HEMISPHERIC ASYMMETRY AND INFORMATION PROCESSING IN POST-TRAUMATIC STRESS DISORDER

Thesis submitted for the degree of
Doctor of Philosophy

Therese Mayo

The University of Adelaide
School of Population Health and Clinical Practice

March 2008
Table of Contents

List of Tables ......................................................................................................................... ix
List of Figures........................................................................................................................ xi
Abstract ................................................................................................................................ xiii
Declaration ........................................................................................................................... xv
Acknowledgements ............................................................................................................. xvi

1. Overview and Outline of the Thesis ............................................................................ 1
   1.1. General Introduction ............................................................................................... 1
   1.2. Aims of the Study .................................................................................................... 1
   1.3. Significance of the Study ........................................................................................ 3
   1.4. Overview of the Thesis ........................................................................................... 4

2. Literature Review: Learning and Memory in PTSD Research ............................... 7
   2.1. Introduction ............................................................................................................. 7
       2.1.1. Focus of the Literature Review ........................................................................ 8
       2.1.2. PTSD Symptoms and Memory Functioning .................................................... 8
   2.2. The Phenomenology of PTSD and Information Processing Models ................. 11
       2.2.1. Synaptic Plasticity as an Adaptive Mechanism ............................................. 15
       2.2.2. Abnormal Tonic States in PTSD .................................................................... 17
   2.3. Distributed Parallel Cortical Systems in PTSD ..................................................... 20
       2.3.1. Summary: Stress Reactions and Memory Functioning .................................. 21

3. Empirical Findings in PTSD and Literature Review Synthesis ............................. 23
   3.1. Brain Organisation: Function and Structure .......................................................... 23
       3.1.1. Lateralisation Hypothesis in PTSD Research ................................................ 25
       3.1.2. Specialisation Theories in Cognition ............................................................. 30
       3.1.3. Microcircuitry in Cognition: Spatial and Temporal Coding .......................... 32
   3.2. Executive Functions and Attention Modulation .................................................... 37
       3.2.1. Link between Neuroanatomical and Neuropsychological Functions ............. 38
       3.2.2. Fear and Knowledge Structures .................................................................. 43
       3.2.3. Right-lateralised Effects of Neurochemical Pathways .................................. 46
       3.2.4. The Secondary Effects of Shared Resources ............................................... 50
       3.2.5. Functional Reorganisation in PTSD ............................................................. 52
   3.3. Compensatory Mechanisms in PTSD ................................................................. 54
       3.3.1. Summary: Functional Cerebral Asymmetry ................................................. 58

4. Objectives and General Methodology ...................................................................... 61
   4.1. Objectives and Scope of Study .............................................................................. 61
4.2. General Methodology ............................................................................................. 62
  4.2.1. Standardised Measures and Procedures .......................................................... 63

4.3. Participants ............................................................................................................. 63
  4.3.1. Inclusion and Exclusion Criteria for Non-Clinical Participants ..................... 63
  4.3.2. Standardised Inclusion and Exclusion Criteria for PTSD ............................... 63
  4.3.3. Characteristics of Participant Sample ............................................................. 66

4.4. Materials and Procedures ....................................................................................... 67
  4.4.1. Web Questionnaire .......................................................................................... 67
    4.4.1.1. Trauma Experience Questionnaire ........................................................... 68
    4.4.1.2. Mood States ............................................................................................. 69
  4.4.2. Electrophysiological Data Collection and Reduction ..................................... 70
    4.4.2.1. Quantitative Electroencephalographic Recordings ................................ 70
  4.4.3. Cognitive Measures ......................................................................................... 72
    4.4.3.1. The Motor Tapping Test .......................................................................... 72
    4.4.3.2. Verbal Fluency Task ................................................................................ 73
    4.4.3.3. The Maze Task ......................................................................................... 74

4.5. Data Analysis ......................................................................................................... 75
  4.5.1. Design of the study ......................................................................................... 75
  4.5.2. Statistics .......................................................................................................... 76

5. Preliminary Investigations of Lateralised Functioning in PTSD............................ 77
  5.1. Rationale ................................................................................................................ 77
  5.2. Study 1: Attentional Capacity for Memory and Retrieval Processes ............ 78
    5.2.1. Introduction ..................................................................................................... 78
    5.2.2. Method ............................................................................................................ 79
      5.2.2.1. Participants ............................................................................................... 79
      5.2.2.2. Instruments and Procedure ....................................................................... 79
      5.2.2.3. Behavioural Measures .............................................................................. 80
    5.2.3. Data Analysis .................................................................................................. 80
    5.2.4. Results ............................................................................................................. 81
      5.2.4.1. Verbal Fluency Task ................................................................................ 81
      5.2.4.2. Summary: Retrieval of Verbal Information ............................................. 81
      5.2.4.3. Executive Maze Task ............................................................................... 82
      5.2.4.4. Summary: Retrieval of Visuospatial Information .................................... 82
      5.2.4.5. Sensori-Motor Tapping Task ................................................................... 83
      5.2.4.6. Summary: Tapping Task and Speed of Processing .................................. 83
    5.2.5. Discussion ....................................................................................................... 83
5.3. **Study 2: Baseline Cortical Arousal**

5.3.1. Introduction

5.3.2. Method

5.3.2.1. Participants

5.3.2.2. Instruments and Procedure

5.3.3. Data Analysis

5.3.4. Results

5.3.4.1. Alpha Peak Frequency Patterns

5.3.4.2. Summary: Alpha Peak Frequency

5.3.4.3. Spectral Power qEEG Characteristics

5.3.4.4. Summary: qEEG Recordings

5.3.4.5. Alpha Frequency

5.3.4.6. Summary: Alpha Spectral Power

5.3.5. Discussion

5.4. **Study 3: Relationship between Memory Retrieval Patterns and Baseline Rhythms**

5.4.1. Introduction

5.4.2. Method

5.4.2.1. Participants

5.4.2.2. Instruments and Procedure

5.4.2.3. Data Analysis

5.4.3. Results

5.4.3.1. Alpha Peak Frequency and Behavioural Data

5.4.3.2. Spectral Power and Behavioural Data

5.4.3.3. Asymmetry and Cognitive Performance

5.4.3.4. Summary: Correlation Patterns - Resting EEG and Cognitive Performance

5.4.4. Discussion

5.5. **General Discussion and Conclusions**

5.5.1. Attention and Self-regulation Associated with Local Processing Structures

5.5.2. Speed of Processing in PTSD

5.5.3. Rehearsal Mechanisms and the Capacity for Intentional Behaviour

5.5.4. Plasticity as a Compensatory Mechanism

5.5.5. Limitations

6. **Reactions to Stress and Resting EEG Patterns**

6.1. Rationale
7. Neuropsychological and Electrophysiological Asymmetry Patterns in PTSD ... 201

7.1. Rationale ......................................................................................................................... 201

7.2. Study 1: Behavioural Tasks and Local Processing Networks .................................. 202

7.2.1. Introduction ............................................................................................................... 202

7.2.2. Method ....................................................................................................................... 203

7.2.2.1. Participants ........................................................................................................... 203

7.2.3. Instruments and Procedures ...................................................................................... 204

7.2.3.1. Modality-specific Cognitive Tasks .................................................................... 204

7.2.3.2. Resting Electrophysiological Measures .......................................................... 204

7.2.4. Data Analysis ........................................................................................................... 204

7.2.5. Results ....................................................................................................................... 205

7.2.5.1. Verbal Performance Measures ......................................................................... 205

7.2.5.2. Nonverbal Performance Measures .................................................................... 206

7.2.5.3. Region-specific Spectral Alpha Amplitude Activity ...................................... 207

7.2.5.4. Summary: Cognitive Performance and Alpha Power .................................. 210

7.2.6. Discussion ................................................................................................................. 211

7.3. Study 2: Cortical Asymmetry Patterns and Traumatic Stressor Events .................. 213

7.3.1. Introduction ............................................................................................................... 213

7.3.2. Method ....................................................................................................................... 214

7.3.2.1. Participants ........................................................................................................... 214

7.3.3. Instruments and Procedures ...................................................................................... 215

7.3.3.1. Responses to CIDI Trauma Scales .................................................................... 215

7.3.3.2. Current Mood Scores ....................................................................................... 215

7.3.3.3. Asymmetry Scores ............................................................................................ 215

7.3.4. Data Analysis ........................................................................................................... 217

7.3.5. Results ....................................................................................................................... 217

7.3.6. Discussion ................................................................................................................. 221

7.4. Study 3: Predictor Variables Associated with Cortical Asymmetry Patterns .... 226

7.4.1. Introduction ............................................................................................................... 226

7.4.2. Method ....................................................................................................................... 227

7.4.2.1. Participants ........................................................................................................... 227

7.4.3. Instruments and Procedures ...................................................................................... 227

7.4.3.1. Self-report Responses to Trauma Events ......................................................... 227

7.4.3.2. Modality-specific Cognitive Tasks .................................................................... 228

7.4.3.3. Alpha Asymmetry Index .................................................................................... 228

7.4.4. Data Analysis ........................................................................................................... 228
7.4.5. Results ................................................................................................................................. 229
  7.4.5.1. Numbing and Avoidance as Predictors of Alpha Asymmetry .................. 229
  7.4.5.2. Numbing-avoidance and Cognitive Performance .................................. 234
7.4.6. Discussion ......................................................................................................................... 240
7.5. General Discussion and Conclusions.................................................................................. 243
  7.5.1. Behavioural Performance for Specialised Modality-specific Processes ....... 248
  7.5.2. Alpha Asymmetry Patterns ......................................................................................... 252
  7.5.3. Local Differences for the Organisation of Knowledge Structures ............... 257
  7.5.4. Uncertainty Hypothesis ................................................................................................. 263
8. Overview and General Conclusions ..................................................................................... 271
  8.1. Complex Neural Interactions in PTSD ................................................................. 271
  8.2. Methodological Limitations of the Study ............................................................... 273
  8.3. Implications and Relevance of Findings ................................................................. 285
  8.4. Future Directions ............................................................................................................. 289
    8.4.1. Learning as a Change Process .............................................................................. 290
    8.4.2. The Role of Stress in Memory Structures ......................................................... 291
References ..................................................................................................................................... 295
List of Tables

Table 4.1. Demographic characteristics of participant sample .................................................. 66
Table 5.1. Descriptive data for verbal fluency task: letter and category words ...................... 81
Table 5.2. Descriptive data for maze task – time to completion, average errors and average overruns ............................................................................................................. 82
Table 5.3. Descriptive data for sensori-motor task as number of taps with each hand....... 83
Table 5.4. Results of ANOVA assessing alpha peak frequency effects at midline sites ..... 95
Table 5.5. Results of ANOVA assessing alpha peak frequency inter-hemispheric effects . 96
Table 5.6. Results of ANOVA assessing APF anterior and posterior region effects............. 98
Table 5.7 Results of ANOVA assessing aggregated global power in each frequency band for left and right hemisphere in each of the eyes open and eyes closed conditions between groups............................................................. 104
Table 5.8 Results of simple effects ANOVA assessing delta aggregated power for each hemisphere and in each condition between groups ....................................................... 105
Table 5.9 Results of simple effects ANOVA assessing theta aggregated power for each hemisphere and in each condition between groups....................................................... 106
Table 5.10 Results of simple effects ANOVA assessing alpha aggregated power ............. 106
Table 5.11 Results of simple effects ANOVA assessing beta aggregated power for each hemisphere and in each condition between groups....................................................... 107
Table 5.12 Results of simple effects ANOVA assessing alpha anterior region power for each hemisphere between groups .................................................................................. 109
Table 5.13 Results of ANOVA assessing alpha frequency for each hemisphere in posterior brain regions between groups ................................................................. 111
Table 5.14. Correlation matrix for PTSD participants and control participants showing relationships between anterior and posterior region APF and behavioural indices ...... 121
Table 5.15. Correlation matrix for eyes closed spectral frequencies and behavioural performance measures .................................................................................................................. 123
Table 5.16. Correlation matrix between eyes closed alpha anterior and posterior region asymmetry index and cognitive performance scores ..................................................... 125
Table 6.1. Group descriptor variables ................................................................................ 154
Table 6.2. Self-reported criterion A1 qualifying traumatic events as percentages in each group ........................................................................................................................................ 156
Table 6.3. Summary of most frequently reported events in each group ......................... 157
Table 6.4. DASS descriptive data .............................................................................. 159
Table 6.5. Summary statistics based on self-report items ............................................. 160
Table 6.6. Results of ANOVA assessing sensori-motor task for number of taps with each hand between groups............................................................................................. 161
Table 6.7. Results of ANOVA assessing APF midline and condition effects between groups ................................................................................................................................. 161
Table 6.8. Results of ANOVA assessing global spectral power by each frequency, hemisphere and condition effects between groups..................................................................................... 163
Table 6.9. Descriptive data for left hemisphere total power ............................................. 164
Table 6.10. Descriptive data for right hemisphere total power.................................165
Table 6.11. Results of ANOVA for anterior and posterior regions in global spectral power by frequency band effects between groups.........................................................169
Table 7.1. Verbal fluency descriptive data .................................................................206
Table 7.2. Executive maze task descriptive data .......................................................206
Table 7.3. Results for ANOVA assessing anterior eyes closed alpha power hemisphere and electrode site effects between groups.........................................................207
Table 7.4. Results for ANOVA assessing posterior eyes closed alpha power hemisphere and electrode site effects between groups........................................................209
Table 7.5. Characteristic demographic and mood data for a reduced number of participants......................................................................................................................214
Table 7.6. Self-report responses to CIDI detailing previous trauma experiences assessed by criterion A2 and reaction patterns.................................................................216
Table 7.7. Correlation profile for each group between alpha anterior and posterior asymmetry, avoidance, numbing, and mood states..................................................218
Table 7.8. Regression coefficients: numbing as a predictor of alpha anterior asymmetry.230
Table 7.9. Regression coefficients: avoidance as a predictor of alpha anterior asymmetry......................................................................................................................231
Table 7.10. Regression coefficients: avoidance as a predictor of posterior anterior asymmetry..............................................................................................................233
Table 7.11. Regression coefficients: avoidance as a predictor of verbal fluency (FAS)....235
List of Figures

Figure 2-1. Conceptualisation of attention and memory processes as might apply to models of brain-behaviour functions and PTSD symptoms ................................................................. 22

Figure 3-1. Executive functions have been shown to involve an overlap in cortical processing systems. .......................................................................................................................... 60

Figure 5-1: Results of ANOVA at midline APF eyes closed condition ........................................... 97

Figure 5-2: Results of ANOVA at midline APF eyes open condition ........................................... 97

Figure 5-3. Boxplot of anterior APF means for the eyes closed condition shows a trend toward higher APF in the PTSD group but little variation between groups at each site. ........................................................................................................................................ 100

Figure 5-4. Boxplot of posterior APF means for the eyes closed condition indicates greater variation in the control group for a pattern of higher and lower APF but still within the normal range of 8-13 Hz. .......................................................................................................................... 100

Figure 5-5. Aggregated means for each frequency bandwidth for left hemisphere (eyes closed condition) shows a trend toward variation in the alpha frequency band between groups. .......................................................................................................................... 101

Figure 5-6. Aggregated means for each frequency at right hemisphere (eyes closed condition) shows an overall similar pattern of means between groups but with differences indicated in the alpha frequency band. .......................................................................................................................... 101

Figure 5-7. Boxplot graph showing aggregated means for each frequency for left hemisphere (eyes open condition) indicates a similar pattern of total power for both groups. .......................................................................................................................... 102

Figure 5-8. Aggregated means for each frequency for right hemisphere (eyes open condition) shows little variation in total power between groups. ................................................................. 102

Figure 5-9. Left hemisphere anterior alpha power .............................................................................. 110

Figure 5-10. Right hemisphere anterior alpha power ........................................................................ 110

Figure 5-11. PTSD within-group posterior regions alpha power is significantly different at the T6 (right hemisphere) site for higher power compared to the T5 (left hemisphere) site .......................................................................................................................... 112

Figure 5-12. Control within-group posterior region alpha power indicates greater variability between hemispheres at temporal and parietal sites for higher right hemisphere amplitudes .......................................................................................................................... 112

Figure 6-1. Global power analysis (eyes closed) left hemisphere frequency patterns with confidence intervals .......................................................................................................................... 164

Figure 6-2. Global power analysis (eyes closed) right hemisphere frequency patterns with confidence intervals .......................................................................................................................... 165

Figure 6-3. Total beta power in the eyes closed condition for both left and right hemispheres .......................................................................................................................... 168

Figure 6-4. Total beta power in the eyes open condition for both left and right hemispheres .......................................................................................................................... 168

Figure 6-5. Global power analysis (eyes closed) in frontal regions indicated a significant difference in alpha power in the TEC group for higher amplitudes compared to the PTSD group and the NC group .......................................................................................................................... 171
Figure 6-6. Global power analysis (eyes closed) in posterior regions indicated no significant differences between groups in any of the amplitude frequency patterns. ....171

Figure 7-1. Scatterplot of alpha asymmetry patterns with anxiety items. ...............................219

Figure 7-2. Scatterplot of alpha asymmetry patterns with anxiety items in posterior regions. ...........................................................................................................................................219

Figure 7-3. Scatterplot of alpha asymmetry patterns in anterior regions with depression items. ...........................................................................................................................................220

Figure 7-4. Scatterplot of alpha asymmetry posterior region patterns with depression items. ...........................................................................................................................................220

Figure 7-5. Alpha asymmetry index with high and low numbing regression lines. .......230

Figure 7-6. Anterior alpha asymmetry (AAA) index with high and low avoidance regression lines. ...........................................................................................................................................232

Figure 7-7. Alpha posterior asymmetry with high and low avoidance regression lines. ....233

Figure 7-8. Avoidance as a predictor of verbal fluency (FAS). ...............................................235

Figure 7-9. PTSD model based on numbing as a predictor of alpha anterior asymmetry and the interaction with anxiety for a loss of inhibitory functions associated with incoming stimuli and loss of integrative functions in posterior left hemisphere regions. ...........................................................................................................................................238

Figure 7-10. Model of TEC group data representing avoidance as a predictor of posterior alpha asymmetry and plausible pathways for processing incoming stimuli - emotionally valent and neutral. ...........................................................................................................................................239
Abstract

Previous studies have suggested that mechanisms for neural compensation involve a reorganisation to right hemisphere processing in people with post-traumatic stress disorder (PTSD), and are associated with functional alterations in the capacity for behavioural flexibility. However, research has not established a direct relationship between the complex physiological and psychological processes of the heterogeneous disorder and right hemisphere cortical activity. The present study examined cognitive information processing in people with PTSD, reaction patterns associated with perceived traumatic stressors, and quantitative electroencephalographic (qEEG) indices of hemispheric asymmetry.

Individuals with PTSD (N=34) and age and sex-matched normal controls (N=136) completed standardised web-based self-report questionnaires assessing traumatic stressor events and reaction patterns to those events. Neuropsychological indices of verbal, visuospatial, sensorimotor performance, and electrophysiological recordings, were examined for right hemisphere coding. The relationships among traumatic characteristic reaction patterns of numbing and avoidance, cognitive performance, and frontal and posterior EEG alpha asymmetry were also investigated.

Structural and functional alterations were shown in those with PTSD, using indices of working memory for the retrieval of verbal and psychomotor information, indicating a reduced speed of processing and alterations to background cortical arousal in left hemisphere frontal regions. The study supported and extended previous findings of verbal working memory abnormality, alterations to left frontal cortical rhythmic oscillations, and low EEG alpha amplitudes in those diagnosed with PTSD. Results indicated a pattern of compensatory mechanisms associated with reduced speed of information processing and right-sided activation patterns in PTSD participants and control participants who experienced strong reactions to perceived traumatic events.

Findings support the impact of traumatic events on psychobiological health in high-risk populations, implicating an association with specific patterns of neural and cognitive functioning in characteristic numbing and avoidance behaviours.
Declaration

This work contains no material which has been accepted for the award of any other degree or diploma in any university and to the best of my knowledge and belief the thesis contains no material previously published or written by another person, except where due reference is made in the text.

I give consent to this copy of my thesis, when deposited in the University Library, being available for loan and photocopying.

Therese Mayo
March 2008
Acknowledgements

I would like to acknowledge and sincerely thank Professor Alexander McFarlane for his guidance and continued support throughout the project. Professor McFarlane provided the opportunity to carry out this research and set the platform for a high benchmark that was made possible only with his supervision, direction, helpful comments, and positive encouragement in our quest to further the field of knowledge in this area of research.

I would like to acknowledge and thank the Brain Resource Company (www.brainresource.com) for permission to use the Brain Resource International Database. I would also sincerely like to thank Professor C. Richard Clark for his support, provision of training, expertise, and facilitation with data collection as part of a larger undertaking in neurocognitive research. The study participants, and the support and encouragement of the staff at Flinders University Cognitive Neuroscience Laboratory and School of Psychology, are also duly acknowledged.

I would like to thank Dr Geoff Schrader and staff of the Psychiatry Department, Queen Elizabeth Hospital, Woodville, for their support and professional inclusion. I would particularly like to thank Irene Paull for psychology training, expert advice during therapy session with PTSD patients, and for her encouragement to finish.

In general there are numerous other acknowledgements of assistance, encouragement and support I wish to make. Special mention and thanks to the staff at the University of Adelaide, Department of Public Health for specialist statistical advice, and to colleagues and friends, in particular, Alan Burns, who provided training in Microsoft Excel and technical computing assistance. A special acknowledgement also goes to those who were brave enough to attempt to proof-read draft copies – they know who they are. Finally I would like to thank my family for being so patient.
1. Overview and Outline of the Thesis

1.1. General Introduction

Since the implementation of standardised instruments, for the assessment of reaction patterns after severe psychological trauma, a significant body of evidence has accumulated indicating working memory deficits in people with post-traumatic stress disorder (PTSD). The question of why some people are emotionally unable to cope with traumatic experiences, while others seem to become emotionally more resilient from the same experiences, has been an ongoing issue of investigation. The complex interactions between physiological and psychological processes in PTSD have been studied with the aim of identifying the functional mechanisms associated with psychopathology. Dysregulation in cognitive processes, particularly for verbally derived information associated with working memory and attention, has raised interest in the PTSD literature about the neurocircuitry underlying emotional and memory functions.

Empirical studies have supported the proposal that chronic functional disturbances in people with PTSD are related to working memory and executive functions, as secondary responses involved with learning and memory, and associated with a dysregulation in parallel distributed information processing (Clark et al., 2003; McFarlane et al., 2002). Recently, the idea that information processing can be biased to a unilateral locus in either the left or right hemisphere and linked to patterns of behavioural responses has been proposed as a hypothesis for abnormal functioning associated with psychopathology (Lanius et al., 2005; Metzger et al., 2004; Vasterling et al., 2004).

An important question that remains unanswered in the PTSD literature is whether compensatory mechanisms are linked to lateralised brain functions for behavioural adaptation after severe psychological traumatic experiences. Based on various lines of evidence, a lateralisation hypothesis suggests the right hemisphere is overactive in people with chronic PTSD (Bremner et al., 1999; Clark et al., 2003; Vasterling et al., 2004). The present study investigated whether resting tonic states of electrical brain activity might indicate differences in cognitive functioning between a PTSD group and a gender and age matched control group.

1.2. Aims of the Study

The primary aim of this study was to examine whether cognitive information processing in people with PTSD is associated with functional alterations in the capacity for behavioural flexibility if cerebral resting oscillation patterns are lateralised to the right hemisphere. In
general terms, the current study focused on a compensatory mechanisms hypothesis in PTSD, proposing that electrocortical asymmetry patterns alter cognitive functioning. This analysis was based on local level processing to include the following investigations:

1. Differences between clinically diagnosed PTSD participants matched to control participants in speed of processing, as a function of working memory systems. Differences in patterns of resting tonic cortical rhythms, as a function of preparatory states for attention modulation.

2. The relationship between behavioural and electrocortical indices for the functional reorganisation of hemisphere-specific verbal and nonverbal processes, and the respective differences between PTSD participants and control participants.

3. The general pattern of reactivity associated with self-reported stress in community samples, compared with self-reported stress reactivity in PTSD participants, and behavioural and electrocortical characteristics of groups.

4. The relationship between mood states, cognitive and electrophysiological profiles in the PTSD group and control group.

5. The relationship between frontal and posterior cortical arousal states, and the respective asymmetry patterns associated with reactivity as PTSD symptom characteristics of appetitive functioning (such as numbing) and aversive functioning (such as avoidance).

6. Predictors of asymmetry patterns in clinical and non-clinical participants - specifically, whether cognitive performance best predicted asymmetry in frontal and posterior regions and whether emotional reaction patterns best predicted cognitive functioning.

To achieve these aims, analyses were conducted on data from the Brain Resource International Database (BRID), which provided the choice of lateralisation indices. These included electrophysiological activation patterns representing background activity and a tonic preparedness for information processing of sensory stimuli. Neuropsychological measures of cognitive functioning in verbal and nonverbal tasks, selected from a battery of IntegNeuro standardised functional indices, were used to assess lateralised deficits in the conscious retrieval of information for left- and right-hemisphere specific working memory systems. Web-based self-report questionnaires provided demographic details, measures of wellbeing,
and an automated version of the Composite International Diagnostic Interview (CIDI; World Health Organisation, 1993) provided perceptions of traumatic stressor events and reactivity to those events, which were used to examine the relationships between cortical asymmetry patterns and cognitive functioning.

1.3. Significance of the Study

Within the framework of parallel distributed processing, an understanding of the role of compensatory mechanisms in psychopathology is relevant to treatment options and treatment outcomes. Various models and theories have proposed that a reorganisation of brain function is associated with PTSD, with suggestions of perceptual asymmetry and psychological differences between verbal and nonverbal information processing. Empirical findings have also indicated that there may be unique patterns of brain activation in frontal, temporal, and parietal regions, with reciprocal links to subcortical regions for specific symptoms associated with anxiety and depression.

However, psychological processes and physiological responses linked to the mechanisms for active and remitted symptoms in PTSD remain to be clarified - thus, leaving sufferers with past stressor events a characteristic profile of memory functioning associated with complex patterns of behaviours that are often resistant to treatment. The efficacy of treatment programs, and reasons why some people are resilient to traumatic stressor events while others develop a chronic pattern of functional disability, are important considerations in understanding the mechanisms underlying brain cortical activation patterns and the distinct neural circuitry in psychopathology. The role of compensatory mechanisms, associated with hemispheric asymmetry patterns, is a useful starting point to an understanding of the development and maintenance of PTSD symptoms and to an understanding of why symptoms overlap.
1.4. Overview of the Thesis

Information processing models and theories for optimal functioning have provided a wide knowledge base for the complex behavioural patterns and cognitive sub-processes associated with attention, memory, and learning. From the wider literature, Chapter 2 presents a brief review of theoretical models outlining psychological and cognitive processes. This review of the literature synthesises issues that are still in contention and remain to be clarified. Suggestions from the literature, for plausible hypotheses that might be associated with hemispheric asymmetry patterns and brain-behaviour compensatory mechanisms of cognitive functioning, are summarised. Many of the models presented represent theoretical constructs of brain-behaviour relationships that are relevant to the conceptual background that informs the discussion for the current study. Chapter 3 presents a selection of the empirical research in PTSD, with evidence for right hemisphere compensatory mechanisms and perceptual asymmetry. The findings are synthesised within the framework of a brain-behaviour model proposed by McFarlane and colleagues (2002), arguing for PTSD biomarkers of neural plasticity over time. The model proposes various mechanisms for alterations to brain organisation patterns, as secondary characteristics of traumatic experiences, and postulates a role for higher-order cognitive processes in PTSD behavioural patterns of functioning.

Chapter 4 outlines the data collection procedures, the role of standardised databases in this field of research, and the study design, together with methods and procedures used in this study. Chapter 5 is a preliminary analysis of the data, examining cognitive processes and cortical asymmetry patterns, in two samples representing a clinical PTSD sample matched to controls for gender and age, before the control sample is divided into subgroups based on their perceived traumatic stressor experiences and reactivity to those experiences. Chapter 6 compares speed of information processing in clinical and non-clinical samples based on their reported reactivity patterns to previous traumatic stressor events. The respective behavioural performance and tonic resting EEG patterns are compared for asymmetry patterns.

To gain an understanding of how psychological and physiological patterns of functioning are linked to reactivity patterns, Chapter 7 compares resting EEG alpha frequency, as asymmetry patterns associated with perceived trauma reactivity, between groups. Predictors for asymmetry in cortical arousal patterns and cognitive functioning were investigated with appetitive and aversive functioning indices, as specific items in category C symptoms (as per DSM-IV, American Psychiatric Association, 1994) linked to numbing and avoidance behaviours.
Chapter 8 presents the limitations of the study, together with key findings for cortical asymmetry as a compensatory adaptive mechanism associated with secondary characteristics in learning and memory. The implications for future research and for clinical applications and treatment interventions are also discussed.
2. Literature Review: Learning and Memory in PTSD Research

2.1. Introduction

The introduction of standardised diagnostic instruments (since 1980), recognising the impact of traumatic events on psychobiological health, has led to an increase in research investigating the mechanisms associated with high risk populations. A difference between objective and subjective characteristics of fear and psychological disturbances is a recent topic of investigation that seeks answers at the neural level of analysis. Post-traumatic stress disorder has been conceptualised as a complex stress response, with a pattern of symptom presentation at specific time points, or alternatively a pattern of oscillation between active PTSD and remitted symptoms (McFarlane et al., 2002). Emotional responses, thoughts and memories of extremely stressful events are, in themselves, not considered to be pathological. However, the psychological sequelae that provide temporary relief from internal states after the traumatic event may, in fact, represent phenomena that have been functionally conceptualised as compensatory and associated with secondary difficulties in learning and memory (McFarlane et al., 2002; Lanius et al., 2005).

Of particular interest to this review are the processes associated with memory functioning because they can help illuminate individual differences associated with the perceptual and conceptual integration of knowledge. Furthermore, the numerous reaction patterns that may occur following traumatic experiences can be represented by memory processes as knowledge structures (Conway and Pleydell-Pearce, 2000; McFarlane et al., 2002; Svoboda et al., 2006) and may be explained by tonic or characteristic implementation styles consistent with learning and working memory processing (Anderson, 1987; David et al., 2004; Galletly et al., 2001). Tonic characteristics, or implementation styles in information processing, have been recently linked to mechanisms in distributed neural networks (Basar, 2004; Fuster, 1997; Klimesch, 1999).

To understand how functional cerebral asymmetry is linked to PTSD symptoms, an analysis of reaction patterns to extreme traumatic events is increasingly being investigated in the PTSD literature using different methodologies and the neurobiological correlates of memory processes (Clark et al., 2003; Felmingham et al., 2002; Veltmeyer et al., 2006). An examination of the specific neural circuitry underlying differences in emotional states, with an emphasis on spatial parameters (Bremner et al., 1999; Lanius et al., 2003), or temporal parameters (Neylan et al., 2003; Protopopescu et al., 2005), has provided evidence for a functional role of hemispheric asymmetry in memory processes and symptom patterns.
2.1.1. Focus of the Literature Review

The purpose of this review is to outline the functional significance of brain neuronal circuitry mapped, to date, in the PTSD literature. An emphasis in this review is on the functional role of hemispheric asymmetry, and hypotheses for memory impairments and associated cognitive changes, or neuropsychological impairments linked to PTSD. The literature is reviewed with a focus on perspectives using a working memory model (Baddeley, 1986) for hemispheric specialisation of verbal and nonverbal information processing, which includes models and hypotheses from the wider literature.

The study of human memory has a wide literature base within the framework of information processing theories. Therefore, the scope of the present review will be limited to issues relevant to the transfer of information between hemispheres associated with modality-specific processing. Using a brain-behaviour model applied to PTSD (McFarlane et al., 2002), as a framework for understanding compensatory mechanisms associated with psychological disturbances after traumatic events, the specialisation of verbal and nonverbal information processing and the mechanisms for integrated functional relationships of brain organisation are reviewed in light of a brain lateralisation hypothesis in PTSD (Bremner et al., 1999; Clark et al., 2003; Vasterling et al., 2004; Lanius et al., 2004).

PTSD studies providing evidence for differences between self-regulation and emotion reposiveness are reviewed within a framework of parallel distributed processing theory (Basar, 2004; Goldman-Rakic, 1996). The classical concepts of memory functioning, such as short-term memory, long-term memory, and the various sub-processes in these systems, are considered from the perspective of associative cortical networks that reflect memory as a dynamic process and as a distributed property of the whole brain (Basar, 2004). How such models and theories might apply to the complex cognitive architecture of PTSD is presented in this chapter in order to familiarise the reader with the conceptual framework that is the focus of this study. The empirical findings in PTSD research are presented as the focus of the next chapter and a synthesis of the two chapters will consolidate evidence of right hemisphere lateralisation in people with PTSD.

2.1.2. PTSD Symptoms and Memory Functioning

Criterion A of the disorder applies to the exposure of the specific traumatic stressors and to the initial reaction patterns involving “fear, helplessness, or horror”. The three separate symptom clusters define behavioural descriptors of functional alterations in cognition and emotion (American Psychiatric Association, 1994). The first group of symptoms (criterion B) includes the persistent re-experiencing of the traumatic event, with an intrusive memory and flashback condition associated with distractibility and high anxiety during reminders of the event. A second group of symptoms (criterion C) includes numbing of responses to external stimulation and a detachment from social situations accompanied by loss of interest, restricted affect and a shortened sense of future. The third group of symptoms (criterion D) are often behaviourally recognised by physiological responses such as exaggerated startle reaction, insomnia, irritability, and defence reflex phenomena associated with the “fight or flight” response (Yehuda, 2002). Group D symptoms are also associated with persistent increased arousal or anxiety, and are considered to be common among people who have a high exposure to extreme stressors (Nemeroff et al., 2006).

PTSD diagnostically requires full symptoms from each category to be present for more than one month after the event (criterion E) and sufferers must show clinically significant distress and impairment that interferes with functioning (criterion F) (American Psychiatric Association, 1994).

A shift from DSM-III-R criteria emphasising the severity of the trauma experienced, to DSM-IV criteria (American Psychiatric Association, 1994) emphasising reaction patterns to trauma (Group A criteria), has led to the development of theories about what functions are altered in the individual as a result of exposure to traumatic stressors (Gold et al., 2005; Schore, 2002; Wilson, 1994). Thus, the idiosyncratic meaning assigned to intrusive and repetitive thoughts, images, memories and impulses, is critical to voluntary control mechanisms for self-regulation (Thatcher, 1997; Williams and Moulds, 2007). High levels of distress caused by intrusive responses to the memory of traumatic events have been linked to chronic stress response patterns, particularly if the intrusions are interpreted as either unwanted or uncontrollable (Litz et al., 2000; Stein et al., 2002; Elzinga and Bremner, 2002). Accordingly, questions have been raised about the cognitive processes or sets of processes that underpin PTSD.

Memory function in PTSD has been explored using various theoretical notions. Recently it has been postulated that the psychopathology associated with the three categories of
symptoms that define the disorder as an anxiety reaction may have specific patterns of brain organisation (Metzger et al., 2004; Nemeroff et al., 2006), with some researchers proposing that certain features are unique to PTSD (Kashdan et al., 2006; Litz et al., 2000). For example, category B symptoms are described as central to the disorder and automatically retrieved as modality-specific sensations (McFarlane et al., 2002). Such symptoms may be particular to the interpretation of the exogenous or endogenous stimuli associated with the traumatic event (Buckley et al., 2000; Litz et al., 2000; Ehlers and Clark, 2000). On the other hand, category C symptoms have been demonstrated to show two neural pathways for emotion regulation (Metzger et al., 2004; Nemeroff et al., 2006). That is, avoidance and numbing symptoms have been shown to be specific toward people, places or thoughts, and involving reaction patterns based on either appetitive functioning or aversive functioning (Kashdan et al., 2006; Litz et al., 2000), implicating control processes for behavioural implementation.

In the case of category D symptoms associated with hyperarousal, these have been linked to impaired concentration (Metzger et al., 2004; Galletly et al., 2001), attention and memory deficits (Vasterling et al., 1998; Bryant et al., 2005), and more recently to avoidance behaviours associated with aversive functioning (Nemeroff et al., 2006; Kashdan et al., 2006). Furthermore, it has been suggested that difficulties in understanding the mechanisms associated with behavioural reactions are largely due to the overlap in symptom categories (Kashdan et al., 2006), and the instability of brain function in PTSD is related to phasic and tonic modes of neural activity (Neylan et al., 2003).

Cognitive deficits associated with PTSD have been linked with both a reduced capacity to encode new information and a disruption in the ability to mentally manipulate information (Clark et al., 2003). These deficits have been linked to attention and memory processes and the respective roles of vigilance and arousal in frontal and subcortical systems (Bryant et al., 2005; McFarlane et al., 1993; Vasterling et al., 1998). Furthermore, it has been suggested that the ensuing emotional dysregulation is associated with an inability to accurately predict context information relating to threat stimuli (Britton et al., 2005; Layton and Krikorian, 2002; Litz et al., 2000).

However, evidence also supports the view that symptom categories in PTSD, as defined by DSM-IV criteria (American Psychiatric Association, 1994), are associated with patterns of behaviour that are evident in resting or tonic electroencephalogram (EEG) states and linked to a stylistic tendency toward reaction (Galletly et al., 2001; Metzger et al., 2004). For example, the numbing and avoidance, type C features, have been associated with depressive strategies
and left hemisphere dysfunction (Nemeroff et al., 2006). It has also been suggested that these features are resistant to change, leading to vulnerability factors for compensatory strategies and to brain reorganisation patterns in the development of PTSD (Yehuda, 2004). On the other hand, type D features, or hypervigilant behaviours, have been associated with anxiety and cognitive deficits related to right hemisphere dysfunction (Bryant et al., 2005; Vasterling et al., 2004). In addition, symptom patterns have been implicated in functional cerebral asymmetry favouring right hemisphere activation patterns (Lanius et al., 2003; 2004).

2.2. The Phenomenology of PTSD and Information Processing Models

Memory functioning is a pivotal conceptual and phenomenological aspect of a number of symptom categories associated with PTSD and various hypotheses have been advanced to understand the relationship between perception and cognition in such complex systems (Galletly et al., 2001; Layton and Krikorian, 2002; McFarlane et al., 1993). Memory has been defined as a functional property of the whole brain, with a dominant view proposing that memory is a distributed property of cortical systems (Basar, 2004; Fuster, 1997). According to Basar (2004), a simple sensory response that is defined by the smallest memory network is a cortical cell group or module. A parallel-distributed model of cognition posits that information processing takes place through the interactions of a large number of simple processing elements (Basar, 2004). Therefore, a potentially useful proposal for the conceptualisation of dysfunction after severe traumatic events is that the abnormal tonic states in PTSD can be linked to the loss of normal flexible modulation of parallel distributed processing in cortical networks (Galletly et al., 2001; McFarlane et al., 2002), and a disruption in working memory processes (Clark et al., 2003) for the formation of specific templates representing objects and memories (Litz et al., 2000; Fuster, 1997).

Fundamental to complex dynamic systems of cortical organisation in human memory are associative networks for the perceived representations of objects as memory in cognitive architecture (Basar, 2004; Mesulam, 1998; Holscher, 2001; Ramos and Savage, 2003). At a general level of analysis, it is important to understand which representations will be implemented on a moment-to-moment basis, and how information is integrated in cortical networks (Ward, 2003). According to Banich (2004), the nature of the interactions between the hemispheres is not a unitary phenomenon. Rather, communication between the various cortical and sub-cortical anatomical channels occurs in parallel via temporal codes and is based on a number of factors. These include the meaning assigned to information, the type of information carried, and the speed at which the information is to be transmitted (Banich,
Therefore, one hypothesis for lateralisation, at a local level of information processing, is through the timing of activity for object and spatial information, dependent on task demands (Sakagami et al., 2006; Ungerleider et al., 1998).

Cognitive processes have been consistently shown to be both spatially and temporally dynamic and do not correspond to brain regions in a fixed and stable way (Courtney et al., 1998a; Landau et al., 2004; Van Horn et al., 1998). Furthermore, Kiernan (1981), and later Fuster (1997), postulated that an attempt to explain physiological phenomena in psychological terms might result in conceptual confusion unless the adaptive significance of the cognitive processes is defined in terms of their behavioural counterparts. Theoretical models and experimental evidence have supported the notion that the same cortical systems that encode sensory information about the world are also involved in the retrieval of stored representations (Linden et al., 2003; Fuster, 1997). In addressing the physiological and psychological concepts relevant to brain-behaviour relationships, it has been suggested, from a philosophical perspective, that “localisation of function” is not the literal physical location of an entity, but the conceptual representation of neurological functions (Kiernan, 1981). Thus, different brain regions are involved in the regulation of emotional behaviour through the dynamic integration of spatial and temporal parameters (Berntson et al., 2003; Sotres-Bayon et al., 2006; Hoge and Kesner, 2007).

Models based on the temporal transmission properties of neural networks for synchronised firing have not been empirically validated and remain as unresolved questions of how information is transferred in the brain. However, such models are theoretically useful in understanding the mechanism(s) for the integration of information and posit that learning-induced changes cause a reorganisation of neuronal assembly coded information (McFarlane et al., 2002; Holscher, 2001; Knyazev et al., 2006b).

One such model is a “temporal binding” hypothesis. Engel and colleagues (1999) argue that the synchronisation of neuronal discharges in distributed memory systems in the brain is a mechanism for the integration of spatially distinct neurons into cell assemblies. Other models have been proposed to suggest that oscillations may be induced into a neuronal network by pacemaker cells and/or endogenous membrane properties of individual cells (Rowe, 2005; Ward, 2003). According to such models, the frequency and/or phase shifts of large populations of oscillators can become progressively uncoupled and exhibit a synchronous discharge pattern within small cortical areas, spreading within distributed cell assemblies in response to cognitive demands (Herrmann, 2003; Munk, 2001; Thatcher, 1997).
At a mechanistic level, research endeavours have demonstrated that different cognitive processes vary according to long-range synchronisation patterns associated with mnemonic functions in various frequency ranges, or with perceptual objects and motor programs (Belger et al., 1998; Sauseng et al., 2005; Schack et al., 2001; Ward, 2003; Schutter and van Honk, 2006). One group of researchers (Klimesch et al., 1997) has argued that thalamo-cortical feedback loops, oscillating within the alpha frequency range, allow searching and identification of encoded information. Based on this assumption, it has also been suggested that capacity differences are reflected as power differences among individuals (Posthuma et al., 2001). That is, the faster oscillating feedback loops correspond to faster access of encoded information and to working memory processes (Klimesch, 1999; Ward, 2003; Knyazev et al., 2006b).

Hemispheric differences in functional connectivity have been related to differences in EEG power and associated with the resting frequency or the dominant alpha rhythm (Cooper et al., 2003; Klimesch, 1999). The alpha frequency has also been associated with trait characteristics as a stable or tonic pattern for the implementation of functional responses to stimuli (Niedermeyer, 2005; Smit et al., 2005). Furthermore, studies by Klimesch (1999) and his group have led to the notion that the dominant alpha rhythm is the “idling” state for mental activity in humans and is measured by the synchronisation of very large populations of neurons oscillating with the same phase and frequency to reflect a state in which no information is transmitted. Based on this theory, the alpha rhythm becomes blocked or desynchronised by attention and/or mental effort, which means that different oscillators within the alpha band are no longer coupled but oscillate with different phase lags and with different frequencies (Klimesch, 1999). In different research endeavours, the resting EEG has been used as a quantitative descriptor of individual differences (Hugdahl and Davidson, 2004; Smit et al., 2005; Zietsch et al., 2007), and used to indicate relationships between affect and asymmetries in anterior cortical regions (Blackhart et al., 2006; Bruder, 2004; Smit et al., 2007; Knyazev et al., 2005).

However, it has also been argued that neural speed does not indicate better cognitive performance, but rather, the degree of connectivity between cerebral regions may be of greater importance to understanding behaviour (Ribary, 2005; Sauseng et al., 2002; Singer, 1999). Distributed networks have been shown to represent the perceptual and executive components of decision processes and the networks for working memory processes associated with the categorisation of information, interpretation of information, short-term memory, and
attentional control (Bastiaansen and Hagoort, 2003; Fuster, 2003; Klimesch et al., 2005). Therefore, within the framework of parallel distributed processing, working memory is considered a “top-down” feedback projection that modulates the activity of feed-forward cells activated through the synchronisation and interaction of network assemblies in different spectral frequencies (Tanaka, 2001; Thatcher, 1997; Ward, 2003) or by humoral neuromodulators (Gold, 2005; Nieuwenhuis et al., 2005).

There is considerable evidence for the view that working memory links together regions in the cortex holding perceptual and response representations relevant to controlled goal-directed behaviour (Goldman-Rakic, 1999; Jensen and Tesche, 2002; Sauseng et al., 2002). Working memory systems have been conceptualised as mechanisms used to hold mental representations active long enough to make the appropriate responses to stimulus configurations or contingencies (Fuster, 1997; Glassman, 1999; Goldman-Rakic, 1999). The bi-directional links between the brain and behaviour implicate anatomical, physiological, and psychological connections at a more subtle level of analysis. Thus, using a mechanistic analysis, the multi-dimensional elements associated with encoding new information, the storage and retrieval of information, and the conceptual differences between self-registration and self-regulation are incorporated in the combinatorial nature of adaptive functioning.

PTSD brain dysfunction and neuropsychological impairment has been associated with both cognitive and emotional disturbances (Bremner et al., 1999; Litz et al., 2000; Clark et al., 2003; Williams et al., 2006; Vasterling et al. 2006). In earlier research, the neurobiological correlates of PTSD collectively provided evidence of dysfunction related to frontal-limbic neural circuits (McFarlane et al., 1993; Vasterling et al., 1998). However, to address the difficulties associated with separating the functional interactions of emotional and conscious processing of information, an understanding of the neural processes involved in reaction patterns and coping mechanisms has been the focus of more recent PTSD research (Williams et al., 2006; Bryant et al., 2005; Felmingham et al., 2002; Jatzko et al., 2006; Vasterling et al., 2006; Lanius et al., 2003). As a framework for investigating differences in clinical and non-clinical populations, a working memory model (Baddeley, 1986) has been one paradigm used to provide evidence of structural and functional abnormalities in people with PTSD (Clark et al., 2003). The importance of understanding the response patterns after severe trauma events has led some research endeavours to focus on the networks for the neuronal organisation of attention, and the associated memory and information processing mechanisms, activated for verbal and nonverbal processes (McFarlane et al., 2002; Vasterling et al., 1998; Lanius et al., 2002, 2005; Protopopescu et al., 2005; Jatzko et al., 2006).
In addition, as previously discussed, the behavioural differences manifest in people with PTSD, and the complexity of reaction patterns, have led to proposals that different symptom combinations may have specific brain organisational patterns for responses to anxiety associated with traumatic events, and may represent a number of subtypes in the heterogeneous syndrome (Begic et al., 2001; Metzger et al., 2004; Kashdan et al., 2006; Nemeroff et al., 2006). Such proposals suggest that specific patterns of symptoms are associated with hemispheric asymmetry (Metzger et al., 2004; Veltmeyer et al., 2006). On the other hand, other researchers have argued that memory abnormalities in people with PTSD are associated with verbal working memory deficits, thus biasing brain organisation patterns to right hemisphere coding (Jatzko et al., 2006; Clark et al., 2003; Bremner et al., 1999).

Consistent with both arguments, and to account for neuropsychological and emotional impairments, one PTSD model posits that changes to memory occur over time by mechanisms of synaptic pruning (McFarlane et al, 2002). According to McFarlane and colleagues, compensatory mechanisms for neural plasticity in PTSD are of an adaptive nature and include the activity-dependent refinement of neuronal connections. The brain’s ability through learning, to reorganise functional mechanisms for adaptive behaviour, has been defined as synaptic plasticity (McFarlane et al., 2002; Sejnowski and Paulsen, 2006). The idea that PTSD is a secondary and general characteristic of learning and memory (Galletly et al., 2001; McFarlane et al., 1993) poses questions about the fundamental difference between the dispersed neuronal assemblies.

2.2.1. Synaptic Plasticity as an Adaptive Mechanism

Changes in neuronal circuitry for learning and memory can include mechanisms of plasticity that change the balance of excitation and inhibition, a long-term potentiation (LTP), or long-term depression (LTD), a change in neuronal membrane excitability, and the anatomical changes over a longer time period (McFarlane et al., 2002; Holscher, 2001). Synaptic plasticity as an adaptive mechanism has been proposed to be activity-dependent and influences the balance of excitation and inhibition as a result of learning processes (McFarlane et al., 2002; Sejnowski and Paulsen, 2006; Knyazev et al., 2006b). More specific to the discussion of adjustments to neuronal patterns in people who have experienced traumatic stressors, the idea of LTP, defined as a mechanism for neuronal plasticity, introduces questions about the multi-dimensional process of associative learning (Mesulam, 1998; Matzel and Shors, 2001) and the dynamic processes that include changes to memory in
an ordered way from moment to moment (Banich, 2004; Fuster, 2003; Hoge and Kesner, 2007); thus implicating temporal parameters.

