Gilbertson, Gurvits, Lasko et al., 2001). It is possible to be more selective about recruiting participants with PTSD, but important findings may then be missed, for example, if depression is part of PTSD symptomatology, or substance abuse is a common outcome of PTSD. It is also difficult to draw firm conclusions about the effects of PTSD because of differences in power, methodology, trauma etiology, and chronicity between the studies that have been conducted. The present research would have been improved by having more homogenous groups, who were unmedicated, and age matched. Participants were asked about substance use and dependence, and handedness but assessment with standardized scales might have improved the validity of those assessments. The use of a dedicated laboratory to which participants could have been brought would have reduced the possibility of confounding variables affecting the data.

Eye-movements were not assessed in this study, and may have contributed to some of the group differences observed (e.g. Broomfield & Turpin, 2005). The lack of IOR in the Control group at the long SOA in the left visual field might have been related to the control participants not listening to the instructions, not maintaining fixation, and trying to predict where the target was going to appear.

It is unfortunate that the small sample sizes constrain the confidence with which the findings can be expected to generalize, but they appear to provide an interesting starting point for further research. In addition, it was not possible to assess the impact of any demographic or questionnaire information on scores, for example, by using regression analyses, because of the small sample sizes. It might also have been interesting to look at practice effects within the emotional cueing task, which might reflect repeated exposure to threat stimuli, as this may be more applicable to normal living (Brewin & Holmes, 2003).

The present study would have been improved if there had been enough time and participants to conduct a pilot study. This might have enabled detection of the time limit on responses that impeded data collection in some of the older PTSD participants in particular. The original design used a traumatized control group rather than separating these into a traumatized control group and a Psychosis group. It was felt that these were too dissimilar to keep in one group, although the Psychosis group do also provide an interesting comparison to the PTSD participants.

Regarding use of the IOR task in clinical groups, it is interesting to see how varied the abnormal results on this apparently straightforward task can be. The interpretation of behavioural findings is informed by an understanding of the neural mechanisms underlying the effect, and reciprocally informs understanding of these mechanisms. Neuroimaging techniques are important to establish how attention processes really work, as behavioural measures are limited on their own. In conducting this research, there is a need to consider all aspects of the specific IOR task very carefully, as there are many variants of the task that can have unexpected effects. The IOR task would work better as a tool if there were standards for stimulus presentation and norms.

Implications for clinical practice

Assuming that performance on the selective attention task reflects general cognitive patterns, awareness of the cognitive pattern of a client is important in terms of interacting with the client and in terms of formulation. A client with psychosis may have a lot of difficulty following a train of thought or maintaining attention in a session. A client with PTSD may also have difficulty concentrating. In both cases, communication with the client will be compromised. The psychotic client may focus on a word, and not inhibit alternative word meanings and become distracted by what

would appear to be irrelevancies. The PTSD client may similarly focus on a word and fail to process the rest of the sentence. In both cases, repetition is likely to help. The psychotic client might benefit from clarification. The PTSD client might need information to be given slowly, to enable them to disengage from any irrelevant stimulus that has captured attention. The Depressed group was very sensitive to cues. These clients may also be sensitive to nuances in the clinician's demeanor.

In addition to considering how the information processing deficits might impact on the client's ability to cope with cognitive therapy, it might be worth considering the extent to which the information processing deficit represents a symptom to target through 'rehabilitation', perhaps in the form of practice and with psychoeducation. This is one way of describing what happens during therapeutic interventions such as eye movement desensitization and reprocessing (EMDR; Shapiro, 1995), and exposure work (e.g. Foa, Riggs, Massie & Yarczower, 1995), both evidence-based treatments.

Process/Personal Issues Arising from the Conduct of the Research

Motivation & Support. As mentioned above, the initial incentive for starting this research was to find a measure of hypervigilance. This arose from attending a talk on the topic by Matt Kimble, then a lecturer in the department, and it was a bonus to find he was happy to supervise the project. Steve Tipper was also approached to be a supervisor, as an expert in selective attention with whom the author had previously worked for many years. Having been employed previously as a researcher it seemed to be a luxury to be able to work on a project of the author's own choosing.

Nevertheless, it soon became apparent that the project was too ambitious, and a lesser goal would be more appropriate. It might have been useful to look at the abstracts of previous large-scale research projects (LSRPs) to get a sense of their scope.

It was a surprise when Matt Kimble said he was returning to the USA.

Another supervisor, Paul Gardner, was recruited for clinical emergencies as part of the conditions for ethical approval. In fact, plenty of support was available when needed, but there were times it seemed that none of it could be counted on, especially given the author's 'last minute' style of working.

Writing the review of IOR was quite enjoyable once a structure had been decided on, and it is intended that a version of this paper will be published. It also seems that there is a lot of scope for developing the IOR as a clinical test, even if not as a test of hypervigilance. The results from the emotional cueing task are also very promising and it would be worth trying to develop this work at a future date.

Time Management. The preparation for the study took place in a rush: producing a proposal, then sending it through a number of ethics and research committees, without the opportunity to really look into the field enough, nor to establish feasibility, e.g. to check the size of the computer that was to be carried around, or the availability of a laptop. It was a shame that the time and effort involved in smoothing the path of the proposal through the committees impacted heavily on the time available for more practical preparations for the project, especially as this took place during the second year of the course, when there was little time allocated to the LSRP.

A lot of committees met monthly, and needed to receive documents two weeks earlier than that, and periodically took a month off. Managing in this system required forward planning. Similarly, when visiting Community Mental Health Teams (CMHTs) there were issues about attending busy monthly meetings when the agendas were already full.