In PTSD research, it has been proposed that early traumatic experiences may shift functional connectivity among brain regions, which later become conditioned responses and reflect abnormal behavioural functioning when the context changes (Teicher et al., 2006; Litz et al., 2000; Lanius et al., 2003; 2005). Such theories propose that neuromodulation alters the timing and flow of information processing by interrupting prefrontal cortex functions through attentional bias dependent on expectations, or predictions, from previous experience (Felmingham et al., 2002; Galletly et al., 2001; McFarlane et al., 1993). Converging evidence supports the above views, indicating that there is a breakdown in functional connectivity in people with PTSD (Shaw et al., 2002; Teicher, 2003; Lanius et al., 2004). This has been demonstrated both in imaging studies and in electrophysiological studies using trauma-neutral (Clark et al., 2003; Veltmeyer et al., 2006) and trauma-activation paradigms (Lanius et al., 2002, 2004).

In determining why there might be an asymmetry between memory for content and memory for context in PTSD, it has been argued that the prefrontal cortex can be conceptualised as a dynamic filtering mechanism, where working memory processes are characterised as more than a passive sustaining of representations, but involve an attentional component in which personal goals modify the salience of different sources of cognitive information (McFarlane et al., 1993; Bryant et al., 2005). These ideas incorporate arguments presented in theoretical models for the optimisation of behavioural goals (Anderson, 1987; David et al., 2004; Dayan and Yu, 2003). Purportedly, differences between self-regulation and self-registration suggest the interaction of emotional and cognitive processing in choices involving attentional bias (Litz et al., 2000; Dayan et al., 2000; Lou et al., 2005; Posner, 2005; Sarter et al., 2005; Vogt and Laureys, 2005). A basic conceptual understanding of optimisation functions is represented in Figure 2-1 to show memory and attention as separate influences on decision processes.

A conceptualisation of brain functions, as multi-dimensional hierarchical structures, has been linked to laterality effects that are influenced by a number of factors, depending on the extent to which symbolic or verbal encoding and rehearsal take place (Petrides, 2000; Glassman, 1999; Ungerleider et al., 1998). Accordingly, for the purposes of this study, the theoretical underpinnings of emotion regulation are subsumed under the cognitive or functional
asymmetry principle, as well as the contralateral control principle of hemispheric function (Kolb and Whishaw, 1996; Gazzaniga et al., 2002; Banich, 2004).

Furthermore, in PTSD research, structural abnormalities associated with information processing have been linked to storage functions for knowledge implementation (Schore, 2002; Clark et al., 2003; Buckley et al., 2000). Circuits underlying emotional and mnemonic processes between cortical networks are the mechanistic basis of conscious behaviour (Basar, 2004; McFarlane et al., 2002; Mesulam, 1998) and can be reorganised by a number of factors, including genetic and environmental (Posner, 2005; Zietsch et al., 2007). According to some authors, channels that break down through early abuse are the limbic system, the corpus callosum and hemispheric laterality, and the hippocampus (Teicher et al 2006). On the other hand, different psychological defence mechanisms, as a strategy for avoiding unwanted feelings, have been proposed in the literature and include the compartmentalisation of cognitions for sensory inputs (John, 2005; Litz et al., 2000; Scaer, 2001). Both a break in communication channels, and the conscious and deliberate strategies for blocking unwanted feelings, implicates an interruption in interhemispheric communication by altering the timing of attentional mechanisms for higher level cognitive processing (Singer, 1999; Glassman, 1999; Rowe, 2005). These notions may be applicable to the overlap in symptoms; particularly numbing and avoidance symptoms associated with PTSD (Kashdan et al., 2006).

2.2.2. Abnormal Tonic States in PTSD

As currently defined, PTSD consists of a combination of phasic and tonic features (McFarlane et al., 2002). Phasic features are transitory and can be easily evoked by salient environmental or internal cues (Pitman, 1993). On the other hand, tonic attributes have been defined as those that reflect the baseline mental functioning of the individual and have been linked to individual differences (Galletly et al., 2001; Pitman, 1993), including differences in the activity of reticulo-cortical pathways (Aston-Jones and Cohen, 2005), and vulnerability to risk of physiological changes accompanying information processing (Southwick et al., 1999; van der Kolk., 2001; Yu and Dayan, 2002).

As previously mentioned, a psychobiological model for chronic PTSD has been conceptualised by McFarlane and others (2002) as falling within the framework of information processing, and has been associated with working memory abnormalities and a functional reorganisation of brain patterns. Theoretical models of PTSD have related symptom categories to a general information-processing alteration in cognitive functioning.
(McFarlane and Yehuda, 2000; Buckley et al., 2000). Based on this argument, one suggestion in the PTSD literature is that compensatory mechanisms involve an increased reliance on visuospatial processing, with concomitant reduction in verbal processing and a reduced left hemisphere neural activation pattern (Clark et al., 2003; Bremner et al., 1999). Linked to this idea, the behavioural features of the disorder are characterised by tonic and phasic symptoms contributing to disturbances in memory functioning (Galletly et al., 2001), with empirical findings supporting arguments suggesting that the traumatic experience is not integrated into existing memory structures (Buckley et al., 2000; Shaw et al., 2002).

Learning processes, as electro-cortical activity patterns, represent the physiological and psychological processes linked to behavioural flexibility (Basar, 2004; Sejnowski and Paulsen, 2006). Furthermore, asymmetry patterns associated with arousal states have been investigated using EEG methodology (Hugdahl and Davidson, 2004). One of the most important parameters of the EEG is frequency. The basic EEG-phenomenon of synchronisation and desynchronisation is referred to as the mechanism by which information is processed in the brain (Niedermeyer and Lopes da Silva, 2005). Temporal as well as spatial changes can be monitored using this technique. That is, a frequency analysis of neuronal activity is represented by oscillations within local or widespread neuronal networks, where each frequency is purported to reflect a different degree of brain activity (Klimesch, 1999). The general computational roles for network oscillations have been defined as comprising a number of functions, with three main possibilities listed. These include sensory information representation, the regulation of the flow of information, and learning and memory functions comprising storage and retrieval processes (Sejnowski and Paulsen, 2006). Relating the above ideas to working memory functioning and cognitive abnormalities associated with PTSD, neural signals have the potential to define the centralised and decentralised processing functions of cognitive architecture.

Different theoretical notions have been proposed for the conceptualisation of brain organisation, particularly for events that occur with a complex sequence of internal states, both in the PTSD literature (Joseph et al., 1997; Nemeroff et al., 2006), and in the wider literature (Baddeley; 2003; Basar, 2004). Accordingly, in brain organisation, it is thought that the phasic mode of activity promotes focused or selective attention, whereas the tonic mode leads to attentional scanning with associated effects on behavioural flexibility (McFarlane et al., 2002; Aston-Jones et al., 1999).
One mechanism proposed for coping with increased stress is the narrowing of attention (Aston-Jones et al., 1999; Cooper et al., 2003). Empirical findings have indicated that attentional demands such as alertness and expectancy are reflected by different electrocortical frequency compositions (Klimesch et al., 1998). A number of researchers have also suggested that EEG oscillations in different frequency ranges play an important role in the functional coupling between posterior and anterior regions of the brain (Basar, 2004; Sauseng et al., 2005). Accordingly, one theory proposes that phasic shifts in attentional capacity in people with PTSD may be linked to a different pattern of cortical oscillations between symptoms and remitted symptoms (McFarlane et al., 2002). Disruption to attention mechanisms may also be consistent with theories proposing that the permanent activation of the fear network in PTSD is related to symptoms of hyperarousal (Bryant et al., 2005; Joseph et al., 1997; Vasterling et al., 1998).

The investigation of cortical arousal in PTSD using electrophysiology is a comparatively recent research endeavour and data accumulated to date has provided biological evidence of differences in functioning for people diagnosed with PTSD (Begic et al., 2001; Jokic-Begic and Begic, 2003; Neylan et al., 2003; Veltmeyer et al., 2006; McFarlane et al 1993). Specifically, these differences relate to increased tonic or baseline physiologic activity (Begic et al., 2001) and attention mechanisms as correlates of neural oscillation patterns, particularly in prefrontal cortex regions (Galletly et al., 2001).

Various models of PTSD hypothesise that the neural correlates of symptoms, such as emotional numbing and hyperarousal, reflect patterns of dysfunctional connectivity among brain regions that may be responsible for alterations in structure or function (McFarlane et al., 2002; Lanius et al., 2005). However, such compensatory mechanisms for higher-order processing may occur without significantly compromising the behavioural capacity for certain other kinds of tasks (Munk, 2001; Elzinga and Bremner, 2002). This notion is particularly relevant to an understanding of the different processes involving the voluntary or conscious retrieval of information from memory as opposed to those processes that initiate the fast and automatic retrieval of information from memory based on early recognition cues (Landau et al., 2004; Brewin, 2003). Furthermore, a consideration that some processes remain intact after trauma, while others alter, may be highly relevant to an understanding of the various inconsistencies found in the literature for functional networks and an association with cognitive proficiency scores. The inconsistencies and inadequacies in mapping the functional roles of the peripheral and central nervous systems, reported in the PTSD literature, have fuelled a current debate focused on whether cognitive problems in PTSD sufferers are trauma-
related, due to premorbid factors, or related to symptom and comorbid presentations (Buckley et al., 2000; McFarlane and Yehuda, 2000; Nemeroff et al., 2006).

2.3. Distributed Parallel Cortical Systems in PTSD

The question of whether asymmetry patterns serve as compensatory mechanisms in the PTSD literature has been raised by different researchers (Bremner et al., 1999; Britton et al., 2005; Clark et al., 2003; Lanius et al., 2002; Vasterling et al., 2004). However, the specific functional role of cerebral asymmetry in compensatory behaviours has not been clarified. This represents a gap in the knowledge required for treatment options and the efficacy of treatment programs, and for understanding why some people are resilient to traumatic stressor events while others develop a chronic pattern of functional disability. Hemispheric specialisation theories have focused on the specialisation of brain structures and have been a useful framework for understanding localised behavioural deficits (Lezak et al., 2004; Vasterling et al., 2004). At the neural level of assessment, research investigations into two complimentary binding strategies have been previously mentioned. These have included mechanisms associated with distributed neuronal activity for storage and integration in memory, and for internally generated synchronised responses; specifically, spatial as well as temporal constraints of brain functioning (Singer, 1999).

As discussed above, investigations in PTSD using EEG measures have predominantly focused on the time domain of the EEG recording to elucidate changes in cortical activation associated with the behavioural symptoms and cognitive disturbances of PTSD (McFarlane et al., 1993; Galletly et al., 2001; Felmingham et al., 2002; Attias et al, 1996). On the other hand, fewer studies in PTSD research have focused on the frequency domain to investigate differences in quantitative and qualitative EEG parameters (Begie et al., 2001; Jokic-Begic and Begic, 2003; Veltmeyer et al., 2006). According to Smit and colleagues (2005), an understanding of individual variance in EEG power could provide clues to the underlying neurobiology of mental disorders. Smit and others posit that both genetic and environmental contributions to individual differences may be reflected in the background EEG power spectrum. The authors also provide data to demonstrate that, generally, heritability is associated with alpha peak frequency and that environmental factors have more impact on low frequency EEG power than on power in the higher frequency bands (Smit et al., 2005).

The next chapter of this review will address the theoretical underpinnings related to these issues and will constrain arguments for cognitive functioning by focusing on processes.
relevant to the encoding, storage, and retrieval of memories within the theoretical framework of hemispheric specialisation. Two mechanisms associated with brain reorganisation patterns as compensatory factors in coping with stress are addressed. The first is at the functional level of analysis, with proposals that an interruption to inter-hemispheric communication can alter the timing of attention mechanisms and disrupt the coordinated flow of information. The second is at the local level of processing, by conscious and deliberate strategies to block unwanted feelings through the conceptual and perceptual identity of items (Clark et al., 2000). At this level of processing, a perceptual asymmetry or selective attention bias has been proposed as a mechanism for neural plasticity in PTSD (McFarlane et al., 2002). Strategies are based on differences related to the meaning or appraisal of an encoded item, and the spatial aspects or the identity of an item, which are known to include two orthogonal components – spatial and temporal parameters of item specificity (Nadel and Moscovitch, 1998). These proposals have led to the question of what role hemispheric asymmetry serves in the coordinated flow of information in psychopathology.

2.3.1. Summary: Stress Reactions and Memory Functioning

The inconsistencies and controversies in patterns of brain organisation in the wider literature highlight two hypotheses relevant to the study of PTSD. These are firstly the subjective perception of stress and the global and unspecific nature of stress compared to other cognitive information; and, secondly the disposing nature of stress reactions for right hemisphere activation (Hugdahl and Davidson, 2004). The first hypothesis may be considered from the perspective of findings in the PTSD literature suggesting a decoupling of regions specialised for certain functions such as verbal and nonverbal processing (Shaw et al., 2002), which is relevant to hypotheses of a narrowing of attention and reduced behavioural flexibility (McFarlane et al., 2002). The second hypothesis can be viewed from the perspective of prevalence to code information using right hemisphere processes and an asymmetry in structural and functional brain processes associated with memory (Bremner et al., 1999; Clark et al., 2003; Lanius et al., 2004).

These hypotheses will form the starting point for the next chapter. A review of the growing body of evidence accumulating in PTSD research has been conducted with the aim of identifying possible mechanisms for the reorganisation of brain function in people suffering from PTSD. However, the question of whether asymmetry patterns serve as compensatory mechanisms has not been clarified in the PTSD literature. Using the theoretical concepts reviewed in this chapter, evidence of lateralised functioning will be presented in the following chapter, and will be framed within a brain-behaviour model (McFarlane et al., 2002) for an
understanding of the functional mechanisms associated with physiological and psychological disturbances in people with PTSD.

A focus on attention and memory, as separate neuroanatomical functions, may be useful for a conceptualisation of the combinatorial nature of brain processes in models suggesting behaviour is driven by optimal gains (Dayan et al., 2000). Numerous models derived from diverse methodologies have been proposed to account for the neuronal circuitry that has been mapped in PTSD. Various psychological processes, associated with working memory systems and learning, have been investigated by the wide literature base in PTSD research (see Figure 2-1). Working memory as a shared neurobiological substrate has been linked to spatial working memory and mechanisms of attention (Awh and Jonides, 2001; Banich, 2004). This may be an important functional characteristic associated with hemispheric asymmetry patterns and altered neuronal circuitry in people with PTSD. The various models and concepts from the wider literature, in understanding compensatory mechanisms, will be synthesied in a review of the PTSD literature within the framework of parallel distributed systems, with relevance to a brain-behaviour model of PTSD (McFarlane et al., 2002).

Figure 2-1. Conceptualisation of attention and memory processes as might apply to models of brain-behaviour functions and PTSD symptoms.
3. Empirical Findings in PTSD and Literature Review Synthesis

3.1. Brain Organisation: Function and Structure

In the PTSD literature, reviews of studies conducted have reported functional abnormalities in cortical and subcortical regions in people with PTSD (Nemeroff et al., 2006; Nutt and Mazilia, 2004; Buckley et al., 2000). However, in order to constrain the argument of knowledge representation and behavioural flexibility, Fodor’s (1984) conceptualisation of cognitive architecture will be adopted here. His thesis focuses on the differences between function and structure. Functional levels are defined as roles and purposes for events, whereas the physical or structural level is characterised by the electrical or chemical characteristics of events (Fodor, 1984, 1988).

Within the generally reported framework of cognitive architecture, neural signals transfer information in the brain (Anderson et al., 2004; Basar, 2004). Different levels of cognitive architecture distinguish between dynamic or computational systems and focus on whether symbolic processing is specified by a priori rules or relies on the emergent properties of processing units (David et al., 2004). Information processing is purportedly based on mechanisms associated with rates of coding, temporal coding, and the speed of responding, depending on differences between memory codes for manipulated and non-manipulated items (Banich, 2004; Engel et al., 1999; Singer, 1999). By conceptualising information processing at a mechanistic level, an analysis of neuronal ensembles, or pathways, provides an understanding of how the selection of perceptually and behaviourally relevant information is transmitted through distinct circuits (Basar, 2004).

Functional asymmetries in the brain are thought to reflect unique processing demands and provide the capacity for purposeful reorganisation or compensation and adaptive functioning (Basar, 2004; Fuster, 2003; Gazzaniga et al., 2002; Kolb and Whishaw, 1996). Classical theories of hemispheric specialisation conceptualise notions of structure and various forms of cognitive processes that differ between the two cerebral hemispheres (Tucker and Williamson, 1984; Fuster, 1995; Kolb and Whishaw, 1996). Based on this conceptualisation, interhemispheric interaction has been proposed as an emergent property (D'Esposito, 2007), and a flexible and adaptable system for meeting the computational demands imposed upon the brain (Banich, 2004; Fuster, 2003; Petrides, 2000). However, within the wider literature, the debate associated with theoretical constructs of working memory phenomena based on “domain specificity” or “processing specificity” remains unresolved (D'Esposito, 2007; Ungerleider et al., 1998).
Thus the empirical findings presented in this review will be synthesised with an emphasis on evaluating differences in neural connectivity patterns associated with stress and arousal that have been linked theoretically with functions associated with volition and motivation for action (Gold, 2005; Tucker and Williamson, 1984). A review of the empirical PTSD data in this chapter will be considered from the perspective that differences in verbal and nonverbal processing at the functional level of implementation, in people with PTSD, reflect a breakdown in parallel processing systems at the structural level of processing (McFarlane et al., 2002). However, the various mechanisms thought to be associated with the switch from nonverbal or visual representations to verbal representations in psychological functioning, as presented in diverse studies in PTSD, can only be briefly summarised in this review.

Various models of brain organisation posit that information in cortical systems, for memory storage and retrieval, changes not only the importance of contexts but also how phenomena fit into different levels of cognitive architecture for adaptive functioning (David et al., 2004; Svoboda et al., 2006; Fuster, 1997). Of particular relevance to this review are notions suggesting that associations between context information and interpretative functions can be altered by environmental influences (Schore, 2002; Zietsch et al., 2007).

In PTSD research, a connection with increased spatial coding has been implied in the functional patterns of information processing (Clark et al., 2003; Bremner et al., 1999; Jatzko et al., 2006) and proposed as a central mechanism in a theoretical model associated with neuronal acetylcholine-induced functional plasticity (McFarlane et al., 2002). Consequently compensation associated with over-reliance on adaptive mechanisms, that limit self-regulation processes, will have a phasic and heterogeneous presentation, making it difficult to determine a common underlying mechanism in PTSD (Litz et al., 2000; Vasterling et al., 2004).

A review by Hull (2002) provides a profile of cortical activity in people with PTSD of predominantly increased amygdala activity, decreased hippocampal and medial prefrontal cortical activity, and increased activity in the posterior cingulate and visual cortices. However, understanding the functional links between brain regions in experimental research is limited by the inconsistencies and controversies surrounding the specific properties of the environment to which the brain has adapted. The different past histories of learning associated with stimuli, in people with PTSD, make the disturbances linked to a specific traumatic stressor event unique, and the presentation of the disorder heterogeneous (Litz et al., 2000; McFarlane and Yehuda, 2000).
In clinical research, the role of the right hemisphere in mediating various perceptual and motor functions of the left side of the body has had a long history of empirical investigation (Andreassi, 1995; Ley, 1980). Traditional ideas of hemispheric specialisation, related to human performance, have been useful for mapping connectivity patterns between cortical regions in psychopathology (Hugdahl and Davidson, 2004), with evidence in the PTSD literature showing that a decoupling between brain regions leads to psychological disturbances (Shaw et al., 2002; Clark et al., 2003; Lanius et al., 2005). Of particular interest to this study is the suggestion that generalised or heightened vigilance states are pathological processes associated with right hemisphere lateralisation (Arruda et al., 1999; Bearden et al., 2004; Thomsen et al., 2005; Vasterling et al., 2004). However, whether anxiety affects the articulatory loop but not the visuospatial scratchpad rehearsal system of working memory (Clark et al., 2003; Vasterling et al., 2004) represents an area of knowledge that has received limited investigation in the PTSD literature.

3.1.1. Lateralisation Hypothesis in PTSD Research

In reviewing a lateralisation hypothesis in PTSD research, few studies were found in support of a common underlying mechanism for a shift to right hemisphere processing in people with PTSD. A lateralisation hypothesis, as a premorbid trait, has not been conclusively supported in the PTSD literature. However, recent studies in PTSD investigating mixed laterality and dominance theories, have associated handedness preference to cerebral lateralisation for language as a marker for developmental abnormalities, and as a risk factor for developing psychopathology (Chemtob and Taylor, 2003; Saltzman et al 2006).

Saltzman and colleagues (2006) investigated the relationship between a history of interpersonal trauma in children (mean age in years: 10.6, ± 1.9) and mixed laterality with respect to handedness, with the aim of examining how laterality interacts with trauma in the context of the developing brain. The study was designed to examine differences between two clinical groups (subthreshold trauma exposure [PTSD-negative: N=31] and trauma exposure with full PTSD symptoms [PTSD-positive: N=28]) and a control group matched on gender, age, and race (no trauma exposure: N=40). The study found significant differences between the traumatised population and controls on gender and ethnicity. Saltzman and colleagues provided evidence that the degree of lateral preference is related to PTSD in traumatised children. Results indicated that a higher degree of mixed lateral preference was associated with positive correlated PTSD symptom severity, with the more symptomatic children
showing more mixed laterality. The authors interpreted the findings to show that increased rates of mixed handedness, rather than a shift away from right-handedness, were associated with risk for psychopathology, including expression of PTSD symptoms.

Evidence of handedness preference as a possible marker of risk in people with PTSD has also been demonstrated in the adult literature, but has been a contentious area of research in the wider literature (Beaton, 2004; Tremblay et al., 2004). In support of a hemispheric lateralisation hypothesis, Chemtob and Taylor (2003) demonstrated that mixed-handedness and parental left-handedness is associated with an increased risk of severity of PTSD symptoms. The authors posited that the right hemisphere plays a role in emotion regulation and reduced cerebral lateralisation for language functions, likely to be associated with PTSD. The results of mixed handedness studies are suggestive of a reduced capacity to manipulate encoded information during retrieval processes, and a reduced capacity for self-regulation, indicating biased perceptions, in those with mixed laterality preferences. This finding may be in agreement with proposed differences to the coordinated flow of information and diffuse versus long-range connectivity parameters modulated by attention mechanisms (Banich, 2004; Rueda et al., 2005).

Language allows for the rehearsal of perceptions and for biased perceptions to become entrenched through selective attention (Buckley et al., 2000; Hartikainen and Knight, 2003; Nadel and Moscovitch, 1998). Models of self-regulation and optimal behaviour implicate accessing information from memory and a reliance on language (Dayan and Yu, 2003; Rueda et al., 2005), which gives rise to feelings and mood states (Brewin, 2003; Joseph et al., 1997). In the study of psychopathology, there has been much debate on how two systems - self-registration associated with affective states, and self-regulation associated with cognitive states - are linked to differential mechanisms for the processing of cognitive information, and for distinct psychophysiological processes associated with general arousal and the processing of emotional stimuli (Hugdahl and Davidson, 2004; John, 2005; Posner, 2005).

Converging evidence in the wider literature has provided support for theoretical arguments indicating that mechanisms for the regulation of emotional expression involve processes of higher-order cognitive activation associated with prefrontal cortex (PFC) regions (Svoboda et al., 2006; Sotres-Bayon et al., 2006; Dayan et al., 2000; Yu and Dayan, 2002; Aston-Jones and Cohen, 2005). Reciprocal, dynamic interactions of the prefrontal cortex with the sensory cortex are thought to maintain an internal representation for coherent spatial and temporal
interactions during working memory and associated executive functions (Corbetta et al., 2002; Courtney et al., 1998b; Goldman-Rakic, 1996).

The two executive processes of selecting and shifting attention, and the execution of a response to that selection, have been linked to an anatomical overlap between frontal and parietal sites and have been well established in experimental research as important to an understanding of brain function (Awh and Jonides, 2001; Miyake et al., 2001; Nobre et al., 2000; Smith et al., 2003). Arguments advanced for the altered patterns of functioning have included views that lateral prefrontal cortex processing recruits dorsal and ventral areas for object and spatial features as specialised processes (Glassman, 1999; Goldman-Rakic, 1996; Linden et al., 2003). An alternative view is that the PFC is highly modifiable by behavioural training during tasks that alter the properties of neurons within regions (Ungerleider et al., 1998; Van Horn et al., 1998; Landau et al., 2004). Consequently, the two executive processes of attention can be dissociated to show that visual working memory is not supported when memory load affects capacity limits (Linden et al., 2003). This latter argument is in agreement with views that the PFC is not organised according to “domain specificity”, but according to “processing specificity”, and sequences of behavioural responses based on the retrieval of information (Landau et al., 2004; Ungerleider et al., 1998). A diagrammatic representation of overlapping cortical processes (Figure 3-1) has been formulated as a basis for understanding the role of executive function and empirical findings in the PTSD literature.

A model positing right hemisphere dominance, for the control of central functions that support survival and the human stress response in the infant brain, forms the foundation of a theory by Schore (2002). According to this model, the complex structure of PTSD is the result of the biological stress systems that are disrupted by overwhelming stress and maltreatment in childhood. The model postulates that inability to cope with stress results in long-term patterns of autonomic reactivity and affect dysregulation. According to Schore, the right brain is dominant for autobiographical or personal memory. This theory proposes that maltreatment in childhood is associated with an inability of experience-dependent learning strategies that are right hemisphere implemented to generate a coherent strategy for coping with the consequences of emotional stress.

Support for the subjective classification of information into event-specific codes, rather than temporal codes that can be manipulated in different contexts, has been provided by a number of different studies (Howe et al., 2003; Lou et al., 2005). Both animal and human studies have linked early childhood maltreatment to right-brain dysregulation and to a number of possible
mechanisms that are associated with structural alterations in the brain during experiential learning. These have been associated with increased hemispheric laterality and decreased hemispheric integration (Teicher et al., 2006).

In a more recent review, Nemeroff and colleagues (2006) indicate that the neural circuitry that has now been mapped, and that characterises PTSD to date, is thought to involve complex interactions between the thalamus, the hippocampus, the amygdala, the posterior cingulate, parietal and motor cortex, and the medial prefrontal cortex, including the anterior cingulate, orbitofrontal, and subcallosal gyrus. Of particular interest to this review, and as reported in the previous chapter, Nemeroff and others have suggested that category C symptoms are associated with high risk for PTSD, vulnerability toward compensation, and brain reorganisation patterns. Therefore, one question that arises is, how are the functional circuits reorganised? Furthermore, notions of environmental influences and stress resilience strategies are implicated.

Experimental research, from animal and human studies, supports the view that state-dependent learning modifies cortical neurons by mechanisms of cellular conditioning and acetylcholine-dependent potentiation (Shulz et al., 2000). Furthermore, at the local level of neural functioning, it has also been proposed that information is controlled by glutamate for response intensities and is then implemented through phase-locked loops at stable frequencies (Compte et al., 2000; Sauseng et al., 2005; Schack et al., 2001; Shulz et al., 2000; Thatcher, 1997). However, in human brain organisation, the function of thalamocortical loops has not been clearly investigated, particularly following severe traumatic psychological events. In the clinical literature, what is not understood is how a distributed memory system is characterised in the complex neural architecture of PTSD.

On the other hand, some evidence for disruption in the human brain has been provided by early developmental studies in PTSD, indicating that childhood stress and maltreatment experiences have been linked to a change in brain structure and function. Changes have been associated with the dysregulation of the cerebellar vermis, together with a reduced size of the corpus callosum and attenuated development of the left neocortex, hippocampus, and amygdala (Teicher et al., 2003). These findings are consistent with patterns of dysregulation in central processor mechanisms and may be related to pacemaker theories of cognitive implementation (Rowe, 2005; Schutter and van Honk, 2006; Ward, 2003). Various pacemaker theories of neuronal entrainment have argued for the local control of the dominant frequency (~ 10 Hz) by thalamocortical loops (~ 6 Hz) that are efficient at decoding temporally encoded
information (Kahana et al., 2001; Ward, 2003). These frequency oscillations have been shown to correlate with the functional roles of attention and working memory, respectively (Sauseng et al., 2002). At a mechanistic level, indices derived from neural oscillation frequency patterns have been shown to provide a useful method for analysing the parallel-distributed networks in cognition and affect in PTSD studies (Veltmeyer et al., 2006; Begic et al., 2001), and the dynamic processes involved in working memory functions (Attias et al., 1996; McFarlane et al., 1993; Veltmeyer et al., 2006) that may implicate neural plasticity (McFarlane et al., 2002; Sejnowski and Tesauro, 1989).

As previously mentioned, electrical or chemical characteristics of events alter the structural patterns in brain organisation (Dayan and Yu, 2003; Gazzaniga et al., 2002; Singer, 1999). This idea has been supported by recent evidence, with the suggestion that cerebellar involvement in cognition and emotion may be linked to the modulation of core electrophysiological frequencies (Schutter and van Honk, 2006). From the wider literature, a hypothesis for an integrated functioning of brain regions involves the cerebellum in a “cerebellar cognitive affective syndrome”. Schmahmann and Sherman (1998) proposed a model of neural circuitry supporting the idea that the cerebellum participates in the organisation of higher-order function by integrating multiple internal representations with external stimuli and self-generated responses in cognitive processing and emotion. Models implicating the cerebellum in higher-order cognitive functions may be useful in tying together evidence for emotional regulation and temporal relationships, associated with responding to stimuli, within theories of cognitive architecture for centralised and distributed processing mechanisms (Anderson et al., 2004; Aston-Jones and Cohen, 2005). Such models may address gaps in theoretical constructs, such as working memory, for the storage and manipulation of encoded information (Aron et al., 2004; Chein et al., 2003; Glassman, 1999), and represent important considerations in an analysis of lateralised cognitive functions in specific prefrontal regions in the study of psychopathology.

However, as discussed previously, cognitive processes are both spatially and temporally dynamic and do not correspond to brain regions in a fixed and stable way (D'Esposito, 2007; Fuster, 2003; Landau et al., 2004). Two regions of the prefrontal cortex that have been implicated in differences between cognitive functions include the dorsal and ventral streams (Clark et al., 2003; Jatzko et al., 2006; Aron et al., 2004; Sakagami et al., 2006). Various mechanisms correspond, not only to alterations in electro-chemical patterns for a disruption to structural channels for communication in neural networks, but also to a reduced ability to
optimise behaviour as a top-down function associated with the selection of goals and adaptive responses to stimuli (Yehuda, 2004; Litz et al., 2000; Dayan and Yu, 2003).

3.1.2. Specialisation Theories in Cognition

Specialisation theories have been linked to efficiency and the speed of processing in cognition, and as a compensatory mechanism in psychopathology, clinical studies have shown that hemispheric specialisation is advantageous to information processing efficiency (Kolb and Whishaw, 1996; Lezak et al., 2004; Hugdahl and Davidson, 2004). In a seminal thesis, Skinner (1950) postulated that alterations to efficient routines were linked to emotional arousal, implicating a temporal relationship between a stimulus and a response. In contrast, more recent research endeavours have demonstrated that different aspects of time-experience involve different functionalities and are experienced as entities with different meanings or emotions and involving anticipatory responses from fragments of information (Ramos and Savage, 2004; David et al., 2004; Nadel and Moscovitch, 1998; Fuster et al., 2000; Brewin, 2003). However, the temporal binding properties associated with the role of lateralisation in information processing, as a functional mechanism for coping with extreme stress, has not been accounted for to date.

Following this line of argument, theories of hemispheric specialisation proposing a left hemisphere interpretative function and a right hemisphere sensory integration function generally consider that memory for context is secondary to memory for content (Dhond et al., 2001; Nadel and Moscovitch, 1998). According to Gazzaniga and colleagues (2002), the process of ignoring content in an attempt to retrieve context requires greater processing cognitive resources. Furthermore, specialisation theories posit that interference from interpretative left hemisphere functions results in reduced functional organisation for right hemisphere perceptual memory (Gazzaniga et al., 2002).

In addition, based on the theoretical multi-component model of working memory (Baddeley, 1986), Courtney and colleagues (1998b) argue that laterality effects in working memory processes might be influenced by a variety of factors. In particular, they suggest that for visual working memory the distinction may not be as clear cut as a division based on object versus spatial processing, but may be better described as a division based on analytical versus image processing. Different researchers have advanced similar proposals, suggesting functional specificity might aid processing strategies (Linden et al., 2003), or differences in interpretative functions might be related to the categorisation of information (Nadel and
Moscovitch, 1998). In other words, the temporary storage of information can be incorporated into a conscious image after processing segments of information. In addition, Courtney and colleagues (1998b) hypothesise that some procedures are implemented automatically and in parallel, such as regenerating or sub-vocally rehearsing to assist the storage processes to maintain the contents.

An alternative theoretical framework, from the wider research literature, proposes that differences in the ability to rehearse and maintain information for further modality-specific processing are associated with specific components of the working memory model (Chein et al., 2003). Empirical studies have shown that auditory information has obligatory access to executive processes compared to verbal rehearsal (Chein et al., 2003); suggesting different pathways for the implementation and coordination of information, with greater efficiency in the auditory processing of information compared to visual or verbal information. These ideas have similarly been modelled by other researchers (Lisman et al., 2001; Logie, 1995; Mesulam, 1998), providing useful paradigms for the functional reliance on associative networks, between short-term storage and long-term memory.

Re-interpreted at a mechanistic level, and from a distributed-processing theoretical perspective, a supplementary view proposes that, depending on the strength of the input pattern of various stimuli and the maximum time constant of neuronal fatigue, neural structures can support the coexistence of both modes of short-term memory and long-term memory to allow for the persistence of a stimulated spatial pattern (Vollmer and Sommer, 2001). In modelling a neuronal parallel processing dynamic system, repeated exposure to a stimulus has been demonstrated to change cognitions as learning emerges over time, implicating episodic memory and hippocampal function (Eichenbaum and Fortin, 2005; Howard et al., 2006; Dayan and Yu, 2003; McFarlane et al., 2002).

However, these differences have not been made explicit in the empirical studies in the PTSD literature. For example, in an early study, modelled on the failure of extinction of fear responsiveness to trauma-related cues, Bremner and colleagues (1999) found evidence for the hypothesis that functional alteration in specific cortical and subcortical brain areas formed the basis of symptom categories of patients with PTSD and involved disruptions to memory, visuospatial processing, and emotional processing. Furthermore, understanding the shift in patterns of activation with task demands and practice effects is limited by the methodological techniques in research studies. Evidence that linguistic and visuospatial information may be processed by different systems and lateralised to different regions of the brain is abundant.
(Ungerleider et al., 1998; Fuster, 2003), yet there is limited insight into how these processing systems may actually differ in brain organisation (D’Esposito, 2007; Glassman, 2000; Courtney et al., 1998b). Memory retrieval, particularly visual long-term memory, is thought to be under the executive control of the prefrontal cortex, implicating reciprocal connections between the temporal and prefrontal cortices (Hasegawa et al., 1998).

From a psychological perspective, the study of memory mechanisms in PTSD enables an understanding of how past knowledge can influence and constrain behaviour. An understanding of the role of hippocampal structures in supporting the short-term maintenance of encoded information, and the retrieval of information corresponding to neural mechanisms of learning and plasticity, now forms an important area of investigation in PTSD research. However, the specific mechanisms of hippocampal dysfunction, and an assessment of the biochemical markers of medial temporal lobe neuronal integrity, have been inconclusive and inconsistent in the PTSD literature (Lindauer et al., 2005). The different approaches to studying hippocampal function in PTSD research (Davidson et al., 2004; Southwick et al., 1999) are outside the scope of information processing theories covered by this review.

3.1.3. Microcircuitry in Cognition: Spatial and Temporal Coding

Attempts to differentiate the impact of psychological stress from that of biological stress in traumatised populations have yielded inconsistent results due to a range of factors (McFarlane and Yehuda, 2000; Teicher et al., 2006). Conversely, a useful model demonstrating the loss of inhibitory functions and disruptions to learning and memory, in people with PTSD, has been associated with the idea that storage functions of working memory depend on distributed prefrontal-inferotemporal networks (Bryant et al., 2005; Galletly et al., 2001; McFarlane et al., 1993; Vasterling et al., 1998). Therefore, a critical component of the working memory model is the short-term storage buffers for verbal and nonverbal information (D’Esposito, 2007; Clark et al., 2000; Nadel and Moscovitch, 1998). Understanding the role of hemispheric asymmetry patterns is integral to functional differences in a fronto-parietal network (Linden et al., 2003).

In a study using PET neuroimaging techniques, Clark and colleagues (2003) found evidence of differences in the neurocircuitry between 10 PTSD participants and 10 age-matched healthy controls on a visuo-verbal task requiring the updating of working memory content. Specific regions for differences associated with executive function alterations between the clinical and non-clinical participants were identified. Cortical areas in the control group,
associated with significant increases in relative regional cerebral blood flow (rrCBF), were identified in the left precentral gyrus and the left middle frontal gyrus (MFG). Areas recruited by the PTSD subjects included the right supramarginal gyrus of the inferior parietal lobe (IPL), the right MFG of the posterior superior parietal lobe (SPL), bilaterally, and the right supplementary motor area. Differences between groups in brain regions recruited during the updating of working memory content included the supramarginal gyrus of the IPL, bilaterally and bilateral regions of the dorsolateral prefrontal cortex (DLPFC).

The results were significant in demonstrating a pattern of lateralised cortical activity in people with PTSD. Furthermore, the findings indicated specific regions of alteration in people with PTSD compared to normal participants. Clark and others (2003) interpreted a pattern of bilateral superior parietal lobe activation in the PTSD group as a dependence on visuospatial coding for working memory representations of trauma-neutral verbal information. On the other hand, according to Clark and colleagues, the reduced dependence on the left DLPFC for the monitoring and manipulation of working memory content in posterior regions of the brain was taken to mean ineffective use of executive control systems in people with PTSD. The secondary effects of disturbances in working memory processes, in people with PTSD, were proposed as the decoupling of frontal and parietal neural circuits, which are important for the selective encoding and retrieval of information (Clark et al., 2003).

Corbetta and colleagues (2002) have argued that the function of the prefrontal areas may be to retrieve stored information, and to provide the content of an expectation that is linked to visual areas via the parietal cortex. This hypothesis suggests that the neural physiological and psychological processes of alerting and vigilance play an important role in spatial working memory, modulated by attention (Corbetta et al., 2002). Of particular relevance to this review are suggestions, from the wider literature, that there are inconsistencies in the mapping of the phonological store and the speech-based rehearsal system to certain brain regions, which appear to have an overlapping representation of verbal and non-verbal information (Chein et al., 2003; Duzel et al., 1999; Linden et al., 2003). Furthermore, Chein and colleagues propose that these regions include the parietal cortex for the phonological storage properties, and Broca’s area for the speech-based rehearsal strategies. These are two regions that have been identified in the PTSD literature associated with working memory abnormalities and executive control functions (Clark et al., 2003) and implicate alterations to both spatial and temporal relationships for signal transmission and response binding.
In contrasting views, the functional role of retrieval processes has also been proposed as a framework within which to understand memory processes (D’Esposito, 2007; Nadel and Moscovitch, 1998). Such views have focused on the functional specificity of retrieval and differences between processing required for items that are manipulated compared to items that are maintained or not manipulated (Linden et al., 2003). Hence, the subjective components of memory incorporate tonic individual differences in memory processing, linked to the categorisation of stimuli, particularly stimuli with a self-referential base (Conway and Pleydell-Pearce, 2000; Nadel and Moscovitch, 1998). An examination of cerebral asymmetry patterns based on such models has provided a different framework for understanding the integrated flow of information processing in brain organisation.

Furthermore, a number of empirical studies support the hypothesis that cognitive difficulties in PTSD are not directly related to the trauma event, but rather suggest that the organisation of information may have altered in functional significance over time, implicating a dysfunction of frontal-limbic neural circuits (Bremner et al., 1993; Clark et al., 2003; Felmingham et al., 2002; McFarlane et al., 1993; Vasterling et al., 2006). However, the disparity of interpretations may be methodologically related to the specialised functional selectivity and anatomical localisation of functions in a distributed cortical system, indicating that an assessment of neuropsychological performance in people with PTSD does not provide conclusive evidence of the connectivity patterns between anatomical structures associated with learning.

A review of the evidence, and the theoretical basis for understanding compensatory mechanisms in people with PTSD, has raised questions concerning attention modulation, spatial attention mechanisms, and the involvement of specific cortical regions linked to cognitive deficits. Deficits in working memory systems may be related both to storage processes and to the manipulation and coordinated flow of information. Therefore, from the wider perspective of hemispheric specialisation theory, it will be argued in this review that processes of self-regulation subsume constructs applicable to emotional memories. Sub-processes associated with fear and anxiety, such as a selective attention bias, involving functions including awareness, alertness or vigilance can be conceptualised as response mechanisms in a distributed system (Dayan and Yu, 2003).

In a more recent PTSD study, Bryant and colleagues (2005) similarly concluded that a reduced capacity for executive functioning might be associated with a common mechanism associated with response to threat and involving cortical and sub-cortical systems. In a study
designed to assess cognitive functioning using a trauma-neutral selective attention task, Bryant and colleagues (2005) examined the question of whether medial prefrontal-amygdala dysregulation in PTSD is specific to threat-related stimuli, or generalises to more generically salient stimuli. Information processing was investigated using a non-verbal auditory oddball task, in which infrequent task-relevant stimuli are presented among frequent task-irrelevant stimuli. The fMRI study was conducted on community sample participants (N=14 PTSD, N=14 age and sex-matched non-traumatised controls). Results converged with previous findings of specialised functions in subregions and indicated lateralised brain functional connectivity patterns in people with PTSD. However, there were contradictory findings in the functional patterns associated with emotional responses.

Findings by Bryant and colleagues (2005) indicated that, in contrast to previous reports of diminished rostral anterior cingulate cortex (ACC) recruitment during threat/emotional processing in PTSD, cognitive processing of salient, non-threat stimuli involves enhanced dorsal and rostral ACC and left amygdala activity in people with PTSD. The authors concluded that responses pertaining to the dorsal ACC activations were associated with cognitive components and ventral-rostral activation was associated with affective components, indicating localised anatomical specificity of functions. In addition, the authors suggested that the relative reduction in DLPFC in PTSD indicates a reduced capacity for working memory and executive functioning and that activation of rostral and dorsal anterior cingulate, left amygdala and posterior parietal networks may reflect generalised hypervigilance.

Similarly, in a study by Britton and colleagues (2005), data from functional neuroimaging of trauma-specific personal emotional events in people with PTSD were compared with data from trauma-exposed control subjects. The study was designed to reveal altered patterns of neural substrates underlying functional changes resulting from the traumatic experience and aid in the detection of compensatory changes in brain function. To identify PTSD-specific and trauma-specific patterns of activation, Britton and colleagues (2005) used PET scanning during script-driven imagery of emotionally evocative and neutral autobiographic events. Data were collected on (N=16) combat veterans with PTSD, (N=15) combat veterans without PTSD and (N=14) healthy, age-matched non-combat control subjects.

Findings by Britton and colleagues (2005) of right insula activation in all three groups were consistent with previous research indicating the role of this region in personal recall of emotional events (Lanius et al., 2004). However, Britton and colleagues were not able to
explain a laterality difference of left insula correlation with skin conductance. Their results demonstrated differential patterns of activation between groups and were consistent with hypothesised predictions that post-trauma experience was associated with decreased amygdala activity. PTSD patients had greater rostral anterior cingulate deactivation compared with control groups, who deactivated ventro-medial prefrontal cortex (vmPFC). Additionally, according to the authors, findings of significantly less amygdala activation in both trauma-exposed groups, in response to traumatic/stressful scripts for trauma-specific investigations, were unexpected. This finding by Britton and colleagues was interpreted as a compensatory response in trauma-exposed individuals that was associated with a mechanism for successful emotion regulation.

Furthermore, according to Britton and colleagues (2005), failure to deactivate the vmPFC may reflect increased self-referential processing, consistent with models suggesting that the medial prefrontal cortex deactivates more extensively when attending to externally cued tasks relative to self-internal processes. This notion of secondary response strategies to retrieve context information may be similar to proposals discussed earlier, by Gazzaniga and colleagues (2002), who argued that reduced functional organisation of right hemisphere perceptual memory results from interference of interpretive functions.

However, PTSD symptoms have also been related to mechanisms of fear conditioning, extinction and sensitisation (Joseph et al., 1997), and associated with altered neurochemical systems (Davidson et al., 2004; Southwick et al., 1999; Yehuda, 2002). Relevant to such findings, one theory posits that fear is a knowledge structure for the implementation of mental algorithms in adaptive functioning (Anderson et al., 2004; Vogt and Laureys, 2005). Conclusions by Williams and colleagues (2006), suggest that the ACC is typically defined as part of the mPFC, and as suggested by Bryant and colleagues (2005), is distinguished by two separate systems of functioning for ventral and dorsal regions. Thus, as previously indicated by the above PTSD studies, evidence for right mPFC activity during fear processing has also been demonstrated (Williams et al., 2006).

Such models can be incorporated into an evolutionary framework, suggesting that knowledge structures are shaped by previous experience, and implemented through learning algorithms (McFarlane et al., 2002; Nadel and Moscovitch, 1998), via neurotransmitter and intracellular synaptic efficacies (Dayan and Yu, 2003; Shulz et al., 2000). This proposal can be linked to subjective event-specific autobiographical memory processes (Conway and Pleydell-Pearce, 2001; Shutter and van Honk, 2006; Svoboda et al., 2006), as a plausible chemical or electrical
characteristic of events, and as a mechanism for the interruption of integrated memories (Aston-Jones et al., 1994; Southwick et al., 1999; Banich, 2004).

3.2. Executive Functions and Attention Modulation

As a common underlying mechanism, both physiological and psychological alterations to the pattern of functioning in people with PTSD have implied alterations to attention mechanisms (Felmingham et al., 2002; McFarlane et al., 2002). As suggested in the previous chapter, attention mechanisms modulate functional implementation (Fan and Posner, 2004; Botvinick et al., 2004; Callejas et al., 2004). In the PTSD literature, a more specific hypothesis of attention deficits - as a threat perception bias (Litz et al., 2000; Buckley et al., 2000; Ehlers and Clark, 2000) - has been associated with vigilance and memory disturbances (McFarlane et al., 1993) and has been linked to frontal system arousal dysfunction for initial acquisition of information (Vasterling et al., 1998).

Attention is posited to be an essential element of self-regulatory mechanisms associated with the microcircuitry linking specialised regions for information transfer (Callejas et al., 2004; Fan et al., 2005; Thomsen et al., 2004; Ward, 2003). Attention control has also been linked to cognitive effort and to the extraction of information from stimuli (Cooper et al., 2003; Hanslmayr et al., 2005; Klimesch et al., 1998; Sauseng et al., 2005), particularly aversive or threatening stimuli (Kensinger and Corkin, 2004). Attention processing has been implicated as a shared resource and purportedly involves the same circuits as alertness (Felmingham et al., 2002; Hartikagen and Knight, 2003), with additional processing in frontoparietal association cortices (Clark et al., 2003; Linden et al., 2003).

Attention modulation and support for the multi-component model of working memory function is based on the fractionation of the frontal cortex into regions that perform specific processes (Goldman-Rakic, 1996). The interruption to the normal transmission of information through fronto-striatal circuitry has been postulated to be altered by the timing and flow of information, hindering processing and causing interference if redundant information arrives within a narrow time window (Rowe, 2005; Banich, 2004; Thatcher, 1997).

In agreement with previous literature, suggesting that PTSD is associated with impaired attention and mnemonic processes, the results presented by Vasterling and colleagues (1998), were consistent with disruption to the maintenance of information implicating attention and learning. The authors hypothesised that cognitive deficits linked to attention modulation
would be related to vigilance and hyper-arousal states, and that memory deficits would be related to disorders of arousal in frontal-subcortical systems associated with frontal system dysfunction, such as inefficient initial acquisition and errors of self monitoring. Results indicated distinctions for attention and memory mechanisms, suggesting that these deficits in people with PTSD were specific.

In a structural and functional analysis, Vasterling and colleagues (1998) included an assessment of intervening cognitive processes and error types rather than just proficiency performance. Community-recruited Gulf War veterans who met criteria for PTSD (N=19) were compared with a sample that did not meet criteria for current mental disorder (N=24). Their findings indicated that performance on sustained attention and vigilance revealed little evidence of PTSD-related attentional fatigue or loss of vigilance but did reveal limited ability by the PTSD sample to inhibit responses to irrelevant information.

Thus, the two distinct processes of attention and memory may be relevant to a lateralisation hypothesis of PTSD. Respectively, the first is an alteration to strategy and implementation style. The second is related to alterations to knowledge structures, which are updated and change over time through learning. However, the distinction between these processes in people with PTSD may be obscured by presentations of premorbid risk factors and with chronic patterns of reactivity, and remain an issue of debate in the literature (McFarlane and Yehuda, 2000). To understand the characteristics of PTSD symptoms at a mechanistic level, the role of neural plasticity has been proposed as a possible mechanism for the reorganisation of neural patterns over time, associated with the neuromodulatory effects of neurotransmitter systems (McFarlane et al., 2002).

3.2.1. Link between Neuroanatomical and Neuropsychological Functions

Various models provide a link between behaviour and functional brain patterns. At a mechanistic level, a brain-behaviour model by McFarlane and colleagues (2002) posits that compensatory mechanisms alter neural pathways in a distributed cortical system of memory functioning. As previously discussed, a general principle of cognitive architecture is the bi-directional flow of information along feed-forward and feedback connections, defined as a distributed system (Singer, 1999; Hoescher, 2001; Basar, 2004). Accordingly, memory is a dynamic property of the whole brain (Fuster, 1977), and the idea that hemispheric differences mediate emotional experiences and regulate behaviour has been linked to the role of working memory as a “top-down” feedback projection mechanism (Clark et al., 2000; Cabezza and Nyberg, 2000; Courtney et al., 1998a; Ward, 2003; Sotres-Bayon et al., 2006).
Cognitive processes involve integrating perceptual and conceptual information with stored knowledge (Nadel and Moscovitch, 1998; Gazzaniga et al., 2002), and relevant to rehearsal mechanisms, are the different conceptualisations of memory organisation (Fuster, 1997). These include the categorisation or classification of information into components and the sequential parsing of information based on chronology (Svoboda et al., 2006). However, the relevance of information is the central element in organising and unifying ideas and limiting incoming stimuli for rehearsal. Furthermore, words or phrases indicate a change over time through transitional organisation (Glassman, 1999; Gazzaniga et al., 2002; Nadel and Moscovitch, 1998). Thus, cognitive learning theories implicate cognitive structures, processes, and representations that mediate between experience and learning, where learning is defined as an internal process through memory and the retrieval of memories rather than environmental influences (McFarlane et al., 2002; Buckley et al., 2000; Nadel and Moscovitch, 1998). In other words, at a conceptual psychological level, what is important is not what happens, but how the event is interpreted.

One way of conceptualising the various models applied to cognition, which permits a synthesis of the different processes in a distributed system, is that the focus of mental activity moves from precepts or things, to rules that govern the behaviour of things (David et al., 2004; Dayan et al., 2000; Nadel and Moscovitch, 1998). This model allows for cognition to be defined as the behavioural outcomes of intermediary processing in the cerebral cortex (Mesulam, 1998), where functional selectivity exists in anatomically differentiated localisations (Basar, 2004). In distributed systems, when context information changes, the algorithms for functional implementation are updated (Fuster et al., 2000; Schutter and van Honk, 2006). Based on this reasoning, through learning, anatomical structural channels, for the representation of information or knowledge, can be altered by sensory stimuli or by interneurons (that is, cells which have inputs only from and outputs only to other neurons) (Basar, 1980; Munk, 2001).

In an assessment of the role of compensatory mechanisms in PTSD, one proposal is that neural plasticity is dependent on the establishment of a number of factors associated with learning or information processing (McFarlane et al., 2002). Furthermore, various theoretical models posit that molecular mechanisms or interneurons can alter the synapse (Sejnowski and Tesauro, 1989; Shulz et al., 2000) by transient circuits that can be formed by subplates of different pyramidal cell sectors (Munk, 2001; Basar, 1980). It has also been hypothesised that cortical reorganisation is linked to basic mechanisms that are known to mediate and modulate
synaptic plasticity (Nadel and Moscovitch, 1998). However, the precise mechanisms associated with the computational role of feedback connections, particularly for the operations of the prefrontal cortical circuit and the role of interneurons in transforming endogenous or exogenous inputs into behavioural outputs, are largely unknown (Tanaka, 2001; Sotres-Bayon et al., 2006).

Relevant to categorising information, mental resources for the processing of novel stimuli and the coordinated flow of information have also been modelled within the constraints of statistical probability for the ability to predict stimuli, involving attention mechanisms in decision processes, particularly important in the modulation of threat responding (Dayan et al., 2000). Optimisation models add another dimension to functional information processing components, and may be conceptually helpful to an understanding of how cognitive and affective information is integrated in distributed systems (Litz et al., 2000; Layton and Krikorian, 2002; Brewin, 2003).

Furthermore, the idea that uncertainty is associated with the switching between neurotransmitter systems for a shift between spatial and temporal codes (Dayan and Yu, 2003) is relevant to the findings presented in this review. For example, Dayan and colleagues (2000) propose that working memory functions rely on the statistical probability that an event will occur, and that the ability to make correct predictions about an event or an external stimulus will maintain confidence or certainty in decision processes. This model addresses, to some extent, a rationale for how the brain compensates when information is not available. In other words, based on appraisal mechanisms, the notion of statistical probability is calculated retrospectively as a timing device with which the brain can guess or predict how fragments of information will be integrated into memory structures (Litz et al., 2000).

In a parallel processing neuro-dynamic system, repeated exposure to a stimulus has been posited to change cognitions as learning emerges over time (McFarlane et al., 2002). Various studies have examined this hypothesis in the PTSD literature. In one study, reduced measures of neuropsychological performance were interpreted as specific deficits rather than global deficits (Stein et al., 2002).

Stein and colleagues (2002) investigated learning and memory problems in female victims of intimate partner violence (IVP). Three groups were measured, (N=39) victims of IVP (N=17 with current PTSD; N=22 without lifetime PTSD) and (N=22) non-traumatised comparison subjects recruited through community samples. Results of performance measures on tests of
attention, working memory, and executive function demonstrated reduced performance for the IVP subjects, regardless of PTSD status, on tasks of speeded, sustained auditory attention and working memory, and response inhibition. IVP with PTSD performed worse than non-traumatised comparison subjects on a set-shifting task. The results of the various aspects of neuropsychological functioning were taken to suggest that the cognitive deficits in IVP subjects were consistent with frontal-subcortical dysfunction in traumatised women. The findings for performance measures on tasks of verbal learning and memory demonstrated no evidence of deficits, irrespective of PTSD status. However, this conclusion by Stein and colleagues is in contrast to the differential deficits associated with attention and memory, as suggested by Vasterling and colleagues (1998).

One plausible hypothesis for the inconsistencies in the literature may be that the behaviour observed, and characterised by relationships inferred from post-perceptual responses, do not provide information about the rate of learning facilitated by the synaptic expression of memories in a complex system (McFarlane et al., 2002; Svoboda et al., 2006; Matzel and Shors, 2001). A limited capacity model of information processing is dependent on the timing of the relationships between presynaptic and postsynaptic signals (Basar, 2004; Thatcher, 1997). A consistent body of evidence has accumulated indicating that differences between novel and repetitive tasks are implemented with learning strategies via neural circuits for executive control and with the recruitment of specialised component working memory systems (Landau et al., 2004; Fuster, 2003). These views are in agreement with models proposing that learning algorithms influence neurotransmitter systems for the speed of processing, depending on confidence in stimulus appraisals (Dayan and Yu, 2003). Such theoretical models of learning have predicted that acetylcholine (Ach) is associated with faster learning in a dual-modularity system involving both Ach and norepinephrine (NE) (Berntson et al., 2003; Dayan and Yu, 2003; Gold, 2005; Nelson et al., 2005; Owen, 1997).

Alternatively, notions of temporal coding can also be interpreted as being convergent with dual-process theories of memory, which include models for understanding the nature of recollection and familiarity (Owen et al., 1996). Recollection has been described as an analytic, consciously controlled process, and familiarity as an automatic process, or a process described as perceptual information supporting awareness for “knowing” something (Nobre et al., 1997). Differences between familiar stimuli and recognised stimuli have not been conceptually examined in this review, but may be relevant to the timing mechanisms underlying the alterations to neural patterns in people with PTSD. Therefore, the relevance of the above-mentioned PTSD findings, and the respective interpretations, can also be
considered from the perspective that memory codes have an association with context information that is linked to a specific point in time (Hasegawa et al., 1998; Howard et al., 2006). According to this theory, the frequent retrieval of the same information alters the pattern of when information was first encoded (Howard et al., 2006; Lisman et al., 2001). This proposition is in agreement with a PTSD model proposed by McFarlane and colleagues (2002), who argue that over time, the knowledge base for functional implementation is altered and may represent a mechanism that, cumulatively, results in specific effects.

In addition, information related to context that influences the recall and reconstruction of memories for an item, may be congruent with biological theories hypothesising that alterations between “top-down” and “bottom-up” processes are based on the sensitivity and sensitisation of the noradrenergic neurotransmitter system (Southwick et al., 1999; Dayan and Yu, 2003). It has been suggested in the PTSD literature that differences in the pattern of responding, based on uncertainty of contextual information, imply alterations to the speed of information processing (Stein et al., 2002). These ideas are relevant to an understanding of why some people are at greater risk of developing PTSD symptoms and whether symptom types are associated with different neural patterns.

In an examination of emotional provocation, Jatzko and colleagues (2006) found an altered neuronal circuitry pattern in chronic PTSD sufferers. The findings were from data using fMRI, as a pilot study to investigate brain regions mediating positive information processing in chronic PTSD male subjects (N=8), compared to male controls (N=8). The study found an increase in right posterior middle temporal, precentral and superior frontal areas in chronic PTSD subjects, while the controls demonstrated a pattern of increased activation in emotional-related regions, bilateral in the temporal pole, and left in fusiform and parahippocampal gyrus.

Based on the hypothesis of right hemisphere dominance for emotional processing, and of the modulation of the ventral pathway by emotionally valent visual cues, the results reported by Jatzko and colleagues supported a compensatory pattern of emotional responses in PTSD. The authors interpreted their findings to suggest that the circuit for emotional processing, activated by the PTSD subjects, was consistent with increased effort in dorsal visuospatial information processing. This interpretation was based on the finding that the posterior middle temporal area is part of the dorsal visual stream, a region important in detecting biological motion.
Alterations to emotional processing in PTSD were demonstrated by frontal activation patterns and by less activation in regions generally activated by positive emotional film clips. In addition, the study by Jatzko and colleagues (2006) demonstrated a reduced left-sided fusiform and parahippocampal gyrus pattern of activation compared to controls. According to Jatzko and colleagues, a bilateral temporopolar activation pattern in the control subjects, but not in the PTSD group, indicated that the PTSD subjects had a different pattern of emotional and mnemonic processing and gating input to the amygdala.

Furthermore, Jatzko and others (2006) suggested that their results were convergent with previous results. These were taken to mean a reliance on a visuospatial processing circuit in PTSD, as described by Bremner and colleagues (1999), for a reorganisation of patterns during exposure to trauma pictures and sounds; and also convergent with results by Clark and colleagues (2003), for a reorganisation of circuits during verbal information processing. Jatzko and colleagues also suggested that the numbing of emotional responses in PTSD might be consistent with more effort on dorsal visuospatial information processing in PTSD.

Based on notions of content and context information processing, and event-specific knowledge (as discussed previously), the hypothesis by Jatzko and others (2006) posits a role for spatial working memory in understanding why some people are resistant to improvement after traumatic experiences, while others recover. The authors suggested that an alternative neural pathway might be associated with specific symptom types.

3.2.2. Fear and Knowledge Structures

The literature in a number of disciplines suggests that threat-related stimuli have a special propensity to attract visual attention processing (Fox et al., 2001; Sarter et al., 2005). A role for attention in the speed of responding to stimuli has been related to one hypothesis positing that the perception of fear is related to the timing of the event (Williams et al., 2006; Dayan and Yu, 2003). Fear has been defined as a basic emotion for the mobilisation of energy and is commonly associated with the principle of “fight or flight” (Cannon, 1914, cited in Yehuda, 2002). According to Gold (2005), adaptive forces can become stressors on the immune system and disturbing events that are particularly well encoded in emotional memory alter the physiology and phenomenology of the stress response and of self-perception. Furthermore, Gold (2005) posits that life-threatening situations are well remembered because the brain has evolved to consciously experience fear through the coordinated behavioural and physiological systems that generate anxiety and promote survival. Hence, anxiety and fear-related
phenomena emerge reflexively during threatening situations and attention is selectively biased toward the perceived threat, at the expense of maintaining attention on other elements in the environment (Corbetta et al., 2002; Gold, 2005).

In a recent study, Vasterling and colleagues (2006) conducted an epidemiological prospective controlled cohort study of active-duty military personnel, categorised by deployment status. The study was designed to measure both subjective and objective neuropsychological outcomes in US male and female Army soldiers deployed to Iraq, matched with non-deployed soldiers. Performance-based neuropsychological measures were used as continuous outcomes to identify relationships in populations between exposures and performance patterns indicative of brain dysfunction. Results revealed a significant deployment effect, in the primary outcome measures, as a function of deployment.

The study by Vasterling and others (2006) found that deployment was associated, at least transiently, with subtle alterations in neural functioning, as indicated by neuropsychological performance measures. The findings were interpreted by the authors to mean that negative neuropsychological outcomes, following Iraq deployment, could not be attributable to pre-existing dysfunction or intervening variables because of the relatively short time-span between war-zone return and assessment.

Furthermore, findings from the abovementioned study suggested that deployment is associated with neuro-behavioural advantage in reacting quickly and efficiently to simple targets, which was reported as a finding in contrast to the decrements in memory and attentional outcomes revealed by the performance measures. This finding was interpreted by the authors within an evolutionary framework to mean that, when confronted with life threat, physiological responses occur in preparation for life-preserving action and are associated with increased arousal in neurotransmitter systems, while neuroendocrine responses become altered via the hypothalamic-pituitary-adrenal (HPA) axis, possibly resulting in heightened behavioural reactivity but dampened attention, learning, and memory for non-threat-relevant stimuli and events. Furthermore, the authors concluded that emotional responses did not fully account for associations between deployment status and neuro-behavioural outcomes.

The suggestion by Vasterling and colleagues (2006), that alterations to cognitive function related to military deployment include secondary effects of dysregulation in brain function affecting day-to-day functioning, is a conclusion that converges with existing evidence in PTSD research for the disruption to working memory systems and adaptive functioning as a
generalised effect of trauma exposure (Galletly et al., 2001). Furthermore, the interpretation of results by Vasterling and colleagues (2006) may be a hypothesis in agreement with what is known about the neural physiological arousal mechanisms of perception (Shulz et al., 2000).

At the mechanistic level, neural models posit that the entrainment of neuronal patterns to rate-code representations of input frequency, through thalamocortical loops, is a mechanism whereby comparisons between cortical timing expectations and the actual input timing are detected through phase-locked loops (Munk, 2001; McFarlane et al., 2002; Rowe, 2005; Singer, 1999). From a psychological perspective, early responses to fear processing in people with PTSD have been proposed as a possible mechanism associated with disruption to cognitive functions (Vasterling et al., 2006; Williams et al., 2006). In agreement with the above views, and with empirical findings of a breakdown in frontal systems (Clark et al., 2003), alterations to the HPA axis have also been a topic extensively studied in the PTSD literature, with a disruption to these systems repeatedly demonstrated (Southwick et al., 1999; Yehuda, 2002). To interpret such changes within a working memory framework, the different levels of cognitive architecture for differences between function and structure (David et al., 2004; Banich, 2004), are useful conceptualisations for understanding alterations to neural pathways that are associated within specific regional and temporal parameters.

Uncontrollable and chronic stress reactions, mediated by the HPA axis cascade, have been associated with alterations to multiple neuroendocrine systems and linked to impaired hippocampal structures in PTSD sufferers (Bremner et al., 1992; Davidson et al., 2004; Southwick et al., 1999). Therefore, pathways mediating the stress response, that involve the release of catecholamines such as adrenaline or the activation of the locus ceruleus-noradrenaline (LC-NE) system for the release of noradrenaline, are central to an understanding of functional relationships between brain regions (McFarlane et al., 2002; Aston-Jones et al., 1994). However, a full discussion of these systems is outside the scope of this review.

Catecholamine innervation is thought to regulate the signal-to-noise ratio of information processing in prefrontal cortex neurons, consistent with goal-directed activity (McFarlane et al., 2002; Aston-Jones and Cohen, 2005). Noradrenergic projections in the prefrontal cortex are mediated by the modification of prefrontal connections with the sensory association cortices, to regulate the signal-to-noise ratio and to inhibit distracting and irrelevant sensory processing (McFarlane et al., 2002; Nelson et al., 2005; Yu and Dayan, 2002). According to a
model proposed by McFarlane and colleagues (2002), the hyper-catecholaminergic state in people with PTSD releases the inhibition typically maintained by the prefrontal cortex.

Convergent with the above views, Dayan and Yu (2003) argue that the glutamatergic and cholinergic transmission on the PFC is not bi-directional in nature and does not link regions in the same way that working memory pathways are linked. This conceptualisation of the loss of inhibitory mechanisms has been associated with efferent neural projections involving cholinergic neurons that are thought to be functionally associated with neuromodulation rather than neurotransmission (Gold, 2005). According to theoretical modelling by Dayan and Yu (2003), the release of ACh has been demonstrated to be unidirectional, so that ACh acts to deplete NE and to boost bottom-up connections, while suppressing top-down connections via nicotinic receptors, in the face of uncertainty or unfamiliarity (Nelson et al., 2005; Sarter et al., 2005). One suggestion regarding behavioural outcomes of decision processes is that ACh is involved in metaplasticity, depending on the neuronal structures that alter associations of stimuli when retrieved (Dayan and Yu, 2003). Thus, the theoretical basis of the Dayan and Yu (2003) optimisation model proposes neurochemical adjustments based on attention mechanisms and certainty in decision processes. These ideas might be relevant to tying together different explanations offered by the disparate studies involving activation patterns associated with connectivity patterns in different anatomical regions in people with PTSD.

3.2.3. Right-lateralised Effects of Neurochemical Pathways

The two pathways (HPA and LC-NE) are regulated through complex multiple feedback loops for the normal functioning of hippocampal cells and for autonomic arousal (Aston-Jones et al., 1994; Southwick et al., 1999; Sarter et al., 2005; Nieuwenhuis et al., 2005; Berntson et al., 2003). A review of the PTSD literature on catecholamine and HPA axis abnormalities by van der Kolk (2001), provides further support to changes in structure and function caused by stress and anxiety in people with PTSD. However, as reported by Hull (2002), identifiable trait effects were missing from research investigations – consequently right-hemisphere lateralisation of post-trauma symptoms could not be conclusively shown (Hull, 2002). Therefore, arguments for the allocation of processing resources to left or right hemisphere, depending on verbal or nonverbal processing demands, do not account for neuromodulation based on spatial and temporal information as two separate parameters in nonverbal information processing. This notion has received little attention in the PTSD literature and is an area of contention in the wider literature, as has been previously discussed.
Emotionally valent imagery as a cognitive function, compared to perceptual experiences associated with automatic emotional experiences, has been found to activate different neuronal circuits (Fletcher et al., 1995). Emotional information has been characterised along two dimensions, arousal and valence; where the former is excitatory or inhibitory and the latter is positive or negative (Kensinger and Corkin, 2004). These distinctions are relevant to underlying mechanisms for specific response patterns associated with imagery and visual perception, particularly in autobiographical memory retrieval.

The differences in cognitive processing, involving spatial or nonverbal working memory, by accessing two different neuronal pathways have been demonstrated in a study by Lanius and colleagues (2004). Evidence was provided for a right lateralisation in PTSD and a reliance on nonverbal coding during the retrieval of trauma memories. Lanius and colleagues used a script-driven imagery procedure adapted to fMRI to measure functional connectivity in regions of interest. The PTSD group, (N=11) subjects meeting full PTSD criteria, were compared with (N=13) subjects who never developed PTSD but met DSM-IV criterion A for trauma exposure. Brain activation patterns during script-driven imagery demonstrated activation in the middle frontal gyrus and various regions of the cingulate gyrus in both groups. Baseline brain activation did not differ significantly between the PTSD group and the comparison group, but significant between groups differences in functional connectivity were found.

PTSD subjects demonstrated greater correlation in the right posterior cingulate gyrus, right caudate, right parietal lobe, and right occipital lobe. In comparison, the non-PTSD subjects demonstrated greater correlations in the left superior frontal gyrus, left anterior cingulate gyrus, left striatum (caudate), left parietal lobe and left insula. Reciprocal connections between the cognitive and affective divisions of the anterior cingulate gyrus, during traumatic recall, was interpreted by Lanius and colleagues (2004) as indicating the linking of autonomic changes to specific emotional stimuli. Lanius and others hypothesised that the differences in functional connectivity patterns between the groups were related to variations in episodic memory retrieval, with flashbacks representing a different form of memory compared to autobiographical memories. The authors suggested that flashbacks are highly perceptual memories, primarily imaged-based, and are consistent with prefrontal activations during episodic memory retrieval with a clear tendency for right lateralisation. On the other hand, anterior cingulate activations during memory recall were associated with language processes, and it was hypothesised that the sensory elements of autobiographical memories were integrated into a personal narrative (Lanius et al., 2004).
In a later study by Lanius and colleagues (2005), the fMRI functional connectivity patterns of dissociative responses in PTSD were compared to a control group based on autobiographical script-driven imagery. The right anterior cingulate gyrus was a region postulated by Lanius and colleagues to be important in the pathophysiology of PTSD. The study was designed to examine personal memory retrieval and the differences between groups in neuronal networks for circuits involved in bodily states and in verbal autobiographical recall. The findings indicated a more right-lateralised pattern in the dissociative PTSD group compared to controls. For the dissociative PTSD group, functional connectivity maps were found for right insula, left parietal, right middle frontal, superior temporal gyrus and right cuneus. In comparison, the control group’s functional connectivity patterns were left ventrolateral thalamus, right parahippocampal gyrus, and right superior occipital gyrus.

The anterior cingulate and the insula have been implicated in the pathology of PTSD and have been shown to play a role in the subjective experience of the integration of bodily responses with behavioural demands and emotion. Lanius and others (2005) have argued that their findings were linked to a right hemisphere function associated with self-registration compared to self-regulation processing and left hemisphere function. The findings were interpreted to mean that people in dissociated states often have difficulties with perception of internal bodily states or self-awareness (Lanius et al 2005). The results and conclusions converge with those by Williams and colleagues (2006), who also argued that there are differences between classes of perceptions. That is, subjective interpretations related to bodily perception and emotional perceptions. As discussed previously, these conclusions are consistent with definitions of imagery and visual perception in the wider literature, and the allocation of the former in cognitive tasks, and the latter in emotional tasks (Fletcher et al., 1995).

Within the context of the working memory model, the results reported by Lanius and others (2004, 2005) are in agreement with studies in the wider literature arguing that the activation of processes in visuospatial buffering systems is suggestive of less capacity for verbal processing (Courtney et al., 1998a). This notion may be relevant to response patterns that are based on the underlying knowledge structures for functional implementation and for tonic or characteristic patterns of functioning. Preliminary evidence congruent with these ideas has been presented in the PTSD literature (Jatzko et al., 2006).

In addition to the above views, Bryant and colleagues (2005) concluded that the role of the ACC was essential in detecting patterns of incongruent stimuli, as a general function for
flexible behaviour. Furthermore, according to Gazzaniga and colleagues (2002), the categorisation of information often results in the false recognition of new items because information is interpreted and assimilated into a comprehensible whole by elaboration and integration with previously encoded information. Thus, the organisation of perceptual information can be compromised by interpretative functions (Brewin, 2003; Conway and Pleydell-Pearce, 2000; Svoboda et al., 2006; Nadel and Moscovitch, 1998). Included in this conceptualisation of functional specialisation, are the specific metrics of sensory stimuli, together with the rules that govern their implementation (David et al., 2004; Gazzaniga et al., 2002). However, the ability to detect accurate patterns in content and context information depends on various factors, including neurotransmitter modulation of neural assemblies; thus, confidence in appraisals remains to be clarified in people with exposure to extreme traumatic stressors.

Memory performance has been associated with greater involvement of executive functioning in visual memory tasks compared to verbal memory tasks (Linden et al., 2003; Glassman, 1999). In addition, as previously discussed, there is a known overlap in some regions that support working memory processes for verbal and spatial information (Awh and Jonides, 2001; Chein et al., 2003). Furthermore, different pathways, as a function of the type of visual information being stored, have been demonstrated using a working memory model for the processing of object and spatial information (Courtney et al., 1998a; Owen et al., 1996). As previously discussed, the functional attributes of items include processes such as whether an item can be manipulated, or not, in which case it is maintained (Duzel et al., 1999; Linden et al., 2003). To add to the complexity of understanding memory functions, processes of visual imagery, visual knowledge, and visual perception have been found to be dissociable in the wider literature (Fletcher et al., 1995; Miyake et al., 2001). However, these differences have not been made explicit in the empirical studies in the PTSD literature (Vasterling et al., 2004).

Brain-behaviour relationships, or learning at the local level, can be understood by quantifying the underlying cognitive processes in behavioural activation tasks (Lezak et al., 2004). A key question is whether behaviour can index neural changes.

In a review of the PTSD literature, Hull (2002) concluded that abnormalities reported in limbic and paralimbic structures during symptom provocation and cognitive activation studies may not be specific to PTSD, but rather implicate the effects of neuronal plasticity mediated by N-methyl-D-aspartate (NMDA) pathways activated by stress-induced chronic trauma memories. According to Hull, studies have shown that increased automatic amygdala responsiveness to stimuli is accompanied by decreased activity of the prefrontal cortex, which
has a role in the encoding and retrieval of verbal memories that are modality-specific and region-specific based on the re-experiencing phenomena. Conclusions drawn by Hull (2002) suggested that PTSD is a progressive state of modification, as demonstrated by biological and neurobiological changes associated with alterations in brain function and structure.

Convergent with the ideas presented for the spatial and temporal coding of information in brain organisation, conclusions drawn by Neylan and colleagues (2003) suggest that it is possible that PTSD states are not tonically hypervigilant or hyper-responsive, but rather have an unstable neurocognitive system, involving attention modulation related to environmental context and other factors that may influence the allocation of attentional resources.

Neylan and colleagues (2003) used ERP components to examine the temporal stability of auditory and visual P300 measures in PTSD and control subjects to determine if the relationship of changes in ERPs was related to changes in symptom levels. Male subjects with combat-related PTSD (N=25) and male combat-exposed normal controls (N=15) completed a three-condition novelty oddball task at two time points separated by 6-12 months. The findings revealed no significant differences in P300 amplitude or latency in PTSD in comparison to control subjects at either time point, regardless of stimulus type or modality. According to the authors, variability of P300 measures over time was not associated with fluctuations in symptoms of depression or PTSD. The results were interpreted as a deficit in attention modulation in people with PTSD.

Thus, converging evidence in the PTSD literature suggests that neural patterns can be reorganised over time for the implementation of tonic, or stable, mental algorithms based on knowledge structures and subjective interpretations of stimuli (Clark et al., 2003; Vasterling et al., 2006; Lanius et al., 2005; Williams et al., 2006). An increasing body of evidence is accumulating to show that the timing and flow of information in people with PTSD is altered by attention modulation (Attias et al., 1996; Bryant et al., 2005; Felmingham et al., 2002; Vasterling et al., 1998).

3.2.4. The Secondary Effects of Shared Resources

Associated with traumatic experiences, and the meanings assigned to those experiences, two neural pathways contributing to the reorganisation of function in PTSD were reviewed in the previous chapter. It has also been hypothesised that with the progression of time, after the traumatic event, self-regulation of conceptual and perceptual parallel pathways may become
biased through processes of selective attention and secondary characteristics for the voluntary control of cognitive processes and psychological performance (McFarlane et al., 2002). From an evolutionary perspective, responses to fear were demonstrated as cognitive structures for the implementation of knowledge gained from experience (Bryant et al., 2005; Vasterling et al., 2006). Arousal mechanisms for the maintenance of inhibitory functions, and a selective attention bias, have been linked to associative networks for retrieval from long-term memory (Dayan et al., 2000; Fuster et al., 2000; Vasterling et al., 2002).

In a PTSD study, based on the idea of associative network models, Litz and colleagues (2000) predicted that a trauma prime would lead to a cascading, spreading activation process, influencing emotional behaviour in a mood-consistent manner. As has been discussed, both fear and selective attention mechanisms have since been associated with the loss of inhibitory functions in PTSD, contributing to reaction patterns and a disruption to cognitive functioning (Vasterling et al., 2006; Bryant et al., 2005).

Litz and colleagues (2000) assessed emotional experience and expressive behaviour in (N=61) Vietnam war-zone exposed veterans assigned to PTSD diagnosis (N=32) and well-adjusted veterans (N=29). The authors predicted that expressive behaviour and evaluations or judgements of affective states are linked, and that reactivity and associated re-experiencing problems in posttraumatic adjustment are based on the primacy of the conditioned aversive context eliciting a state-specific differential emotional-processing deficit. However, the authors found that results were inconsistent with the generally accepted concept of emotional numbing in PTSD. Rather, the findings by Litz and others demonstrated that Vietnam War veterans with PTSD and well-adjusted veterans responded comparably and distinctly to three categories of emotionally valenced stimuli (positive, neutral, and negative).

According to Litz and colleagues (2000), the results were consistent with a model suggesting that emotional-processing deficits in PTSD are secondary, at least in part, to reactions to trauma reminders. The PTSD participants, in contrast with control participants, were less responsive to positively valenced emotional stimuli only after they were exposed to a trauma-related priming challenge. Litz and colleagues suggested that the degree of ambiguity and uncertainty of contextual cues were a possible factor in the unexpected result of the PTSD rating for hedonically neutral stimuli. The finding of increased heart rate in the PTSD group, to all emotional stimuli, was also interpreted as an inability to anticipate and predict the emotional demands of each trial.
However, the finding of increased heart rate in the PTSD group by Litz and colleagues (2000) was not interpreted as a rhythmic shift in arousal mechanisms associated with functions of pattern-matching and selective attention. Rather, Litz and others questioned the validity of the symptom class of emotional numbing in PTSD. The authors proposed that PTSD is associated with a “vigilant readiness” to respond to uncertain contexts, arguing that this state does not alter the capacity for expressive behaviour. This view concurs, in part, with a model for behavioural optimisation proposed by Dayan and Yu (2003); also proposing that differences in neural activation are dependent on appraisals based on certainty or uncertainty, with consequent alterations to neurotransmitter systems.

Further support for distinct regional activation patterns was provided in an extension of the above models. Kashdan and colleagues (2006) sought to clarify how symptom categories in PTSD overlap, and more specifically, the functional role for anhedonia in psychopathology. Their study found that anhedonia had a unique positive relationship with PTSD emotional numbing symptoms and minimal relationships with other negatively valenced and avoidance-related PTSD symptoms. In a sample of veterans with military-related trauma, the results by Kashdan and others revealed an independence of symptoms reflecting aversive functioning (high levels of negative affect) and appetitive functioning (low levels of positive affect). Kashdan and colleagues argued that a distinction between appetitive and aversive functioning might provide insight into the nature of PTSD, and emotion regulation deficits.

Therefore, it remains unclear whether maladaptive coping strategies contribute to the secondary cognitive impairments identified in PTSD (McFarlane et al., 1993; Galletly et al., 2001), or whether compensatory mechanisms, consistent with increased recruitment of certain cortical regions, contribute to the expression of pathology in PTSD associated with deficits in left prefrontal regions (Clark et al., 2003; Bremner et al., 1999; Lianus et al., 2004).

### 3.2.5. Functional Reorganisation in PTSD

There is now a significant body of evidence that PTSD sufferers demonstrate deficits in memory for trauma-neutral information, which has been associated with hemispheric asymmetry patterns at the neural level of analysis (Clark et al., 2003; Bryant et al., 2005). In addition, evidence is also emerging that scalp electrical and neuroimaging indices of brain activity in PTSD are associated with deficiencies in working memory operations that have been hypothesised to be associated with a functional decoupling in the frontoparietal neuraxis (Galletly et al., 2001; Shaw et al, 2002). These findings implicate overlapping neurobiological
substrates in memory processing, which has been a controversial topic of investigation in the wider literature in understanding storage and retrieval processes in working memory systems (Linden et al., 2003; Nadel and Moscovitch, 1998).

In a study investigating the efficacy of a combined multivariate/resampling procedure for the analysis of positron emission tomography (PET) activation studies, Shaw and colleagues (2002) examined distributed brain systems for (N=10) PTSD community sample participants and (N=10) healthy controls matched for age, sex, premorbid intelligence, and years of education, by using an updating working memory paradigm. The study provided direct evidence of a difference in functional connectivity between PTSD patients and controls.

Findings by Shaw and colleagues (2002) demonstrated differences between the groups in the cerebellum, right gyrus rectus (overlapping the cingulate gyrus), precentral gyrus, right inferior temporal gyrus (overlapping middle temporal gyrus) and the right inferior parietal lobe. Shaw and colleagues found increased activity in the parietal and motor regions, which was taken to reflect increased dependence on visuospatial memory and preparation for action during the stress response. Furthermore, a decrease in the orbitofrontal gyrus/gyrus rectus was interpreted as a region involved in the recovery of extinguished fear; while the finding of a decrease in the right middle temporal gyrus was interpreted as a region with a role in declarative memory. The results presented by Shaw and colleagues were convergent with findings by Bremner and others (1999) of an increased dependence on nonverbal working memory areas in PTSD patients, as a strategy for coping with decreased verbal memory abilities.

However, Shaw and colleagues (2002) extended the model to interpret the results for the decreased bilateral activation of the middle frontal gyri, suggesting that these areas play a role in executive functions concerned with working memory processing. In addition, the increased cerebellum activity in the PTSD group was interpreted as the detection of a disparity between intention and action, based on the measured verbal target stimulus. As indicated by the authors, this interpretation was in agreement with previous working memory studies suggesting an association of the cerebellum with evaluation functions in verbal coding as opposed to nonverbal coding. However, few studies have included the role of the cerebellum in PTSD research and it is possible that this region plays a key role in altered rates of coding, corresponding to the loss of inhibitory frontal system mechanisms, as has also been suggested in recent research in the wider literature (Shutter and van Honk, 2006).
A more recent area of investigation in the PTSD literature has focused on understanding the specific patterns of neural activity associated with subtypes of symptoms and the cortical pathways involved in compensatory strategies (Metzger et al., 2004; Kashdan et al., 2006). The various studies presented in this review, as examples of lateralised functioning in people with PTSD, have illustrated common areas of dysfunction and various alternative hypotheses for the compensatory mechanisms associated with the interruption to the flow of information in a distributed memory system.

In PTSD research, biased perceptions of trauma-related triggers and a decrease in attention to non-trauma related stimuli have been associated with disturbed concentration, and general emotional numbing responses (Felmingham et al., 2002; McFarlane et al., 2002). In various analyses, using functional magnetic resonance imaging (fMRI) and script-driven symptom provocation paradigms, Lanius and colleagues (2001, 2002, 2003, 2004, and 2005) have also demonstrated differences in PTSD brain connectivity patterns of inter-regional brain activity. Evidence of connectivity patterns involving networks implicating a nonverbal pattern of memory recall were found in patients who had a hyperarousal response to traumatic transcript-driven imagery (Lanius et al 2004). The authors hypothesised that abnormal connectivity among regions involved in the recall of traumatic material may be an important mechanism, not only for understanding the encoding of traumatic memories, but also in distinguishing neural networks underlying the different anxiety disorders.

3.3. Compensatory Mechanisms in PTSD

As a summary, the empirical evidence reviewed in this chapter, reveals an accumulating body of knowledge, from neuroimaging and activation studies, demonstrating that distinct cortical pathways, involving regions of the prefrontal, medial prefrontal, and cingulate cortices, are disrupted in people with PTSD (Lanius et al., 2005; Jatzko et al., 2006; Britton et al., 2005). In addressing the question of what leads to compensatory mechanisms in people with PTSD, one line of evidence indicates that control processes for the inhibition of irrelevant information are disrupted through attention mechanisms (Clark et al., 2003; Bryant et al., 2005; Vasterling et al., 1998). Alterations to executive control functions, and in verbal coding of information, have been implicated as common mechanisms in the disorder (Clark et al., 2003; Bremner et al., 1999; Jatzko et al., 2006).

One interpretation of this finding is that functional changes may relate to an increasing number of new sensory triggers in people with PTSD (Bryant et al., 2005), and according to
some researchers, a decreased threshold for amygdala activation as a result of dysfunctional medial prefrontal cortex and anterior cingulate gyrus modulation (Bryant et al., 2005; Hull, 2002; Nutt and Malizia, 2004; Williams et al., 2006). On the other hand, related to attention modulation, the role of neurotransmitter pathways and the speed of processing associated with the interaction of cholinergic systems have also been proposed as a plausible mechanism for compensation in people with PTSD (McFarlane et al., 2002; Aston-Jones et al., 1994; Southwick et al., 1999).

However, distinct biological markers that define the disorder as “unique” have not been validated. Empirical findings have suggested disturbed memory in PTSD is consistent with a common environmental cause. On the other hand, PTSD symptoms have also been referred to as the secondary characteristics of traumatic events (McFarlane et al., 2002), with one proposal suggesting that numbing responses are linked to cognitive processes (Felmingham et al., 2002; Kashdan et al., 2006; Litz et al., 2000), and activated in different environmental contexts (Neylan et al., 2003).

From a psychophysiological perspective, Felmingham and colleagues (2002) demonstrated that specific patterns of functioning, associated with attention disturbances and executive deficits, were relevant to symptom types. The authors examined the relationship between disturbance in event-related potentials (ERPs) and symptom clusters in PTSD. ERPs were recorded in (N=17) unmedicated civilian PTSD patients and (N=17) age- and sex-matched controls during a conventional auditory oddball task. The results replicated findings of disturbed N200 and P300 ERP components in PTSD, and provided converging evidence for a stimulus discrimination and attention impairment in PTSD (McFarlane et al., 1993). Numbing symptoms were associated with reduced P300 amplitude. The authors interpreted this result as a reduced attentional response, consistent with models positing a relationship between disordered arousal and attention in PTSD.

In a more recent examination of this proposal, Kashdan and colleagues (2006), also argue that avoidance responses are associated with a disruption to arousal and attention mechanisms. However, Kashdan and others propose that numbing is associated with appetitive functioning, or low positive affect, compared to avoidance, which is associated with aversive functioning. This interpretation of attention disturbances and numbing responses posits two distinct pathways for information processing and response binding, implicating differences depending on subtypes.
In a methodologically different study, the hypothesis that uncertainty or apprehensive arousal is linked to different cortical patterns has also been demonstrated in the PTSD literature. Metzger and colleagues (2004) examined the relationship among PTSD, anxiety, and depressive symptoms and frontal, temporal, and parietal EEG alpha asymmetry in female Vietnam War nurse veterans. Three groups were assessed: (N=18) current-PTSD, (N=14) past-PTSD, and (N=18) non-PTSD. In an attempt to maintain a homogeneous sample, a number of other measures were taken, including criteria for major depressive disorder (MDD), and the CAPS subscale scores, which provided measures of severity of PTSD symptoms. The results demonstrated that PTSD arousal symptoms are associated with increased right-sided parietal activation. Results also suggested that the combination of arousal, depression, and their interaction in right-sided posterior activation is specific to the anxious arousal subtype. This study demonstrated a more specific pattern of regional alteration in people with PTSD.

The results presented by Metzger and colleagues (2004) support previous models (McFarlane et al., 2002), that the pattern of functioning changes over time through learning and through neural plasticity to become a stable trait. Biological markers of trait patterns have been reliably characterised using EEG methodology (Allen et al., 2004), and the alpha frequency has been posited as a genetic and stable trait (Smit et al., 2005). According to Metzger and colleagues, alterations that occur at the local level of information processing may be detected by regional activation patterns and isolated statistically.

In the wider literature, EEG parameters have been used in an assessment of affective states (Allen and Kline, 2004) for a reliable examination using frontal EEG alpha frequency parameters. However, results of background spontaneous activity in EEG recordings has not been consistent or highly replicable (Jausovec and Jausovec, 2000). On the other hand, in the PTSD literature, the results provided by Begic and colleagues (2001) and Jokic-Begic and Begic (2003) consistently demonstrated increased beta power in people with PTSD. The pattern of results also converge with views presented by McFarlane and others (2002) that alterations occur at the local level of information processing as a result of neural plasticity.

Begic, Hotujac, & Jockic-Begic (2001) investigated the biological basis for PTSD using quantitative EEG methodology to assess differences between patients with PTSD and healthy controls. Veterans with PTSD (N=18) were compared with healthy non-veterans (N=20) for power values of particular frequency bands (delta, theta, alpha1, alpha2, beta1, beta2) with 8 homologous pairs of scalp electrodes. Results revealed increased theta activity in the PTSD
group over central regions, and increased beta1 activity over frontal, central and left occipital regions and increased beta2 activity over frontal regions. There were no differences found between groups in delta and alpha activity. The authors interpreted the results to mean a neurological basis for PTSD in tonic or resting states. Increased theta activity was suggested as a mechanism explaining changes in hippocampal volume. On the other hand, increases in beta activity in PTSD patients were not explained, but related to a number of factors. Suggestions included global cortical hyper-excitability, prolonged wakefulness, or attention disturbances. As an alternative explanation, the authors proposed that medication effects might be a likely hypothesis for the results. In addition, the authors proposed that to understand beta rhythm activity, the classification of PTSD participants into subtypes according to arousal, avoidance, and intrusion phenomena would be useful.

In a later study, Jockic-Begic and Begic (2003) compared two groups of veterans (PTSD (N=79) combat veterans and non-PTSD veterans (N=37)) using qEEG data for differences in cortical activation characteristics. Results revealed decreased alpha power and increased beta power in the PTSD veterans. PTSD subjects demonstrated suppression of alpha1 over frontal, central and occipital regions, more pronounced over the left hemisphere. In the alpha frequency band, data demonstrated a decreased lower alpha1, which was interpreted as an attention deficit characteristic for PTSD. For the beta frequency band, data demonstrated that beta1 activity increased more in patients with PTSD than controls, in particular over frontal and central regions. Results were taken to suggest an altered neurobiology in PTSD that was possibly correlated with emotional reactivity and residual effects of medication use. Furthermore, Jockic-Begic and Begic maintained previous conclusions that the clinical heterogeneity of the participants may have contributed to the inconsistency of results.

In a synthesis of the different studies presented, various hypotheses have provided evidence of lateralised functioning in people with previous traumatic stressor experiences, including an identification of pathways for inter- or intra-hemispheric asymmetry. A growing body of evidence has been presented to suggest that PTSD symptoms and disturbances in memory functioning may be associated with specific asymmetry patterns. These are associated with specialised working memory systems for the implementation of functional states linked to compensatory mechanisms. It has been demonstrated that distinct circuits and regions are disrupted in people with PTSD. However, the exact mechanisms for compensatory shifts in neural activity have not been clarified.
One proposal for a common mechanism suggests attention modulation and the alteration in patterns of verbal and nonverbal working memory structures. This proposal is consistent with suggestions that spatial and temporal constraints in rhythmic oscillation patterns are associated with the implementation of functional codes. At a local level of investigation, the pattern of anticipatory electrocortical resting states enables the characterisation of the stable patterns underlying cognitive operations that will determine connectivity between neural regions (Basar, 2004). Furthermore, a modularity thesis would enable a theoretical and statistical examination of the different dimensions of appraisal mechanisms for a mechanistic interpretation of decision processes, and may provide a useful avenue for clinical research. Such an analysis would improve our understanding of the psychological and physiological properties of attention mechanisms between component stages of processing. This idea might be an important concept in clinical research for an understanding of how to redirect behavioural activation associated with conditioned processing and will contribute to a further understanding of early and late stages of processing in people with PTSD.

3.3.1. Summary: Functional Cerebral Asymmetry

The mechanisms by which information is transferred in neural systems remain to be determined. Perceptual asymmetries, associated with the early stages of encoding, as well as neuromodulatory mechanisms associated with later stages of information processing (Vasterling et al., 1998, 2006), or rehearsal substrates, as well as retrieval processes (Bryant et al., 2005; Jatzko et al., 2006), have been proposed to account for functional deficits in people with PTSD. In support of a lateralisation hypothesis, neural circuits in people with PTSD may be altered through conceptualisations of self-registration and self-regulation (Litz et al., 2000). However, specific subtypes associated with symptom patterns, particularly for differences between numbing and avoidance characteristics, and the neuronal patterns associated with content and context memories that activate certain pathways, remains as an area requiring further investigation.

Therefore, one set of general questions raised from this review of the empirical literature points to localising the deficits at a structural level of analysis (Bremner et al., 1999; Clark et al., 2003; Protopopescu et al., 2005; Williams et al., 2006). Recent directions in PTSD research have focused on the microcircuitry of information processing in distributed networks (Metzger et al., 2004; Jokic-Begic and Begic, 2003). The question of whether lateralised patterns, at the local level of information processing, can be associated with specific regions for disruption to cognitive processes remains an issue open to investigation in PTSD research.
In particular, the integration of content and context information, conceptualised as self-regulation and self-registration, requires further clarification from the perspective that memory of a traumatic event is a secondary characteristic related to neural plasticity (McFarlane et al., 2002). The empirical findings suggest specific disruption to brain regions in PTSD, the dorsolateral prefrontal cortex (DLPFC) and the anterior cingulate cortex (ACC) showing distinctive patterns of disruption to memory and attention, implicating a specific role for working memory (WM) systems in people with PTSD. Learning, memory and attention paradigms have provided converging evidence of cortical and subcortical abnormalities (see Figure 3-1).

In addition, the suggestion that numbing and avoidance responses may be linked to distinct pathways for responses to stress (Kashdan et al., 2006), implicate higher-order information processing mechanisms related to learning and a reorganisation of brain function (McFarlane et al., 2002). To understand the role of compensatory mechanisms in PTSD, the above ideas form the starting point for this study. One question based on these ideas is whether hemispheric asymmetry at the structural level of analysis can predict retrieval processes at the functional level. For an understanding of the role of hemispheric asymmetry in PTSD, the relationship between cognitive processes and local level biological processes is examined using a working memory model and a parallel distributed system perspective of brain organisation.
Figure 3-1. Executive functions have been shown to involve an overlap in cortical processing systems.

Note: Disruption to information processing in PTSD may be specific to a lateralised coding pattern in working memory systems (Clark et al., 2003) and modulated by selective attention mechanisms (Bryant et al., 2005) for a reorganisation of brain function over time (McFarlane et al., 2002).
4. **Objectives and General Methodology**

4.1. **Objectives and Scope of Study**

The general methodology for this research, and the objectives and rationale for the selection of measures for three separate analyses conducted, is contained within this section. However, a more detailed methodology section is presented within each of the relevant chapters.

The main objective of this research was to examine structural and functional indices of hemispheric lateralisation patterns between PTSD participants and normal control participants, matched for age, gender, and years of education. The relationship between cortical resting EEG activation patterns and verbal and nonverbal working memory functions was investigated for an increased reliance on right hemisphere processes in people with PTSD.

Using quantitative electroencephalographic (qEEG) methodology, the first question addressed by this research focuses on differences in localised baseline resting patterns between people with PTSD and age and gender matched controls. Behavioural patterns for specialised left and right hemisphere working memory processes are examined using neuropsychological indices for compensatory asymmetry patterns in PTSD.

A second question examined the influence of high stress responses in both clinical and non-clinical participants by investigating the effect of clinical symptoms on working memory retrieval functions and preparatory resting EEG frequency patterns. By dividing the control group into those participants who had experienced traumatic stressful events, but were not diagnosed with a clinical condition, the differences in lateralised functioning between three groups were compared for localised deficits.

The third question focused on the predictive relationships between cognitive functions, alpha asymmetry, and self-reported reaction patterns to perceived traumatic stressors, as a comparison between groups.

As a retrospective analysis, the variables examined in this study were not explored for intermediary processes or sub-components in working memory systems. Furthermore, self-report questionnaire items, examining reaction patterns to traumatic stressor events, is presented as a general and exploratory investigation of trauma reactivity rather than an objective measure of characteristic response patterns to specific events. The current study was
designed to generate variables of interest, particularly for the DSM-IV category C criteria (American Psychiatric Association, 1994), which may be useful to future endeavours aiming to deepen an understanding of the mechanisms associated with specific symptom characteristics and brain organisational patterns in people with PTSD.

4.2. General Methodology

**Role of Standardised Datasets:**

The role of standardised databases has been discussed in various domains of research, outlining the benefits of providing large sample sizes for statistical testing and an accepted standardised methodology to account for a broad range of individual differences (Gordon et al., 2005; Niedermeyer and Lopes da Silva, 2005). Standardised datasets provide information on a wide diversity of measures, together with large numbers of participants for both normative and clinical population samples. Furthermore, a range of techniques permits specialised analyses using neuropsychological, electrophysiological, and psychometric parameters for an integrative assessment of brain and cognitive function. Another advantage is that datasets enable the matching of control participants for planned analyses with clinical populations on a number of variables, such as age and gender (Gordon et al., 2005).

Data from a recognised international standardised database, Brain Resource International Database (BRID), was accessed for this study and is duly acknowledged. Information on the major operating procedures of the database, the methods, procedures, and equipment employed in the acquisition of data, as well as reliability and validity studies conducted on the different measures collected, can be accessed in the literature (Clark et al., 2006; Gordon, 2003; Gordon et al., 2005; Williams et al., 2005).

Data for BRID is acquired using identical equipment and experimental procedures and test batteries from a number of laboratories, including international research facilities (Gordon et al., 2005). The non-clinical data is collected as a reference population and normative database (for full details see Gordon et al., 2005). Information for the centralised database is recorded, stored, and accessed for research purposes only with participants’ informed consent.
4.2.1. Standardised Measures and Procedures

Data for this study were collected according to standardised procedures implemented by a consortium of laboratories locally and internationally. Data collected by the Adelaide laboratory, for the purposes of this research, were included in the database and then accessed retrospectively for research purposes from a total sample of 2667 participants. Safeguards guaranteeing that participant confidentiality would not be compromised in any way were stringently met. Accessing data from BRID provides all participants with protection under a number of ethical guidelines, including approval for testing and for the release of de-identified data.