Some of the time spent was on work that does not appear anywhere in this report, such as getting the equipment portable appliance tested in order to conform with the Trust requirements, and attending CMHT meetings to try to recruit participants. It was often difficult to balance the relative demands of different aspects of the project, most especially during data collection when an effort was made to increase participant numbers, even though in retrospect the time might have been better spent writing.

Communication. The author did not know how the Conwy & Denbighshire Trust was organized or how to set about recruiting there. When e-mails were not answered, the assumption was made that the trust was much like the local trust, and that participants for the study could be recruited in the same way when the time came. In fact, there were problems in the Trust of which the author was unaware. This proved to be a problem later when it came to data collection.

Only when it was too late did it become apparent that there was a trauma center in the North East Wales trust (pointed out by a former trainee). Also, that in the local trust primary care counselors saw the majority of the PTSD clients (pointed out by a CMHT member). Without appropriate ethical approval it was not possible to make use of these sources. It is unfortunate that nobody involved in the clinical training was in a position to know about these sources, as it would have improved the project enormously to have a reliable source of suitable participants. The inclusion of so many participants that it had been intended to exclude complicated the presentation of the project both in terms of explaining why they were included, and interpreting the results.

Discomfort. Some of the potential recruiters for the project expressed reluctance to ask their clients to take part. This seemed to be because they felt

protective towards them and were concerned about asking them to do something out of the ordinary.

Sometimes the recruiters made recommendations, for example, about seeing a client on a particular day, which were difficult to meet. Constraints on the researcher's time concerned use of study days, and the availability of a room, the client, and the equipment, which was shared. It was not easy to accommodate any other constraints and it seemed that by trying to come across in an approachable way to the recruiters, the researcher might also have seemed to be more relaxed and carefree than she really was.

The protocol and information sheets stated that participants would not need to talk about their traumatic experiences. Nevertheless, a lot of the participants in all groups did talk about them, to the extent that the sessions often went over time. This was a little difficult to handle, as the researcher did not want to seem disinterested, but also needed to keep focused on the information required, and not function as a therapist. In most cases questions were not used to prompt further disclosure, but this often seemed to help them to open up more. However, in no case was it considered that the participant had been negatively affected by talking about a traumatic experience. It is probable that the participants who are prepared to take part in a study of trauma are those who are comfortable enough about their experiences to be willing to talk about them.

There were concerns about how participants might be affected by doing the research. When planning the study, it was not expected that anyone would be affected. While conducting it, it was less clear. EMDR became more familiar, which involves desensitization by talking about a trauma while moving the eyes back and forward to stimulate different sides of the brain. As study participants were often

talking about trauma, and then sitting watching left and right cues, it did not seem so very dissimilar. However, there was no evidence that participants were made either better or worse by taking part.

The 800 ms cut off for RTs was also a cause for concern. While most participants were unaffected, some of them did have a high number of responses exceeding the cut-off. It is difficult to know how this might have affected their mood or confidence. There was an error tone on each late response, but individual participants do not know how their performance compares with anyone else's, so they will not necessarily interpret this negatively. During debriefing they were asked how they had found the tasks, and care was taken not to imply they had performed badly in any way. There has been no indication that anyone found participating in the experiment a negative experience.

References

Brewin, C. R., & Holmes, E. A. (2003). Psychological theories of posttraumatic stress disorder. *Clinical Psychology Review, 23*, 339-376.

Broomfield, N. M., & Turpin, G. (2005). Covert and overt attention in trait anxiety: a cognitive psychophysiological analysis. *Biological Psychology, 68*, 179-200.

Buckley, T. C., Blanchard, E. B., & Neill, W. T. (2000). Information processing and PTSD: A review of the empirical literature. *Clinical Psychology Review, 20*, 1041-1065.

Kimbrell, T. A., & Freeman, T. W. (2003). Clinical care of veterans seeking compensation (letter). *Psychiatric Services, 54*, 6.

Foa, E. B., Riggs, D. S., Massie, E. D., & Yarczower, M. (1995). The impact of fear activation and anger on the efficacy of exposure treatment for posttraumatic stress disorder. *Behavior Therapy, 26*, 487-499.

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*, 413-432.

Horner, M. D., & Hamner, M. B. (2002). Neurocognitive functioning in posttraumatic stress disorder. *Neuropsychology Review, 12*, 15-30.

Mueser, K. T., Rosenberg, S. D., Goodman, L. A., & Trumbetta, S. L. (2002). Trauma, PTSD, and the course of severe mental illness: an interactive model. *Schizophrenia Research, 53*, 123-143.

Posner, M. I., & Cohen Y. (1984). Components of visual orienting. In H. Bouma, & D. Bouwhuis (Eds.), *Attention and Performance X* (pp. 55-66). Hillsdale, NJ: Erlbaum.

Poy, R., del Carmen Eixarch, M., Avila, C. (2004). On the relationship between attention and personality: Covert visual orienting of attention in anxiety and impulsivity. *Personality and Individual Differences*, 36, 1471-1481.

Shapiro, F. (1995). *Eye movement desensitization and reprocessing*. Guilford: NY.

Appendix O

Statement of Word Count

Abstract	290
Section 1	
COREC Form (online version: unknown)	-
C&D Risk Checklist	366
Section 2	4937
Section 3	6377
Section 4	2920
Total:	14,890

Appendices

Section 1	
A Printed tests (unknown)	-
PSS-I	378
Patient Information sheet (English)	991
Recruiter Information sheet	867
Consent form	153
H Changes to Protocol	188
Section 2	
References	983
Table 2.1	366
Section 3	
References	1242
Tables 3.1-3.3	464
Figures 3.1-3.4	187
J Total Scores for Questionnaires	353
K Prescribed Medication	79
L Error Rate Analyses (Experiment 1)	513
M Error Rate Analyses (Experiment 2)	548
Section 4	
References	273

Total: 2585

Grand Total: 22,475