4.3. Participants

The present study examined data from 170 participants participating in BRID. As part of the testing procedure, all participants signed written informed consent prior to participation and also for the subsequent release of de-identified data for research purposes. In addition, this research study obtained ethics approval from the Adelaide University Human Research Ethics Committee, separately to the consent requirements already in place, so that data could be retrospectively accessed for statistical testing.

4.3.1. Inclusion and Exclusion Criteria for Non-Clinical Participants

All participants included in the adult normative database met stringent inclusion and exclusion criteria. All were over 18 years of age and were pre-screened for a personal or family history of mental illness, physical brain injury, neurological disorder, or other serious medical condition and depressive or anxiety disorder. Participants were excluded if they had a history of loss of consciousness due to head injury lasting more than ten minutes (within the last five years). Participants were also excluded if they had a personal or family history of Attention Deficit Hyperactivity Disorder (ADHD), psychotic or genetic disorder, sleep disorder, learning disability, or drug and alcohol addiction.

4.3.2. Standardised Inclusion and Exclusion Criteria for PTSD

An extensive evaluation of the psychological status for PTSD participants was conducted by self-report as well as diagnostic clinical interview measures. All participants predominantly spoke English, and had normal or corrected vision, hearing, and dexterity. To meet the criteria for inclusion, PTSD participants were chronic sufferers (symptomatic for at least 6 months),
and had experienced the precipitating trauma incident within the last 10 years. Inclusion in the PTSD clinical database was processed using two phases.

The first stage of eligibility for inclusion in the BRID clinical PTSD sample was ascertained when patients presented for treatment and medico-legal assessment with a treating psychiatrist. A clinical diagnosis, confirming the requisite diagnostic symptoms as defined by the DSM-IV (American Psychiatric Association, 1994), provided an initial assessment of PTSD. Following this assessment, all presenting patients who had been given a diagnosis of PTSD, and who volunteered to participate in the research, were assessed using the Clinician Administered PTSD Scale (CAPS) (Blake et al., 1995), as well as the web measures for inclusion into the database, and the PTSD scales listed below. A psychologist or an appropriately trained master-degree student administered the tests.

In the second phase, the standardised procedures ensured that all clinical participants were also pre-screened for a history of serious medical conditions, intellectual disability, psychosis, past or present psychiatric or neurological disorder or injury, and a family history of ADHD, Schizophrenia, or Bipolar Depressive Disorder. Participants were also excluded from the PTSD sample if they had a hepatic illness (HIV, Hepatitis B, Hepatitis C), a current comorbidity for drug and alcohol dependence, and comorbid Axis I anxiety disorder, if it developed prior to the onset of PTSD. Exclusion criteria included a history of substance dependence or current psychotropic medication use other than selective serotonin reuptake inhibitors (SSRIs).

Pre-screening PTSD Diagnostic Instruments:

The following list of instruments was used as a validation of clinical status, prior to participant inclusion in the database. For the purposes of this study, these measures are included here as pre-requisite inclusion criteria for selection into a clinical sample in the standardised database.

PTSD DSM-IV Diagnostic Criteria:

The American Psychiatric Association (1994) criteria for PTSD stipulate the threshold minimum criteria in each symptom cluster. This must include at least one event on the Trauma Event Checklist and then a score of two for criterion A2 (fear, helplessness or horror). For each symptom category, a response to one or more of the criteria for cluster B
(re-experiencing), a response to three or more of the criteria for cluster C (avoidance and numbing), a response of two or more to the criteria for cluster D (hyperarousal), duration of each of the symptoms (B, C, and D) for more than one month, and clinically significant distress or impairment.

*The Clinician-Administered PTSD Scale (CAPS):*

The CAPS (Blake et al., 1995) is a standardised screening instrument designed to diagnose and assess adults for the severity of PTSD symptoms using a psychometrically sound interview-based rating scale (Weathers et al., 2001).

The CAPS as a screening instrument, is described as having clear behavioural indicators, has a time frame concordant with that of DSM diagnostic criteria, and separates frequency and intensity ratings. It is a highly reliable instrument, for items, raters, and testing occasions, with strong evidence of both convergent and divergent validity, diagnostic utility, and sensitivity to clinical change. Flexibility in scoring the CAPS permits an assessment of individual frequency, intensity or severity ratings for individual PTSD symptoms, for the three PTSD symptom clusters (re-experiencing, avoidance and numbing, and hyperarousal), and for the PTSD syndrome (Weathers et al., 2001).

The scoring rule using the CAPS assessment was consistent with the \( F1/I2/TSEV45 \) rule for a rating of 1 or higher for frequency and a 2 or higher for intensity, and a total severity score of greater than 45 (Weathers et al., 2001). However, for inclusion of participants into the BRID dataset, the cut-off was elevated to a total severity score of greater than 50 to confirm PTSD diagnosis.

*The PTSD Checklist-Civilian Version for DSM-IV (PCL-C):*

The PTSD Checklist-Civilian Version for DSM-IV (Weathers et al., 1994) was designed to measure posttraumatic distress symptoms corresponding to the 17 symptoms associated with the DSM-IV criteria. This instrument was used as a self-report inventory to confirm the clinical diagnosis, with the requirement of a minimum number of symptoms from each of the DSM-IV Criteria B, C, and D, used as cut-off scores.
The Clinician-Administered Dissociative States Scale (CADSS):

The Clinician-Administered Dissociative States Scale (Bremner et al., 1998) was designed for the assessment of “present-state” dissociation symptoms and to discriminate patients with dissociative disorders from patients with other psychiatric disorders. Subscales of the CADSS for the assessment of individual symptom areas include items for amnesia, depersonalisation, and derealization. The instrument has been reported to have good interrater reliability and construct validity (Bremner et al., 1998).

The Alcohol Use Disorders Identification Test (AUDIT):

The Alcohol Use Disorders Identification Test (Babor et al., 1992; Saunders et al., 1993) was designed to screen for a range of drinking problems, and to identify hazardous and harmful alcohol use.

4.3.3. Characteristics of Participant Sample

The present study examined data for 34 clinical PTSD participants and 136 normal participants, who were matched on variables of gender, age, and as close as possible on years of education. There were equal numbers of males and females in both groups (PTSD: N=17; control: N=68). For both groups, ages ranged from 19-58 years and years of education ranged from 8 – 18 years. Characteristics for each group, with means and standard deviations, are presented in Table 4.1. Parametric and non-parametric statistical significance, calculated for each of the demographic variables, indicated less years of education in the PTSD group (t = -2.20, df(166), p = 0.03), with no significant differences on gender or age variables between groups (p > 0.05).

Table 4.1. Demographic characteristics of participant sample

<table>
<thead>
<tr>
<th>Demographic Characteristics</th>
<th>PTSD (N=34)</th>
<th>Control (N=136)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (F/M)</td>
<td>17/17</td>
<td>68/68</td>
</tr>
<tr>
<td>Age</td>
<td>40.67 ± 11.14</td>
<td>40.23 ± 11.33</td>
</tr>
<tr>
<td>Education *</td>
<td>13.12 ± 2.87</td>
<td>14.47 ± 3.00</td>
</tr>
</tbody>
</table>

Note: * P < 0.05
4.4. Materials and Procedures

A brief summary of the methodological procedures and paradigms for the current study is presented below. However, a more detailed discussion on the role of BRID standardised database measures can be found in Gordon (2003), with additional information on methodological and administration procedures in Gordon and colleagues (2005). Three sections are covered as methods for the collection of data. The first is an anonymous (digit identified) web questionnaire, which all participants complete as a screening tool prior to further testing. Participants then complete two testing sessions when they present for the psychophysiological assessment.

4.4.1. Web Questionnaire

As a screening tool, the standardised BRID web-based questionnaire was administered to all participants. This instrument is divided into three sections, a Standard section, a Core section, and an Optional section. Participants are required to provide self-report answers to all questions in the Standard section. The Standard section includes participant’s previous medical history, general health and wellbeing, and information related to demographic data. As part of the health and wellbeing information, the computerised Composite International Diagnostic Interview (CIDI) (World Health Organization, 1993) is included as a self-report measure for possible diagnostic symptoms of psychiatric conditions. Dichotomous responses to a Core section of the web questionnaire (“yes” or “no” answers) provide further information related to medical and experiential data, and are used for the assessment of health and emotional functioning associated with daily living. Based on affirmative responses, provided in the Core section, participants then selectively answered questions from an Optional section, relating to various indices of behavioural and psychological functioning.

Only a small selection of the information provided in the web-based battery was accessed and examined in the current study. Demographic information, with additional information relevant to trauma experience and related functioning, as well as reaction patterns following traumatic stressors, was accessed to examine type of traumatic experiences reported by all participants in the normal sample as well as the clinical PTSD sample. A brief description of other data accessed, from the web-based screening instrument that were relevant to this study, is provided below.
**Self-report: Medication Use**

Medication use, for the purposes of this study, was not controlled. Using an initial screening battery of web-questionnaires, participants were asked to volunteer information about their current medication regime and use. A high percentage of the PTSD participants (42%) responded affirmatively to current SSRI medication use. None of the control group reported taking psychotropic medication, but 23.5% responded affirmatively to taking prescription and non-prescription medication. As suggested by Begic and colleagues (2001), medication use presents as a confounding variable in the analysis of cortical arousal and must be defined as a characteristic of the sample selected.

**Self-report: Handedness Laterality Quotient and Direction of Handedness**

Based on the Edinburgh Handedness Inventory (Oldfield, 1971), one of the standard and core sections in the BRID web-questionnaire, answered by all participants, was a self-report measure of lateral preference or the degree of right- or left-handedness used to complete unilateral tasks. A fifteen-item questionnaire on hand use was answered using response choices for each item indicating “left”, “right”, “either”, or “don’t know”. Using the same response choices, two of the questionnaire items assessed handedness of the participant’s biological parents. Scores could range from −1.00 (extreme left lateral preference) to +1.00 (extreme right lateral preference), a score of 0 representing the midpoint between the two extremes and a mixed laterality quotient (LQ).

Handedness preference for both groups ranged from −100 to +100. The degree and direction of handedness, which provided information about dominant hand or ambidextrousness was used to ascertain a right-handed sample for the statistical analyses using electrophysiological variables. Participants with extreme left-handedness preference or extreme mixed lateral preference were removed from analyses. These are detailed in the relevant sections.

4.4.1.1. Trauma Experience Questionnaire

Measures of self-report trauma experience were collected using the computerised CIDI (World Health Organization, 1993). The PTSD-section CIDI checklist of events questionnaire listed nine traumatic events and included two general questions relating to trauma exposure. The remainder of the scale listed responses to events based on reactions to the nominated traumatic experiences; the questions consistent with DSM-IV symptom categories, including
the age at which the event occurred, and criterion-A, indicating responses such as “terrified” and “helpless”. All responses to items were indicated by “yes” or “no” answers. In the case of criterion E items, respondents nominated duration of problems by ticking a specified temporal scale. For the present analysis, scores for each category of responses were tallied, and the “yes” responses were used as the dependent variables.

The CIDI is a comprehensive computer-aided instrument for assessment of psychiatric symptoms and diagnoses, determined according to criteria defined in the DSM-IV (American Psychiatric Association, 1994). The CIDI is both valid and reliable for epidemiological and cross-cultural studies, as well as for clinical and research purposes (Paul et al., 2005). As a standardised index of subjective experiences, this instrument can be used to assess variation in prevalence and in reported lifetime prevalence for PTSD, the retrospective age of the first-onset of the event and peaks in occurrence of traumatic events, the most common type of trauma, the frequency of specific traumatic events, as well as the gender-specific trauma type, and severity of PTSD symptomatology.

The rationale for selecting this instrument for the current study was based on the collection of trauma reactivity information in both the PTSD group and the control group using the same instrument and method. Inclusion of those in the normal sample, who responded to the optional section of the web-questionnaire as having experienced negative responses to traumatic stressor events (Checklist of Events), allowed for an assessment of trauma reactivity to be conducted on three groups of participants, and hence a finer detailed investigation of reactions to perceived traumatic events in participants without a clinical diagnosis.

4.4.1.2. Mood States

A comparison of current mood states was used as a measure of wellbeing, or psychological health, defined by depression, anxiety and stress (7 questions for each state). The DASS-21, an abbreviated version of the Depression Anxiety Stress Scales (DASS) (Lovibond and Lovibond, 1995), was used in the current study to examine the core symptoms of the negative emotional states and any comorbid presentations for depression, anxiety and stress. The DASS-21 is rated on a 4-point Likert-type response scale to assess the severity and frequency of negative emotional symptoms experienced “over the past week”.
4.4.2. Electrophysiological Data Collection and Reduction

Data collection:

Participants sat in a quiet, sound-attenuated, testing room of ambient temperature, facing a 15” colour monitor screen (at a distance of 60 centimetres from the eyes) and wore a pair of headphones. Auditory information (tones and instructions) was presented diatonically via headphones. The EEG was recorded continuously from 26 scalp electrode sites (Fp1, Fp2, Fz, F3, F4, F7, F8, FC3, FCz, FC4, Cz, C3, C4, T3, T4, CPz, CP3, CP4, T5, T6, Pz, P3, P4, O1, O2, and Oz) (Compumedics Neuroscan Nuamps; 10-20 International System; but see Gordon et al., 2005), using electrodes embedded in a flexible nylon cap (Quikcap). Horizontal eye movements were recorded with electrodes placed 1.5cm lateral to the outer canthus of each eye. Vertical eye movements were recorded with electrodes placed 3mm above the middle of the left eyebrow and 1.5cm below the middle of the left bottom eye-lid. Additional physiological data were obtained from the orbicularis oculus and the masseter. EEG data were acquired continuously relative to virtual ground and referenced offline to linked mastoids. Electrode impedance at each site was kept below 5 kOhms. The sampling rate was 500 Hz using a 22 bit analog-to-digital converter (NuAmps). A low pass filter with attenuation of 40 dB per decade above 100 Hz was implemented before digitisation. EEG data were screened offline for artifacts. Electro-oculogram (EOG) correction was based on a method following Gratton and colleagues (1983).

4.4.2.1. Quantitative Electroencephalographic Recordings

Baseline resting EEG power measures were recorded in the two conditions of eyes open and eyes closed. In the eyes open condition, participants were instructed to fixate on a red dot presented on the computer monitor for 3 minutes, and during the eyes closed condition participants were asked to rest quietly with their eyes closed. Both conditions were recorded for 2 minutes and divided into adjacent intervals of 4 seconds for Fast Fourier Transformation (FFT). A Welch (sliding) window was used to taper the cosine function and attenuate spectral side lobes in the calculation of frequency band power before FFT to the signal. The resulting power spectra were computed for 28 epochs and averaged for each paradigm using FFT auto-spectral and cross-spectral analysis, yielding a single eyes-open and a single eyes-closed average power spectrum for each electrode position across all frequency bands.

The EEG was decomposed into bands defined on the basis of lower and upper frequency bins. Each band power was square-root transformed to approximate the normal distribution
assumptions required for parametric statistical analysis. Quantitative EEG (qEEG) data are reported in terms of microvolts squared ($\mu V^2$), or power, and the parameters used to describe the characteristics of the EEG are frequency and amplitude. Core EEG rhythms are expressed as cycles per second or Hz. Frequency bands reported for this study were delta (1.5-3.5 Hz), theta (4-7.5 Hz), alpha (8-13 Hz) and beta (14.5-30 Hz). For the purposes of the current study only absolute spectral power analysis was used (following Pivik et al., 1993), which provides information on the contributions of each frequency to the entire EEG spectrum. In addition, each of the components can be measured by their peak frequency, as well as power. Alpha peak frequency (APF) is defined as power exceeding the magnitude of the EEG spectra at a point where it crosses the lower band limit (8 Hz).

Asymmetry Index:

Asymmetry is derived as a ratio of amplitude and was calculated by using the percentage of the left-right difference in average reference power spectra. A number of indices have been proposed to express a metric for the magnitude and direction of asymmetry (Davidson et al., 2000). Interlead differences in power have been denoted a measure of amplitude asymmetry (Pivik et al., 1993) and can be computed by subtracting the spectral power recorded at the left electrode from that recorded at the right electrode site for each homologous right- and left-hemisphere region. The percentage of left-right difference in average reference power spectra $(\text{Right} - \text{Left})/(\text{Right} + \text{Left})$ for the homologous left and right recording sites provides a measure of the relative activity of lateralised differences and is reported as the mathematical difference between the natural logarithm of left hemisphere alpha power from that of the right hemisphere $(\ln(\text{Right})-\ln(\text{Left}))$ spectral power.

One benefit of using the difference score metric is that the scores are normalised for individual differences in the overall alpha power (Allen and Kline, 2004). Because alpha power has been taken as an index of the inverse of cortical activity, positive scores are indicative of greater alpha power at the right compared to the left electrode site, which is assumed to reflect greater left-sided brain activation. Conversely, negative scores are indicative of greater right-sided brain activation.
4.4.3. Cognitive Measures

Procedure:

The standardised experimental design and procedure for electrophysiological data collection included the assessment of cognitive psychomotor and attentional functions. An IntegNeuro battery was administered after a short break following the electrophysiological data collection. The cognitive tests were administered using standardised task instructions, in a sound-attenuated testing room. Participants were seated in front of a touch-screen computer (NEC MultiSync LCD 1530V) and pre-recorded instructions were provided (via headphones and visual screen display). The touch-screen computer was used to record non-verbal responses. Task instructions included computerised visual demonstrations followed by a test trial prior to data acquisition.

The test battery covers five cognitive domains, and consists of 12 tasks that take approximately 50 minutes to complete (Clark et al., 2006; Gordon et al., 2005; Paul et al., 2005). Tests were designed to explore a profile of sensori-motor, language fluency, attention, memory, new learning, executive function, and estimated intelligence. The battery included the following tests: Motor Tapping, Choice Reaction Time, Timing, Span of Visual Memory, Digit Span, Memory Recall and Recognition, Verbal Interference, Spot the Real Word, Word Generation, Sustained Attention, Switching of Attention, and Executive Maze (Gordon et al., 2005).

The battery of tests has been validated as a computerised battery of cognitive tests for neuropsychological assessment (Paul et al., 2005). Correlation analyses by Paul and colleagues (2005) revealed strong relationships between the IntegNeuro tests and standard measures of cognitive function. However, only measures used in this study will be described in detail. The three tasks examined in the current study are listed below.

Materials:

4.4.3.1. The Motor Tapping Test

From the IntegNeuro sensori-motor domain, the Motor Tapping Test was selected as a test of simple motor function. The participant is required to place the palm of the hand on the touchscreen and tap as fast as possible with the index finger of each hand for a period of 60 seconds. The measures of tapping frequency from each hand are recorded. A tapping score is
computed for each hand separately as an index of motor control and speed of manual dexterity (Paul et al., 2005; Williams et al., 2005).

Simple tests of motor performance have been used as reliable indicators of the integrity of brain functions (Lezak et al., 2004) and have shown convergent validity with the variable for choice reaction time (Williams et al., 2005).

4.4.3.2. Verbal Fluency Task

From the IntegNeuro language domain, the Word Generation task comprised two parts. The Letter Fluency sub-test is a variant of the Controlled Oral Word Association (COWA) test, or FAS (Benton, 1968, cited in Tombaugh et al., 1999). The test requires participants to orally generate as many words as possible that begin with the letters F, A, and S. Each letter was timed for 60 seconds and recorded via microphone in „wav” files. Scoring by hand excluded proper nouns, intrusive or perseverative responses. The dependent measure was the total number of correct words generated across the three trials as an index of phonemic verbal fluency.

The Animal Fluency sub-test was also timed for 60 seconds and participants were required to recall as many animal words as they could within the allocated time. Scoring by hand excluded intrusions and perseverations. The dependent measure was the total number of correct responses as an index of semantic verbal fluency.

Verbal fluency reflects strategies for the planning and retrieval from long-term memory (verbal association fluency), the maintenance of words selected in short term store, and the ability to spontaneously generate words beginning with a given letter or a given class within a limited time frame (Spreen and Strauss, 1998). Lezak and colleagues (2004) have classified this test as representing executive function and suggest that productivity measures are related to the production of words, but the capacity for mental flexibility is measured by the ability to shift between clusters and to switch within phonetic and semantic subcategories. Norms for the outcomes of the test have been shown to be sensitive to the effects of years of education and age, but relatively insensitive to gender effects (Tombaugh et al., 1999). For the purposes of this study, accuracy scores were used as an index of executive function, in conjunction with speed of processing and the capacity for self-regulation.
4.4.3.3. The Maze Task

From the IntegNeuro executive function domain, the Maze Task is a computerised adaptation of the Austin Maze (Walsh, 1985). The computerised version of the maze is presented on an 8 x 8 grid of red circles and is displayed across the screen in a square array. The task reflects planning and executing actions to find a path through a hidden maze and involves encoding, the maintenance of visited points in short-term store and the retrieval of learned information on subsequent trials (Clark et al., 2006). The hidden path for the task commences at a marked circle at the bottom of the screen (in yellow) and finishes at a marked circle (in blue) at the top of the screen. The participant is able to navigate around the grid by pressing arrow keys (up, down, left, right). A total of 24 consecutive correct moves are required to complete the maze. Feedback cues are presented with auditory tones and visual cues – a red cross at the bottom of the screen for an incorrect move, and a green tick at the bottom of the screen for a correct move. Each learning trial begins at the yellow start circle and ends when the blue circle is reached. Participants are required to correctly complete the maze on two consecutive trials or until timed-out after 7 minutes.

The test was used as a measure of visuospatial attention, the executive function of planning, the ability to control impulse, and to incorporate feedback cues into planned responses to achieve the correct solution with minimal time. Maze scores were averaged for time to completion, number of errors, and number of overrun scores. For an assessment of nonverbal (visuospatial and visuomotor) information processing, the productivity measure was the time taken to complete the maze accurately on two consecutive occasions. These measures are also associated with speed of processing and learning functions. This task has been associated with a number of distributed neural networks and functions involving episodic memory retrieval and right hemisphere processes (Lezak et al., 2004). Memory was measured by using accuracy and overrun scores. For the purpose of the present study, the time taken to complete the maze was used as an index of specialised nonverbal processing for executive functions of attentional control. The dependent variable is the total time taken to complete the maze, with the total number of errors and number of overruns indexing subcomponents such as error correction, visuospatial learning and memory, abstraction and foresight (Williams et al., 2005).
4.5. Data Analysis

4.5.1. Design of the study

Data from a clinical PTSD sample and a normal age- and gender-matched sample, for the present study, primarily focused on baseline measures of functioning, utilising a cross-sectional design and standardised database methods and procedures. All participants (N=170) were included in the initial analyses but left-handed participants were excluded from subsequent analyses conducted using EEG data. For all variables, missing values and outliers (beyond 1.5 times the inter-quartile range greater than the upper or lower quartile cut-off) are reported for each of the studies conducted. Participant numbers, as well as means and standard deviations for each statistical analysis, are detailed in the relevant chapters.

In general, initial analyses were designed to compare standardised psychological and physiological measures between clinical and non-clinical groups. Data analyses examined EEG interhemispheric and intrahemispheric asymmetry in PTSD participants with control participants. As a preliminary comparison, resting qEEG profiles were examined using spectral power split into four bandwidths (delta (1.5-3.5 Hz), theta (4-7.5 Hz), alpha (8-13 Hz), and beta (14.5-30 Hz)). Measures included EEG resting activity for eyes open and eyes closed. Hemispheric and regional comparisons for aggregated left/right and frontal/posterior electrodes were compared in separate analyses and detailed in Chapter 5.

Behavioural measures from the IntegNeuro data yielded 8 dependent variables for analysis. Correlational analyses were computed to investigate relationships between resting cortical patterns and scoring on standard cognitive tests for verbal and nonverbal function as a comparison between clinical and non-clinical participants. Data analysis details are described in Chapter 5.

In a second study, to achieve manipulation of the independent variables, the control sample was sub-grouped according to data for retrospective, self-reported endorsement of traumatic experiences. Analyses of behavioural and resting EEG measures were conducted with three groups to include a non-clinical sample with previous traumatic stressor exposure. Measures of alpha asymmetry were conducted using an asymmetry index (following Pivik et al., 1993). The analyses are described in detail in Chapter 6.

In a final analysis, asymmetry scores were computed for the combined anterior (Fp2-1, F4-3 and F8-7) and combined parieto-temporal (P4-3, T4-3 and T6-5) sites by averaging
asymmetry scores from each homologous site for each region. A third study examined zero-
order correlations, means, and standard deviations for category C symptoms of numbing and 
avoidance, and cognitive verbal and nonverbal function. Separate regression analyses were 
conducted to predict residualized anterior and posterior EEG alpha asymmetry.

4.5.2. Statistics

The sizes of the sample groups for this study were based on a ratio of one PTSD participant to 
four control participants and matched for age and gender, thus meeting statistical standards 
using methods reported by Gordon and colleagues (2005). All power measures were log 
transformed to reduce distribution skewness and checked to meet parametric tests of 
assumptions after transformation. All aggregated data were checked for internal consistency 
and only used if Cronbach’s alpha was > 0.70.

Data were analysed using the Statistical Package for Social Sciences (SPSS) version 12.0 and 
version 14.0. Non-parametric analyses were used where appropriate after inspection of the 
distributions. Differences between groups were analysed separately for behavioural indices, 
using independent samples t-tests and the Analysis of Variance (ANOVA) procedures. Data 
were statistically treated using the General Linear Model (GLM) multi-level full factorial 
repeated measures procedure to test the null hypotheses, separately, for measures derived 
from spectrally analysed EEGs for absolute power, frequency and asymmetry measures. One-
way ANOVA, with post hoc Bonferroni multiple comparisons, or an equivalent non-
parametric test, examined null hypotheses about the effects of a group, as a between-subjects factor, and effects of frequency bands, as a within-subjects factor, on absolute power or mean frequency variables. Data analyses compared and contrasted the correlations between EEG power measures, behavioural measures, and various indices of psychological functioning. Hierarchical regression analyses were computed to investigate if EEG asymmetry predicts cognitive performance or avoidance and numbing responses. A full description of procedures, for each analysis conducted, is provided in each of the relevant chapters.
5. Preliminary Investigations of Lateralised Functioning in PTSD

5.1. Rationale

The purpose of this study was to investigate the role of hemispheric lateralisation in PTSD by addressing the question of whether localised functioning for verbal and nonverbal tasks has been interrupted by a common neuroanatomical pathway, as a compensatory shift to right hemisphere processing in people with PTSD.

A compensatory mechanisms hypothesis has been suggested for information processing disturbances in people with PTSD, indicating an alteration to brain structure and function (Lanius et al., 2005; Clark et al., 2003). The narrowing of attention, or focusing mechanisms, may serve to reduce information complexity by inhibiting prefrontal cortex functions via compensatory mechanisms and via an alternative pathway for well-rehearsed responses to anticipated stress stimuli (McFarlane et al., 2002). One line of evidence in the PTSD literature indicates that an asymmetry in modality-specific working memory systems underpins disruptions to executive functions (Clark et al., 2003), thus modulating attentional control (Vasterling et al., 1998).

The primary aim of this preliminary investigation was to examine whether resting EEG activity can predict vulnerability for a shift to right hemisphere functioning, and to assess whether cognitive functions of retrieval, implicated in the linking of frontal and parietal regions, are correlated with EEG resting arousal patterns. Three separate studies were conducted.

The first study focused on neuropsychological functions, investigating modality-specific processes of retrieval for verbal and nonverbal tasks. This analysis provides a behavioural index of ability to extract meaning from stimuli, based on previous experiences, and an index of localised functioning (Kolb and Whishaw, 1996; Lezak et al., 2004).

The second component investigated the characteristic EEG patterns of brain activity during the resting state, which has been shown to affect the nature of task-induced changes. The localisation of function has been defined in terms of certain areas of the brain having a functional role associated with major brain rhythms in posterior and frontal areas (Basar, 2004).
The third study examined the relationship between cognitive functions of retrieval in verbal and nonverbal tasks with EEG resting arousal patterns. The analysis was aimed at establishing factors associated with local level information processing, in an attempt to find patterns that may elucidate mechanisms for compensation after extreme psychological trauma, and which may be related to attention, learning, and memory operations.

5.2. **Study 1: Attentional Capacity for Memory and Retrieval Processes**

5.2.1. **Introduction**

Traditionally, neuropsychological measures of behaviour performance have been used as an index to assess the locus of any underlying neural deficit in left or right hemisphere specialised domain-specific processing (Lezak et al., 2004). Furthermore, the speed of processing, also an efficiency construct, has been used to indicate volition or the capacity for intentional behaviour associated with executive function and behavioural flexibility (Lezak et al., 2004). The verbal fluency task and the executive maze task have been widely used as measures of executive function by assessing the retrieval of stored verbal information from long-term memory and the retrieval of visuospatial information stored as episodic memory; thus, indicating the structural integrity of left- and right-hemisphere functions respectively (Lezak et al., 2004; Spreen and Strauss, 1998). The Motor Tapping task has also been widely used as an index of the speed of information processing and of procedural or skill memory (Christianson and Leathem, 2004). In addition, when used in assessing neurological and psychological pathology, simple tests of motor function have been shown to index not only motor speed, but also performance in other functional areas, such as alertness or orientation, selective attention or the ability to focus attention, and sustained attention (Spreen and Strauss, 1998).

A central question for this study was whether memory difficulties in PTSD were associated with an asymmetry in specialised hemispheric processing of verbal and nonverbal codes. The current study focused on the voluntary or conscious retrieval of stored information, which was examined using methodology based on accuracy and speed of processing (Landau et al., 2004). However, the various sub-processes in verbal and nonverbal tasks would not be evident using this methodology. Therefore, based on previous literature findings of cortical and sub-cortical disruptions to attention and executive function processes in people with PTSD (McFarlane et al., 1993; Clark et al., 2003; Vasterling et al., 1998), it was predicted that patterns for the retrieval of stored information would not be significantly different for items that were stored in long-term memory and well rehearsed.
However, localised deficits for modality-specific processes could be inferred from an increased effort in attentional capacity and reduced efficiency in performance of specialised left- and right-hemisphere working memory tasks, particularly for tasks representing manipulated items involving left hemisphere motor regions. In people with PTSD, it was expected that this would be associated with a disruption to the manipulation and maintenance of information and related to vigilance and hyper-arousal states, as indicated by previous research (Bryant et al., 2005; Clark et al., 2003; Bremner et al., 1999; Vasterling et al., 1998). Therefore, it was expected that compensatory mechanisms for the rehearsal of non-manipulated items would interfere with spatial attention and executive functions, thus reducing the capacity for attentional scanning and speed of processing, as measured by accuracy scores and simple tests of motor function.

5.2.2. Method

5.2.2.1. Participants

Two groups were selected for inclusion in this study and all participants met stringent inclusion and exclusion criteria as described in Chapter 4. The clinical group were 34 PTSD participants (17 male, 17 female), whose ages ranged from 19 to 57 years (M = 40.67, SD = 11.14). The PTSD participants were matched for gender and age with 136 controls (68 male, 68 female) whose ages ranged from 19 to 58 years (M = 40.23, SD = 11.33).

5.2.2.2. Instruments and Procedure

*IntegNeuro Battery:*

The standardised procedure for the administration of the battery of computerised cognitive measures was outlined in Chapter 4. From the IntegNeuro battery of tests, three tests were selected as representative measures of the cognitive modalities for psychomotor response, and verbal and nonverbal information processing. Procedures and materials for the neuropsychological tests included in this analysis are described in Chapter 4.

Tests were selected to investigate memory performance by measuring either the automatic or active retrieval of information. Accuracy scores were used to infer capacity or load and to assess the efficiency of lateralised local processing for left and right hemisphere processes. All tests were timed and this measure was used to assess the speed of information processing.
Speed of processing, for the purposes of this study, was also used to assess cognitive flexibility. In the current study the tapping score was taken as a measure of cognitive flexibility for procedural memory. The capacity for intentional behaviour, as well as the coordination and flow of information for self-regulation, was indirectly assessed using the Motor Tapping task (Spreen and Strauss, 1998; Christianson and Leathem, 2004).

5.2.2.3. Behavioural Measures

Verbal Fluency:

The „IntegNeuro“ battery included, as a test from the language domain, the word fluency task involving phonemic fluency (FAS) and semantic (animal words) fluency sub-tests. The dependent variable was the correct number of words, averaged across the three trials for the letter fluency task, and the average number of correct words generated in the semantic category task. Details can be found in Chapter 4.

Maze Task:

The Maze task was included in the „IntegNeuro“ battery as an executive function task (see Chapter 4 for details). For the purposes of this study, as a measure of nonverbal visuospatial function, the dependent variable in the Maze Task is the total time taken to complete the maze. The number of errors and number of overruns were used to index subcomponents such as error correction, visuospatial learning and memory.

Motor Tapping Task:

The Motor Tapping Task was included in the „IntegNeuro“ battery as a test of sensori-motor function (see Chapter 4 for details). A tapping score is computed for each hand separately to measure motor control and speed of manual dexterity. The dependent variable was the total number of taps with each hand.

5.2.3. Data Analysis

IntegNeuro data were scored using standardised and automated algorithms (Gordon et al., 2005). The three tasks under investigation were analysed separately, comparing PTSD participants with control participants. For an investigation into the retrieval and the oral production of words a one-way ANOVA was conducted. Data for the maze task were
assessed for between group differences using an independent samples t-test for each of the subtests. A repeated measures ANOVA was conducted for the sensori-motor task. The between-subjects factor was group (PTSD, control) and the within-subjects factor was the hand used for tapping (left/right). All tasks were analysed using 2-tailed significance at the \( p = 0.05 \) level.

5.2.4. Results

5.2.4.1. Verbal Fluency Task

The descriptive data (Table 5.1) for the verbal fluency tasks (FAS and animal words) indicated little variation between groups. However, the statistical analysis indicated a significant difference between groups, only for the FAS subtask.

| Table 5.1. Descriptive data for verbal fluency task: letter and category words |
|-----------------|-----------------|-----------------|-----------------|-----------------|
|                 | PTSD            | Control         |                 |                 |
|                 | N   | Mean | SD  | N   | Mean | SD  |
| FAS             | 31  | 14.14 | 4.5 | 122 | 16.24 | 4.17 |
| Animal          | 31  | 21.94 | 5.37| 122 | 23.54 | 5.1 |

There was a significant difference between the PTSD participants and control participants for the phonemic subtest - words beginning with the letters FAS \( [F(1,152) = 6.04, \ p = 0.02] \), indicating a reduced capacity for this task by those with PTSD compared with control participants.

There was no significant difference between the groups in the production of words in the semantic category \( [F(1,152) = 2.04, \ p = 0.12] \). The results for this subtest were interpreted to indicate that the capacity to retrieve semantic words for animal names was the same for both groups.

5.2.4.2. Summary: Retrieval of Verbal Information

Overall, memory retrieval capacity was not equal for the two verbal subtests. There was a significant difference between groups for the phonemic subtest but no differences between groups for the animal word task. The data could be interpreted with reference to both storage capacity functions and the speed of processing. Thus, differing attentional control processes in the phonemic task may underpin the retrieval of information using different network
structures, suggesting an increased memory load in the PTSD group as evidenced by the lower scores. The higher number of words produced in the semantic task indicated a more practised, or more organised, structure for the automatic retrieval of animal words compared to the phonemic category of words, suggesting an equal capacity for memory control and volition for the production of animal words in both groups of participants. The differences in the FAS task compared to the animal category task in both groups support the hypothesis of manipulated items associated with analytic processes (FAS) and non-manipulated items associated with image processing (animal words).

5.2.4.3. Executive Maze Task

The descriptive data for the maze task (Table 5.2) indicated minimal differences between groups. There were no significant differences between groups for any of the maze paradigm subtests - average time to completion (MCT); p = 0.61; average number of errors (M Av E); p = 0.38; average number of overruns (M Av O); p = 0.92.

### Table 5.2. Descriptive data for maze task – time to completion, average errors and average overruns

<table>
<thead>
<tr>
<th></th>
<th>PTSD</th>
<th></th>
<th></th>
<th>Control</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>Mean</td>
<td>SD</td>
<td>N</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>M CT</td>
<td>31</td>
<td>4.27</td>
<td>1.35</td>
<td>131</td>
<td>4.41</td>
<td>1.37</td>
</tr>
<tr>
<td>M Av E</td>
<td>30</td>
<td>4.59</td>
<td>2.50</td>
<td>130</td>
<td>4.28</td>
<td>1.48</td>
</tr>
<tr>
<td>M Av O</td>
<td>30</td>
<td>1.94</td>
<td>0.78</td>
<td>130</td>
<td>1.91</td>
<td>0.76</td>
</tr>
</tbody>
</table>

Note: MCT = average time to completion; M Av E = average number of errors; M Av O = average number of overruns

5.2.4.4. Summary: Retrieval of Visuospatial Information

Spatial attention, as measured by the speed of information processing and the accuracy of retrieved episodic information in a right-hemisphere specialised processing task, was not statistically different between groups. Results were interpreted to mean that attention mechanisms indicated efficient encoding strategies for right hemisphere parallel processing and episodic memory retrieval in the PTSD group. However, further investigation is required to assess neuroanatomical overlaps between encoding and retrieval in visuospatial functioning in people with PTSD.
5.2.4.5.  Sensori-Motor Tapping Task

The descriptive data for the Motor Tapping Task (Table 5.3) indicated a slower mean for both left- and right-hand taps for the PTSD group. This was confirmed statistically.

<table>
<thead>
<tr>
<th></th>
<th>PTSD</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>Mean</td>
</tr>
<tr>
<td>LHT</td>
<td>29</td>
<td>144</td>
</tr>
<tr>
<td>RHT</td>
<td>29</td>
<td>160.38</td>
</tr>
</tbody>
</table>

Note: LHT = left-hand taps; RHT = right-hand taps

A repeated measures ANOVA revealed a significant main effect for Hand \( [F(1,152) = 80.31, p < 0.001] \) and a main effect for Group \( [F(1, 152) = 9.91, p = < 0.01] \). A significant effect for hand indicated a dominant right-hand performance for both groups. However, the PTSD group produced fewer taps for both the left- and the right-hand, compared to the control group, reflecting lower psychomotor performance and speed of processing in the PTSD group.

5.2.4.6.  Summary: Tapping Task and Speed of Processing

Statistical analysis revealed no interaction effect in the Motor Tapping task to support a hypothesis of hemispheric asymmetry in the ability to process information in the PTSD group. The data indicated that attentional control in the PTSD group was not lateralised at the structural level of cognitive architecture. A significant lower tapping score for both hands in the PTSD group was interpreted as a slower information processing speed. The reduced speed of information processing was suggestive of a disruption to executive function in the PTSD group, implicating frontal lobe neural differences and interpreted as reduced capacity for self-regulation and volition.

5.2.5.  Discussion

The hypothesis of lateralised hemispheric processing, indicating disruption to local specialised working memory systems in PTSD, was not supported by the data. There were no consistent laterality effects across the three tasks to suggest a local deficit and an altered pattern of memory retrieval in the PTSD group for modality-specific information. However, the hypothesis of reduced speed of processing in PTSD was supported, thus indicating an
attentional disturbance associated with volition or executive control, and a shared resource function, implicating spatial working memory processes and selective attention mechanisms involving manipulated items.

It has been demonstrated in the PTSD literature that attention and memory functioning are typically associated with frontal system dysfunction, and involve errors of self monitoring and a reduced ability to inhibit responses to irrelevant information (Vasterling et al., 1998; Bryant et al., 2005). On the other hand, in an alternative hypothesis, one group of researchers has proposed that increased activity in parietal and motor regions represents a preparation for action during a stress response (Shaw et al., 2002). The significance of this proposal is relevant to an understanding of a hypothesised link between spatial working memory and affective processing during memory retrieval processes (Jatzko et al., 2006; Svoboda et al., 2006).

Furthermore, a decoupling between frontoparietal regions has been reported in the PTSD literature (Shaw et al., 2002; Clark et al., 2003; Lanius et al., 2005). Together, these findings indicate a role for the right hemisphere in mediating various perceptual and motor functions for the decoding or reconstruction of events as memory codes (Linden et al., 2003; Nadel and Moscovitch, 1998). Spatial working memory, as a right hemisphere process, is thought to be a shared resource with attention mechanisms (Awh and Jonides, 2001; Miyake et al., 2001).

Consequently, a shift in attention mechanisms may modulate the speed of processing for local processes that represent an overlap in neurobiological substrates (Banich, 2004). Such interactions between functions may form the basis for a right hemisphere compensatory mechanisms hypothesis in people with PTSD (Vasterling et al., 2004). Alternatively, a decoupling of frontal and parietal regions implicates a spatial shift in patterns of functioning, with an over-reliance on perception and the saliency of features influencing decision processes (Clark et al., 2003).

As discussed previously (Chapters 2 & 3), attention definitions are very broad (Fan et al., 2005), but for the purposes of this study have been conceptualised as those processes that enable perceptual or motor responses to be made voluntarily and selectively to one stimulus category or domain in preference to others. In addition, volition, or the capacity for intentional behaviour, includes a number of separate sub-processes and has been defined as an executive function (Lezak et al., 2004). Two fundamental processes of executive function include selective attention and task management (Smith and Jonides, 1999). Accordingly,
attention can be viewed as a neuroanatomical process (Fan and Posner, 2004; Banich, 2004), whilst the retrieval of stored information can be considered as a functional behaviour that can be measured as a memory process (Landau et al., 2004; Rockstroh and Schweizer, 2001).

Theories of memory hypothesise that encoding is an active process requiring attention related to learning, whereas retrieval is more automatic and is associated with attention related to memory functioning (Landau et al., 2004). This leads to the conclusion that, while impaired memory may be connected with attention processes, the underlying neural deficit may be related to executive functions of selective attention and task management, or executive control mechanisms associated with working memory systems involving the updating of information, and/or the maintenance of information, or any combination of these.

The current data for the three tasks provided evidence for disturbances at a functional level of information processing, involving the combinatorial and emergent properties of dynamic binding in a distributed system. However, at the structural, or implementation, level of information processing, the data did not indicate a difference in memory codes for modality-specific information between the two groups examined. This interpretation is based on results that show specific deficits in the PTSD group data. Overall, the data indicated a general pattern of slower responses in verbal fluency and motor tapping in the PTSD group, with minimal differences between groups in the visuospatial maze task. Thus, results were interpreted as a strategy shift associated with selective attention mechanisms in people with PTSD and not an algorithm for the structural over-reliance of one memory code in preference to another. On the other hand, this interpretation must be cautiously accepted because the subprocesses for the manipulation and maintenance of information in each task were not assessed using the current methodology, making it difficult to draw conclusions at the computational level. Therefore, based on this argument, an explanation for differences in behavioural performance, particularly for the phonemic verbal task and the motor tapping task, will be provided at the functional level of memory processing, and related to symptom responses associated with vigilance in people with PTSD.

The data demonstrated that memory retrieval capacity for the verbal fluency task was different for the subtasks in both groups. As an argument for differences in memory processing, this finding can be interpreted using models proposing spatial and temporal mechanisms of brain activity (Singer, 1999; Banich, 2004). The first interpretation is that specific neurological substrates convey signals to represent information that can be manipulated, or not manipulated, as categories of information that are associated with the
most salient features in a given modality (or schema representations) (Mesulam, 1998; Gazzaniga et al., 2002; Linden et al., 2003). A second, or alternative, explanation is that an overlap in some regions of the brain, which support specialised working memory processing for verbal and spatial information, are modulated by the speed at which selectively grouped information is transmitted (Singer, 1999; Banich, 2004).

Using the above models, it can be argued that the lower performance for the verbal fluency task, suggesting a meaning based on left hemisphere functional and motor attributes for objects beginning with the letters FAS, can be interpreted as an attentional deficit in both groups. This interpretation is consistent with models that propose a generative lexicon is accessed for articulatory coding by internal processes via efferent projections and interneurons that signal interpretative functions (Munk, 2001; Schwartz et al., 2005; Gallese and Lakoff, 2005; Nadel and Moscovitch, 1998; Khader and Rosler, 2004).

On the other hand, if the production of animal words for both groups is interpreted as information that is not manipulated, but is accessed by processes of association and selectively maintained, then the higher performance scores in this subtask, for both groups, can be interpreted from the perspective that a visual lexicon is more widely distributed and accessed by both hemispheres as a function of speed from the right hemisphere to the left (Banich, 2004). This interpretation of the data, for the animal word task, implies afferent projections for sensory stimuli (visual brain areas to frontal areas), attentional scanning mechanisms (Aston-Jones et al., 1999; Linden et al., 2003), and faster responses for the generation of animal words that depend on visual attributes as a single neural grouping feature for the maintenance of information. Furthermore, this interpretation is in agreement with evidence supporting the view that the category of animal words can be classified as a familiar task and can be retrieved automatically (Landau et al., 2004). Based on this argument, the results are also in agreement with interpretations suggesting that the capacity for intentional behaviour is intact in people with PTSD (Twamley et al., 2004), because, as a function of language and memory, the production of animal words did not differ significantly between groups. However, an assessment of capacity differences in people with PTSD must be qualified by whether items in memory are manipulated or maintained (Linden et al., 2003).

On the other hand, an argument for differences between the two word tasks, modulated by attention mechanisms and involving differences between manipulated and maintained information, can be analysed in relation to whether different neural substrates are involved in behavioural performance. As discussed in the literature review chapters, context information
allows for the categorisation of words to include general, rather than event-specific memory codes, unless context is associated with novelty and mechanisms of plasticity (Gazzaniga et al., 2002). Therefore, based on this interpretation, the current results suggest that different retrieval mechanisms, and spatial parameters for coding information, may be associated with performance scores in the phonemic task, between groups. Furthermore, symptoms of hyper-arousal may be linked to the retrieval of context information, and a likely explanation for the lower scores in the PTSD group (Litz et al., 2000; Vasterling et al., 1998).

In people with PTSD, specific deficits have been linked to different mechanisms for attention and memory, with evidence now supporting a capacity to remain vigilant, concomitant with the loss of inhibitory responses to irrelevant information (Vasterling et al., 1998; Bryant et al., 2005). Thus, based on the argument that some items are not manipulated, but remain as event-specific items, hence altering the temporal coding for such items, the current results would indicate greater search activation strategies by the PTSD group and reduced performance scores on manipulated items.

Extending this proposal to one of spatial coding, and making certain inferences from evidence supporting the decoupling between frontal and parietal regions in PTSD (Shaw et al., 2002; Lanius et al., 2004, 2005); a reduced capacity for executive function, or volition, would be consistent with a disruption to attention resources following a need to shift or engage in a new way of responding during the retrieval of phonemic words from long-term memory. That is, information that does not arrive within a specific time window, or information that requires the shifting of attention, is likely to interrupt the maintenance of spatial and temporal parameters of the content of an object (Banich, 2004; Howard et al., 2006; Hartikagen and Knight, 2003; Compte et al., 2000).

Because attention is a single resource, faster processing and more attentional control is possible with representations that fit certain patterns of organisation and do not require serial processing search strategies (Vasterling et al., 2006; Gazzaniga et al., 2002). For specified categories of words, particularly those associated with salient features in a given modality, such as animal words distinguished by vision, the capacity to retrieve information is increased as less search time is involved by using such strategies as priming, chunking and/or automatic retrieval for highly practiced classes of information. For the animal words subtest, this interpretation could be applied to the results for both groups. However, for the phonemic subtest, the PTSD group data indicate an alteration in attentional scanning and consequent
reduction in behavioural flexibility associated with categories of words that depend largely on left hemisphere functional or motor attributes and the manipulation of information.

In agreement with the above argument, for differences between groups based on attentional differences, the results of the maze task can be interpreted as clinically rather than statistically significant. That is, the results of the maze task, representing visuospatial processing, indicated that spatial attention, as measured by the speed of information processing and the accuracy of retrieved episodic information was not statistically different between groups. One interpretation for this result suggests a similar capacity for volitional control in the PTSD group and in the control group. In addition, the level of control for updating and maintaining information, as measured by errors and overruns in the maze task, and interpreted as attention processes linking frontal and posterior regions, indicated a similar capacity for alertness and awareness in both groups. Based on this interpretation, the results are in agreement with findings by Vasterling and colleagues (1998), proposing that the capacity for sustained attention and vigilance in people with PTSD are not disrupted. However, this interpretation will require a more detailed analysis and further research to be fully supported. Therefore, the current results examining specialised tasks, based on the premise for an increased functional role in right hemisphere processing in people with PTSD, could not be confidently interpreted using the current analysis; and, it could not be concluded that compensatory mechanisms associated with spatially distinct structures are activated in people with PTSD.

In contrast to the above arguments, maze processing requires a complex composite of cognitive operations and non-specific control processes (Caplan et al., 2001; Fletcher et al., 1995; Flitman et al., 1995). Thus, the different sub-processes involved in spatial information processing require further testing to determine whether compensatory mechanisms alter differences in speed of processing or accuracy levels in people with hyper-arousal symptoms. For example, it would need to be established whether individual differences arise from the conscious or automatic implementation of procedures used for the regeneration of images or sub-vocal rehearsal, particularly during storage processes to maintain the contents of executing a maze task. However, it remains to be clarified whether the generation of images for specific events is processed separately or differently to linguistic material in storage processes in the working memory model (Chein et al., 2003; Glassman, 1999). Conversely, differences in episodic memory may be influenced by tonic states or semantic memories (Nadel and Moscovitch, 1998) and may be in agreement with one hypothesis arguing for reduced language functions associated with handedness dominance (Chemtob and Taylor, 2003).
As discussed in Chapter 2, the role of spatial working memory in human cognition is not well understood. One proposal that has not been evaluated in the PTSD literature is the timing of information processing. Specifically, how frequency signals may carry novel information for further processing, or, transfer information to interneuron loops for a consolidation of information in long-term memory stores. For example, if information is reconstructed for retrieval and is integrated only with selective fragments of incoming stimuli, then speculatively, the detail for different items is lost over time, as modelled by Howard and colleagues (2006) and by Nadel and Moscovitch (1998). Furthermore, as proposed by Dayan and colleagues (2000), context information is likely to play a key role in predicting anticipatory responses to stimuli, or during heightened activity, in selective brain regions that alter facilitation or inhibition mechanisms depending on predicted meanings assigned to the early recognition of stimuli.

The two specialised tasks of verbal and nonverbal information processing have provided evidence of a disruption in people with PTSD at the implementation level associated with executive functions. Alterations to the meaning of stimuli may result in loss of confidence in decision processes (Dayan et al., 2000), thus altering spatial and temporal parameters in the coordinated flow of information. To confirm disruptions to control processes at a functional level, the results of the sensori-motor task demonstrated lower psychomotor performance in the PTSD group for both pathways. The reduced speed of processing was interpreted as a disruption to executive functions in the PTSD group, suggesting frontal lobe neural differences in the capacity for self-regulation and volition between the groups. This interpretation is in agreement with models proposing disruptions to executive function in PTSD (Clark et al., 2003; Felmingham et al., 2002; Vasterling et al., 2004), and as discussed, results of the verbal fluency task were consistent with an explanation for a slower rate of phonemic retrieval and decreased left frontal region activation.

The adjustments or alterations in mental processing in people with PTSD have been linked to compensatory mechanisms for the integration of information (Clark et al., 2003; Lanius et al., 2004), and, as proposed by McFarlane and colleagues (2002), may represent secondary characteristics of learning. The current data are in accord with models proposing that specific deficits are associated with PTSD and that through learning and synaptic plasticity, the speed of responding to certain classes of stimuli is altered as a result of the traumatic event (McFarlane et al., 2002; Vasterling et al., 2006). The current findings indicate a disruption associated with event-general concepts that can be manipulated as functional and motor...
attributes of higher-order cognitive processing. The disruption to information processing in PTSD participants was also interpreted as consistent with alterations to the speed of processing, or attention modulation, rather than specialised knowledge codes.

Thus, in agreement with Shaw and colleagues (2002), a common neuroanatomical pathway may be plausibly associated with anxiety reactions, which involves motor actions and dorsal parietal networks for the early detection of stimuli with ascribed meanings. However, of critical importance is how the meanings are derived from context information, or nonverbal automatically retrieved cues, for time and place, defining event-general or event-specific information. This difference is speculatively interpreted as a difference between items that can be manipulated or updated, and items that cannot be manipulated and are automatically retrieved. Furthermore, this difference in meanings is not readily examined using methodology based on accuracy scores, and thus requires further investigation and clarification. Consistent with arguments proposed by Dayan and Yu (2003), certainty or uncertainty in decision processes is relevant to an assessment of whether information is transferred through afferent loops or efferent loops in decision processes and the consequent modulation of attention through neurochemical processes.

The current findings did not support an increased reliance on visuospatial coding or right hemisphere processing, with concomitant reduced verbal working memory left hemisphere functioning, in PTSD participants. Alternatively, the results may be consistent with an interpretation that suggests a disruption to the flow of information processing associated with faulty gating or inhibitory mechanisms and the selective attention to certain classes of information. This latter explanation would involve greater search time and increased arousal. Tentatively, it is concluded from the pattern of results that the transfer of information between hemispheres, in people with PTSD, may not be disrupted by mechanisms of spatial coding but by mechanisms altering temporal channels of communication. This can be examined using baseline resting EEG states for the preparatory processing of sensory stimuli, which can be associated with temporal codes for the mapping of psychological functions to brain organisation patterns.
5.3. **Study 2: Baseline Cortical Arousal**

5.3.1. **Introduction**

Previous studies using EEG methodology in PTSD research (Jokic-Begic and Begic, 2003; Veltmeyer et al., 2006) have shown that the alpha frequency band is abnormal in people with PTSD, with some indication of laterality effects found with both ERP components and EEG resting patterns (Veltmeyer et al., 2006). On the other hand, there has been an inconsistency in findings, with one group of researchers arguing that the higher beta frequency is a possible biological marker for PTSD (Begic et al., 2001), while another group have linked cognitive disturbances in PTSD to lower alpha and theta frequencies (Veltmeyer et al., 2006). However, there is also a paucity of studies measuring activity patterns of the spontaneous EEG in the PTSD literature.

The localisation of function has been defined in terms of certain areas of the brain having a functional role in coherent neurological patterns associated with core frequencies in posterior and frontal areas (Basar, 2004). The resting or spontaneous EEG is most commonly characterised with respect to frequency and amplitude parameters (Davidson et al., 2000). This is a useful index for an examination of lateralised functioning associated with inter- and intra-hemispheric regional differences and synchrony patterns in core frequencies for the coherent transfer of signals between cortical regions (Basar, 2004). At a local or structural level of analysis, EEG power or spectral amplitude has been reported as a possible marker for detecting a biological difference in neural activation patterns in PTSD (Begic et al., 2001), and as a characterisation of the underlying stability of brain function (Neylan et al., 2003; Veltmeyer et al., 2006). In addition, core frequencies have been associated with functional specialisation, genetic traits, and the coherent transfer of information between brain regions (Hanslmayr et al., 2005; Klimesch et al., 1998; Smit et al., 2005; Zietsch et al., 2007). Alpha peak frequency has also been associated with the speed of processing and functional patterns for the coordinated flow of information (Angelakis et al., 2004; Clark et al., 2004; Posthuma et al., 2001).

The primary focus of this investigation was to investigate whether specialised processing, associated with working memory systems for left- and right-hemisphere localised functions, is associated with altered or lateralised resting neural activation patterns in PTSD participants. Based on previous studies, it was expected that asymmetry patterns of resting cortical activation would indicate a shift in local processing for memory storage, and would also characterise an unstable pattern of functioning associated with affective states.
From the previous behavioural analysis in this study, it was expected that there would be a
difference in the speed of processing in PTSD participants compared to the control
participants. This is typically examined using alpha peak frequency (APF). However, because
there have been no known studies investigating this parameter in PTSD research, the direction
of the difference was not predicted.

Based on previous findings in the literature, it was expected that maintenance of a vigilant
state in PTSD would reduce working memory resources for executive control functions
associated with the prefrontal regions. Furthermore, a higher activation pattern in the right
hemisphere, particularly for the alpha frequency in posterior regions, was expected to
represent disturbances in cognitive functioning associated with visual brain areas (occipital
and temporal cortex) and the functional role of perception. Thus, it was predicted that
associations with the faster frequencies (alpha and beta frequency bands), for binding
information, would be disrupted in the PTSD group.

Based on the behavioural performance results in this chapter, it was also expected that there
would be differences in resting amplitude patterns between the PTSD participants and the
control participants, particularly for the alpha band of frequencies. Furthermore, it was
expected that shifts in amplitude or frequency would be localised to frontal brain regions in
PTSD participants. On the other hand, because PTSD is conceptualised as heterogeneous in
its neuropathology, and previous research has argued for the identification of subtypes, it was
also expected that a characteristic profile of synchronisation patterns in PTSD would not be
readily detected using a global power analysis.

5.3.2. Method

5.3.2.1. Participants

As in the previous analysis, the PTSD data comprised 34 participants, equal number of male
and female, whose ages ranged from 19 to 57 years. They were matched for gender and age
with 136 healthy controls.
**5.3.2.2. Instruments and Procedure**

*EEG Measures and Data Reduction:*

Resting EEG data were collected for both the eyes closed and eyes open conditions and treated according to procedures and methods described in Chapter 4. A FFT was performed on 2-second epochs of artifact-free data to obtain estimates of absolute spectral power in each of the frequency bands delta (1.5-3.5 Hz), theta (4-7.5 Hz), alpha, APF (8-13 Hz), and beta (14.5-30 Hz). EEG data for each frequency were log transformed prior to analysis following methods according to Pivik and colleagues (1993).

**5.3.3. Data Analysis**

*Alpha Peak Frequency:*

The EEG data for the current study were measured using the following anterior, central, and posterior midline leads (Fz, Cz, Pz), left hemisphere (anterior: F3, F7; temporal: T5; posterior: P3) and right hemisphere (anterior: F4, F8; temporal: T6; posterior: P4) electrode site leads. Because of missing data, participant numbers are reported for each analysis. GLM repeated measures ANOVA was used to examine APF values between groups for changes to frequency, region, and condition in three separate analyses.

The first analysis compared midline sites for anterior, central, and posterior positions in both the eyes closed and eyes open conditions. A three-way ANOVA was conducted with Group [PTSD (N = 21), control (N = 81)] as the between-subjects factor, and Site (Fz, Cz, Pz) and Condition (EC, EO) as within-subjects factors.

The second analysis compared hemispheric differences between groups [Group (PTSD N=31, control N=112) x Hemisphere (Left, Right) x Site (F3, T5, P3; F4, T6, P4)]. Because there were no differences between groups in condition, and due to the higher percentage of missing data in the eyes open condition, only the eyes closed condition was examined.

The third analysis examined mean differences in APF between groups for anterior and posterior regions. A three-way ANOVA was conducted with the between-subjects factor Group (PTSD N=27, control N=105) and within-subjects factors Region (Anterior, Posterior) and Site [(FZ, F3, F4, F7, F8), (PZ, P3, P4, T5, T6)].
QEEG Characteristics: Global Absolute Power:

To reduce the data for statistical analyses, hemispheric asymmetries were examined by pooling homologous electrode sites within each hemisphere (following a method by Davidson et al., 2000), thus creating mean global (whole-head) eyes closed or eyes open variables for left and right hemisphere for each frequency band. All aggregated variables were > 0.80 on Cronbach’s Alpha.

As an initial investigation, and to explore the characteristics of global rhythmic patterns, two statistical models were computed as a manipulation check (Gueorguieva and Krystal, 2004). The first model, using the SPSS Mixed procedure, was used because it accounts for any differences in covariance structures using aggregated variables. The purpose of using the SPSS Mixed procedure was to examine high order categories using a large number of parameters. However, to maintain consistency in reporting findings for this study, the same analysis was then repeated using the SPSS GLM model.

Differences in amplitude for each frequency band were evaluated using GLM repeated measures ANOVA for a between-subjects factor of Group (PTSD N=34, control N=132) and within-subjects factors of frequency Band (delta, theta, alpha, beta), Hemisphere (left, right) and Condition (eyes closed, eyes open).

All other analyses were computed using repeated measures ANOVA. Significance levels were two-tailed and set at the alpha p = 0.05 level. Post hoc analyses were computed where necessary. A Greenhouse-Geisser correction was used where sphericity assumptions were violated.

Midline Absolute Alpha Power:

To investigate characteristic midline arousal, a between groups (PTSD N=33, control N=132) analysis for the effects of Site (Fz, Cz, Pz) and Condition (eyes closed, eyes open) was computed using repeated measures ANOVA.

EEG Alpha: Inter-hemispheric and Intra-hemispheric Asymmetries:

Two separate analyses were computed to assess differences between groups for intra-hemisphere (lateral inter-areal: anterior and posterior) and inter-hemisphere (horizontal: left
and right) absolute power in the resting alpha band in the eyes closed condition. To investigate frontal asymmetry, a GLM ANOVA for a between-subjects factor Group (PTSD, control) and within-subjects factors Hemisphere (left; right) and Site (left: Fp1, F3, F7; right: Fp2, F4, F8) was computed. Similarly, to investigate posterior asymmetry in the resting alpha band between groups a separate ANOVA was computed for the between subjects factor Group and within-subjects factors Hemisphere and Site (left: T5, P3, O1; right: T6, P4, O2).

5.3.4. Results

5.3.4.1. Alpha Peak Frequency Patterns

*Midline APF:*

The results are presented in Table 5.4, indicating a significant difference between groups for APF [F(1,100) = 4.62, p = 0.03]. There was also a main effect for site [F(G-G: 2,164) = 5.67, p = 0.01], indicating a difference in peak frequency at each midline site, with the means showing a consistent trend for lower anterior to higher posterior frequency for both groups in both the eyes closed and eyes open conditions.

The pattern for APF at midline sites indicated higher mean APF amplitudes in both the eyes closed and eyes open conditions in the PTSD group. This difference is represented in Figures 5-1 and 5-2.

*Table 5.4. Results of ANOVA assessing alpha peak frequency effects at midline sites*

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site</td>
<td>2,164 (G-G)</td>
<td>5.67**</td>
</tr>
<tr>
<td>Condition</td>
<td>1,100</td>
<td>1.04</td>
</tr>
<tr>
<td>Group</td>
<td>1,100</td>
<td>4.62*</td>
</tr>
<tr>
<td><strong>Interaction Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site x Group</td>
<td>2,164 (G-G)</td>
<td>1.35</td>
</tr>
<tr>
<td>Condition x Group</td>
<td>1,100</td>
<td>0.53</td>
</tr>
<tr>
<td>Site x Condition</td>
<td>2,177 (G-G)</td>
<td>1.56</td>
</tr>
<tr>
<td>Site x Condition x Group</td>
<td>2,177 (G-G)</td>
<td>0.83</td>
</tr>
</tbody>
</table>

*Note:* (G-G) = Greenhouse-Geisser correction; *p < 0.05; **p ≤ 0.01
Interhemispheric Differences in APF:

Results of the three-way ANOVA examining hemisphere effects are presented in Table 5.5. As expected, a main effect for site was significant \([F(2,245) = 9.04, p = < 0.001]\), indicating differences in frequency at each site, but with the same consistent pattern of peak frequency at each site for both groups. A hemisphere effect was not significant between groups \([F(GG: 1,141) = 0.74, p = 0.39]\), indicating the same pattern of means between groups for both left and right hemisphere.

<p>| Table 5.5. Results of ANOVA assessing alpha peak frequency inter-hemispheric effects |
|--------------------------------------|-----------------|</p>
<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisphere</td>
<td>1,141</td>
<td>0.01</td>
</tr>
<tr>
<td>Site</td>
<td>2,245 (G-G)</td>
<td>9.04**</td>
</tr>
<tr>
<td>Group</td>
<td>1,141</td>
<td>0.74</td>
</tr>
<tr>
<td><strong>Interaction Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisphere x Group</td>
<td>1,141</td>
<td>0.38</td>
</tr>
<tr>
<td>Site x Group</td>
<td>2,245 (G-G)</td>
<td>1.94</td>
</tr>
<tr>
<td>Hemisphere x Site</td>
<td>2,258 (G-G)</td>
<td>0.37</td>
</tr>
<tr>
<td>Hemisphere x Site x Group</td>
<td>2,258 (G-G)</td>
<td>0.69</td>
</tr>
</tbody>
</table>

Note: **p < 0.01
Figure 5-1: Results of ANOVA at midline APF eyes closed condition.

Figure 5-2: Results of ANOVA at midline APF eyes open condition.
Anterior and Posterior APF:

A region by group interaction was on the cusp of significance \[ F(1,130) = 3.86, p = 0.05 \], and taken to indicate differences in mean APF between groups. As presented in Table 5.6, the only other significant effect was for region \[ F(1,130) = 14.13, p = < 0.001 \], indicating a different pattern of APF oscillations between anterior and posterior regions for both groups.

A post hoc analysis did not reach statistical significance for the interaction effect. A visual inspection of the means for the APF anterior and posterior leads indicated small differences between groups, with APF for both regions just below the normal range of 10 Hz (Anterior: PTSD = 9.82 ± 0.86; control = 9.49 ± 0.93; Posterior: PTSD = 9.84 ± 0.83; control = 9.81 ± 0.87). These are represented as graphs in Figures 5-3 and 5-4.

### Table 5.6. Results of ANOVA assessing APF anterior and posterior region effects

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Region</td>
<td>1,130 (G-G)</td>
<td>14.13**</td>
</tr>
<tr>
<td>Site</td>
<td>3,407 (G-G)</td>
<td>0.22</td>
</tr>
<tr>
<td>Group</td>
<td>1,130</td>
<td>1.40</td>
</tr>
<tr>
<td><strong>Interaction effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Region x Group</td>
<td>1,130 (G-G)</td>
<td>3.86‡</td>
</tr>
<tr>
<td>Site x Group</td>
<td>3,407 (G-G)</td>
<td>0.50</td>
</tr>
<tr>
<td>Region x Site</td>
<td>3,407 (G-G)</td>
<td>0.70</td>
</tr>
<tr>
<td>Region x Site x Group</td>
<td>3,407 (G-G)</td>
<td>0.50</td>
</tr>
</tbody>
</table>

Note: **p < 0.01; ‡ p = 0.051

5.3.4.2. Summary: Alpha Peak Frequency

In summary, significant differences in midline APF were found between the two groups, with a higher frequency in the PTSD group compared to the control group. There were no significant differences between eyes closed or eyes open conditions between groups, with both groups showing a higher pattern of APF in posterior regions compared to anterior regions. An interaction effect for between-group differences in anterior and posterior regions was almost significant, with a trend toward higher APF in frontal regions in PTSD participants compared to the control group. Overall, the results are indicative of an altered pattern of APF in PTSD participants.
5.3.4.3. Spectral Power qEEG Characteristics

Using whole-head residualized power values, a visual representation of the data provides a summary of median global power values for each of the frequency patterns between groups. The box plot graphs, Figures 5-5 and 5-6, show greater variability in the pattern of amplitudes for the alpha bandwidth between groups and greater variation in left hemisphere aggregated power means compared to right hemisphere means, indicating interaction effects in the eyes closed condition.

In the eyes open condition, Figures 5-7 and 5-8, the boxplot graphs reveal a similar pattern of aggregated means for each frequency in both groups. There is less indication of variation in each of the frequency band pattern of means with eyes open between groups. Overall, the graphs demonstrate a pattern of minimal differences between groups in resting spontaneous EEG patterns but suggest there may be some differences associated with the alpha frequency band, particularly for left hemisphere eyes closed condition, indicative of interaction effects.
Figure 5-3. Boxplot of anterior APF means for the eyes closed condition shows a trend toward higher APF in the PTSD group but little variation between groups at each site.

Figure 5-4. Boxplot of posterior APF means for the eyes closed condition indicates greater variation in the control group for a pattern of higher and lower APF but still within the normal range of 8-13 Hz.
Figure 5-5. Aggregated means for each frequency bandwidth for left hemisphere (eyes closed condition) shows a trend toward variation in the alpha frequency band between groups.

Figure 5-6. Aggregated means for each frequency at right hemisphere (eyes closed condition) shows an overall similar pattern of means between groups but with differences indicated in the alpha frequency band.
Figure 5-7. Boxplot graph showing aggregated means for each frequency for left hemisphere (eyes open condition) indicates a similar pattern of total power for both groups.

Figure 5-8. Aggregated means for each frequency for right hemisphere (eyes open condition) shows little variation in total power between groups.
**Global Power Mixed Model Analysis:**

Using a mixed model analysis, a Compound Symmetry covariance model was initially tested against a more constrained Heterogeneous Compound Symmetry model. The Goodness of Fit statistic (-2 Restricted Log Likelihood) was reduced to a 35 unit drop in the Chi square distance (35,3) and not significant (p > 0.05) for the Heterogeneous Compound Symmetry model. Therefore, the more constrained model (17 parameters compared to 34) is reported.

A Group by Band interaction was significant [F(3,246) = 4.97, p = < 0.01], indicating that there were differences between groups in frequency amplitudes, with differences in the pattern of amplitudes in one or more frequency bands. There was also a significant interaction of Band by Condition [F(3,321) = 87.38, p = < 0.001]. This was confirmed by a main effect of Band [F(3,246) = 191.93, p = < 0.001], which as expected, revealed that for both groups each frequency was oscillating with a different mean power amplitude pattern. A main effect of Condition [F(1,383) = 286.35, p = < 0.001] served as a manipulation check and indicated that there were differences in absolute power between the eyes closed and eyes open conditions for both groups. The estimates of fixed effects indicated that there were significant differences between conditions, with lower amplitudes for the eyes open condition [t = 15.81, df(201), p = < 0.001].

A post hoc analysis, using the mixed model procedure for the simple effects of the band by group interaction, revealed that the pattern of results remained stable within the various covariance structures. Differences between frequency patterns were estimated and referenced against the beta frequency band to show that there was a significant difference between beta and delta [t = -8.33, df(222), p = < 0.001]. There was a significant difference between beta and alpha [t = 0.30, df(249), p = < 0.01], and no difference between beta and theta [t = 0.89, df(239), p = 0.38]. Using the beta band as a reference, the results also demonstrated that there were no differences in amplitude between groups for this band or for the delta band (p = 0.26). There were no significant differences between groups for the theta band [t = 1.88, df(239), p = 0.06]. However, there was a statistically significant difference in lower alpha amplitudes in the PTSD group [t = 2.33, df(249), p = 0.02], despite the overall topographic pattern remaining the same between groups. Results indicated that for each frequency band there were no amplitude differences between hemispheres [t = -0.37, df(259), p = 0.71].
Global Power GLM ANOVA:

The above analysis was repeated using the more commonly reported GLM model assuming fixed parameters. The results for this analysis are presented in Table 5.7.

A significant three-way interaction of Band by Hemisphere by Group \([F(G-G: 3,345) = 4.36, p = < 0.01]\), indicated differences in the pattern of absolute power between groups for frequency and laterality. A significant Band by Condition interaction \([F(G-G: 3,323) = 217.18, p = < 0.001]\) was confirmed as per the mixed model above. As expected, there was a main effect for Band \([F(G-G: 3,372) = 59.18, p = < 0.001]\), indicating a difference in frequency patterns for each bandwidth for both groups. Similarly, as expected, there was a main effect for Condition \([F(1,164) = 167.11, p = < 0.001]\), indicating reduced amplitude patterns for eyes open compared to eyes closed states in both groups.

**Table 5.7** Results of ANOVA assessing aggregated global power in each frequency band for left and right hemisphere in each of the eyes open and eyes closed conditions between groups

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Band</td>
<td>3,373 (G-G)</td>
<td>59.18**</td>
</tr>
<tr>
<td>Hemisphere</td>
<td>1,164</td>
<td>1.95</td>
</tr>
<tr>
<td>Condition</td>
<td>1,164</td>
<td>167.11**</td>
</tr>
<tr>
<td>Group</td>
<td>1,164</td>
<td>0.95</td>
</tr>
<tr>
<td><strong>Interaction Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Band x Group</td>
<td>3,373 (G-G)</td>
<td>1.96</td>
</tr>
<tr>
<td>Hemisphere x Group</td>
<td>1,164</td>
<td>0.09</td>
</tr>
<tr>
<td>Condition x Group</td>
<td>1,164</td>
<td>0.34</td>
</tr>
<tr>
<td>Band x Hemisphere</td>
<td>3,345 (G-G)</td>
<td>1.29</td>
</tr>
<tr>
<td>Band x Condition</td>
<td>3,323 (G-G)</td>
<td>217.18**</td>
</tr>
<tr>
<td>Hemisphere x Condition</td>
<td>1,164</td>
<td>0.66</td>
</tr>
<tr>
<td>Band x Hemisphere x Condition</td>
<td>3,427 (G-G)</td>
<td>2.73‡</td>
</tr>
<tr>
<td>Hemisphere x Condition x Group</td>
<td>1,164</td>
<td>0.89</td>
</tr>
<tr>
<td>Band x Condition x Group</td>
<td>3,323 (G-G)</td>
<td>0.07</td>
</tr>
<tr>
<td>Band x Hemisphere x Group</td>
<td>3,345 (G-G)</td>
<td>4.36**</td>
</tr>
<tr>
<td>Band x Hemisphere x Condition x Group</td>
<td>3,427 (G-G)</td>
<td>1.49</td>
</tr>
</tbody>
</table>

Note: **p < 0.01; ‡ p = 0.052
Post Hoc Analysis: Three-way Interaction of Band x Hemisphere x Group:

To explore the three-way interaction further, the simple effects for each frequency band were investigated for hemisphere and group effects. The four frequency bands were examined using separate ANOVAs for a between-subjects factor Group (PTSD, control), and within-subjects factors Hemisphere (left, right) and Condition (eyes closed, eyes open).

**Delta Bandwidth (1.5-3.5 Hz):**

Table 5.8 demonstrates that there were no significant main effects in the delta bandwidth and no interaction effects.

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main Effect</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisphere</td>
<td>1,164</td>
<td>0.75</td>
</tr>
<tr>
<td>Condition</td>
<td>1,164</td>
<td>0.07</td>
</tr>
<tr>
<td>Group</td>
<td>1,164</td>
<td>0.00</td>
</tr>
<tr>
<td>Interaction Effect</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Condition x Group</td>
<td>1,164</td>
<td>0.01</td>
</tr>
<tr>
<td>Hemisphere x Condition</td>
<td>1,164</td>
<td>0.96</td>
</tr>
<tr>
<td>Hemisphere x Group</td>
<td>1,164</td>
<td>0.56</td>
</tr>
<tr>
<td>Hemisphere x Condition x Group</td>
<td>1,164</td>
<td>0.12</td>
</tr>
</tbody>
</table>

**Theta Bandwidth (4.0-7.5 Hz):**

Results for the theta bandwidth are presented in Table 5.9. The only statistically significant effect was for Condition \( [F(1,164) = 78.85, p = < 0.001] \), indicating lower theta amplitude, for both groups, in the eyes closed condition compared to the eyes open condition.
Table 5.9 Results of simple effects ANOVA assessing theta aggregated power for each hemisphere and in each condition between groups

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main Effect</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisphere</td>
<td>1,164</td>
<td>0.08</td>
</tr>
<tr>
<td>Condition</td>
<td>1,164</td>
<td>78.85**</td>
</tr>
<tr>
<td>Group</td>
<td>1,164</td>
<td>0.99</td>
</tr>
<tr>
<td>Interaction Effect</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Condition x Group</td>
<td>1,164</td>
<td>0.46</td>
</tr>
<tr>
<td>Hemisphere x Group</td>
<td>1,164</td>
<td>1.41</td>
</tr>
<tr>
<td>Hemisphere x Condition</td>
<td>1,164</td>
<td>0.11</td>
</tr>
<tr>
<td>Hemisphere x Condition x Group</td>
<td>1,164</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Note: **p < 0.01

Alpha Bandwidth (8-13Hz):

The results for the alpha bandwidth are presented in Table 5.10. There was a significant interaction for Hemisphere by Condition [F(1,164) = 4.42, p = 0.04], which was confirmed by a significant main effect for Hemisphere [F(1,164) = 4.89, p = 0.03] and a significant main effect for Condition [F(1,164) = 346.75, p < 0.001]. The significant effects are explored in separate analyses and as further investigations in the subsequent chapters.

Table 5.10 Results of simple effects ANOVA assessing alpha aggregated power for each hemisphere and in each condition between groups

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main Effect</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisphere</td>
<td>1,164</td>
<td>4.89*</td>
</tr>
<tr>
<td>Condition</td>
<td>1,164</td>
<td>346.75**</td>
</tr>
<tr>
<td>Group</td>
<td>1,164</td>
<td>2.80</td>
</tr>
<tr>
<td>Interaction Effect</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisphere x Group</td>
<td>1,164</td>
<td>0.50</td>
</tr>
<tr>
<td>Condition x Group</td>
<td>1,164</td>
<td>0.12</td>
</tr>
<tr>
<td>Hemisphere x Condition</td>
<td>1,164</td>
<td>4.42*</td>
</tr>
<tr>
<td>Hemisphere x Condition x Group</td>
<td>1,164</td>
<td>1.59</td>
</tr>
</tbody>
</table>

Note: *p < 0.05; **p < 0.01

Beta Bandwidth (14.5-30 Hz):

The main effects for the beta bandwidth are presented in Table 5.11. There was a significant interaction effect for Hemisphere by Condition [F(1,164) = 21.41, p = < 0.001], which was
confirmed by a significant main effect for Hemisphere $[F(1,164) = 11.63, p = 0.001]$ and a significant main effect for Condition $[F(1,164) = 37.80, p < 0.001]$.

**Table 5.11** Results of simple effects ANOVA assessing beta aggregated power for each hemisphere and in each condition between groups

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisphere</td>
<td>1,164</td>
<td>11.63**</td>
</tr>
<tr>
<td>Condition</td>
<td>1,164</td>
<td>37.80**</td>
</tr>
<tr>
<td>Group</td>
<td>1,164</td>
<td>0.58</td>
</tr>
<tr>
<td><strong>Interaction Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisphere x Group</td>
<td>1,164</td>
<td>1.20</td>
</tr>
<tr>
<td>Condition x Group</td>
<td>1,164</td>
<td>1.83</td>
</tr>
<tr>
<td>Hemisphere x Condition</td>
<td>1,164</td>
<td>21.41**</td>
</tr>
<tr>
<td>Hemisphere x Condition x Group</td>
<td>1,164</td>
<td>2.82</td>
</tr>
</tbody>
</table>

Note: **p < 0.01

5.3.4.4. **Summary: qEEG Recordings**

As a manipulation check, results of the mixed model interaction indicated group differences between the beta bandwidth and the alpha bandwidth, which was consistent with results of the ANOVA models. Global power (aggregated means) for each frequency, and for amplitude differences, indicated alterations in cortical resting patterns between groups and interaction effects for the higher (alpha and beta) frequencies in both groups. Both sets of results revealed a reliable trend toward the same pattern of cortical arousal between groups.

The ANOVA pattern of results demonstrated a three-way interaction effect (Band by Hemisphere by Group) in the alpha and beta frequencies. Main effects for Hemisphere and Condition were found for the alpha and beta frequencies. Data for both groups demonstrated alterations to the pattern of amplitudes with reactivity (eyes open condition) for the theta, alpha, and beta bandwidths. For both groups all data indicated consistently lower amplitudes in the eyes open condition compared to the eyes closed condition. As a simple effects analysis, reactivity between eyes closed and eyes open for the beta frequency was not significantly different between groups. Similarly, cortical resting patterns for the beta frequency indicated no significant differences between groups.

However, the PTSD data revealed a pattern of consistently lower alpha power in all analyses compared to the control group. The interaction effect was explored further as simple effects for the alpha frequency in the following post hoc analyses.
5.3.4.5. Alpha Frequency

*Eyes Closed Alpha Intra-hemispheric Analysis:*

Differences between groups, examining the pattern of eyes closed alpha spectral power amplitudes, were conducted for anterior and posterior regions with a Region by Group ANOVA. The analysis for anterior and posterior regions was conducted with pooled left- and right-hemisphere sites for each region. As expected, there was a main effect for Region \[F(1,164) = 35.75, p = < 0.001\], indicating differences between anterior and posterior regions in alpha absolute power. There were no other significant effects, indicating that both groups demonstrated the same pattern of alpha amplitudes for the anterior and posterior regions, in the expected direction of higher posterior resting alpha amplitudes. Separate post hoc analyses were conducted for each region to investigate the three-way interaction of Hemisphere by Site by Group from the previous analysis.

*Eyes Closed Anterior Alpha Frequency Patterns:*

Frontal asymmetry was investigated using ANOVA for a between-subjects factor Group (PTSD N=28; control N=129) and within-subjects factors Hemisphere (left; right) and Site (left: Fp1, F3, F7; right: Fp2, F4, F8). Results are presented in Table 5.12.

For anterior regions, there was a significant interaction effect of Hemisphere by Site by Group \[F(G-G: 2,256) = 4.69, p = 0.02\], indicating differences between groups in resting anterior alpha amplitudes. A laterality effect was confirmed with a Hemisphere by Group interaction \[F(1,155) = 4.46, p = 0.04\]. A main effect for Site \[F(G-G: 2,296) = 351.70, p = < 0.001\], demonstrated differences in power amplitudes at each of the electrode sites measured.

For the left hemisphere, a Site by Group interaction \[F(G-G: 2,282) = 3.57, p = 0.03\] was not significant when adjusted for multiple comparisons at the \(p = 0.01\) criterion level. Using Bonferroni corrections the differences in alpha amplitudes in the left hemisphere were not confirmed between groups.
Table 5.12 Results of simple effects ANOVA assessing alpha anterior region power for each hemisphere between groups

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main Effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisphere</td>
<td>1,155</td>
<td>.114</td>
</tr>
<tr>
<td>Site</td>
<td>2,296 (G-G)</td>
<td>351.70**</td>
</tr>
<tr>
<td>Group</td>
<td>1,155</td>
<td>1.41</td>
</tr>
<tr>
<td><strong>Interaction Effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisphere x Group</td>
<td>1,155</td>
<td>4.46*</td>
</tr>
<tr>
<td>Site x Group</td>
<td>2,296 (G-G)</td>
<td>1.20</td>
</tr>
<tr>
<td>Hemisphere x Site</td>
<td>2,256 (G-G)</td>
<td>1.54</td>
</tr>
<tr>
<td>Hemisphere x Site x Group</td>
<td>2,256 (G-G)</td>
<td>4.69*</td>
</tr>
</tbody>
</table>

Note: * p < 0.05; **p < 0.01

For the right hemisphere, the post hoc analyses demonstrated that there was a simple main effect for Site \([F(G-G: 2,320) = 343.83, p = < 0.001]\), indicating differences in amplitude patterns between each electrode site. As expected, for anterior resting alpha patterns there were no other significant effects. The consistently low alpha amplitudes at each site for the PTSD group were not confirmed statistically between groups in frontal right hemisphere regions \(p = 0.08\).

_Eyes Closed Posterior Alpha Frequency Patterns:_

A separate ANOVA was computed for the between subjects factor Group (PTSD N=32; control N=130) and within-subjects factors Hemisphere and Site (left: T5, P3, O1; right: T6, P4, O2) for the eyes closed condition. The results are presented in Table 5.13. There was a significant interaction effect of Hemisphere by Site \([F(G-G: 2,265) = 26.73, p = < 0.001]\), indicating that the pattern of amplitudes between hemispheres was not consistent at all sites for posterior regions. This was confirmed by a main effect for Hemisphere \(p = < 0.001\), and a main effect by Site \(p = < 0.001\) but there was no significant main effect by Group, indicating the same pattern for both groups.
Figure 5-9. Left hemisphere anterior alpha power.

Note: Graph shows differences in the pattern of means between groups.

Figure 5-10. Right hemisphere anterior alpha power.

Note: Graph shows a similar pattern of means for alpha power at each side between groups but with lower alpha power in the PTSD group.
Table 5.13 Results of ANOVA assessing alpha frequency for each hemisphere in posterior brain regions between groups

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisphere</td>
<td>1,162</td>
<td>14.28**</td>
</tr>
<tr>
<td>Site</td>
<td>2,282 (G-G)</td>
<td>40.73**</td>
</tr>
<tr>
<td>Group</td>
<td>1,162</td>
<td>2.05</td>
</tr>
<tr>
<td><strong>Interaction Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisphere x Group</td>
<td>1,162</td>
<td>0.14</td>
</tr>
<tr>
<td>Site x Group</td>
<td>2,282 (G-G)</td>
<td>0.14</td>
</tr>
<tr>
<td>Hemisphere x Site</td>
<td>2,265 (G-G)</td>
<td>26.73**</td>
</tr>
<tr>
<td>Hemisphere x Site x Group</td>
<td>2,265 (G-G)</td>
<td>1.01</td>
</tr>
</tbody>
</table>

*Note:* **p < 0.01

**Post-hoc Analysis: Hemisphere by Site Interaction Effect in Posterior Regions:**

The mean amplitude patterns for both groups indicated a trend towards higher right hemisphere amplitudes in posterior regions. A simple effects analysis to investigate the interaction of Hemisphere by Site was conducted for each group separately.

**PTSD Group:**

For the PTSD group, the post-hoc interaction of Hemisphere by Site was significant [F(G-G: 2,60) = 12.87, p = < 0.001], indicating a difference in amplitudes between the left and right hemisphere. The interaction was confirmed by a significant main effect for Hemisphere [F(1,32) = 4.34, p = 0.05], and a significant main effect for Site [F(G-G: 2,49) = 13.43, p = < 0.001]. As demonstrated by Figure 5-11, the results of a paired samples t-test indicated a difference for higher right hemisphere alpha amplitudes at T6 electrode sites [t = -3.80, df(1,33), p = 0.001] in posterior regions for the PTSD group.
Figure 5-11. PTSD within-group posterior regions alpha power is significantly different at the T6 (right hemisphere) site for higher power compared to the T5 (left hemisphere) site.

Figure 5-12. Control within-group posterior region alpha power indicates greater variability between hemispheres at temporal and parietal sites for higher right hemisphere amplitudes.
Control Group:

For the control group, the post-hoc analysis for the interaction effect of Hemisphere by Site was also significant \(F(G-G: 2,206) = 36.26, p = < 0.001\], as indicated by the variance distributions in the boxplot graphs for alpha power. A main effect for Hemisphere \(F(1,130) = 20.58, p = < 0.001\] confirmed a laterality effect for alpha amplitudes. A main effect for Site \(F(G-G: 2,231) = 46.05, p = < 0.001\], indicated a difference in amplitudes between sites for each hemisphere. To explore this difference, a paired samples t-test indicated that differences in alpha amplitudes, for the control group, were at temporal T5-T6 sites \(t = -6.31, df(1,130), p = < 0.001\], but also at parietal P3-P4 sites \(t = -3.26, df(1,131), p = 0.001\].

5.3.4.6. Summary: Alpha Spectral Power

The results for alpha frequency band amplitudes, in anterior regions, revealed that overall there were no significant differences between groups in asymmetry or shifts in power at specific topographic anterior locations. The significant interaction effect (Hemisphere by Site by Group) was not statistically significant when reduced to simple effects and corrected for multiple comparisons. The data revealed only a trend toward lower left hemisphere amplitudes in the PTSD group.

However, for posterior regions, the results revealed a clear laterality effect with a pattern of higher alpha absolute power in the right hemisphere for both groups. Additionally, although interaction effects indicated asymmetry between hemispheres and sites, there were no clear differences between groups in specific hemispheric topographic locations. The results indicated greater variability in the control group in the alpha frequency band, as suggested by the boxplot graphs in Figures 5-5 and 5-6. This finding was localised to temporal and parietal electrode leads in the control group. However, hemisphere differences in posterior regions for the PTSD group were only revealed at temporal electrode leads.

Data for the alpha band of frequencies was consistent with global (whole-head) power findings, which indicated significantly lower alpha absolute power in the PTSD group, but a similar pattern of means across hemispheres, regions, and conditions for both groups. Overall the data revealed the same consistent pattern across sites of lower power, at anterior compared to posterior sites, for both groups. Furthermore, data for both groups revealed a pattern of slightly higher amplitudes for the right hemisphere, which was only significant in posterior regions. This was an unexpected finding for the control group.
5.3.5. Discussion

The hypothesis of a shift to a lateralised pattern of activation, at the local level of processing in people with PTSD, was not supported by the data. An unexpected finding was that both the PTSD group and the normal group data indicated a shift toward right hemisphere increased alpha and beta frequencies in posterior regions. Furthermore, both groups indicated a pattern of spontaneous APF oscillating at just below the normal 10 Hz, but still above the low value alpha range of 8 Hz. Based on previous research, a number of alternative explanations may be relevant to the findings.

EEG waveforms and spectral data are considered an objective measure of the probability and timing of information transfer (Rowe, 2005) and provide a useful analysis of preparedness for information processing (Basar, 2004). In addition, increased occipital activity has been shown to be consistent with an increase in externally directed visual attention (Aston-Jones et al., 1999). The data for the PTSD group and the normal control group indicated a characteristic alpha frequency pattern of greater neural activity over posterior regions, and also a pattern of differences in reactivity with eyes open and eyes closed conditions in the higher beta frequency, consistent with normal patterns. On the other hand, the data for APF indicated a higher frequency pattern for the PTSD group in posterior regions. This finding may be linked to differences in preparatory states. The speed of processing, together with a selective threat perception bias, may be a plausible explanation for the engagement of different neuronal pathways with early recognition cues or the facilitation of awareness of novel stimuli in people with PTSD.

The results of the qEEG analyses converge with the findings for the behavioural analysis. That is, speed of processing, as revealed by the motor tapping task, indicated a general slowing of information processing in the PTSD group rather than a lateralised pattern of deficits. A reduced speed of processing was also indicated by the pattern of APF, with a non-significant trend toward differences between groups in anterior regions. This pattern is suggestive of differences between groups in control mechanisms in anterior regions and the flow of information via feedback loops for working memory processes. Feedback loops for attention, involving content and context information, have not been clarified in the wider literature, but it has been suggested that working memory mechanisms may alter the timing of the transfer of information from left and right hemisphere specialised regions (Banich, 2004; Glassman, 1999). In support of this interpretation, the current data also provided evidence of interaction effects in the alpha and beta frequencies, with simple effects showing a trend toward differences in right hemisphere posterior regions.
The increased activity in right hemisphere posterior regions, for both groups, was an unexpected finding and may account for the non-significant results on behavioural tasks. One plausible explanation is that a similar pattern of cortical arousal may be associated with stress reactivity in both groups, and consequently, reduced performance scores on behavioural measures. Furthermore, an increased pattern of posterior neural activity in the higher frequencies has been associated with increased externally directed visual attention (Aston-Jones et al., 1999; Hanslmayr et al., 2005), and, in the PTSD group, with increased preparation for action during a stress response (Shaw et al., 2002). An increased pattern of externally directed attention may imply reduced attentional resources for combinatorial problem solving when context information is recognised as threat-related. This view accords with previous experimental findings suggesting that conflict monitoring that is not predictable is related to low performance outcomes (Attias et al., 1996; Bryant et al., 2005).

Although not significantly different to the control participants, the neural activation patterns of the PTSD group suggested a trend toward differences between groups for reactivity, as demonstrated by the interaction with condition effects. At the functional level, mental preparation states involve spatial or temporal elements of neural activity for the implementation of cognitive processes (Fuster, 1997). From a psychological perspective, the current data for the behavioural, or structural, elements of cognitive processing indicated a reduced capacity in PTSD participants for self-regulation and for free-recall associated with semantic processing.

From a mechanistic perspective, an alteration to attention mechanisms, associated with increased posterior right hemisphere activation patterns, implies a disruption to spatial working memory processes for a difference between visual perception (externally focused) and imagery (internally manipulated) (Bhattacharya et al., 2001; Linden et al., 2003). Thus, the current results may be suggestive of a pattern of preparedness in the PTSD group involving the engagement of distinct patterns of neural activity prior to stimulus presentation. This difference in attentional scanning implies activity associated with the conscious control of selective attention or switching attention mechanism (Awh and Jonides, 2001; Corbetta et al., 2002; Hartikainen and Knight 2003). The attentional control of retrieval processes, which have been implicated in reducing the storage capacity of spatial working memory and disrupting executive functions for flexible behaviour (Awh and Jonides, 2001; Brewer et al., 1998; Linden et al., 2003), may be one argument for alterations to semantic processing in PTSD participants. This argument supports the hypothesis that local processes for tasks can
be interrupted by a common mechanism, as a compensatory shift to right hemisphere processing in people with PTSD (Clark et al., 2003).

Another plausible hypothesis for alterations in preparatory states in people with PTSD might be linked to spatially distinct neuronal systems, which are coupled by mechanisms involving the transfer of information as long-range or diffuse codes (Holscher, 2001; Rodriguez et al., 1999). Based on the contralateral control principle of hemispheric function (Mesulam, 1998; Kolb and Whishaw, 1996), slower left hemisphere activation is likely to involve an altered pattern of engagement with monitoring mechanisms and activity in mid-frontal regions. This argument is in agreement with findings of an altered pattern of inhibitory mechanisms, in people with PTSD, as proposed by Bryant and colleagues (2005). This argument is also in agreement with the hypothesis that focusing mechanisms may serve to reduce information complexity by inhibiting frontal cortex functions via compensatory mechanisms and via an alternative pathway for well-rehearsed responses to anticipated stress stimulation (McFarlane et al., 2002; Gold, 2005). The explanation is consistent with views based on vigilance theories for a sensory-memory storage space (Arruda et al., 1999; Bearden et al., 2004).

An alternative explanation for increased higher frequency oscillations in right hemisphere posterior regions is related to the functional role of pre-stimulus attention mechanisms. According to a number of authors (Aston-Jones et al., 1999; Sejnowski and Paulsen, 2006; Rowe, 2005; Ward, 2003; Sauseng et al., 2005), the brain has an internal system that controls attention, expectation, and memory for the flexible rearrangement of cognitive operations that serve as adaptive mechanisms. An effective compensatory mechanism for adaptive functioning would require generalised processes or expectations to recognise stimuli, and focusing mechanisms based on an experiential knowledge store, to enhance the speed of pattern matching to threat cues (Herrmann, 2003; Sauseng et al., 2002). Thus, as proposed by previous models, a logical reorganisation of function in acute PTSD becomes pathological and chronic over time as the threat of danger is linked to a memory reference (McFarlane et al., 2002; Lanius et al., 2005).

The current data indicated differences in EEG topography, between groups, that may be suggestive of altered preparatory mechanisms for stimulus processing in PTSD participants, which change depending on task demands during pre-processing stages. This interpretation was based on findings suggestive of specific differences in the pattern of oscillatory activity between groups, rather than a clear hemispheric asymmetry pattern at the local level of rhythmic activity in people with PTSD. However, the above arguments do not explain the
increased alpha and beta activity in right hemisphere posterior regions in the control group, and the findings raise questions concerning lateralised shifts in posterior regions between groups. In particular, the relationship between cognitive behavioural indices and mental preparation states raises questions about characteristic rhythmic patterns between groups that are related to behavioural flexibility.

5.4. **Study 3: Relationship between Memory Retrieval Patterns and Baseline Rhythms**

5.4.1. **Introduction**

The previous two analyses investigated a shift to right hemisphere compensatory mechanisms in people with PTSD by examining local functioning for patterns of lateralisation. There were indications of altered patterns of functioning in the PTSD participants for both behavioural indices and biological indices. However a right-lateralised profile of behavioural and biological characteristics in people with PTSD was not fully supported by the data. The findings indicated alterations in PTSD participants for resting cortical arousal states; with data supporting a pattern of higher APF and lower alpha frequency amplitudes. At a functional level, differences in alpha frequency background resting states have been associated with attention mechanisms (Cooper et al., 2003; Ward, 2003). Furthermore, behavioural performance measures indicated differences between groups on the phonemic verbal production task but not the semantic production task, implying specific regional differences, possibly associated with striatal loops in the left frontal cortex (Hasegawa et al., 1998; Khader and Rosler, 2004), and perceptual differences between manipulated items and non-manipulated items (Linden et al., 2003).

Memory tests have been shown to correlate with cognitive abilities (Vasterling et al., 1998; Rockstroh and Schweizer, 2001). On the other hand, attention processes modulate cognitive abilities and are linked to arousal patterns (Felmingham et al., 2002; Rockstroh and Schweizer, 2001; Banich, 2004). In models of attention, it is proposed that there are different functions associated with interrelated neural networks (Callejas et al., 2004; Fan et al., 2005; Herrmann, 2003; Ward, 2003). These include the ability to develop and maintain an alert state, to orient to sensory input, and to execute control and planning (Fan and Posner, 2004). In the context of vigilance, arousal functions associated with attention mechanisms have been correlated with biological parameters (Rockstroh and Schweizer, 2001). However, in the PTSD literature, the relationship between memory and cognitive abilities requiring modality-specific processing has not been clarified, particularly for perceptions that remain constant over time.
The current study aimed to investigate how particular neuroanatomical regions (left hemisphere versus right hemisphere and anterior versus posterior regions), are spatially and temporally correlated with cognitive abilities and resting rhythmic states. The primary objective of this analysis was to examine the relationship among EEG measures (mean frequency, mean power, and asymmetry) and specialised verbal and nonverbal memory retrieval performance between groups. Based on the preceding analyses, it was expected that tasks involving greater attention, such as the verbal phonemic (FAS) task, would be associated with frontal regions and show a different relationship with resting oscillation patterns in the PTSD group compared to the control group. In addition, it was expected that PTSD participants would have a reduced capacity for memory tasks, as indicated by the pattern of general slowing of information processing, derived from the behavioural data. On the other hand, it was expected that both the animal category of words and the maze task would be processed as familiar tasks, in both groups, and would be associated with posterior regions for a similar pattern of associations with resting oscillation patterns between groups. However, the direction of the relationships was not predicted.

5.4.2. Method

5.4.2.1. Participants

As previously described, data was drawn from 34 participants in the PTSD group, age range from 19 to 57 years, who were age- and gender-matched with 136 control participants.

5.4.2.2. Instruments and Procedure

Data from in the preceding two analyses, as previously described, was used to explore the relationship between groups for behavioural performance measures and EEG oscillation patterns.

*Behavioural Indices:*

Behavioural performance scores, for each subtest of the verbal (word generation) and nonverbal (maze) tasks, as described in Study 1 of this chapter, were used as cognitive indices requiring modality-specific processing.
**EEG Recordings:**

As previously described, spectral power data from electrode leads (Fp1, Fp2, F3, F4, F7, F8, Fz, FC3, FC4, C3, C4, Cz, T3, T4, CP3, CP4, P3, P4, Pz, T5, T6, O1, O2, Oz) in each of the four broad frequency bands (delta, theta, alpha, beta), and from APF, for both the eyes closed and eyes open conditions, were reduced by aggregating leads in each hemisphere to correspond to left (Fp1, F3, F7, FC3, T3, C3, CP3, T5, P3, O1) and right (Fp2, F4, F8, FC4, T4, C4, CP4, T6, P4, O2) hemisphere and anterior (Fp1, Fp2, F3, F4, F7, F8, FC3, FC4, C3, C4) and posterior (T3, T4, CP3, CP4, P3, P4, T5, T6, O1, O2) regions. Data were aggregated to minimise the possibility of a Type 1 error. The mean inter-item correlations were measured using Cronbach alpha, all coefficient values were > .96. Both conditions (eyes closed/open) were examined following a method used by Jausovec and Jausovec (2000).

**Asymmetry Index:**

Of interest to the current analysis were asymmetry patterns between frontal and parietal regions. Two pairs of electrodes were chosen to measure amplitude asymmetry for intrahemispheric regions (Right: P4/F4, Left: P3/F3), providing a measure of relative activity between frontal and posterior regions within each hemisphere. As a measure of interhemispheric asymmetry, separate anterior and posterior analyses were computed for comparison by aggregating homologous leads in each region (anterior: Fp2-1, F4-3, F8-7; posterior: P4-3, T4-3, T6-5). As described in Chapter 4, alpha asymmetry indices of left- or right-brain activity can be examined by computing an asymmetry score (following Pivik et al., 1993). Because alpha power has been taken as an index of the inverse of cortical activity, positive scores are indicative of greater alpha power at the right compared to the left electrode sites, which is assumed to reflect greater left-sided brain activation. Conversely, negative scores are indicative of greater right-sided brain activation.

5.4.2.3. Data Analysis

Three separate correlation analyses were computed – APF, spectral power for four frequency bandwidths (delta, theta, alpha, beta), and an asymmetry index for separate anterior and posterior regions in each frequency, were correlated with behavioural performance measures. The relationships between the different EEG measures and behavioural performance measures were examined by using zero-order correlations (Pearson’s Bivariate correlation coefficient) for two-tailed significance. Because of missing data, all correlations are reported with participant numbers.
5.4.3. Results

5.4.3.1. Alpha Peak Frequency and Behavioural Data

Anterior/Posterior APF and Behavioural Indices:

Table 5.14 provides the correlation patterns for the PTSD group and the control group for anterior and posterior APF and the subtasks for the verbal and nonverbal behavioural data. There were intercorrelations between the performance tasks, but no significant correlations between the eyes closed condition of APF and the memory performance tasks for the PTSD group.

For the control group, results indicated a pattern of significant positive correlations between eyes closed anterior APF and animal word scores ($r = .24$, $p = 0.01$, $N = 116$), a positive correlation pattern between posterior APF with animal word scores ($r = .27$, $p = < 0.01$, $N = 116$), and a negative correlation pattern between posterior APF and average number of errors in the maze task ($r = -.18$, $p = 0.05$, $N = 123$).

There were no significant correlations in the eyes open condition between APF and cognitive tasks for either the PTSD group or the control group.

Left/Right Hemisphere APF and Behavioural Indices:

There were no significant correlations between left or right APF in the eyes closed or eyes open conditions and cognitive tasks for the PTSD group.

For the control group, the only significant correlation patterns were in the eyes closed condition for a bilateral (left and right hemisphere) pattern of significant correlations between APF and cognitive sub-tasks – animal words and maze average number of errors. There was a significant positive correlation between left hemisphere eyes closed APF ($r = .26$, $p = < 0.01$, $N = 118$) and animal words. Similarly, for right hemisphere eyes closed APF there was a significant positive correlation ($r = .25$, $p = < 0.01$, $N = 118$) with animal words. There was an inverse relationship between left hemisphere eyes closed APF and maze average number of errors ($r = -.20$, $p = 0.03$, $N = 126$), and an inverse relationship between right hemisphere eyes closed APF and maze average number of errors ($r = -.20$, $p = 0.03$, $N = 126$).
Table 5.14. Correlation matrix for PTSD participants and control participants showing relationships between anterior and posterior region APF and behavioural indices

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PTSD</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Eyes closed Anterior APF</td>
<td>-</td>
<td>.92(***)</td>
<td>-.09</td>
<td>-.06</td>
<td>.20</td>
<td>.13</td>
<td>.04</td>
</tr>
<tr>
<td>2. Eyes closed Posterior APF</td>
<td>-</td>
<td>.01</td>
<td>-.06</td>
<td>.09</td>
<td>.19</td>
<td>.11</td>
<td></td>
</tr>
<tr>
<td>3. Animal words generated</td>
<td>-</td>
<td></td>
<td>.68(***)</td>
<td>-.00</td>
<td>-.16</td>
<td>-.01</td>
<td></td>
</tr>
<tr>
<td>4. Letter words (FAS)</td>
<td></td>
<td></td>
<td>.28</td>
<td>-.23</td>
<td>-.34</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Maze completion time</td>
<td></td>
<td></td>
<td></td>
<td>-.08</td>
<td></td>
<td></td>
<td>.81(**)</td>
</tr>
<tr>
<td>6. Maze average errors</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Maze average overruns</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Eyes closed Anterior APF</td>
<td>-</td>
<td>.91(***)</td>
<td>.24(***)</td>
<td>.06</td>
<td>.09</td>
<td>-.17</td>
<td>-.05</td>
</tr>
<tr>
<td>2. Eyes closed Posterior APF</td>
<td>-</td>
<td>.27(***)</td>
<td>.07</td>
<td>.10</td>
<td>-.18(*)</td>
<td>-.06</td>
<td></td>
</tr>
<tr>
<td>3. Animal words generated</td>
<td>-</td>
<td></td>
<td>.34(***)</td>
<td>.16</td>
<td>-.27(**)</td>
<td>-.20(*)</td>
<td></td>
</tr>
<tr>
<td>4. Letter words (FAS)</td>
<td></td>
<td></td>
<td></td>
<td>-.10</td>
<td>.06</td>
<td>.10</td>
<td></td>
</tr>
<tr>
<td>5. Maze completion time</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.46(**)</td>
<td>-.44(**)</td>
<td></td>
</tr>
<tr>
<td>6. Maze average errors</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.85(**)</td>
</tr>
<tr>
<td>7. Maze average overruns</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).
5.4.3.2. Spectral Power and Behavioural Data

Eyes Closed Spectral Power and Behavioural Indices:

Table 5.15 provides details of correlation patterns for each group, showing that there were no significant correlation patterns between the four frequency bands (aggregated whole-head data) and the behavioural performance indices for the PTSD group or for the control group. The table indicates that there were fewer inter-correlations between the behavioural tasks and between the spectral power data in the PTSD group compared to the control group. While there were moderate positive correlations between the lower frequencies and the beta frequency in the control group, there were no significant correlations between the lower frequencies and the beta frequency in the PTSD group.

In separate analyses for the eyes closed condition (and frequency with region), there was only one significant correlation between cognitive tasks and oscillation patterns in the PTSD group. Average time to complete the maze correlated positively with the eyes closed alpha frequency at right hemisphere leads \( r = .37, p = 0.04, N = 31 \).

For the control group, there was a significant negative correlation in the right hemisphere beta frequency band with the phonemic words subtask \( r = -.18, p = 0.05, N = 118 \). There were no other significant findings of an association between cognitive tasks and eyes closed frequency bands in anterior or posterior regions.
Table 5.15. Correlation matrix for eyes closed spectral frequencies and behavioural performance measures

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PTSD</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Eyes closed delta</td>
<td>-</td>
<td>.73(**)</td>
<td>.39(*)</td>
<td>.30</td>
<td>-.10</td>
<td>-.15</td>
<td>.10</td>
<td>-.00</td>
<td>-.15</td>
</tr>
<tr>
<td>2. Eyes closed theta</td>
<td>-</td>
<td></td>
<td>.68(**)</td>
<td>.33</td>
<td>.05</td>
<td>-.09</td>
<td>.22</td>
<td>-.01</td>
<td>-.09</td>
</tr>
<tr>
<td>3. Eyes closed alpha</td>
<td>-</td>
<td>-.26</td>
<td>.24</td>
<td>.22</td>
<td>.24</td>
<td>.20</td>
<td>.10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Eyes closed beta</td>
<td>-</td>
<td>-.08</td>
<td>-.13</td>
<td>-.15</td>
<td>-.28</td>
<td>-.20</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Animal words generated</td>
<td>-</td>
<td></td>
<td></td>
<td>.68(**)</td>
<td>-.00</td>
<td>-.16</td>
<td>-.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Letter words (FAS)</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td>.28</td>
<td>-.23</td>
<td>-.34</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Maze time to completion</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.08</td>
<td>-.31</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Maze number of errors</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.81(**)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Maze number of overruns</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Control</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Eyes closed delta</td>
<td>-</td>
<td>.76(**)</td>
<td>.38(**)</td>
<td>.30(**)</td>
<td>.05</td>
<td>-.11</td>
<td>.08</td>
<td>-.04</td>
<td>.02</td>
</tr>
<tr>
<td>2. Eyes closed theta</td>
<td>-</td>
<td></td>
<td>.66(**)</td>
<td>.38(**)</td>
<td>-.04</td>
<td>-.09</td>
<td>-.02</td>
<td>.09</td>
<td>.11</td>
</tr>
<tr>
<td>3. Eyes closed alpha</td>
<td>-</td>
<td></td>
<td></td>
<td>.42(**)</td>
<td>.01</td>
<td>-.09</td>
<td>-.02</td>
<td>.02</td>
<td>.00</td>
</tr>
<tr>
<td>4. Eyes closed beta</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td>.00</td>
<td>-.17</td>
<td>-.09</td>
<td>.04</td>
<td>.04</td>
</tr>
<tr>
<td>5. Animal words generated</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.34(**)</td>
<td>.16</td>
<td>-.27(**)</td>
<td>-.20(*)</td>
</tr>
<tr>
<td>6. Letter words (FAS)</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.10</td>
<td>.06</td>
<td>.10</td>
</tr>
<tr>
<td>7. Maze time to completion</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.46(**)</td>
<td>-.44(**)</td>
<td></td>
</tr>
<tr>
<td>8. Maze number of errors</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.85(**)</td>
<td></td>
</tr>
<tr>
<td>9. Maze number of overruns</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
**Eyes Open Spectral Power and Behavioural Indices:**

For the PTSD group, the only significant correlation pattern for the eyes open condition was a positive correlation between the delta frequency in anterior regions and average time to complete the maze ($r = .43, p = 0.02, N = 30$).

For the control group, the only significant correlation in the eyes open condition was a negative correlation between beta frequency in the right hemisphere and the phonemic words task ($r = -.20, p = 0.03, N = 119$).

**5.4.3.3. Asymmetry and Cognitive Performance**

**Eyes Closed Correlation Patterns:**

Table 5.16 shows that there were no significant correlation patterns in the alpha asymmetry indices (in anterior and posterior regions) and cognitive performance scores for the PTSD group or the control group in the eyes closed condition.

However, correlation patterns for the control group were significant between theta asymmetry index and the maze subtasks. Average time to complete the maze was significantly and negatively correlated with theta in a bi-lateral derivation P4-F4 ($r = -.21, p = 0.02, N = 126$) and also P3-F3 ($r = -.18, p = 0.04, N = 126$). There was a significant positive correlation for theta asymmetry index with average number of errors (P4-F4: $r = .28, p = < 0.01, N = 125$), and theta asymmetry with average number of overruns (P4-F4: $r = .19, p = 0.03, N = 125$), for a relatively right hemisphere derivation.

**Eyes Open Correlation Patterns:**

There were no significant correlations between the cognitive subtasks and frequency pattern asymmetry indices in the eyes open condition for the PTSD group.

For the control group, there was only one significant correlation for the alpha asymmetry index in the eyes open condition. Animal word scores correlated positively with a left derivation at P3-F3 ($r = 20, p = 0.03, N = 119$). For the beta asymmetry index there was a positive correlation also with a left derivation at P3-F3 ($r = .19, p = 0.04, N = 119$) and animal words.
Table 5.16. Correlation matrix between eyes closed alpha anterior and posterior region asymmetry index and cognitive performance scores

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PTSD</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Alpha anterior asymmetry</td>
<td>-</td>
<td>-.19</td>
<td>-.00</td>
<td>-.05</td>
<td>.13</td>
<td>-.24</td>
<td>-.06</td>
</tr>
<tr>
<td>2. Alpha posterior asymmetry</td>
<td>-</td>
<td>-.14</td>
<td>.19</td>
<td>.03</td>
<td>-.04</td>
<td>-.17</td>
<td></td>
</tr>
<tr>
<td>3. Animal words generated</td>
<td>-</td>
<td></td>
<td>.68(**)</td>
<td>-.00</td>
<td>-.16</td>
<td>-.01</td>
<td></td>
</tr>
<tr>
<td>4. Letter words (FAS)</td>
<td>-</td>
<td>-.14</td>
<td>.19</td>
<td>.03</td>
<td>-.04</td>
<td>-.17</td>
<td></td>
</tr>
<tr>
<td>5. Maze completion time</td>
<td>-</td>
<td>-.04</td>
<td>-.08</td>
<td>.81(**)</td>
<td>-.31</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Maze average errors</td>
<td>-</td>
<td>-.34(**)</td>
<td>.16</td>
<td>-.27(**)</td>
<td>-.20(*)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Maze average overruns</td>
<td>-</td>
<td>-.10</td>
<td>-.10</td>
<td>.06</td>
<td>.10</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| **Control** |        |        |        |        |        |        |        |
| 1. Alpha anterior asymmetry | -      | -.13   | -.14   | .02    | .10    | .01    | -.01   |
| 2. Alpha posterior asymmetry | -      | -.04   | -.08   | -.16   | .03    | .05    |        |
| 3. Animal words generated | -      |        | .34(**)| .16    | -.27(**)| -.20(*)|        |
| 4. Letter words (FAS) | -      | -.10   | -.10   | .06    | .10    |        |        |
| 5. Maze completion time | -      | -.46(**)| -.46(**)| -.44(**)| .85(**) |        |        |
| 6. Maze average errors | -      |        |        |        |        |        |        |
| 7. Maze average overruns | -      |        |        |        |        |        |        |

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
APF and Cognitive Performance:

There were no significant correlation patterns for the PTSD group between APF and cognitive tasks (verbal and nonverbal).

On the other hand, correlation patterns for APF and cognitive tasks in the control group indicated a bilateral positive association with animal words, while the bilateral association between APF and the number of errors in the maze task was negative. Examining anterior and posterior region differences, the pattern of significant correlations in the control group was of a positive association in both anterior and posterior regions with animal words. In contrast, a negative correlation pattern was demonstrated for APF and maze errors in posterior regions.

Spectral Power and Cognitive Performance:

The only statistically significant cognitive task associated with frequency patterns in the PTSD group was average time to complete the maze. For this task there was a positive right hemisphere correlation pattern in the alpha frequency with eyes closed. However, this was different to the pattern for the eyes open condition, which revealed a positive association between time to complete the maze and delta frequency in anterior regions.

On the other hand, for the control group, the only task to correlate in the eyes closed condition was FAS words with a negative right hemisphere pattern in the beta frequency. This pattern was repeated in the eyes open condition. The results indicated a consistent frequency pattern was associated with FAS words in the control group.

Asymmetry and Cognitive Performance:

There were no significant correlation patterns for asymmetry indices in the four frequencies (eyes closed or eyes open) and cognitive tasks in the PTSD group.

On the other hand, the control group data indicated significant correlation patterns between theta asymmetry in the eyes closed condition and the maze subtasks. For time to complete the maze there was a bilateral negative correlation with theta asymmetry patterns for posterior and anterior regions. For the error monitoring subtasks (average errors and average overruns),
there was a positive right hemisphere significant correlation with theta asymmetry in posterior and anterior regions.

In the eyes open condition, the control group data indicated a pattern of posterior and anterior asymmetry positively associated with the higher frequencies (alpha and beta) and animal words for a left hemisphere derivation.

5.4.4. Discussion

Together with the behavioural and electrophysiological data for this chapter, the correlation results may be consistent with the hypothesis of a disruption to memory codes involving visual representations and compensatory mechanisms associated with rehearsal processes (Clark et al., 2003). However, the argument for a lateralisation hypothesis, based on the storage of visuospatial and object features in the activation of prefrontal cortex functions, was not supported by the current pattern of results.

As previously discussed (Chapters 2 and 3 of this study), a spatio-temporal hypothesis has been proposed as a possible mechanism for the interruption to specialised coding processes and has been linked to structural abnormality in people with PTSD (Shaw et al., 2002). Alterations to knowledge structures, consistent with hypotheses suggesting a decoupling of anterior and posterior regions, have been associated with processes of visual acuity, or the capacity for enhanced visual memory that would be necessary for encoding new information (Lanius et al., 2005). Therefore, this argument proposes that a change in context information, incorporating the order of items, or the functional role of specific items, would be consistent with a modulation of attention processes for sensory stimuli in preparatory or anticipatory states (Dayan et al., 2000; Nadel and Moscovitch, 1998). However, dynamic processes indicating alterations in arousal states were not measured by the current analysis, thus limiting an interpretation of tonic patterns in neuro-physiological functions to differences in storage functions at the local level of processing.

Eyes Closed APF and Behavioural Tasks:

Results for the PTSD group indicated no significant correlations between behavioural task scores and APF. The lack of a relationship between these measures partly supported models proposing that a pattern of general slowing of information processing is associated with reduced memory capacity in people with PTSD (McFarlane et al., 1993; Vasterling et al., 2004). Thus, APF indices of functioning may be suggestive of altered temporal coding in
people with PTSD and the modulation of attention mechanisms for visual acuity. That is, as one interpretation, this result is consistent with notions that perceptual speed correlates with mental ability and altered attentional mechanisms in PTSD participants (Vasterling et al., 2004, 2006). Based on this argument, it can be inferred that an alteration to the speed of processing in people with PTSD may be associated with altered attention mechanisms in preparatory states.

On the other hand, results for the control group may be suggestive of a link between speed of processing and connectivity between posterior and anterior cortical regions. The APF pattern of correlations between anterior and posterior regions and memory retrieval of non-manipulated stored items (animal words and error monitoring) in the control group was consistent with models predicting that behavioural flexibility is associated with attentional scanning (Aston-Jones et al., 1999).

Significant relationships between APF (eyes closed condition) and cognitive tasks for the control group partly supported the hypothesis predicting that the processing of familiar tasks would be linked to posterior regions. Various researchers have proposed that information retrieved from memory can be accessed using different pathways based on familiarity or recollection; where familiarity has been referred to as past experience, irrespective of how the items were encoded or the order of events (Van Horn et al., 1998; Lou et al., 2005; Mesulam, 1998; Niebauer, 2004). On the other hand, recollection has been referred to as events that have occurred in the recent past, characterised by all information associated with the experience (Nadel and Moscovitch, 1998; Eichenbaum and Fortin, 2005; Howard et al., 2006). Hence different mechanisms will prime networks for memory codes. The important distinction between the two forms of retrieval for stimuli is the time that has passed since the experience and the order of the events (Eichenbaum and Fortin, 2005). Therefore, proposing an argument of differences in APF, in people with PTSD, may be consistent with notions of differences in the signal strength for encoding new information.

Only two behavioural measures (oral production of animal words and average number of errors in the maze task) were correlated with APF in the control group. The results were interpreted from a working memory model perspective. One possible explanation for this pattern of correlations is related to the degree of manipulation required in working memory systems for animal words and error monitoring. It can be speculated that the relationship between these variables is linked to memory functioning and the ability to retrieve this information automatically, without manipulation. That is, it can be surmised that animal
words and maze errors in retrieval tasks are associated with rapid perceptual processing and are suggestive of a relationship for higher capacity in visual memory in the control group, compared to the PTSD group. Error monitoring, particularly associated with a negative correlation pattern in posterior regions, would be consistent with models proposing that perceptual codes can be maintained in transitory states (Glassman, 1999; Singer, 1999). Based on this argument, the current data support the hypothesis for efficiency and connectivity between regions, in the control group, with increased attentional scanning related to the ability to maintain imagery for cognitive task processing and self-regulation.

In addition, the current results could also be interpreted from a framework arguing that connectivity between cortical regions, particularly between frontal and posterior regions is consistent with thalamo-cortical responsiveness (Basar, 2004; Hoeschler, 2001). For the control group, as expected, the results for the pattern of APF and cognitive tasks were interpreted based on models suggesting that relationships between neural activity and memory codes are indicative of effective synaptic connectivity for the functional involvement of long-term memory access and the updating of information (Basar, 2004; Munk, 2001). The results for the association between APF and behavioural tasks also converge with the results for the previous two studies, in this chapter. Based on indicators of a slower speed of processing in the PTSD group, and lower performance scores compared to the control group, the differences in the correlation patterns between groups support arguments for alterations in neural coding in people with PTSD.

However, another plausible explanation for the pattern of correlations for the current study is that there was insufficient statistical power to find a significant result. In the eyes open condition there were a reduced number of participants in the PTSD group from which data was drawn. Thus the results may be spurious, and, therefore, must be cautiously interpreted. This possibility leaves open the question of whether reduced information processing capacity in people with PTSD is linked to specific spatial coding mechanisms, temporal mechanisms associated with differences in function, or spatio-temporal mechanisms that are uniquely related to compensatory functioning.

*Spectral Power and Memory Retrieval Performance:*

The hypothesis that familiar tasks would be associated with the beta frequency was not supported by the data. However, the results of correlation analyses between spectral frequencies and cognitive tasks supported the hypothesis of an altered pattern of resting
activation patterns in PTSD participants compared to control participants. Furthermore, the results may be relevant to proposals of compensatory mechanisms in people with PTSD (Clark et al., 2003; Lanius et al., 2005) and with proposals arguing that neural patterns in people with PTSD are unstable (Neylan et al., 2003). In addition, alpha synchrony, as an active process to dynamically regulate the system, may be altered in people with high anxiety (Knyazev et al., 2005).

In the PTSD group, the significant correlation pattern in the eyes closed condition, between alpha power in the right hemisphere and time taken to complete the maze (an index of learning), is a particularly relevant finding when compared to the pattern of significant correlations in the eyes open condition. The most unusual finding in the PTSD group pattern of correlations was the positive association between time to complete the maze and the delta frequency (eyes open) in anterior regions. This finding may be consistent with models suggesting a different pattern of resting frequencies in people with PTSD (Metzger et al., 2004), or in people with anxious apprehension (Knyazev et al., 2006b).

The pattern of correlations for the PTSD group, for both the eyes closed condition and the eyes open condition, may be linked to arguments of an alteration to the pattern of rehearsal mechanisms in working memory models (Chein et al., 2003; Courtney et al., 1998a; Glassman, 1999). Models proposing that spatial working memory and selective attention are associated with a shared neurobiological substrate argue for a disruption to the coordinated flow of information when one signal arrives within a faster time window (Thatcher, 1997; Banich, 2004). This pattern may be in agreement with models predicting alterations to cognitive functioning at the structural level in people with PTSD (Vasterling et al., 2004; Lanius et al., 2005; Jatzko et al., 2006; Metzger et al., 2004), and suggestive of compensatory mechanisms that alter tonic states, (McFarlane et al., 2002).

To understand how the above arguments are in agreement with the finding of a positive correlation pattern between alpha right hemisphere (eyes closed) and time taken to complete the maze, the various elements of this relationship will be considered separately. Based on models reviewed in Chapter 2, alpha synchrony has been shown to be associated with attention mechanisms and the maintenance of an alert state (Klimesch, 1999; Knyazev et al., 2005). Therefore, one plausible hypothesis for the current finding may be related to dual-task representations in right hemisphere processing for the differential involvement of controlled attention mechanisms (Linden et al., 2003; Petrides, 2000). If this hypothesis is correct, it would suggest that sustaining focused attention in the maze task for PTSD participants would
require increased effort when there is an overlap in neurobiological substrates for spatial working memory processes and selective attention.

Furthermore, the sub-processes involved in learning a maze path are dynamic and involve many regions of the cortex (Fletcher et al., 1995; Van Horn et al., 1998), including sub-vocal rehearsal of the different sections of the path traversed and identified as correct. Models of list learning have shown that the temporal order of items is important in memory mechanisms (Lisman et al., 2001). This hypothesis is relevant to the finding of a positive correlation pattern between the maze time to completion and anterior brain regions in the delta frequency for the eyes open condition in PTSD participants. Therefore, it is possible that there is an interruption to selective attention mechanisms in visual representations for preparedness of external stimulus processing (Knyazev et al., 2005). This argument may be relevant to an interaction effect in people with PTSD with processing of the maze task in spatial working memory (right hemisphere posterior regions) and the rehearsal of items for updating information (left hemisphere anterior regions).

In the PTSD group, the link between the slower delta frequency pattern and cognitive processing may be suggestive of structural abnormality and reactivity effects with eyes open (Knyazev et al., 2006b; Basar, 2004; Niedermeyer, 2005; Vasterling et al., 2004). Rehearsal mechanisms have been linked to the low delta frequency as compensatory mechanisms in memory functioning (Klimesch, 2003). Therefore, to take the above arguments further, if the phenomenon of delta synchrony, as a rehearsal strategy is correct, it may be a plausible mechanism for shifts between tonic and phasic states, and for a pattern of unstable synchronisation in people with PTSD. As suggested by Neylan and colleagues (2003), people with PTSD may not have disturbed arousal patterns but may have an altered pattern of structural functioning. The pattern of correlations between alpha right hemisphere power (eyes closed) and delta anterior power (eyes open), and time taken to learn a maze task, may be partial support for a model proposing altered temporal mechanisms in people with PTSD that are associated with shared neurobiological substrates. Furthermore, a disruption to theta synchrony has been a consistent pattern in the PTSD literature and associated with the disruption to working memory processes (Veltmeyer et al., 2006). Thus, from the perspective of a working memory model, a lateralisation hypothesis in people with PTSD (Clark et al., 2003), based on mechanisms for the alteration to spatial coding, can be interpreted as a disruption to rehearsal mechanisms through altered synchrony patterns.
In contrast, patterns of neural activity and cognitive performance, from the control group data, indicated an association between cognitive tasks and the higher beta frequency. This pattern was negatively correlated for FAS words and right hemisphere beta power, in both the eyes closed and eyes open conditions. The results were interpreted as suggestive of different timing mechanisms for memory patterns between the PTSD group and the control group. This hypothesis may be linked to more recent suggestions of a connection between spectral changes, cerebellar activity, and sensory motor patterns (Shaw et al., 2002; Schutter and van Honk, 2006). Thus, implicating emotional responses in people with PTSD, and associated with an interaction effect between norepinephrine and acetylcholine systems in decision processes linked to uncertainty (Dayan and Yu, 2003). However, this interpretation must be considered speculative without further testing and replication.

The relationship between the FAS subtask of verbal memory and beta frequency, in right hemisphere anterior and posterior regions in the control group, may be suggestive of a functional connectivity pattern for variables that involve long-range connections for the retrieval from long-term memory. That is, it is possible that the significant correlation between the retrieval and production of phonemic words in the control group is associated with increased arousal levels for this task. On the other hand, arguing from the perspective that a visual lexicon of right hemisphere processing supports a generative lexicon (Gazzaniga et al., 2002) associated with left hemisphere processing; the current results may be in agreement with models that propose specialised hemispheric processing for tasks, depending on task demands (Gevins and Smith, 2000; Sauseng et al., 2002). The current findings can be interpreted as a plausible argument for a lateralised effect in specialised verbal and nonverbal tasks at the global level of processing, and the different correlation patterns for the PTSD group and the control group are suggestive of altered synchrony patterns in people with PTSD for task demands. This argument is in agreement with previous evidence in the PTSD literature, of a disruption to verbal working memory processes and frontal system networks (Clark et al., 2003), and provides partial support for functional cerebral asymmetry favouring right hemisphere processing.

Asymmetry Correlation Pattern with Behavioural Tasks:

Relationships between neural activity and cognitive processes were examined for differences between groups using an asymmetry index of relative frontal and posterior derivations in left and right hemisphere neural activity patterns. In the PTSD group, there were no significant findings of relationships between asymmetry patterns in frontal-parietal derivations and
cognitive tasks. This finding supported the data of non-significant correlations between APF and cognitive tasks in people with PTSD. Therefore, the results were interpreted as suggestive of altered patterns of oscillations in preparatory states, and associated with altered connectivity patterns in people with PTSD.

On the other hand, the pattern of correlations for the control group (in the eyes open and eyes closed conditions) was consistent with connectivity between left and right hemispheres and between frontal and posterior regions for the integration of verbal and nonverbal processes. This interpretation of the results was based on models proposing that synchrony patterns are associated with spatial and temporal connectivity (Singer, 1999) and supports earlier conclusions drawn from the correlation patterns of APF data and spectral frequency data.

One hypothesis has linked altered patterns of core frequencies, particularly theta activity in hippocampal regions, to structural knowledge in memory functioning (Rowe, 2005; Klimesch, 2003). Networks associated with theta synchrony have also been linked to hippocampal functioning and working memory processes (Klimesch, 2003; Munk, 2001). The asymmetry patterns (in the eyes closed condition) suggested a bilateral pattern of negative associations with time to complete the maze, and a positive right hemisphere association with error monitoring, but only for the theta frequency in the control group. In contrast, the pattern of asymmetry correlations for the eyes open condition indicated an association with attention and binding mechanisms. The higher alpha and beta frequencies were associated positively with animal words, but only for left hemisphere derivations. Thus, the asymmetry in fronto-parietal networks associated with animal words and the higher frequencies in the control group were taken to represent alterations to relative synchrony patterns, depending on task demands. This interpretation is in agreement with models proposing a disruption to controlled attention and the maintenance of a visual representation through left hemisphere processes (Linden et al., 2003; Miyake et al., 2001; Ungerleider et al., 1998).

Based on this model, the relationship between alpha and beta frequencies and memory codes, for the control participants, was interpreted as one suggestive of attentional scanning in posterior regions associated with long-range functional connectivity between regions. As an alternative hypothesis, the correlation pattern between asymmetry scores and performance scores in the higher beta frequency may be associated with thresholds of recognition and false alarm rates for the correct identification of items, and may also be linked to confidence in decision processes, implicating emotion regulation (Dayan and Yu, 2003). However, the
pattern of associations between frequency asymmetries and animal words was only weakly correlated and further investigation is required for a plausible interpretation of the data.

5.5. General Discussion and Conclusions

The question of whether working memory systems in people with PTSD are lateralised as compensatory mechanisms that support emotion regulation, was investigated in this chapter, by examining psychobiological functioning using three separate analyses. The main objective of this chapter was to investigate the proposal for cortical lateralisation in PTSD (Clark et al., 2003; Vasterling et al., 2004) by comparing relationships between tonic, resting qEEG patterns with behavioural measures of cognitive performance. The speed of processing, as a general function, associated with connectivity between structures based on relatively fixed networks of local processing for specialised verbal and nonverbal tasks, formed the defining variable for the functional roles of neural and behavioural patterns.

The hypothesis of compensatory mechanisms in people with PTSD, for increased coding in right hemisphere processing, was only partly supported by the data. Behavioural and qEEG analyses indicated two pathways for specific functioning in people with PTSD. These related to visual memory systems, associated with right hemisphere processes and posterior regions, and rehearsal systems, associated with left hemisphere anterior regions. Similarly, the hypothesis of a functional reorganisation of neural patterns in people with PTSD was also partly supported by the current data. The results from the PTSD data indicated no associations between cognitive tasks and measures of the speed of frequency (APF), or asymmetry patterns for a relative shift in connectivity associated with frontal and parietal regions. But more importantly, the significant correlation patterns indicated altered frequencies for an association between resting states and cognitive tasks. Findings from the separate analyses in this chapter are summarised from the perspective of four characteristics associated with memory functioning, for an analysis of possible alterations associated with psychobiological indices, in PTSD participants compared to control participants.

Based on interpretations offered in the preceding sections, these are described as follows: (1) Core frequencies in resting anticipatory states are associated with alterations to the preparation for encoding sensory stimuli and implicate the modulation of attention mechanisms. (2) Alterations to synchrony patterns associated with connectivity between regions are associated with the speed of processing and APF rhythms for altered timing mechanisms in core frequencies. (3) Relationships between cognitive processes and synchrony patterns are associated with rehearsal mechanisms during retrieval processes and
may depend on temporal mechanisms, spatial coding mechanisms, or an interaction of spatio-temporal mechanisms for connectivity between regions. (4) Plasticity, as a compensatory mechanism may be associated with rehearsal mechanisms and altered synchronisation patterns at a structural level of cognitive architecture and linked to timing mechanisms involving cortical and subcortical regions. This mechanism implies a change to knowledge structures and an altered pattern of functioning possibly associated with a defining event from environmental influences. Alternatively, plasticity as a compensatory mechanism altering executive functioning may be associated with an instability of neural network paths depending on the processing of context and content information during phasic states. This mechanism implies changes at the implementation level of cognitive architecture through attention and learning, the modulation of electro-chemical balance, and genetic as well as environmental influences.

Arguments supporting the above hypotheses are briefly reviewed. However, the above conclusions have been formulated, in part, on data derived from correlation analyses, which are exploratory variables for examining directions for future research, rather than causal explanations for a functional analysis.

5.5.1. Attention and Self-regulation Associated with Local Processing Structures

Electrophysiological indices of tonic preparatory states, for stimulus processing and for information processing functions, were not predicted a priori in the current study. In contrast to findings by Begic and colleagues (2001), the current results demonstrated little variability between groups in resting EEG cortical activation patterns. The qEEG data for this study indicated a consistent pattern of cortical activation in both groups with significant differences between groups in the alpha frequency. The lower pattern of alpha power in people with PTSD was in agreement with findings by Jokic-Begic and Begic (2003). Alpha resting frequency has been associated with attention modulation and perceptual memory processes (Aston-Jones et al., 1999; Cooper et al., 2003). In frontal regions, depending on asymmetry patterns, alpha synchrony has been associated with affective states (Blackhart et al., 2006; Bruder, 2004; Davidson et al., 1990).

In the current study, interaction effects for frequency signals, in laterality and reactivity, indicated not only altered patterns of anticipatory states in the PTSD participants compared to the control participants, but also suggested a shift in tonic characteristics. The results were in agreement with models suggesting instability in cortical arousal in PTSD, not related to symptom categories (Neylan et al., 2003), but to alterations in functioning associated with the
secondary characteristics of learning after traumatic events (McFarlane et al., 1993; Galletly et al., 2001). The current study also examined asymmetries in baseline mental preparation states, for both anterior and posterior regions as well as laterality effects for modality-specific memory processing. There were no lateralised patterns evident for the PTSD group. However, interaction effects supported regional differences in the pattern of oscillations for left and right hemisphere activity. The data were significant for alterations to the pattern of frontal activity in people with PTSD, but not for posterior patterns of alpha oscillations. However, there was an unexpected finding indicating that both groups shifted to a lateralised pattern of alpha oscillations in posterior regions. Therefore, a plausible interpretation of the data suggested a shift in the pattern of baseline cortical arousal for tonic features in the PTSD group.

A pattern of altered theta activation has been found in previous studies of resting EEG patterns in PTSD (Begic et al., 2001; Jokic-Begic and Begic, 2003; Veltmeyer et al., 2006). However, there were no significant results in the current study for activation or laterality associated with the theta frequency bandwidth. Therefore, further clarification, using cognitive processes, that are dynamic not static, is suggested for an understanding of how different neural circuits integrate information during memory processes in phasic states.

As complementary effects of theta and alpha, in different cognitive operations occurring in cortico-thalamic circuits (Basar, 2004; Ward, 2003), it is plausible that the lower alpha and theta patterns for the PTSD group are associated with a mechanism involving a faster oscillation feedback loop for searching and pattern matching to encoded information relevant to threat perception. Furthermore, an association between cognitive tasks and the delta frequency, in anterior regions, may be a significant finding, and useful in understanding models suggesting a reorganisation of neural pathways as a compensatory mechanism in PTSD (McFarlane et al., 2002).

Different models have demonstrated that anticipation of a stimulus is associated with the entrainment of interneurons in higher frequency networks for better memory performance (Klimesch et al., 2005; Schwartz et al., 2005). Arguments supporting these models suggest that lower alpha activity enhances perceptual performance because the cortex is activated and prepared to process a specific stimulus (Aston-Jones et al., 1999). On the other hand, higher alpha frequency activation is thought to inhibit irrelevant information for controlled attention and the flexible storage and manipulation of information (Klimesch et al., 2005). This view is consistent with those arguing that alterations to neural network pathways in PTSD are
associated with cholinergic neuromodulatory effects and structural abnormality (McFarlane et al., 2002).

Accordingly, different models in the PTSD literature have been proposed for a disruption to attention mechanisms in people with PTSD (Buckley et al., 2000; Felmingham et al., 2002; Vasterling et al., 1998), and a link between attention mechanisms and executive processing in the discrimination of stimuli (McFarlane et al., 1993). The various models were reviewed in the first two chapters of this study. The current results provided evidence of alterations to the alpha frequency in resting states in the PTSD group, supporting an inference that a disruption to attention mechanisms alters voluntary and conscious processing of information, in people with PTSD, which was presented as a plausible explanation for the difference in cognitive performance measures between groups in this study.

From the perspective that preparatory resting states are linked to attention mechanisms, the pattern of preliminary results for the current study may be consistent with reduced capacity for mental preparation in people with PTSD. Disturbed processes of encoding, retention, and manipulation may be indicative of the low alpha power and the lower performance scores in the PTSD group and associated with reduced behavioural flexibility (Aston-Jones et al., 1999). The results suggest a hypothesis consistent with proposals that language-based interpretations of objects and stimuli are associated with emotion regulation at the encoding stage of information processing (Brewin, 2003; Ehlers and Clark, 2000; Nadel and Moscovitch, 1998), and with priming by specific context cues at the retrieval stage (Dayan and Yu, 2003; Glassman, 1999). This interpretation is in agreement with suggestions that uncertainty in decision processes, or discrimination of stimuli, is associated with attention mechanisms and a reduced speed of processing, as reported in an early study by McFarlane and colleagues (1993) and later by Vasterling and colleagues (2004). A mental state of preparation for anticipated stimuli is likely to involve various reference codes for the identification of stimuli and temporal codes associated with meaningful material (Sejnowski and Paulsen, 2006; Svoboda et al., 2006; Wagner et al., 1999) for fast early recognition of perceived threat (Williams et al., 2006).

5.5.2. Speed of Processing in PTSD

In an assessment of the speed of information processing, measuring the capacity for intentional behaviour, there were significant differences for the sensori-motor task, indicating general slowed information processing in the PTSD group. Furthermore, the results for the verbal task also indicated slower speed of processing in verbal fluency in the PTSD group.
Results involving relationships between neural resting patterns and cognitive tasks indicated a pattern of alterations to core frequencies was associated with differences between groups for asymmetry patterns and regional connectivity patterns. Thus, it can be speculated that in people with PTSD, an asymmetry for the transfer of information between hemispheres, or the integration of information in parallel processing systems, may be associated with perceptual speed and the meaning assigned to categories of context information.

This interpretation of the current pattern of results is also in agreement with models positing that temporal channels are a functional strategy for the modulation of attention mechanisms (Banich, 2004). Arguments for this hypothesis are also based on models suggesting that dedicated units of neural activity achieve sensory processing, which can be altered by modifications of sensory-motor integration based on time-dependent binding, which impair cognitive processes and conscious perceptions (Ribary, 2005), particularly if associated with changes to cerebellar activity (Schutter and van Honk, 2006).

The current results are partly in agreement with previous findings by Clark and colleagues (2003) for an alteration in verbal working memory systems, and are in agreement with previous evidence of a disruption to executive functioning associated with frontal systems. Based on a hypothesis implicating alterations to the speed of processing, the current data may be plausibly interpreted as a shift in executive functions, in people with PTSD, and an alteration in prefrontal networks affecting the capacity for intentional behaviour; in particular, for verbal working memory and left hemisphere processing. On the other hand, it is also plausible that all participants processed the nonverbal maze task as a familiar task, and the lack of significant differences between groups can be interpreted as a similar capacity for efficiency involving executive functioning for specific task demands.

However, of particular interest to altered functioning in people with PTSD, is the notion that episodic memories are altered by hippocampal functions, particularly associated with selective attention and retrieval processes (Nadel and Moscovitch, 1998). The role of the hippocampus in shifting attention may be useful in understanding how visual information is integrated. Behavioural measures in this study were more consistent with an alteration in the speed of information processing and a relationship between synchronisation and visual memory for items that are not manipulated. This pattern is in agreement with hypotheses proposing a link between self-registration and selective attention (Svoboda et al., 2006). This hypothesis was also supported by the electrophysiological data; results indicating a trend toward lower alpha power in left hemisphere frontal regions for resting cortical arousal.
patterns in participants with PTSD. This finding implicates alterations to spatial and temporal parameters of information processing in PTSD.

The data for the behavioural pattern of functioning in people with PTSD were interpreted as suggesting a shift in the capacity for self-regulation and it is speculated that this is possibly linked to compensatory mechanisms at the global level for the integration of information. The specificity of efficient processing, compared to slowed information processing in people with PTSD, may be plausibly related to disruptions in sensory-motor pathways and the rhythmic activation patterns of neural networks, as proposed by Shaw and colleagues (2002). Thus, the current results also implicate alterations to theta frequency rhythms, as suggested by previous PTSD research (Veltmeyer et al., 2006).

5.5.3. Rehearsal Mechanisms and the Capacity for Intentional Behaviour

The current results did not support the hypothesis of a consistent laterality effect in local processing and, therefore, do not suggest a consistent difference in attentional control between groups for specialised hemispheric processing functions associated with memory coding. However, the current data, indicated sensori-motor deficits in people with PTSD, and lend support to conclusions presented by previous research suggesting that reflexive stress responses increase activation in parietal and motor association cortex regions and inhibit frontal cortex functions (Shaw et al., 2002). This pattern of cortical reorganisation may be associated with compensatory mechanisms that underpin working memory storage processes for attentional control (Clark et al., 2003; Bremner et al., 1999).

As a mechanism for compensatory behaviour, it is plausible that emotion is a mediator variable in the relationship between the speed of processing and the capacity for intentional behaviour. However, an unexpected finding in the current data was a shift to right hemisphere posterior alpha spectral activity in both groups. This lateralisation effect was unexplained, but, speculatively, was interpreted as an alteration in tonic states for those in the control group who may have experienced reactivity to prior traumatic stressor events. If this hypothesis is correct, the results are congruent with those presented by Vasterling and colleagues (2006), arguing that alterations in cognitive states are a result of innate processes after experiencing perceptions of threat. As found by Vasterling and others, the speed of processing on simple sets of information is altered in people who have been exposed to extreme stress.

Evidence suggesting a right hemisphere neural circuit, involving the occipital, dorsal parietal and superior frontal cortex regions, is accumulating in the PTSD literature (Clark et al., 2003;
Jatzko et al., 2006; Lanius et al., 2005). In addition, the data for the current study indicated a consistent trend in the PTSD group to show altered reactivity patterns from the eyes closed to the eyes open condition, suggesting phasic alterations as well as tonic alterations. However, a number of theoretical issues remain unresolved, particularly pertaining to questions of capacity limits, shared resources for attention and spatial working memory, and mnemonic representations defining the dimensional relations of objects, temporal parameters and location of stimuli. These issues may be related to the subjective definition of context information, based on focused or selective attention bias toward threat-stimuli in people with PTSD (Bryant et al., 2005), and higher-order expectations (Conway and Pleydell-Pearce, 2000; Dayan et al., 2000; Mesulam, 1998).

This conclusion leads to the question of whether the voluntary control of attention influences the speed of processing. This can be dissociated from attention processing that alters the coordination and flow of information for a decoupling of localised connectivity patterns, to a reorganisation of communication channels that link different cortical regions at a global level. According to a number of authors (Sejnowski and Paulsen, 2006; Rowe, 2005; Ward, 2003; Sauseng et al., 2002; Aston-Jones et al., 1999), the brain has an internal system that controls attention, expectation, and memory for the flexible rearrangement of cognitive operations that serve as adaptive mechanisms. However, as previously discussed, neural codes can alter depending on a number of parameters (Singer, 1999).

Patterns of memory for the classification of objects are established through learning and are subsequently altered by learning and changes to perception (Nadel and Moscovitch, 1998). In people with PTSD, changes after traumatic events have been associated with a decreased capacity to process early information (Attias et al., 1996; Bryant et al., 2005). Thus, low alpha power in resting states could suggest a decreased capacity to process early information as a compensatory mechanism for the maintenance of controlled attention in local networks for rehearsal processes (Veltmeyer et al., 2006).

5.5.4. Plasticity as a Compensatory Mechanism

The findings of the current study indicated a relationship between different networks for cognition and adaptive functioning. For the PTSD group, the results were consistent with a functional abnormality of lower amplitudes, which implied the disruption to the maintenance and coordination of information (Knyazev et al., 2006b). Retrieval of stored information, associated with functions of memory processes, also indicated that structural abnormality was associated with attention processes, as revealed by the pattern of results for alpha peak...
frequency (Angelakis et al., 2004; Clark et al., 2004) and by the lower performance scores in the PTSD group. For the control group, there were unexpected results, the data indicating higher alpha frequency activity in right hemisphere posterior regions. It is possible that compensatory mechanisms are related to patterns of heterogeneity in both groups, the differences in cortical arousal masked by individual trait characteristics. This finding suggests implications associated with working memory functioning; specifically, the capacity to inhibit or facilitate the organisation of representations for information processing involving global networks in functional connectivity or synchrony.

Alterations at the structural level of cognitive architecture:

The data for the maze-learning task in people with PTSD suggested cognitive abnormalities linked to performance measures that were positively correlated with the lower frequency delta. As a task requiring greater attention for the retrieval of stored information about correct moves, and the switching of attention to encode new correct moves, the maze task was not expected to be associated with delta frequency patterns in people with PTSD. Therefore, the relationship between time to complete the maze task and lower frequencies in people with PTSD suggested a different pattern of information processing for certain categories of information. This conclusion was inferred from the pattern of results in behavioural performance measures, resting EEG patterns, and correlation analyses, which suggested a plausible argument for a response-set in PTSD as a tonic state. The correlation patterns revealed by the current analysis, between the delta frequency and alpha frequency with nonverbal memory codes, lend support to the idea that an abnormal activity in oscillation patterns may be associated with episodic memory, the coding of new information, and hippocampal dysfunction in people with PTSD. Hence, optimisation goals for the retrieval of information are more consistent with processing specificity rather than domain specificity.

Episodic memory is vital for encoding context information and associative representations for the unique conjunction of stimuli and their combinatorial flexibility in every-day functions (Eichenbaum and Fortin, 2005). Time is an important dimension of spatial representations and essential for the updating and storage of episodic information (Fuster et al., 2000; Lisman et al., 2001). If this function is disrupted in PTSD, the implications are of an abnormal integration of coded information and for instability of neural global network connectivity (Hoge and Kesner, 2007). Speculatively, it can be argued that the anticipatory pre-synaptic responses for incoming stimuli may be formulated on selective outcomes rather than on the perceived stimuli, thus acting as a mechanism for focusing attention to goal-directed
responses and inhibiting irrelevant information (Aston-Jones et al., 1994; David et al., 2004). However, this interpretation is in contrast to views in the PTSD literature arguing for a loss of inhibitory functions in people with PTSD (Bryant et al., 2005). The notion of preparatory processing in resting states is relevant to the direction and flow of information. Such a system implicates feed-forward and feed-backward loops, and re-entrant activity in thalamo-cortical and limbic-cortical pathways, for the generation of alpha and theta oscillation patterns (Jones and Wilson, 2005), which drive plastic changes and higher information exchange between populations of neurons in different oscillatory systems (Knyazev et al., 2006b).

Therefore, if theta synchronisation is altered to a delta pattern of synchrony in people with PTSD, as a compensatory mechanism for rehearsal maintenance in visual memory systems, then it is plausible that the current results support arguments for plasticity as a secondary characteristic of altered knowledge structures (McFarlane et al., 2002). Furthermore, if the stability of theta frequency in PTSD has been disrupted, it can be inferred that the highly selective excitatory network connections in the hippocampal formation have also altered. Some evidence of alterations to episodic memory functioning were supported by the current results, particularly lower accuracy scores in the behavioural data. A reorganised pattern for processing incoming stimuli through interneuron loops may be linked to a shift in timing channels and changes to frequency patterns of cerebellar activity (Schutter and van Honk, 2006). This argument may be a plausible model for the alterations in APF and the altered alpha rhythms found in PTSD participants in this study.

Alpha peak frequency has been used as a measure of the speed of central nervous system processing (Posthuma et al., 2001), with suggestions that this is a heritable trait involving physiological mechanisms responsible for brain oscillations and processing capacity in working memory systems (Clark et al., 2004). The lower APF values for both groups could be suggestive of a different mechanism for the control of anticipatory responses, possibly associated with heritable traits for the implementation of memory codes according to stylistic processing strategies.

On the other hand, the functional role of the speed of processing, in people diagnosed with PTSD, has been described as consistent with models proposing that a negative attention bias to stimulus processing is an attempt to avoid perceived threat or danger (Felmingham et al., 2002; Ehlers and Clark, 2000; Buckley et al., 2000). Based on this argument, the reorganisation of cognitive processing in PTSD becomes a functional goal-directed behaviour to reduce distress and a secondary characteristic following psychological trauma (McFarlane...
et al., 2002). In view of this argument, the current results of correlation patterns for the PTSD participants may indicate an alteration to long-range frequency patterns and the disruption of information processing in the higher beta frequency.

Alterations at the implementation level of cognitive architecture:

The current data were also suggestive of discrimination difficulties in participants with PTSD, possibly associated with reduced speed of processing in both verbal and nonverbal domains and with an altered pattern of frequencies for memory codes. The pattern of behavioural results was suggestive of abnormalities in executive processes for volition and self-regulation, consistent with altered selective attention and task management control mechanisms. On the other hand, conclusions indicating abnormal control mechanisms in local hemispheric processing networks for the storage of information could not be drawn based on the current analyses. The proposal that differences in structural connectivity at the local level may underlie functional differences in cognitive ability between groups remains unclear using the current analysis.

Furthermore, there was no clear relationship between oscillation patterns and coding for specialised domain-specific mnemonic representations. Speculatively, the pattern of correlations between oscillation patterns and cognitive tasks indicated that differences in the relationship between neural activity and coding for memory representation were of a functional nature associated more with frontal and posterior decoupling (Clark et al., 2003). However, it is plausible that the relative right-hemisphere patterns for memory coding in the PTSD group may be consistent with the behavioural performance measures revealing a reduced verbal working memory performance. This conclusion is in agreement with earlier neuroimaging data by Clark and colleagues (2003) for structural as well as functional abnormalities in PTSD.

On the other hand, the hypothesis of an electrophysiological imbalance, with suggestions that this mechanism provides a modulation effect for global integration differences (Schutter and van Honk, 2006), was more consistent with the pattern of correlations revealed by the data and is suggestive of abnormal attentional processes in control mechanisms. Furthermore, the pattern of correlations was also consistent with differences in the timing of oscillatory activity between groups, the PTSD group showing unexpected abnormal associations with cognitive tasks in the lower delta frequency band rather than in the higher beta frequency range.
In an early thesis aiming to understand the biological effects of trauma, Olton and Wolf (1981) proposed that a transient disruption of hippocampal electrical activity would “reset” working memory and produce a form of retrograde amnesia that is specific to hippocampal storage. The current pattern of results, indicating reduced verbal working memory performance in PTSD, may be linked to transient shifts in timing (Shaw et al., 2002) and an interruption to the coordinated flow of information processing in dissociative states (Lanius et al., 2005). This argument is also in agreement with models suggesting a connection between cerebellum activation and motor coordination (Teicher et al., 2003) involving executive functions and the timing of information flow (Schutter and van Honk, 2006).

Therefore, it may be possible to interpret the results of the current analyses according to suggestions that the capacity for effortful control and regulation in anterior regions is likely to be influenced by a functional overlap in right hemisphere networks, particularly the right temporo-parietal junction involved in visual orienting and voluntary attention (Corbetta et al., 2002). This hypothesis is suggestive of mental ability and speed of processing between groups influenced by attentional processes, for both internal and external stimuli, and influenced by the interaction of attention circuits, as suggested by a number of other researchers in the wider literature (Fan et al., 2005; Gevins and Smith, 2000; Linden et al., 2003).

However, in PTSD participants, at a local level of information processing, it cannot be ruled out that differences in anterior regions, associated with executive functions and circuits influencing working memory processes for the maintenance of information in a short term buffer and for updating information, were not linked to trait characteristics of premorbid functioning. These differences may be plausibly related to differences in alpha power. Therefore, the statistically significant relationships, indicating a reduced tendency for involvement with anterior regions and greater involvement for central-temporal regions, in memory for verbal and nonverbal codes, in people with PTSD, requires further investigation. The possible alterations to neural pathways, resulting from affective states and linked to frontal EEG rhythms, may indicate laterality effects associated with cognitive functions and require a finer analysis of investigation.

5.5.5. Limitations

The results and conclusions discussed in the preceding sections must be interpreted with caution and are based on a number of limitations, which are now reviewed. In this initial investigation, participant numbers for the PTSD group were small, added to this, missing data in some of the analyses may have reduced statistical power to preclude significant findings.
Furthermore, the use of data accessed from a standardised dataset, in the case of the current study, imposed limits on the potential use of behavioural indicators to assess laterality. Therefore, it is possible that differences between groups were not found because a number of confounding variables may have obscured measurement of variance, particularly in relation to variables associated with the heterogeneous nature of PTSD, individual differences, and an inability to hold constant all task parameters associated with neuropsychological measures of lateralisation.

For example, the CAPS (Weathers et al., 2001) scores were used as an independent clinical assessment criterion for inclusion of PTSD participants in this study. The current analyses did not differentiate within-group differences for the type of trauma, the time since trauma, the severity of the traumatic experience, or the number of traumatic experiences. Similarly, the control group participants, after meeting the strict inclusion and exclusion criteria for entry into the normative database, were selected for a match with the variables of age and gender, but were also included in this study if they responded affirmatively (using self-report assessment) to having experienced traumatic event(s). The heterogeneity of groups for reactivity patterns to traumatic stressors was not controlled. In addition, it has been well documented that different patterns of stress and anxiety alter cognitive functioning (Gold, 2005; Hugdahl and Davidson, 2004; Wager et al., 2003).

Another important variable that was not controlled in the current analyses was handedness preference scores (Oldfield, 1971). This may be an important variable with respect to trait characteristics, particularly in proposals suggesting ambidextrousness is linked to higher risk of PTSD, and may carry implications for the speed of processing and cortical connectivity patterns in working memory functions (Chemtob and Taylor, 2003). All analyses conducted in this chapter were initially repeated with and without the inclusion of participants reporting left-handedness. Because there was little difference between the results, and to avoid a smaller PTSD sample, all participants were included in the analyses presented. However, neural connectivity alterations, based on dominance theories associated with left- or right-handedness, and those with a more diffuse pattern of connectivity influenced by ambidexterity and possible early childhood reorganisation patterns (Saltzman et al., 2006), were not investigated by the current analyses but may be useful to future investigations.

In addition, as presented in Chapter 4, medication status was not controlled in the current study and the results for the PTSD group may have been influenced by the use of SSRI medication. These variables require further investigation and quantification for an analysis of
altered cortical activation patterns with the use of psychotropic medication (Bruder et al., 2001). Therefore, significant effects may have been difficult to detect in this study, particularly for the higher beta frequency.

The current results may have been specific to people drawn from community samples, which included a number of different traumatic stressor events and mixed gender samples. These variables may place limitations on the generalisation of the pattern of current results. It is possible that differences to psychological and physiological functioning depend not only on the type of experience associated with trauma but also how the sample is selected. In addition, subjective perceptions of threat, even when using standardised instruments for assessment, may involve differences with gender-bias (Wager et al., 2003). In the current study, differences associated with gender were not reported.

Furthermore, despite attempts to match education with age and gender in control participants, differences in education may have contributed to the pattern of results in this study. There have been some arguments that IQ influences results for cognitive processing in people with PTSD (Vasterling et al., 2002), as well as the pattern of spontaneous electrophysiological activity (Begic et al., 2001; Jokic-Begic and Begic, 2003). However, previous findings have been reported from studies conducted on combat-exposed war veterans. In the current study there was no control for IQ, and this factor may have contributed to the pattern of brain-behaviour relationships, predominantly associated with the analysis of neuropsychological measures.

Lesion studies have traditionally been useful in detecting cognitive abnormalities (Lezak et al., 2004), principally at the local level of functioning. However, advances in methodological techniques, and more recent empirical evidence, supporting the complex structure of neural connectivity patterns in people with PTSD (Clark et al., 2003; Bryant et al., 2005), highlight a presumptive stance in this research on the use of existing neuropsychological measures to represent lateralised modalities of performance on task demands. Because the current analyses focused on tonic states of arousal, a simplistic analogy of functional analysis is represented by the current behavioural data and further research is recommended to assess the complex relationship between arousal, laterality and task demands in people with PTSD.

Taken together, methodological issues play a large part in understanding complex behavioural systems. Skinner (1950) argued for finding precisely what variables influence behavioural responses before a detailed functional analysis can be made. Even if all attempts are made to
identify the contributing variables for the reduced speed of information processing in PTSD, as identified by the current results, the limitations of cross-sectional methodology and retrospective analysis would still apply. The current methodology limits a functional analysis of behavioural measures that would be better suited to time-series analyses and the extraction of common factors associated with task demands. Consequently, a number of the abovementioned limitations will be addressed further in the following sections of this study, with a primary emphasis on individual differences associated with stress reactivity.

Conclusions:

The current pattern of results provides evidence for two tentative conclusions. The first is that there were no clear patterns of hemispheric lateralisation in resting cortical arousal patterns or behavioural performance accuracy scores in data drawn from PTSD participants. The results indicated that temporal channels of communication in brain organisation patterns in people with PTSD might be disrupted at a number of levels of information processing. Thus, evidence involving a common neuroanatomical pathway for structural alterations to brain organisation patterns and compensatory mechanisms in PTSD was only partly supported by APF patterns and differences between memory retrieval involving perceptions based on familiarity versus recollection.

The second conclusion is in agreement with previous research findings, as was demonstrated by alterations to attention mechanisms in people with PTSD, and indications of alterations to sensory-motor pathways that consequently disrupt frontal networks and executive functions. The relationships between synchronisation of different frequency patterns and sensory-motor pathways indicated a disruption to long-range synchrony associated with the higher beta frequency and an abnormal association with the lower delta frequency. Preliminary evidence was provided for an alternative pathway involving well-rehearsed responses to anticipated stimuli. This finding may be linked to mechanisms of plasticity and altered neural connectivity patterns in people with PTSD.
6. Reactions to Stress and Resting EEG Patterns

6.1. Rationale

This chapter investigates proposals that secondary characteristics of trauma exposure influence information processing in people with PTSD (McFarlane et al., 2002) and that cortical arousal, as measured by spontaneous electrocortical activity, has an identifiable characteristic activation pattern in clinically diagnosed PTSD sufferers (Begic et al., 2001; Jokic-Begic and Begic, 2003).

Following the previous analyses in Chapter 5, this chapter examines two related aims. The first aim is concerned with the subjective reactivity patterns to perceived traumatic stressors and the relationship of those reaction patterns to mood states. The second aim focuses on whether cognitive functioning in people with PTSD, as well as those not diagnosed with PTSD but who reported strong reactivity to stressful events, is lateralised, thus contributing to secondary difficulties with learning and memory. The analyses for behavioural performance examining the speed of processing for the motor tapping task, and resting EEG, from the previous chapter were repeated for three groups.

6.1.1. Introduction

PTSD has been characterised as a secondary response to traumatic experience, although there is some debate about whether the syndrome behavioural patterns are associated with premorbid conditions or whether they are the direct result of traumatic exposure (McFarlane and Yehuda, 2000). Furthermore, various groups have demonstrated that disruptions to information processing occur in those who have experienced extreme traumatic events but have not been diagnosed with PTSD or other mental disorder (Britton et al., 2005; Stein et al., 2002; Vasterling et al., 2006).

In addressing a conceptual dilemma of how to evaluate, appropriately, responses to trauma and how symptom manifestations are to be treated, McFarlane and Yehuda (2000) identified a number of issues related to criterion A of the PTSD diagnostic nosology (DSM-IV, American Psychiatric Association, 1994). Specifically, the authors drew attention to the differences between the response to a traumatic event and the predisposing risk factors for PTSD other than exposure to a traumatic stressor.

As a secondary characteristic to traumatic exposure, evidence has also been provided to suggest that the neural pathways for information processing have altered in PTSD sufferers
Clark et al., 2003; Galletly et al., 2001). This hypothesis was partly supported by the analyses in the previous chapter. As previously discussed, an interruption to the coordinated flow of information and inter-hemispheric integration in memory functions is thought to result in abnormal working memory processes and altered neural connectivity patterns between cortical regions, particularly associated with updating information in regions specialised for verbal and nonverbal processing (Shaw et al., 2002).

Therefore, as a question derived from the analyses in the previous chapter, it was not clear whether those who had suffered severe traumatic stress, but were not diagnosed with PTSD, also developed secondary characteristics for adaptive functioning and whether the speed of processing in these individuals was mediated by anxiety and depression symptoms. Given that trauma exposure is comparatively more common in community samples than PTSD prevalence (Frans et al., 2005; Gold et al., 2005; Rosenman, 2002), it was unclear whether those in the normative control group, who may have had exposure to a traumatic event, without subsequent clinical diagnosis for their reactivity patterns, would show differences in the speed of information processing and in cortical arousal patterns, compared to those who reported no experience with traumatic events.

Based on previous studies, it was expected that there would be a high prevalence of self-reported exposure to traumatic stressors in the control group. In addition, it was expected that psychobiological differences for those from the normal control group, experiencing strong reactivity to previous trauma events, would be mid-way between the PTSD group and those with minimal or no trauma exposure. Based on the findings derived from analyses in the previous chapter, it was also expected that any differences between groups in speed of information processing would be revealed in lower accuracy scores for behavioural performance, in alpha peak frequency, and as differences in cortical activation patterns between frontal and posterior brain regions in those participants reporting previous trauma reactivity.

6.2. Method

6.2.1. Participants

Participant data has been described in chapter 4 and in the previous chapter. For the current analysis all left handed participants were excluded from the original two samples. The remaining PTSD participants (N=33: 16 female, 17 male), ages ranging from 20-57 (M = 40.28, SD = 11.08), with years of education ranging from 8-18 years (M = 13.23, SD = 2.87),
were compared with control participants (N=120) who were split into subgroups based on
self-report measures of trauma experience. The two subgroups comprised a Trauma-Event
control (TEC) group (N=32: 22 female, 10 male), ages ranging from 23-55 (M = 41.77, SD =
8.75), with years of education ranging from 10-18 years (M = 14.06, SD = 2.86), and a
normal control (NC) group (N=88: 42 female, 46 male), ages ranging from 19-59 years (M =
38.34, SD = 11.98), with years of education ranging from 10-18 years (M = 15.46, SD =
2.42). The procedure for assigning subgroups is reported in the following section and the
participant characteristics are presented in Table 6.1.

6.2.2. Instruments and Procedures

6.2.2.1. Demographic and Psychometric Variables

A standardised initial screening tool was administered to all participants prior to testing, as
described in Chapter 4. The web-based questionnaire was used to acquire demographic data
including age, gender, and years of education, but was also used as a screening tool for
general health, and to assess mood states, and handedness preference.

Handedness preference was measured using the Edinburgh Handedness Inventory (Oldfield,
1971), as described in Chapter 4. Those participants reporting a strong left-handed preference,
or mixed laterality preference, were excluded from the current analysis. The Laterality
Quotient (LQ) for the remaining participants is presented in Table 6.1.

Current mood states were assessed using self-report scales for depression, anxiety, and stress
using an abbreviated version of the Depression Anxiety Stress Scale (DASS 21; Lovibond
and Lovibond, 1995).

A standardised CIDI (World Health Organization, 1993) was used as a self-report screen for
symptoms indicative of possible psychopathology. Exposure to traumatic stressors was
assessed using the CIDI Traumatic Event Checklist. All individuals who reported exposure to
a traumatic event for this module went on to complete a schedule of questions based on PTSD
criteria, as defined according to the DSM-IV (American Psychiatric Association, 1994).

6.2.2.2. PTSD Measures

As detailed in Chapter 4, a range of selection criteria was used to assign clinical and non-
clinical participants for inclusion into the BRID clinical and normative datasets. The CAPS
(Blake et al., 1995) was used as an independent measure to confirm minimum diagnostic criteria and the clinical status of the PTSD participants.

However, in the current analysis of subjective responses to traumatic stressors, only the CIDI responses were used because this variable was a standardised measure used with both the PTSD group and the control group. Therefore, analyses that included the CIDI were based on a reduced number of the PTSD participants because not all PTSD participants responded with a full dataset to the CIDI questionnaire.

The control participants were assigned into two subgroups based on their responses to the CIDI Checklist of Events question. The number of reported trauma events, classified as criterion A1, and the reactions to events, classified as criterion A2, were summed to determine the allocation of participants into subgroups. Those participants who reported at least one event on the Trauma Event Checklist and then scored two for criterion A2 were included in the subgroup TEC. All other control participants remained as the normal control (NC) and included a mix of participants who reported experience with traumatic exposure, but did not meet the thresholds for the criterion symptoms, and those who reported having had no experience with traumatic stressor events.

6.2.2.3. Behavioural Performance: Sensori-motor Task

The Motor Tapping Task is one of the subtests in the IntegNeuro battery of neuropsychological tests as described in Chapter 4. It is part of a standardised and automated procedure for assessing a range of neuropsychological indices. For the current study, the test was used to assess sensori-motor skill for left- and right-hemisphere dominance and speed of information processing. In the current study the tapping score was taken as a measure of cognitive flexibility in procedural memory.

6.2.2.4. Resting Electrophysiological Data Collection and Measures

According to procedures detailed in Chapter 4, resting background EEG data was collected for both the eyes closed and eyes open conditions. As previously detailed, data were acquired using an electrode cap from 26 scalp sites (using 10 lateral pairs of electrodes [Fp1, Fp2, F3, F4, F7, F8, FC3, FC4, C3, C4, T3, T4, T5, T6, CP3, CP4, P3, P4, O1, O2] and six midline electrode sites [Fz, FCz, Cz, CPz, Pz, Oz]). Core frequencies examined in the current analysis were delta (1.5-3.5 Hz), theta (4-7.5 Hz), alpha (8-13 Hz), beta (14.5-30 Hz), and alpha peak frequency (8-13 Hz). EEG data for each frequency was log transformed prior to analysis.
following methods according to Pivik et al (1993) and as outlined previously in Chapter 4. To reduce the EEG data for statistical analysis, laterality was examined by aggregating electrode sites into regions of interest (following a method by Davidson et al., 2000). All aggregated variables were assessed for internal consistency and measured > 0.80 on Cronbach’s Alpha.

6.3. Data Analysis

All data analyses were computed using SPSS (version 12 and version 14) and results have been presented as absolute numbers, means and standard deviations or percentages.

Chi-square ($\chi^2$) analyses were used to compare demographic characteristics. One-way ANOVAs examined the differences in self-reported subjective traumatic experiences and mood states between groups.

The sensori-motor task provided a score for the number of taps for left- and right-hand. Speed of processing and hemispheric dominance was examined using repeated measures ANOVA with Groups (PTSD, TEC, NC) as the between-subjects factor and Hand (left-hand, right-hand) as the within-subjects factor.

Alpha peak frequency (APF) was assessed for differences in midline sites using GLM repeated measures ANOVA for a between-subjects factor Group (PTSD, N=20; TEC, N=20; NC, N=42) and within-subjects factors Site (Fz, Cz, Pz, Oz) and Condition (eyes closed, eyes open). Due to missing data a reduced number of participants in each group were available for this analysis.

Aggregated regions of interest (left/right, and anterior: Fz, F7, F3, F4, F8; posterior: Pz, P3, P4, T5, T6) were compared as differences in log amplitude power. Using a series of repeated measures ANOVAs, comparisons included the four frequency bands (delta, theta, alpha, beta), hemisphere (left, right), region (anterior, posterior), and condition (eyes closed, eyes open) as within-group factors, and group (PTSD: N=33, TEC: N=32, NC: N=84) as between-group factors.

Post hoc analyses were conducted using one-way ANOVAs to investigate significant interaction effects. The Greenhouse-Geisser epsilon correction was used where appropriate. Significance levels were set at the $p = 0.05$ level and Bonferroni tests were conducted to correct for multiple testing.
6.4. Results

6.4.1. Selection of Subgroups and Demographic Data

Table 6.1 indicates that the three groups did not differ significantly in age [F(2,150) = 1.21, p = 0.30], but differed significantly in years of education [F(2,148) = 9.57, p = < 0.001]. The PTSD group showed the least number of years of education (M = 13.23, SD = 2.87) and the NC group (M = 15.46, SD = 2.42) the most. Post-hoc Bonferroni tests for multiple comparisons indicated a significant mean difference between the PTSD group and NC group (MD = -.2.23, S.E = .55, p = < 0.001) and a significant mean difference between the TEC group and the NC group (MD = -1.39, SE = .54, p = 0.03).

There were no significant differences in gender in the PTSD group ($\chi^2 = 0.03$, df(1,29), p = 0.86), and no differences in gender in the NC group ($\chi^2 = 0.18$, df(1,120), p = 0.67), but there was a significantly greater number of females in the TEC group ($\chi^2 = 4.50$, df(1,30), p = 0.03).

**Table 6.1.** Group descriptor variables

<table>
<thead>
<tr>
<th>Measure</th>
<th>Age (years)</th>
<th>Education (years)</th>
<th>Laterality Quotient</th>
<th>Trauma events</th>
<th>Time since trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTSD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>33</td>
<td>31</td>
<td>17</td>
<td>22</td>
<td>31</td>
</tr>
<tr>
<td>M</td>
<td>40.28</td>
<td>13.23</td>
<td>0.93</td>
<td>4.18</td>
<td>7.98</td>
</tr>
<tr>
<td>sd</td>
<td>11.08</td>
<td>2.87</td>
<td>0.11</td>
<td>1.76</td>
<td>8.69</td>
</tr>
<tr>
<td>TEC</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>32</td>
<td>32</td>
<td>25</td>
<td>31</td>
<td>32</td>
</tr>
<tr>
<td>M</td>
<td>41.77</td>
<td>14.06</td>
<td>0.88</td>
<td>2.84</td>
<td>23.5</td>
</tr>
<tr>
<td>sd</td>
<td>8.75</td>
<td>2.86</td>
<td>0.14</td>
<td>1.49</td>
<td>12.81</td>
</tr>
<tr>
<td>NC</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>88</td>
<td>88</td>
<td>79</td>
<td>88</td>
<td>51</td>
</tr>
<tr>
<td>M</td>
<td>38.34</td>
<td>15.46</td>
<td>0.92</td>
<td>1.32</td>
<td>19.02</td>
</tr>
<tr>
<td>sd</td>
<td>11.98</td>
<td>2.42</td>
<td>0.14</td>
<td>1.62</td>
<td>12.69</td>
</tr>
</tbody>
</table>

6.4.1.1. Number of Events Reported – Criterion A1

In the PTSD group, 100% (N=22) reported more than one event using the CIDI checklist (of N=33 using the CAPS) and 69% (N=83) of the total control group reported at least one event at some time in their life. Of the total control sample, only 26.7% (TEC: N=32) reported the A2 criterion for responses to both categories of “terrified” and “helpless”.

154
The CIDI checklist of events questionnaire listed nine traumatic events and included two general questions relating to trauma. The two general questions were analysed as separate categories.

The first general question asked about experience with “an extremely stressful or upsetting event”. In response to this general question, 95.5% (N=21) of the PTSD group reported having experienced “an extremely stressful or upsetting event” and 94% (N=30) of the TEC subgroup answered affirmatively to this question. Of the remaining control group (NC), 38% (N=34) reported experiencing an extremely stressful event. The PTSD group and the TEC group did not differ in their reporting of having experienced “any stressful event” (Fisher’s Exact Test = 1.39, df(1,52), p = 0.42). However, in comparison, the NC group reported significantly lower responses to this general question ($\chi^2 = 48.53$, df(2,140), $p = < 0.001$).

The second general question on the checklist related to the witnessing of events and asked whether any of the events reported on the list caused “shock because they happened to someone close”. For responses to witnessing or learning about serious threat, injury or death to someone close, there were more responses across all three groups. In the PTSD group 95.5% (N=21) reported extremely shocking events to others, while 97% (N =32) of the TEC group reported this type of experience, and 46% (N=41) of the NC group responded affirmatively to this question. There were no differences in prevalence or reporting between the PTSD group and the TEC group ($\chi^2$, $p = 0.82$), but as expected, there was a significant difference for the NC group ($\chi^2 = 8.61$, df(2,140), $p = 0.01$).

The number of responses to the nine trauma-checklist questions is presented for each group in Table 6.1. Results indicated that there was wide variability within groups for each of the reported events. For the PTSD group, events experienced per participant ranged from 1-8 (M = 4.18, SD = 1.76). For the TEC group, the number of events per participant ranged from 1-7 (M= 2.84, SD = 1.49). The number of trauma experiences reported by the NC group ranged from 0-7 (M = 1.32, SD = 1.62), despite low responses to feeling “terrified” or “helpless”. A one-way ANOVA revealed that the total number of events reported was significantly different between groups (F(2,138) = 31.96, $p = < 0.001$). Differences for the total number of events were significant between each of the groups, after Bonferroni adjustments (PTSD vs TEC, $p = 0.01$; PTSD vs NC, $p = < 0.001$; TEC vs NC, $p = < 0.001$).
Table 6.2 indicates the percentage frequency with which each item on the checklist for A1 events was reported in each group. The three most commonly reported events in the PTSD group were witnessing traumatic events (68%), life-threatening accidents (63%), and attack or assault (46%). For the TEC group, the three most common events reported were molestation (41%), witnessing traumatic events (31%), and being attacked or assaulted (28%). For the NC group, witnessing traumatic events was the most commonly reported event (18%), followed by being threatened with a weapon (16%), and life threatening accidents (15%). Based on responses to criterion A2 reactivity, Table 6.2 also indicates the non-parametric (Mann-Whitney U) statistical differences in event type that were calculated only between the PTSD group and the TEC group.

Table 6.2. Self-reported criterion A1 qualifying traumatic events as percentages in each group

<table>
<thead>
<tr>
<th>QUALIFYING EVENT</th>
<th>PTSD (%) (N = 22)</th>
<th>TEC (%) (N = 32)</th>
<th>NC (%) (N = 51)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any traumatic stressor (to self)**</td>
<td>90.1</td>
<td>71.0</td>
<td>35.2</td>
</tr>
<tr>
<td>Any witnessed traumatic stressor (to close associate)*</td>
<td>95.5</td>
<td>96.9</td>
<td>46.6</td>
</tr>
<tr>
<td>Direct combat experience</td>
<td>9.1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Life-threatening accident**</td>
<td>63.2</td>
<td>12.5</td>
<td>14.8</td>
</tr>
<tr>
<td>Natural disaster (fire, flood, other)*</td>
<td>36.4</td>
<td>12.9</td>
<td>5.7</td>
</tr>
<tr>
<td>Witness someone badly injured or killed**</td>
<td>68.2</td>
<td>31.3</td>
<td>18.2</td>
</tr>
<tr>
<td>Rape</td>
<td>13.6</td>
<td>12.5</td>
<td>2.3</td>
</tr>
<tr>
<td>Sexual molestation**</td>
<td>9.1</td>
<td>40.6</td>
<td>10.2</td>
</tr>
<tr>
<td>Physically attacked or assaulted</td>
<td>45.5</td>
<td>28.1</td>
<td>12.5</td>
</tr>
<tr>
<td>Threatened (weapon, held captive, kidnapped)</td>
<td>36.4</td>
<td>21.9</td>
<td>15.9</td>
</tr>
<tr>
<td>Tortured or victim of terrorists</td>
<td>4.5</td>
<td>3.1</td>
<td>0</td>
</tr>
</tbody>
</table>

Note: *p < 0.05; **p < 0.01

A summary of the most frequently reported events in each group, ranked as the top three from the nine event checklist items, is presented in Table 6.3. Based on descriptive classifications such as physical events, witnessed events, or personal events, a general pattern is formulated by ranking commonly reported events for each group, and is presented as an overview. Table 6.3 shows that the category of physical attack or assault is common to both the PTSD group and the TEC group. However, both the PTSD group and the NC group ranked witnessed events as the highest category of traumatic stressor.
Table 6.3. Summary of most frequently reported events in each group

<table>
<thead>
<tr>
<th>Event Ranked by Frequency</th>
<th>PTSD</th>
<th>NC</th>
<th>TEC*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Witnessed Event</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Life threatening Attack</td>
<td>2</td>
<td>3</td>
<td>-</td>
</tr>
<tr>
<td>Physical Assault or Attack</td>
<td>3</td>
<td>-</td>
<td>2</td>
</tr>
<tr>
<td>Threat with Weapon</td>
<td>-</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>Molestation</td>
<td>-</td>
<td>-</td>
<td>1</td>
</tr>
</tbody>
</table>

Note: * Ranked by type of event and by frequency of the event, the TEC group indicates a different profile compared to the PTSD and NC groups.

6.4.1.4. Responses to Extreme Stress – Criterion A2

Responses to the checklist of events for feeling “terrified” or “helpless” were totalled for the two categories of responses. For the PTSD group, 82% (N=18) answered to feeling “terrified” compared to 91% (N=20) reporting feeling “helpless” (of N=33 using the CAPS). For the TEC group, all participants, 100% (N=32), answered feeling both “terrified” and “helpless”. For the NC group, 6% (N=5) of the sample reported feeling “terrified” and 30% (N=26) reported feeling “helpless”, indicating a difference in the pattern of responding for those who reported a traumatic experience (terrified: \( \chi^2 = 64.75, \text{df}(1,83), p = < .001 \); helpless: \( \chi^2 = 22.45, \text{df}(1,83), p = < .001 \)). Because of this difference in the NC group, a chi-square analysis revealed a significant difference in responding between groups (\( \chi^2 = 73.80, \text{df}(2,105), p = < 0.001 \)). Differences in responding patterns between the PTSD group and TEC group (Fisher’s Exact Test = 6.28, df(1,54), p = 0.02), were also significant, but were taken to be due principally to missing values on the self-report questionnaire.

Differences in the NC group were more difficult to interpret due to the inclusion of participants reporting no exposure to traumatic events and differences in the size of the groups. Of those in the mixed control group (NC), 42% reported no traumatic stressor experience, while others reported only one initial type of reaction pattern and did not meet the minimum criterion A1, and then reported either no further reactions to the event or reported the full range of symptom pattern reactivity. Therefore inclusion of criterion A2 for reactivity in statistical analyses was representative only for those who reported previous trauma exposure and excluded the remainder of the control participants, thus creating an experimental artefact.
6.4.1.5. Age at the Time of the Traumatic Event

A one-way ANOVA revealed that age at the time of experiencing the event was significantly different between the PTSD group and the two control groups (F(2,111) = 13.80, p = < 0.001). Age at the time of trauma events was reported as later in life in the PTSD group (M = 31.10, SD = 11.40) and before the age of 21 in the two control groups, TEC (M = 18.00, SD = 10.47) and NC (M = 20.12, SD = 10.67). Post hoc Bonferroni comparison tests indicated that the above differences between the PTSD group and both the TEC group (p = < 0.001) and the NC group (p = < 0.001) were significant.

The time elapsed since the traumatic event was calculated for a between-groups analysis using one-way ANOVA. The PTSD group reported a mean of 7.97 years (SD = 8.69, N = 31), the TEC group a mean of 23.50 years (SD = 12.81, N = 32), and a similar pattern was revealed for the NC group (M = 19.02, SD = 12.69, N = 51), indicating a significant difference between groups (F(2,111) = 14.71, p = < 0.001). Post hoc Bonferroni comparison tests revealed the differences between the PTSD group and both the TEC group (p = < 0.001) and the NC group (p = < 0.001) were significant.

Despite both groups indicating severe reactivity to events, there were significant differences (at the p = < 0.01 level) between the two groups on all variables associated with traumatic stressor events, as described above.

6.4.1.6. Relationship of Trauma Experience with Mood States

Table 6.4 presents the DASS scores as descriptive statistics for the three groups. As expected, there were significant differences between groups in mood scores, the PTSD participants scoring highest on each subscale for depression (F(2,139) = 81.96, p = < 0.001), anxiety (F(2,139) = 76.12, p = < 0.001), and stress (F(2,139) = 97.62, p = < 0.001). Bonferroni multiple comparison tests indicated a significant difference between the PTSD group and the two control groups for all three subscales at the p = < 0.001 level, but no difference between the two control groups on any of the mood subscales.
Table 6.4. DASS descriptive data

<table>
<thead>
<tr>
<th>Measure</th>
<th>N</th>
<th>Depression</th>
<th>Anxiety</th>
<th>Stress</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTSD</td>
<td>22</td>
<td>11.36</td>
<td>8.09</td>
<td>12.86</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5.32</td>
<td>5.48</td>
<td>4.93</td>
</tr>
<tr>
<td>TEC</td>
<td>32</td>
<td>2.06</td>
<td>0.88</td>
<td>3.19</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2.87</td>
<td>1.26</td>
<td>3.00</td>
</tr>
<tr>
<td>NC</td>
<td>88</td>
<td>1.99</td>
<td>0.92</td>
<td>2.56</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2.47</td>
<td>1.50</td>
<td>2.58</td>
</tr>
</tbody>
</table>

Summary: Self-Report Appraisals of Traumatic Stressor Events

As predicted, a high number from the control group reported having experienced at least one traumatic stressor event. Responses to the CIDI trauma events checklist indicated differences in the subjective reactivity to event stressors (criterion A2) between the groups. Furthermore, a higher ratio of female participants reported strong reactivity to traumatic events in the TEC group, and a similar pattern of strong reactivity to traumatic stressors for the PTSD group and the TEC group compared to the NC group.

As expected, there was a significant difference between groups for the average number of events reported, with the PTSD participants reporting the highest number of events compared to the two control groups. Time elapsed since the event indicated, on average, a more recent event for the PTSD group (< 10 years) compared to the two control groups (> 19 years). But this measure was interpreted as an effect of selection criteria and representative only of the current sample. A summary of differences for demographic variables, CIDI responses, and DASS items is presented in Table 6.5, with significance ratings for each group presented in a “Difference” column.
Table 6.5. Summary statistics based on self-report items

<table>
<thead>
<tr>
<th>Measure</th>
<th>F</th>
<th>p</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>1.21</td>
<td>0.30</td>
<td>n/s</td>
</tr>
<tr>
<td>Gender</td>
<td>2</td>
<td>1,3</td>
<td></td>
</tr>
<tr>
<td>Education, years</td>
<td>9.57</td>
<td>&lt; .001</td>
<td>1&lt;2&lt;3</td>
</tr>
<tr>
<td>Handedness (LQ)</td>
<td>1.01</td>
<td>0.37</td>
<td>n/s</td>
</tr>
<tr>
<td>Number of events</td>
<td>31.96</td>
<td>&lt;.001</td>
<td>3&lt;2&lt;1</td>
</tr>
<tr>
<td>Time since trauma</td>
<td>14.71</td>
<td>&lt;.001</td>
<td>1&lt;3&lt;2</td>
</tr>
<tr>
<td>PTSD Scale</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A2 Responses</td>
<td>176.16</td>
<td>&lt;.001</td>
<td>3&lt;2,1</td>
</tr>
<tr>
<td>Re-experiencing</td>
<td>56.43</td>
<td>&lt;.001</td>
<td>3&lt;2&lt;1</td>
</tr>
<tr>
<td>Numbing</td>
<td>36.27</td>
<td>&lt;.001</td>
<td>3&lt;2&lt;1</td>
</tr>
<tr>
<td>Avoidance</td>
<td>109.02</td>
<td>&lt;.001</td>
<td>3&lt;2&lt;1</td>
</tr>
<tr>
<td>Hyperarousal</td>
<td>67.10</td>
<td>&lt;.001</td>
<td>3&lt;2&lt;1</td>
</tr>
<tr>
<td>Total severity</td>
<td>98.82</td>
<td>&lt;.001</td>
<td>3&lt;2&lt;1</td>
</tr>
<tr>
<td>DASS-21</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>81.96</td>
<td>&lt;.001</td>
<td>3,2&lt;1</td>
</tr>
<tr>
<td>Anxiety</td>
<td>76.12</td>
<td>&lt;.001</td>
<td>2,3&lt;1</td>
</tr>
<tr>
<td>Stress</td>
<td>97.62</td>
<td>&lt;.001</td>
<td>3,2&lt;1</td>
</tr>
</tbody>
</table>

Note: Difference ratings: PTSD = 1; TEC = 2; NC = 3

6.4.2. Sensori-motor Behavioural Performance Measures

Table 6.6 shows a significant main effect for Hand (F(1,136) = 107.77, p = < 0.001) confirming a difference between the tapping scores for left- and right-hand. There was no interaction effect (p = 0.45). The main effect indicated that all groups were consistent in a higher number of taps produced by the right-hand compared to the left-hand, as expected for a right-handed sample.

There was a significant main effect for Group (F(1,136) = 4.43, p = 0.01), revealing that the lowest tapping scores were for the PTSD group (LHM = 144.57, SD = 23.34; RHM = 161.75, SD = 23.63) and the highest scores were for the NC group (LHM = 155.21, SD = 18.95; RHM = 174.78, SD = 19.31). Post-hoc analyses using Bonferroni adjustments revealed that there was a statistically significant difference between the PTSD group and the NC group (p = 0.01) but not between the two control groups or between the PTSD group and the TEC group (LHM = 153.72, SD = 18.91; RHM = 168.69, SD = 20.30).
Table 6.6. Results of ANOVA assessing sensori-motor task for number of taps with each hand between groups

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hand</td>
<td>1,136</td>
<td>107.77**</td>
</tr>
<tr>
<td>Group</td>
<td>2,136</td>
<td>4.43**</td>
</tr>
<tr>
<td><strong>Interaction Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hand x Group</td>
<td>2,136</td>
<td>0.81</td>
</tr>
</tbody>
</table>

Note: **p < 0.01

Summary: Sensori-motor Functions:

Results supported the hypothesis of a general slowing of information processing in the PTSD group, and a mid-way difference for the TEC group. Data indicated that speed of processing for a procedural motor task is reduced for both left- and right-hand taps in the PTSD group, when compared to the NC group, but not when compared to the TEC group. The data revealed a consistent pattern of higher right-hand taps compared to left-hand taps in each group.

6.4.3. Midline Resting Alpha Peak Frequency

Table 6.7 shows a main effect of Midline leads [F(G-G: 2,184) = 24.47, p = < 0.001], indicating a different pattern of APF at each site (Fz, Cz, Pz, Oz), and a main effect for Condition [F(1,79) = 4.79, p = 0.03], revealing lower APF in the eyes closed condition compared to the eyes open condition for all groups.

Table 6.7. Results of ANOVA assessing APF midline and condition effects between groups

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Midline</td>
<td>2,184(G-G)</td>
<td>24.47**</td>
</tr>
<tr>
<td>Condition</td>
<td>1,79</td>
<td>4.79*</td>
</tr>
<tr>
<td>Group</td>
<td>2,79</td>
<td>2.18</td>
</tr>
<tr>
<td><strong>Interaction Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Midline x Condition</td>
<td>2,201(G-G)</td>
<td>0.79</td>
</tr>
<tr>
<td>Condition x Group</td>
<td>2,79(G-G)</td>
<td>0.62</td>
</tr>
<tr>
<td>Midline x Group</td>
<td>5,184(G-G)</td>
<td>1.87</td>
</tr>
<tr>
<td>Midline x Condition x Group</td>
<td>5,201(G-G)</td>
<td>0.89</td>
</tr>
</tbody>
</table>

Note: *p < 0.05; **p < 0.01
Summary: Alpha Peak Frequency:

Overall, the expected difference between groups for a qualitative shift at frontal sites in APF was not supported by the data. All groups were within the normal range (8-13 Hz) for APF but there was a non-significant pattern of higher APF in the PTSD group, and, contrary to the predicted mid-way difference for the TEC group, there was a pattern of lower APF for the TEC group in both eyes closed and eyes open conditions. However, as previously mentioned, this analysis was conducted with smaller group sizes and requires replication and further investigation with larger groups.

6.4.4. Global Spectral Power Resting Patterns

As presented in Table 6.8, results of a global power analysis using four-way ANOVA revealed a significant interaction effect of frequency Band by Hemisphere by Condition \[F(G-G: 2,373) = 5.12, p = < 0.01\]. This interaction effect indicated not only the expected differences in frequency band rhythms \[F(G-G: 2,327) = 73.12, p = < 0.001\], which served as a manipulation check, but also a shift in amplitude patterns with eyes open and eyes closed conditions \[F(1,146) = 191.05, p = < 0.001\]. A significant main effect for Group \[F(2,146) = 3.58, p = 0.03\] confirmed a difference between groups in amplitude patterns for each frequency band and across conditions.
Table 6.8. Results of ANOVA assessing global spectral power by each frequency, hemisphere and condition effects between groups

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Band</td>
<td>2,327 (G-G)</td>
<td>73.12**</td>
</tr>
<tr>
<td>Hemisphere</td>
<td>1,146 (G-G)</td>
<td>0.81</td>
</tr>
<tr>
<td>Condition</td>
<td>1,146</td>
<td>191.05**</td>
</tr>
<tr>
<td>Group</td>
<td>2,146</td>
<td>3.58*</td>
</tr>
<tr>
<td><strong>Interaction Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Band x Group</td>
<td>4,327 (G-G)</td>
<td>1.34</td>
</tr>
<tr>
<td>Hemisphere x Group</td>
<td>2,146</td>
<td>0.9</td>
</tr>
<tr>
<td>Condition x Group</td>
<td>2,146</td>
<td>0.93</td>
</tr>
<tr>
<td>Band x Hemisphere</td>
<td>2,299 (G-G)</td>
<td>1.29</td>
</tr>
<tr>
<td>Band x Condition</td>
<td>2,289 (G-G)</td>
<td>285.36**</td>
</tr>
<tr>
<td>Hemisphere x Condition</td>
<td>1,146 (G-G)</td>
<td>1.56</td>
</tr>
<tr>
<td>Band x Hemisphere x Condition</td>
<td>2,373 (G-G)</td>
<td>5.12**</td>
</tr>
<tr>
<td>Hemisphere x Condition x Group</td>
<td>2,146</td>
<td>0.48</td>
</tr>
<tr>
<td>Band x Condition x Group</td>
<td>4,289 (G-G)</td>
<td>1.75</td>
</tr>
<tr>
<td>Band x Hemisphere x Group</td>
<td>4,299 (G-G)</td>
<td>2.01</td>
</tr>
<tr>
<td>Band x Hemisphere x Condition x Group</td>
<td>5,373 (G-G)</td>
<td>0.83</td>
</tr>
</tbody>
</table>

*Note: *p < 0.05; **p < 0.01

The global analysis is represented diagrammatically in Figure 6-1 for left hemisphere regions and Figure 6-2 for right hemisphere regions to show each frequency band for the three groups. Confidence intervals (95%) indicate that the alpha frequency is significantly different between groups. Table 6.9 and Table 6.10 show the estimated means (EM), standard error (SE) and 95% confidence intervals (CI) for each bandwidth and for each group in left and right hemispheres respectively.
Table 6.9. Descriptive data for left hemisphere total power

<table>
<thead>
<tr>
<th></th>
<th>delta</th>
<th>theta</th>
<th>alpha</th>
<th>beta</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left Hemisphere - EM</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>3.75</td>
<td>3.52</td>
<td>4.23</td>
<td>3.81</td>
</tr>
<tr>
<td>T-E Control</td>
<td>3.83</td>
<td>3.72</td>
<td>4.72</td>
<td>3.95</td>
</tr>
<tr>
<td>N-Control</td>
<td>3.77</td>
<td>3.57</td>
<td>4.27</td>
<td>3.71</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>delta</th>
<th>theta</th>
<th>alpha</th>
<th>beta</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left Hemisphere - SE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>0.08</td>
<td>0.09</td>
<td>0.14</td>
<td>0.09</td>
</tr>
<tr>
<td>T-E Control</td>
<td>0.08</td>
<td>0.09</td>
<td>0.14</td>
<td>0.09</td>
</tr>
<tr>
<td>N-Control</td>
<td>0.05</td>
<td>0.06</td>
<td>0.09</td>
<td>0.06</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>delta</th>
<th>theta</th>
<th>alpha</th>
<th>beta</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left Hemisphere - 95%CI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>0.15</td>
<td>0.18</td>
<td>0.27</td>
<td>0.17</td>
</tr>
<tr>
<td>T-E Control</td>
<td>0.16</td>
<td>0.18</td>
<td>0.27</td>
<td>0.17</td>
</tr>
<tr>
<td>N-Control</td>
<td>0.10</td>
<td>0.11</td>
<td>0.17</td>
<td>0.11</td>
</tr>
</tbody>
</table>

Figure 6-1. Global power analysis (eyes closed) left hemisphere frequency patterns with confidence intervals.

Note: Graph shows a significant difference for higher alpha amplitudes in the TEC group compared to the NC group and the PTSD group. There was a significant difference in beta power between the TEC group and the NC group.
Table 6.10. Descriptive data for right hemisphere total power

<table>
<thead>
<tr>
<th></th>
<th>delta</th>
<th>theta</th>
<th>alpha</th>
<th>beta</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Right Hemisphere - EM</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>3.57</td>
<td>3.38</td>
<td>4.64</td>
<td>3.78</td>
</tr>
<tr>
<td>T-E Control</td>
<td>3.70</td>
<td>3.68</td>
<td>5.29</td>
<td>4.03</td>
</tr>
<tr>
<td>N-Control</td>
<td>3.58</td>
<td>3.41</td>
<td>4.78</td>
<td>3.74</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>delta</th>
<th>theta</th>
<th>alpha</th>
<th>beta</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Right Hemisphere - SE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>0.09</td>
<td>0.12</td>
<td>0.17</td>
<td>0.09</td>
</tr>
<tr>
<td>T-E Control</td>
<td>0.09</td>
<td>0.12</td>
<td>0.17</td>
<td>0.10</td>
</tr>
<tr>
<td>N-Control</td>
<td>0.06</td>
<td>0.07</td>
<td>0.11</td>
<td>0.06</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>delta</th>
<th>theta</th>
<th>alpha</th>
<th>beta</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Right Hemisphere - 95%CI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>0.18</td>
<td>0.23</td>
<td>0.33</td>
<td>0.19</td>
</tr>
<tr>
<td>T-E Control</td>
<td>0.18</td>
<td>0.23</td>
<td>0.34</td>
<td>0.19</td>
</tr>
<tr>
<td>N-Control</td>
<td>0.11</td>
<td>0.15</td>
<td>0.21</td>
<td>0.12</td>
</tr>
</tbody>
</table>

Figure 6-2. Global power analysis (eyes closed) right hemisphere frequency patterns with confidence intervals.

Note: Graph shows a significant difference in the alpha frequency for higher amplitudes in the TEC group and for lower alpha amplitudes in the PTSD group.
6.4.4.1. Post Hoc Analysis: Band by Hemisphere by Condition

To explore the three-way interaction effect for frequency band, hemisphere, and condition, separate post-hoc analyses for each frequency band were conducted between groups to test for simple main effects of hemisphere and condition.

**Delta:**

There were no statistically significant results for the delta frequency band between groups. However, the pattern of means indicated non-significant higher amplitudes in the TEC group and lower amplitudes in the NC group compared to the PTSD group (as shown in Figure 6-1 and 6-2).

**Theta:**

The only significant main effect in the theta frequency band was for condition \( [F(1,146) = 9.38, p = < 0.001] \) revealing a lower amplitude in the eyes open condition for all three groups.

**Alpha:**

For the alpha frequency band, there was a significant interaction of hemisphere by condition \( [F(1,146) = 8.98, p = < 0.01] \).

A post hoc analysis of hemisphere by condition was conducted using a paired samples t-test that revealed a significantly lower amplitude (\( t = -.4.50, \text{df}(148), p = < 0.001 \)) in the left hemisphere (\( M = 4.44, \text{SD} = .85 \)) compared to the right hemisphere (\( M = 4.49, \text{SD} = .83 \)) only in the eyes closed condition. The results were consistent for all three groups. The analysis for the eyes open condition did not reach statistical significance (\( p = 0.37 \)). The differences in amplitude for eyes open and eyes closed were confirmed by a main effect for condition \( [F(1,146) = 428.16, p = < 0.001] \), the amplitudes for eyes open were lower than for the eyes closed condition. The main effect for hemisphere did not reach statistical significance \( [F(1,146) = 3.14, p = 0.08] \).

There was a main effect for Group \( [F(2,146) = 4.17, p = 0.02] \), indicating differences in alpha frequency amplitudes between groups, with the highest amplitudes in the TEC group and the lowest amplitudes in the PTSD group. Bonferroni post hoc tests revealed a significant difference in alpha amplitudes between PTSD participants and TEC participants (\( p = 0.03 \)).
and between TEC participants and NC participants (p = 0.03), as represented in Figure 6-1 (left hemisphere total power) and Figure 6-2 (right hemisphere total power).

**Beta:**

For the beta frequency band, a significant interaction of Hemisphere by Group [F(2,146) = 3.31, p = 0.04], revealed differences in beta amplitudes between groups across each hemisphere.

A post hoc analysis for the interaction of hemisphere by group was conducted using a one-way ANOVA, which revealed a significant difference between groups for beta amplitudes in the left hemisphere eyes closed condition [F(2,148) = 4.14, p = 0.02), but not for the right hemisphere (p = 0.08).

Pairwise comparisons revealed a significant difference between the TEC group and the NC group (MD = -.21, SE = .103, p = 0.04) in beta amplitudes for hemisphere differences, indicating a shift in both the TEC group beta amplitudes and the PTSD group beta amplitudes. This was confirmed by Bonferroni comparisons (MD = -.30, SE = .104; p = 0.01) revealing slightly lower amplitudes in the NC group (M = 3.67, SD = .53) compared to the TEC group (M = 3.97, SD = .51) and is represented in Figure 6-1 using confidence intervals, and in Figures 6-3 and 6-4, using estimated marginal means. There were no significant differences between groups for a hemisphere effect in the eyes open condition (left hemisphere: p = 0.31; right hemisphere: p = 0.48). The main effect for Condition [F(1,146) = 10.87, p = < 0.01], indicated lower amplitudes in the eyes open condition compared to the eyes closed condition.
Figure 6-3. Total beta power in the eyes closed condition for both left and right hemispheres.

Note: Graph shows an interaction effect between the TEC group and the NC group.

Figure 6-4. Total beta power in the eyes open condition for both left and right hemispheres.

Note: Graph shows higher reactivity in the PTSD group and an interaction effect between the TEC group and the NC group.
Summary: Global Spectral Power Resting Patterns:

The hypothesis of differences in amplitudes for each spectral frequency band between groups was not supported. There were, however, significant differences between groups for the alpha and beta frequency bandwidths. For all groups there was a significant and consistent change among all frequencies toward lower amplitudes in the eyes open condition. There was a consistent pattern in the TEC group toward the highest amplitudes in all frequency bands.

A significant interaction effect for frequency Band by Hemisphere by Condition revealed differences between groups for higher amplitudes in the TEC group compared to the NC group in the alpha and beta bandwidths at left hemisphere eyes closed conditions. These differences suggested greater variability between the control groups in the higher frequencies. There was a pattern of lower amplitudes in all frequencies in the PTSD group and a significant difference in alpha power (eyes closed) compared to the TEC group.

6.4.4.2. Anterior/Posterior EEG Resting Patterns

For an analysis of global frequency patterns associated with frontal and posterior regions, Table 6.11 shows a significant interaction for Band by Region \([F(2,328) = 162.23, p = < 0.001]\), which was confirmed by a significant main effect for frequency Band \([F(4,328) = 68.43, p = < 0.01]\). The interaction effect was explored as a post hoc analysis.

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Band</td>
<td>4,328 (G-G)</td>
<td>68.43**</td>
</tr>
<tr>
<td>Region</td>
<td>1,147 (G-G)</td>
<td>2.18</td>
</tr>
<tr>
<td>Group</td>
<td>2,147</td>
<td>3.75*</td>
</tr>
<tr>
<td><strong>Interaction Effect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Band x Region</td>
<td>2,328 (G-G)</td>
<td>162.23**</td>
</tr>
<tr>
<td>Band x Group</td>
<td>4,328 (G-G)</td>
<td>1.53</td>
</tr>
<tr>
<td>Region x Group</td>
<td>2,147 (G-G)</td>
<td>.029</td>
</tr>
<tr>
<td>Band x Region x Group</td>
<td>5,328 (G-G)</td>
<td>.432</td>
</tr>
</tbody>
</table>

Note: *\(p < 0.05\); **\(p < 0.01\)
6.4.4.3.  Post Hoc Analysis: Band by Region

A post hoc simple main effects analysis for each region, as a separate analysis, revealed that there were differences between groups in frontal regions; the main effect for Band \( F(3,145) = 73.61, \ p = < 0.001 \) and the main effect for Group \( F(2,147) = 4.03, \ p = 0.02 \), were both significant.

For posterior regions post hoc simple main effects revealed a significant effect for Band \( F(3,145) = 188.26, \ p = < 0.001 \). There were no significant differences between groups in posterior regions \( F(2,147) = 2.83, \ p = 0.06 \).

Further post hoc analyses for the frontal region data were assessed using one-way ANOVA. Based on findings of the global analyses, the alpha and beta bandwidths were the only two frequency bands examined for differences between groups in frontal regions. There was a significant difference between groups in the alpha bandwidth \( F(2,147) = 4.71, \ p = 0.01 \) but not in the beta bandwidth \( F(2,147) = 2.42, \ p = 0.09 \). Results for a difference in alpha frequency were adjusted for multiple comparisons (Bonferroni: 0.05/2) and were significant at the \( p < 0.03 \) level. Bonferroni multiple comparisons revealed a significant difference between groups in the alpha frequency band between the PTSD group and the TEC group \( (MD = -.43, \ SE = .15, \ p = 0.02) \) and between the TEC group and the NC group \( (MD = -.34, \ SE = .13, \ p = 0.02) \) for higher alpha amplitudes in the TEC group in frontal regions, as shown in Figure 6-5.

Summary: Anterior/Posterior Resting EEG Patterns:

As expected, data supported the hypothesis of a difference between frontal and posterior regions in resting cortical arousal patterns, with each frequency oscillating at different amplitudes. The TEC group amplitudes in each frequency were consistently higher for both regions. Post hoc analyses revealed significant differences between groups for higher amplitudes in the TEC group compared to the PTSD group and the NC group in the alpha frequency band in frontal regions. There were no significant differences in frequency amplitudes in posterior regions between groups.
Figure 6-5. Global power analysis (eyes closed) in frontal regions indicated a significant difference in alpha power in the TEC group for higher amplitudes compared to the PTSD group and the NC group.

Figure 6-6. Global power analysis (eyes closed) in posterior regions indicated no significant differences between groups in any of the amplitude frequency patterns.
6.5. General Discussion and Conclusions

As a question, derived from the first set of analyses in the current study, it was not clear whether those who had suffered severe traumatic stress, but were not diagnosed with PTSD, developed similar secondary characteristics for adaptive functioning and whether the speed of processing in these individuals was mediated by anxiety and depression symptoms. Using standardised instruments, the two related hypotheses examined by the current study were supported by the data.

The pattern of results indicated differences in subjective reactivity to perceived traumatic stressor events for people in community groups. The pattern of differences, between those experiencing strong reactivity to perceived traumatic stressors, and those who indicated minimal and no reactivity, was consistent. Control group stratification based on this criterion indicated that there were alterations to resting cortical arousal indices and to speed of information processing in sensori-motor functions between groups. The significant findings are also in agreement with a different pattern for speed of processing in people with PTSD, as discussed in the previous chapter, and as indicated in patterns of functioning in studies using a three-group design (Stein et al., 2002; Britton et al., 2005). The current pattern of preliminary results suggests important implications for treatment options and for diagnostic purposes. The findings for the current analyses will be elaborated in the following sections.

6.5.1. Learning Changes Perception through Neural Plasticity

The clinical significance of the current findings is relevant to understanding how learning changes perception over time, particularly from the perspective of theories proposing that neural plasticity is a secondary characteristics in PTSD with cumulative and specific effects (McFarlane et al., 2002), altering patterns of information processing at the structural level of cognitive architecture. The question is whether abnormality, arising as a gradual process of change, can be reversed. One suggestion is that deficit is linked to a disruption in language and left hemisphere functions and associated with an alteration in neuronal pathways, particularly for fronto-parietal regions (Clark et al., 2003). Based on the pattern of current results, this model may be a plausible explanation for the specific or unique psychological and physiological changes in people with PTSD and provides avenues for alternative treatment options. Furthermore, understanding patterns associated with resilient functioning after severe psychological distress is useful in clarifying the mechanisms for shifts between structural and functional abnormalities.
However, the current results were also in agreement with findings presented in a review article by Nemeroff and colleagues (2006). Specifically, the results were in agreement with suggestions that a higher proportion of women are at risk of developing PTSD symptoms. As a preliminary and exploratory analysis, the relationship between emotional reactivity and cortical activation patterns examined in this study indicated alterations to neural pathways for reaction patterns in the TEC group. This finding raises the question of whether premorbid traits are a plausible explanation for the results. On the other hand, the idea that gender differences are associated with differences in brain organisation patterns and with differences in reactivity to trauma exposure was not investigated in the current study and further investigation is required for a full analysis of the pattern of results derived from the current data. This notion may be consistent with differences between perception and interpretation of context information. Of greater significance to understanding behavioural data at a mechanistic level, was the finding that community groups also indicate altered patterns of resting cortical activation that is consistent with effortful control for self-regulation, and the distribution of attentional resources between patterns of stress and arousal.

In the current study, resting patterns for cortical arousal were found to be consistent for a lower alpha amplitude pattern in the PTSD group, with differences in the frontal left hemisphere regions suggesting a common underlying factor for altered cortical oscillation patterns. Based on previous studies, the pattern of brain activity in resting states in people with PTSD (Jokic-Begic and Begic, 2003; Metzger et al., 2004), suggest that resting baseline patterns of attention modulation, as indexed by the alpha band of frequencies, differs in people with PTSD and indicates a desynchronisation of cortical arousal. Greater left hemisphere desynchronisation has been associated with increased reactivity or higher stress, while greater right hemisphere activation has been associated with the modulation of arousal (Basar, 2004; Niedermeyer and Lopes da Silva, 2005). Alpha frequency patterns have also been linked to semantic retrieval functions (Cooper et al., 2003), which may be important for understanding the current pattern of resting states in PTSD participants and in those in the control group with high reactivity to traumatic stressors. In addition, it has been suggested that the binding of information in the higher frequencies can be disrupted in frontal regions with lower alpha power in baseline states (Sauseng et al., 2005; Ward, 2003).

The lower amplitudes, particularly for the alpha frequency band, characteristic of the PTSD group reported so far in the current study, were interpreted to mean desynchronisation in background resting rhythms and instability in cortical arousal patterns in PTSD associated with an altered pattern of activation in frontal regions. This pattern may be consistent with
behavioural findings of increased cognitive effort and slowed information processing. Furthermore, this argument was extended to suggest that the finding of consistently higher amplitudes in the TEC group was an alteration at the local or structural level of processing.

Therefore, linking the above two hypotheses, one plausible suggestion for the preliminary results in this study is that compensatory neural mechanisms, in women with strong reactivity to traumatic events, may be linked to language-based rehearsal mechanisms. This proposal is sample-specific but may account for the current pattern of results, particularly when applied to the wider context of how disruption to rehearsal systems alters the pattern of behavioural performance. That is, the motor-tapping results for the current analysis indicated a more effortful performance by both the PTSD group and the TEC group compared to the NC group, implying no differences in the execution of procedural skills for the two groups having experienced strong stress reactivity to traumatic events. However, there were significant differences between the PTSD group and the TEC group in resting cortical activation patterns, suggesting an altered pattern of self-regulation is associated with selective attention mechanisms, possibly linked to anxiety or comorbid mood states in people with PTSD.

The idea that rehearsal mechanisms are disrupted by selective attention, and by spatial working memory processes, when accessing long-term memory retrieval codes for self-registration or self-regulation, has also been proposed in the wider literature (Chein et al., 2003; Smith et al., 2003). A spatial working memory hypothesis for the organisation of knowledge (Corbetta et al., 2002; Glassman, 1999) is in agreement with models suggesting that autobiographical memory is different to ordinary memories (Svoboda et al., 2006), and with models suggesting that emotions play a role in the facilitation of memory consolidation and retrieval processes (Lanius et al., 2005; Conway and Pleydell-Pearce, 2000; Schutter and van Honk, 2006).

Taken together, the different models implicate an interaction between frontal-lobe activation, semantic memory processes, and cerebellar activity for the updating of a feed-forward code facilitating the phonological loop (Desmond et al., 1997). Such arguments for the coordinated integration of information within different regions of the brain are presented as theoretical proposals for understanding functional mechanisms, and extend the earlier proposal by Clark and colleagues (2003) arguing for a disruption to language systems in people with PTSD. Therefore, at a local level of information processing, how information is strengthened by neural codes, as a mechanism associated with specific regional disruptions to the coordinated
flow of information, may be a more useful hypothesis linking arguments based on the specialisation of language or spatial working memory processes.

In the current analysis, an asymmetry hypothesis based on hemispheric specialisation theories, as local modality-specific processes that have been disrupted in people with PTSD, was not supported by the EEG data for alterations to hemispheric resting cortical patterns. In contrast, the view that a functional shift in attention mechanisms is based on the manipulation of objects versus the maintenance of objects to support the integration of information in memory processes has been linked to different mechanisms in people with PTSD (Jatzko et al., 2006; Bryant et al., 2005; Vasterling et al., 2006). Therefore, the retrieval of information and the reconstruction of information, as a second stage of processing may be important to understanding neural codes in PTSD (Mesulam, 1998). Differences in the TEC group resting frequency patterns in the current analysis support the view that learning and plasticity are influenced by different frequency patterns for a dynamic and emergent shift in neural mechanisms at the functional level of analysis. Furthermore, the results of this study indicated differences in sensori-motor functions in those in the control group with strong reactivity to traumatic stressor events. This finding may be interpreted from the viewpoint of differences between diffuse neural connectivity patterns and more focal patterns of brain organisation (Lezak et al., 2004; Banich, 2004).

Therefore, as an alternative explanation for the pattern of results, and consistent with a hypothesis of diffuse connectivity, models proposing neural interactions of spatio-temporal activation patterns (Singer, 1999) suggest that individual differences in compensatory mechanisms are possible. Based on this interpretation of the current data, it is plausible that alterations to neural patterns, as a secondary characteristic of trauma exposure, are suggestive of shifts in retrieval mechanisms and the connectivity between regions linked by temporal codes. This notion is in agreement with models proposing that different frames of mental reference, for the processing of sensory stimuli, can be mapped by neural mechanisms in the same nonverbal space for different valence and arousal representations (Shelton and Gabrieli, 2004; Nadel and Hardt, 2004) and for different dimensions of time (Fuster, 2003; Singer, 1999; Lisman et al., 2001). In other words, information processing is not just the representation of sensory stimuli, but also the transformation of information consistent with emotional arousal (Mesulam, 1998; Metzger et al., 2004; Shelton and Gabrieli, 2004). Such models implicate interactions between different parameters in the coordinated flow of information and a specific role for the temporal dynamics of frequency patterns in distributed systems (Ward, 2003; Singer, 1999). Thus, whether preparatory cortical resting rhythmic
oscillations, with a lateralised pattern of activation, are predictive of interactions between cognitive function and emotional reactivity remains to be clarified. The results for the current data are discussed in greater detail in the following sections for alternative explanations that may be consistent with previous findings in the PTSD literature and in the wider literature.

6.5.2. Characterisation of Traumatic Stressors in Community Samples

The hypothesis predicting that there would be a high prevalence of reported traumatic stressors in community samples with differing characteristic presentations was supported by the current data. The results indicated that a subgroup (TEC) could be identified from the control sample, for events experienced that were perceived as traumatic and causing a response that included feeling both “terrified” and “helpless”, but that did not result in a clinical diagnosis of PTSD. The data also indicated differences in reactivity to events (criterion A2), particularly in the NC group. Results demonstrated a more specific pattern of responding to traumatic stressor events. That is, a higher percentage of respondents in this subgroup reported feeling “helpless” rather than “terrified”. This latter finding must be cautiously interpreted and requires further investigation because it is likely that the formation of the normal control group, and the resulting heterogeneity of the group, contributed to the result. However, if this result is indicative of a particular response pattern, then it supports other views in the literature suggesting that PTSD subtypes are to be assessed separately for a clearer understanding of the disorder (Metzger et al., 2004; Lanius et al., 2003; Begic et al., 2001).

On the other hand, the findings may be related to heterogeneity of the groups and may be related to chance results. However, this is unlikely because differences in the control group indicated a gender bias associated with the nature of the traumatic event reported. The clinical relevance of this finding is in agreement with models that suggest a trend for greater susceptibility of women experiencing traumatic stressor events compared to men (Nemeroff et al., 2006), and also in the wider literature, that women have a higher ratio of depressive episodes compared to men (Smit et al., 2007). In contrast, Rosenman (2002) has suggested that the type of trauma, rather than gender, is a greater risk factor in the progression to PTSD. Therefore, gender-specific effects in responses to traumatic stressors require further investigation to assess coping strategies and the compensatory mechanisms associated with working memory systems that may be indicative of a different pattern of functioning, depending on the nature of the event, and appraisal systems reflecting social values. In addition, the results indicated that the subjective nature of appraisals for situations or events
might be a difficult construct to measure in heterogeneous samples and the current pattern of significance testing remains to be clarified.

As expected, the current study revealed a significant difference between groups in the average number of events reported, with the PTSD participants reporting the highest number of events. The number of stressor events may be correlated with biological changes associated with the hypothalamic-pituitary-adrenal axis and catecholamine systems, and may be consistent with findings suggesting that PTSD is a syndrome that reflects an atypical stress response (McFarlane and Yehuda, 2000; Southwick et al., 1999; Hull, 2000). This interpretation would imply a threshold effect based on a continuum of traumatic stressor exposure. However, a threshold effect hypothesis based on the number of events experienced is an unlikely account of the current data, because it does not satisfactorily explain data for those people who develop PTSD after one traumatic event exposure. Furthermore, although there was a significant difference between groups, the PTSD group revealing a pattern of multiple stressor events, there were also multiple events reported in the non-clinical TEC group.

The theoretical question that can be posed from this discussion is whether the stress response in people with strong reactivity is a deliberate shift in responding to stimuli rather than a compensatory shift. Therefore, in contrast to a threshold based on the number of events experienced, an alternative, and more likely, explanation is the continuum of patterns associated with a traumatic event that are retrieved and replayed, as per DSM-IV symptom category B (re-experiencing). One model has demonstrated that, over time, the retrieval mechanisms for items that are constantly replayed in memory alter the temporal contiguity of when an event was first encoded (Howard et al., 2006). Based on this model, an asymmetry in working memory patterns associated with a traumatic event is a plausible explanation for shifts in neural connectivity patterns in people with PTSD or with previous traumatic experiences. This interpretation implies a selective attention bias in attentional scanning associated with perceptual asymmetry (Bryant et al., 2005; Felmingham et al., 2002).

6.5.2.1. Reaction Patterns Associated with Learning and Memory

An alternative explanation for an association between stressor events and biological change may include secondary characteristics such as working memory functions implicating psychological appraisals and reactions to the type of event, and a deliberate shift in responses. A shift to right hemisphere processing, as a compensatory adaptive mechanism, may be consistent with emotion regulation based on the processing of contextual cues and internal
representations associated with familiarity and reduced language functions (Chemtob and Taylor, 2003; Niebauer, 2004). This model of information processing is congruent with models suggesting the loss of predictive functions based on increasing levels of uncertainty, and an interaction between neurotransmitter systems (Dayan and Yu, 2003; Berntson et al., 2003). In addition, increased stress and glucocorticoids have been associated with impaired memory retrieval (Aston-Jones et al., 1994; Gold, 2005; Southwick et al., 1999).

The current study found differences between groups in the most commonly reported types of traumatic events (criterion A1). That is, witnessing of events was the highest reported checklist item in both the PTSD group and the NC group. The nature of the event was not the same in the PTSD group and the TEC group, with the latter reporting a higher incidence of sexual molestation compared to a higher incidence of physical attacks reported in the PTSD group. These findings imply social as well as psychological factors, in the appraisal of events and the subsequent reaction pattern involving learning and memory mechanisms, in cortical and subcortical structures.

As previously discussed, abnormality in the short-term or transitory storage of information has been demonstrated in PTSD, principally for visual-verbal working memory at the encoding stage of information processing (Clark et al., 2003). Therefore, a language-based hypothesis for the implementation of information at a functional level may be a plausible explanation for the pattern of findings indicated by the current results. As discussed previously (Chapters 2 & 3), language includes multiple components that are thought to represent syntactic comprehension and the functional-anatomical organisation of internal representations of information, including affective components for right-hemisphere processes (Nadel and Moscovitch, 1998; Gazzaniga et al., 2002). These may be associated with the perception of stimuli in different dimensions, and with perceptual information that might remain in transitory states when accessing long-term memory codes (Brewin, 2003; Chein et al., 2003; Glassman, 2000; Mesulam, 1998).

Based on a language hypothesis, the current findings suggest that differences between the PTSD group and the two control groups may be consistent with explanations of structural alterations at the knowledge or mental algorithm level. Various theories have been proposed to support the idea that structural compensatory mechanisms are linked to PTSD and involve alterations to hemispheric communication channels. For example, Schore (2002) has proposed that the right hemisphere is not developed for affect regulation when maltreatment and overwhelming stress occurs in early childhood, leading to structural alterations in the brain
and poorly integrated information processing. This notion has also been associated with mixed lateral handedness preference models and the higher risk for PTSD (Saltzman et al., 2006). In addition, McFarlane and Yehuda (2000) have proposed that premorbid factors may contribute to reaction patterns. In other words, episodic memories are semantically interpreted and stored in similar circuits that reduce connectivity by losing spatio-temporal specificity (Nadel and Moscovitch, 1998). This latter suggestion is consistent with proposals that general or tonic characteristics, at the computational level of information processing, enable the functional manipulation of information across different situations (Anderson et al., 2004; Galletly et al., 2001).

Consequently, developmental skills or the capacity for problem solving have been shown to depend on the age at which the event was experienced (McFarlane et al., 2002; Teicher et al., 2003; Schore, 2002). The results of the current study demonstrated that “time elapsed since the event” indicated on average a more recent traumatic event for the PTSD group compared to the two control groups. The pattern of differences between groups suggested a later life onset in PTSD, with PTSD participants reporting trauma exposure in adulthood, while the control participants reported trauma in the younger years. However, this result may have been due specifically to group allocation and to the experimental condition and cannot be generalised or taken as an accurate indication of PTSD populations. On the other hand, if this finding is not an experimental artefact, it may be suggestive of alterations to attention mechanisms, which have been consistently reported as impaired in the PTSD literature (Bryant et al., 2005; Buckley et al., 2000; Vasterling et al., 1998). Therefore, time elapsed since the event may be a useful variable in offering an explanation of plausible mechanisms for changed neuronal pathways and requires further investigation.

Attention has been linked to the timing of information flow and the modulation of cortical arousal (Ward, 2003). Attention mechanisms have also been related to both structural and functional changes and to how information is appraised, encoded, and later retrieved (Thomsen et al., 2004; Umilta, 2001; Vasterling et al., 1998). The nature of the event and the age at which the event first occurred have been proposed in earlier studies as highly relevant factors in the development and maintenance of PTSD symptoms (Mineka and Zinbarg, 2006; Nemeroff et al., 2006). However, in contrasting views, numerous studies have also reported that the subjective appraisal of events involves individual differences based on perception of the contextual features that are encoded at the time of the event and subsequently triggered as a result of situational cues, priming mechanisms, and associative memory networks (Brewin, 2003; Svoboda et al., 2006; Glassman, 1999; Kirsch et al., 2004).
Related to attention mechanism is the question of differences between the nature of the event and right hemisphere processing mechanisms. For example, are there differences in right-hemisphere processing mechanisms for physical trauma compared to trauma involving sexual molestation? The above arguments pose another question of whether language-based interpretations of stimuli are differentially related to right hemisphere processes and to affective states (Chemtob and Taylor, 2003), implying differences between self-registration and self-regulation mechanisms for information processing and interaction effects between patterns of context information and content information. Specifically, these categorisations of information involve visual experiences of contextual cues that become organised through content (Van Horn et al., 1998; Hasegawa et al., 1998; Howe et al., 2003; Niebauer, 2004), where context information is not always explicit, but internally imposed (Nadel and Moscovitch, 1998).

Fear has been reported as an emotional reaction that may be represented by two pathways, namely psychological or physiological (Williams et al., 2006; Bryant et al., 2005; Yu and Dayan, 2002). The data for this study indicated that experiences of trauma events later in life, as reported in the PTSD group, compared to the earlier or childhood experiences reported in the control groups, may implicate mechanisms that alter the interactions in distributed parallel networks. In particular, different functions in verbal and nonverbal working memory systems may underlie psychological or physiological pathways. It has been suggested that there are critical variables that mediate and modulate arousal patterns (McFarlane and Yehuda, 2000) and these may be consistent with alterations in neuronal pathways and the reorganisation of brain functions organised for coping with extreme stress (Lanius et al., 2005).

Educational experience was also significantly different between groups, the PTSD group reporting fewer average years of education compared to the two control groups. The protective barriers to stress provided by high intellectual functioning, as well as the availability of intellectual resources for problem solving, have been reported in previous PTSD literature (Vasterling et al., 2002). The implications of lower intellectual capacity for the speed of processing, and for self-regulation and control, may be related to the appraisal of information and the modulation of cortical arousal systems (Yu and Dayan, 2002). Buffers to stress from education and age may be relevant factors associated with the interaction effects of neurotransmitter systems. On the other hand, if time since trauma is a relevant factor, and the number of traumatic events is high, it is plausible that the retrieval of stored information
becomes less accurate over time and the initial reaction becomes generalised to more current stimuli resulting in secondary characteristics (McFarlane et al., 2002; Litz et al., 2000).

### 6.5.2.2. Compensatory Mechanisms Hypothesis

Based on a compensatory mechanism hypothesis, and parallel-distributed processing models of neural organisation (McFarlane et al., 2002), the subjective nature of appraisals for events, together with the number of events perceived as being highly traumatic, may be relevant variables for a selective attention bias in PTSD symptomatology and may be a significant factor contributing to behavioural adjustment after the event. Speculatively, the effects of lower education levels may suggest fewer resources for returning to normal functioning and the persistence of maladaptive symptoms associated with re-experiencing phenomena. As previously discussed, the current data supported previous findings that PTSD may be associated with lower intellectual functioning that may lead to higher stress reactions (Vasterling et al., 2002). Consistent with the latter notion, age at the time of experiencing a traumatic stressor suggests important implications for learning and the allocation of attention resources, particularly for adaptation and the coping strategy of avoiding certain categories of stimuli (Saltzman et al., 2006; Schore, 2002; Teicher et al., 2003).

Selective attention biases have been related to hyperarousal symptoms and to lower intellectual functioning (Bryant et al., 2005; Vasterling et al 2002), with the suggestion that verbal abilities may act as a buffer to the development of PTSD and reduce central nervous system vulnerability to stress (Brewin, 2003; Vasterling et al., 2002). On the other hand, as discussed above, age is also an important variable for mental functioning, and, according to developmental theories, has important implications for adjustments in behavioural outcomes, particularly for older people (Buckley et al., 2000; Nemeroff et al., 2006), as well as the very young (Howe et al., 2003; Schore, 2002).

Furthermore, it has been demonstrated that age-related differences are implicated in models of contextual binding and hippocampal function in episodic memory formation, with modelling results suggesting a decrease in the ability of older people to recover temporal contexts (Thomsen et al., 2004; Howard et al., 2006). Hence, age at the time of experiencing the traumatic stressor may be highly relevant to the coping strategies adopted and to the maintenance of maladaptive behaviours associated with mechanisms for the retrieval of information for content information. The role of spatial working memory is relevant to attention mechanisms and an “asymmetry drift” altering the timing and flow of information (Compte et al., 2000; Howard et al., 2006). This idea implies alterations to rehearsal
mechanisms for the maintenance and coordination of working memory systems (Glassman, 1999; Chein et al., 2003) and has been associated with the loss of spatio-temporal specificity in hippocampal structures (Nadel and Moscovitch, 1998).

The results of the different reaction patterns (based on criterion A2), as detailed in a standardised “checklist of events”, may provide a subjective preliminary evaluation about memories for a particular extreme event. However, the current study found no significant differences between the reaction patterns for the PTSD group and the TEC group. On the other hand, there were differences in the type of traumatic stressor reported between the two groups, differences in the number of events reported, and differences in the age at which the events were experienced. Learning theories posits that a number of stimulus-response associations or response-outcome associations can occur to alter and reinforce a pattern of behaviour (Kirsch et al., 2004). According to Clark and colleagues (2003), differences at the encoding stage, particularly for verbal working memory processes are associated with alterations to cortical functioning in people with PTSD. However, with progressive retrieval and reconstruction of events in memory, the invariant properties of items are subject to changes over time (Howard et al., 2006; Nadel and Moscovitch, 1998). This model may be in agreement with the hypothesis proposed by McFarlane and colleagues (2002) of alterations to cognitive functioning as a result of synaptic plasticity and secondary characteristic of traumatic experiences associated with stress reaction patterns over time.

An alternative plausible suggestion is that subjective appraisals of events have been modelled to reflect patterns of the self in the world (Nadel and Moscovitch, 1998; Conway and Pleydell-Pearce, 2000; Svoboda et al., 2006). These patterns have also been related to spatial working memory and the organisation of mental representations that impose structure and meaning to events and to classes of stimuli for fast recognition and for detection of relationships between items of high importance (Eichenbaum and Fortin, 2005; Nadel and Hardt, 2004; Shelton and Gabrieli, 2004).

In addition, involvement of the cerebellar in feed-forward loops to frontal lobes and the facilitation of rehearsal mechanisms involving the phonological loop have also been implicated in tonic states as patterns of structural knowledge (Desmond et al., 1997; Schutter and van Honk, 2006). Various theories propose that failure to predict anticipated stimuli may serve to reinforce associations between items and restrict connectivity patterns by preventing attentional scanning mechanisms (Dayan et al., 2000; Ramos and Savage, 2003). These
notions may be relevant to the findings in the PTSD literature, and to the current results of alterations at the structural level of information processing, and for neural plasticity over time.

Learning and coping strategies formulated for adaptation during the early developmental years imply the development of self-protective barriers and symbolic or semantic patterns for classifying environmental stimuli (Schore, 2002; Teicher et al., 2003). It is possible that the appraisal of future threat and the direction and allocation of attention mechanisms for the preparation to respond to future possible threats of danger may become disrupted by fast recognition mechanisms associated with self-registration and spatial working memory systems. An inability to spontaneously reorganise memory representations or failure to update working memory systems is likely to result in alterations to the sequencing of memories, particularly for categories of information involving perceptual dimensions (Nadel and Moscovitch, 1998; Howard et al., 2006). Based on an optimisation model by Dayan and colleagues (2000), and a hypothesis suggesting the transitory storage of perceptual information (Glassman, 1999), the results of the current study may be speculatively interpreted to suggest that greater exposure to traumatic events is likely to create uncertainty in the recognition of categories of information and unreliability in categories of stimuli. For the PTSD group, the data suggest that these processes become disrupted later in life, and for the TEC group, the data indicate that different processes were implemented after early life stressors for adaptive functioning. This hypothesis implies two processes - a lack of awareness compared to conscious implementation in stress-management strategies, and the consequent altered patterns of functioning associated with retrieval mechanisms.

Allocentric and egocentric frames of reference have been referred to as mechanisms associated with the perceptual nature of the experience and associated with attention mechanisms linking the posterior cortex to the anterior cortex (Shelton and Gabrieli, 2004; Nadel and Hardt, 2004). The retrieval of long-term memories, linking pathways to autobiographical specific events, rather than episodic memory for cognitive events, has been suggested as a plausible hypothesis for the contextual patterns in retrieval strategies (Svoboda et al., 2006; Glassman, 1999). The distinction between a specific memory and a general class of events has been shown to alter perceptions (Conway and Pleydell-Pearce, 2000; Nadel and Moscovitch, 1998; Ramos and Savage, 2004). Based on such arguments, the events experienced and later recalled may be consistent with an underlying pattern of altered arousal when triggered by contextual information that is linked to an emotional frame of reference (Corbetta et al., 2002; Ehlers and Clark, 2000; Nadel and Hardt, 2004; Shelton and Gabrieli,
A focus on context information involves differences between spatial and visual working memory, modulated by selective attention mechanisms (Miyake et al., 2001).

Thus, studies suggesting that the orienting to sensory information involves spatial working memory pathways prior to target presentation (Eichenbaum and Fortin, 2005) may be relevant to the current results indicating differences between people reporting similar reaction patterns to events, but who did not develop PTSD. As already discussed, differences between groups may depend on the allocation of attention that has emotional significance based on only fragments of visual information. Speculatively, it may be argued that the emotional significance of perceptual information may distort the sequencing of a pattern of associated stimuli in left hemisphere processing and bias interpretations at any one point in time (Jatzko et al., 2006; Munk, 2001).

It is plausible that the data for both the PTSD group and the TEC group are suggestive of subjective responses that imply a pattern of representations coded as visual representations. An association of visual representations with emotional reaction patterns has been suggested in earlier models of PTSD (Brewin, 2003; Joseph et al., 1997). Understanding the mechanisms responsible for symptom patterns and their maintenance requires not only knowledge of how coded information becomes altered as a result of stressor events, but also data about whether the learning-induced changes of neuronal representation alter the response properties of synchronously firing neurons. It has been postulated that the encoding of information caused by learning does not necessarily alter existing response patterns, and this may result in loss of adaptive functioning (Munk, 2001; Lanius et al., 2005). This is an important factor that has received limited investigation in the literature to date.

The above ideas offer a plausible explanation to alterations in functioning associated with PTSD symptomatology. A higher number of stressor events experienced may speculatively be associated with increasing “asymmetry drift” during retrieval of memories (Howard et al., 2006; Landau et al., 2004). Together with theories of uncertainty associated with the prediction of future stimuli (Dayan and Yu, 2003; Niebauer, 2004), these ideas may be applicable to the pattern of active and remitted symptom presentations in people with PTSD (McFarlane et al., 2002).

6.5.2.3. Functional Cerebral Asymmetry

In proposing a functional role for the reaction patterns reported by the TEC group, it may be plausible to suggest that narrative skills may have played a significant role in mediating the
long-term chronic reaction patterns. This proposal is consistent with ideas that suggest that creativity is a right hemisphere function that is also linked to problem solving solutions (Jausovec and Jausovec, 2000). As previously discussed, a switch from right-hemisphere identification and processing of items, to left-hemisphere interpretation of items, may distort response patterns according to a single interpretation and the sequencing of object-level experiences at any one point in time (Nadel and Moscovitch, 1998; Van Horn et al., 1998; Niebauer, 2004). It is plausible, therefore, that problem-solving behaviours, immediately after a traumatic event, lead to individual differences in functional pathways and compensatory strategies that continue to activate the right hemisphere but do not result in chronic PTSD patterns. This interpretation may be applicable to those in the control group, for the current study, who reported strong reactivity to traumatic stressors, and is congruent with empirical evidence provided by Vasterling and colleagues (1998), suggesting a deficit with the early recognition of information in people with PTSD.

Significant differences between groups in the current study, as defined by the nature of the traumatic stressor, the number of events reported, the time elapsed since the traumatic stressor event, and the number of women reporting strong reactions to events, were consistent with findings suggesting that certain variables are linked to the development of risk factors for PTSD (Nemeroff et al., 2006). The complexity of the syndrome has been related to the interaction of a range of the detailed risk factors and the activation of biological systems over time (McFarlane et al., 2002). Coping strategies or mediating variables have been proposed to account for the low prevalence rates of PTSD after extreme traumatic stressor events (Frans et al., 2005; Fullerton et al., 2001; Gold et al., 2005; Perkonigg et al., 2000; Rosenman, 2002; Yehuda, 2004). The findings of the current study are in agreement with proposals indicating that a high rate of exposure to trauma does not lead to the diagnosis of PTSD (McFarlane and Yehuda, 2000; Stein et al., 2002). Therefore, specific variables are implicated in the progression to PTSD symptomatology (Metzger et al., 2004; Kashdan et al., 2006).

However, the pattern of results in the current study is also convergent with models of diffuse connectivity, and disruption of long-range circuits for well-rehearsed routines in people with strong reactivity to traumatic stressors (Britton et al., 2005; Shaw et al., 2002). This pattern may be associated with emotional stimuli and uncertainty in decision processes linked to comorbid mood states. The hypothesis of comorbid depression and anxiety for the PTSD group was supported by the data. The mood scores for the PTSD participants in the current study included high rates of depression, anxiety, and stress compared to the low rates reported by participants in the two control groups. This leads to the question of whether mood states
affect temporal parameters in neural mechanisms or whether specific regions in brain organisation are implicated as “unique” to PTSD characteristics, as suggested by neuroimaging studies (Bryant et al., 2005; Lanius et al., 2005).

From a brain-behaviour perspective (McFarlane et al., 2002), the results of the current study are speculatively associated with a timing mechanism (Rowe, 2005) that alters the flow of information to intra-hemisphere and inter-hemisphere projections. These notions may be relevant to the current PTSD data by suggesting a probable argument for an influence in theta oscillations on long-term potentiation and synaptic plasticity (McFarlane et al., 2002; Rowe, 2005; Matzel and Shors, 2001). It is possible that, through altered frequency oscillations, specific modes of synaptic plasticity can have important implications for learning and memory at the behavioural level when associated with specific regions in medial temporal lobes (Gabrieli et al., 1997; Gaffan, 1994; Singer, 1999).

6.5.3. Slower Information Processing in PTSD: Behavioural Tapping Task

The above conclusion is supported by the results of the sensori-motor task in the current analysis, indicating a reduced speed of processing in both the PTSD group and the TEC group. Furthermore, there is an increasing body of knowledge accumulating that PTSD involves two different pathways; that is, psychological and physiological pathways may engage a number of different mechanisms (Williams et al., 2006; Nemeroff et al., 2006; Vasterling et al., 2006), thereby altering functional connectivity and behavioural outcomes.

Results of the current analysis supported the hypothesis of slower information processing of a general nature in the PTSD group. This finding is in agreement with previous research suggesting disturbance in executive functions in PTSD, such as planning and shifting attention (Clark et al., 2003; Vasterling et al., 1998, 2004). Furthermore, empirical evidence in animal studies has demonstrated that motor behaviour is phase-locked to the theta rhythm and to hippocampal involvement in distinct aspects of memory function (Caplan et al., 2001). This hypothesis may be relevant to the above discussion and the possible involvement of interneurons in the control of autonomic function and visceral functions (Gallese, 2003), with the disruption of theta rhythms for long-range synchronisation in anticipatory states (Jones and Wilson, 2005).

Overall, the scores of the tapping task for the current study were significantly different between groups for speed of tapping; the lowest scores were for the PTSD participants. In addition, the data indicated a difference between the number of taps for each hand; the NC
group showing a much slower rate of left-hand taps (15%) compared to the PTSD group (6% difference in hand tapping scores) and the TEC group (5% difference for a reduced number of left-hand taps). This result was interpreted to mean a more diffuse pattern of hemispheric organisation in the PTSD group and the TEC group compared to the NC group. It is plausible that any psychological functional deficits represented by a 15% difference between left and right hand tap scores in the NC group may be related to the complex relationship between the neural effects of tapping as a procedural skill, or mastery of the motor movement, and the initiation of the task as a cognitive process. Based on a hypothesis that more diffuse connections result in lower performance scores (Landau et al., 2004), it was inferred that the lower scores for the PTSD participants were indicative of a disruption to neural circuitry for connectivity between cortical regions. This interpretation is in agreement with findings of a disruption to spatial parameters in brain organisation in people with PTSD (Lanius et al., 2005; Shaw et al., 2002).

The initiation of a goal or task has been linked to the functional roles involving the engagement of the prefrontal cortex (Clark et al., 2000; Courtney et al., 1998a). Executive functioning encompasses not only the goals for the task but also the implementation of those goals (Fuster, 2003; Galletly et al., 2001). Theories of retrieval, together with theoretical models of working memory, propose that a breakdown is possible in either the active organisation of a sequence of responses during the retrieval of stored information or the active manipulation and monitoring of information (Clark et al., 2003; Galletly et al., 2001). Based on these ideas, it was inferred that a sensori-motor circuit was activated to retrieve a sequence of actions for finger tapping with each hand. An alteration in connectivity patterns between frontal and parietal lobes may be one hypothesis for a shift in executive functioning and for the slower tapping scores in both the PTSD group and the TEC subgroup. This interpretation of a disruption to connectivity between regions in people with PTSD is in agreement with models that propose that anticipatory states can disrupt long-range synchronisation patterns (Ward, 2003; Schwartz et al., 2005).

On the other hand, Lezak and colleagues (2004) argue that a disturbance of psychological motor behaviour can be attributed to different causes and can be cortical or subcortical in origin. In reference to lesion damage and deficits, Lezak and colleagues have suggested that a difference in tapping scores of greater than 10% or lower than 10% between each hand can be interpreted in two different ways for lateralised indicators of loss of function. As discussed in the previous section, and as one possibility, Lezak and colleagues have also suggested that frontal-striatal loops may be involved in the disruption to the planning and maintenance of a
motor rhythm as a sequence of responses. Therefore, speculatively, an emotional bias, at a structural level of cognitive architecture, could imply that the tapping scores were associated with decreased frontal-striatal responsiveness associated with phase-locked motor rhythms during planning and performance (Bastiaansen and Hagoort, 2003). As a second possibility, at a functional level of cognitive architecture, a general slowing of information processing implies an attentional bias, and a difficulty with inhibiting irrelevant information, so that the flow of information processing is disrupted by inhibitory and excitatory mechanisms as previously suggested by Bryant and colleagues (2005).

In handedness preference theories, dominance, as the degree of communication between the two cerebral hemispheres, has been a topic extensively studied, and with consistent findings of a dynamic multi-faceted process that changes according to task demands and performance goals (Beaton, 2004). Motor acts have been associated with left hemisphere dominance for skills that are carried out with either hand or with those that require bimanual coordination and right hemisphere specialised function for the realisation of goal-directed behaviour (Gazzaniga et al., 2002; Serrien et al., 2006). Therefore, one hypothesis for the results of the current data may be related to perceptual-motor disruptions and to altered sequencing functions associated with left hemisphere processing. A disruption to left-hemisphere sequencing of information may be associated with rhythmic discrepancies in the mPFC (Shaw et al., 2002; Sakagami et al., 2006), and may be suggestive of alterations to cerebellum activation patterns, and the timing of information flow (Schutter and van Honk, 2006), particularly influenced by a timing pattern of theta oscillations (Rowe, 2005).

In view of the above arguments, the slower speed of tapping for the PTSD group in the current study was interpreted as being consistent with hypotheses suggesting greater effort or reduced efficiency, implying blocked or structural alterations to channels of communication in left hemisphere sequencing of information (Vasterling et al., 2004). In addition, diffuse connections were also taken to mean a more bilateral connectivity pattern for the interhemispheric coordination of information, altering the timing and coordination of information processing requiring specialised pathways. This hypothesis is consistent with an alteration to physiological pathways for regional connectivity and may be related to the interruption of neural signals, and the specific neuronal shape for signal transmission (Watson et al., 2006), possibly involving regions of the ACC for different connectivity patterns (Bryant et al., 2005; Lanius et al., 2005).
In previous PTSD studies, the thalamus and insula have been suggested as regions relevant to bodily perceptions (Lanius et al., 2005), and regions that are thought to mediate the interaction between attention and arousal (Rowe, 2005). As suggested by Landau and colleagues (2004), the neural effects of enhanced behavioural performance could include (1) organisational patterns for a greater neural efficiency, (2) more precise functional circuitry, or (3) greater endogenous spatial representation for motor movement tasks, as has been indicated by practice effects. In addition, a number of learning patterns for the alteration of biological baseline regulation can be activated by severe psychological reactions to stress (Kim and Gorman, 2005). In the PTSD group, mood data for the current analysis indicated a high level of psychological dysfunction, consistent with biological models of altered subcortical systems (Lanius et al., 2005; Litz et al., 2000). These arguments, together with the current findings, are consistent with the hypothesis that long-range connectivity is disrupted in people with PTSD. However, the precise mechanisms for this disruption cannot be clarified by the results presented. Therefore, further investigation is warranted to understand the different mechanisms proposed in the above discussion.

From a genetic perspective, it is also plausible that the direction of handedness scores may be relevant to a differentiation between premorbid factors and environmental factors (Zietsch et al., 2007) in understanding chronic PTSD. In the current study, the degree and direction of handedness was not investigated and firm conclusions cannot be drawn for any effects resulting from handedness preferences. However, previous data on PTSD populations have suggested a connection between mixed laterality and predominance for right hemisphere processing (Chemtob and Taylor, 2003; Saltzman et al., 2006). Speculatively, it is possible that data for the TEC group, showing a lower pattern of difference between left- and right-hand taps compared to the NC group, is linked to handedness preference. However, this proposal requires clarification and replication of the data.

6.5.3.1. Sensori-motor Functions and the Coordinated Flow of Information

Given the above arguments, the implications of the current findings are for a disruption to the timing of information flow in PTSD participants. This disruption is likely to be consistent with previous findings proposing a decoupling of long-range neural connections (Shaw et al., 2002; Ward, 2003), which are modulated by attentional processing (McFarlane et al., 1993; Vasterling et al., 1998), or LTP alterations (McFarlane et al., 2002) involving the ACC and fronto-insular cortex (Bryant et al., 2005; Lanius et al., 2005; Britton et al., 2005). This preliminary conclusion is also in accord with findings of cognitive efficiency as proposed by information processing theories (Clark et al., 2003; Linden et al., 2003) and a model of
selective attention for predictive relationships associated with anticipatory responses and optimal functioning (Bryant et al., 2005; Dayan et al., 2000).

Furthermore, the current findings and arguments may be consistent with models proposing that severe psychological stress alters the biological regulation of arousal (Yu and Dayan, 2002; Kim and Gorman, 2005), so that shorter pathway connections between cortical regions decreases cerebral sensitivity for specialised tasks involving sequential rule-based learning (Boyer et al., 1998). The reduced speed of tapping in both the PTSD group and the TEC group may be consistent with diffuse connectivity patterns but without necessarily representing a disruption to specialised hemispheric functions for different tasks.

Additionally, a tapping task involves motoric rehearsal that has been associated with functions of the cerebellum (Desmond et al., 1997). The cerebellum has been associated with a functional relationship between attention and emotional memory systems when active maintenance of information is required (Schmahmann and Sherman, 1998). Rehearsal functions for spatial selective attention have been associated with the preparation for movement in fronto-striatal circuitry and the coordinated activity of the cerebellum (Schutter and van Honk, 2006; Wang et al., 2004) and may be relevant to understanding perseveration and an inability to shift attention during task initiation and maintenance processes. The modulation of core electrophysiological frequencies has also been a mechanism associated with a disruption in cerebellar activation for voluntary control functions or self-generated responses associated with frontal theta rhythms (Schutter and van Honk, 2006). Behavioural tasks, such as motor coordination, that are sensitive to cerebellum function, may suggest structural changes associated with reactivity patterns and enduring changes associated with conditioning or procedural memory linked with rule learning (Boyer et al., 1998; Eichenbaum and Fortin, 2005). Structural changes have been linked to spatial and temporal parameters implicating specific alterations in dorsal and ventral PFC streams (Aron et al., 2004; Sakagami, 2006). As suggested previously, the discrepancies in the finger tapping performance data, for the three groups in this study, may be suggestive of disrupted inter-hemispheric connectivity patterns for the specific localisation of functions in people exposed to extreme traumatic stressors, indicating a potential mechanism for memory disturbances.

The ability to plan and conceptualise changes to goals, even for relatively simple tasks, requires good impulse control and intact memory functions (Lezak et al., 2004). Inhibition has been associated with right hemisphere ventrolateral frontal cortex pathways (Aron et al., 2004; Sakagami et al., 2006). At a more general level, but consistent with the above
arguments, it is plausible that differences between the PTSD group and the TEC group may be related to processes of sequential planning and the facilitation versus inhibition of a perceptual-motor memory mechanism (Fuster et al., 2000). These processing strategies would suggest a qualitative difference in selective attention mechanisms for pre-attentive and focal attention stages in an internally generated mental representation of space (Fox et al., 2001; Umilta, 2001).

Different mechanisms in the PTSD group compared to the TEC group may be responsible for similar behavioural results. Speculatively, whereas the results for the PTSD group may be reflective of alterations associated with psychological and physiological mechanisms, the results for the TEC group may be more consistent with disruptions to gating mechanism and alterations to physiological pathways. It is possible that the tapping task scores for the TEC group are also indicative of a pattern of executive function alteration, and a reorganisation of memory representations for motor functions, but the precise cognitive mechanisms responsible are not identical to those in the PTSD group.

This interpretation would suggest that the PTSD group have a focus on selective stimuli and inhibit information based on endogenous representational patterns of rehearsal, whereas the TEC group may have an external focus for the facilitation of incoming information, and, therefore, would be less able to voluntarily suppress irrelevant information for simple tasks. Recent experimental evidence from the clinical literature, based on the investigation of hemispheric asymmetry patterns in people with depression, has suggested a possible link between motor control, cortical inhibition and excitatory activation patterns (Salustri et al., 2007). The authors proposed that structural and functional alterations to the pattern of cortical rhythmic lateralisation imply a change in the balance of interneuron activity between the parietal cortex and the primary sensory cortex in the right hemisphere in people with depression. Accordingly, biased attention processes associated with depressed mood states are likely to increase information processing load and reduce the speed of processing.

This view converges with findings presented by Clark and colleagues (2003) suggesting a right-hemisphere bias in people with PTSD in parietal regions. However, it may be applicable only to people with a comorbid depression and this model may not explain patterns of behaviour in people without depression who may have experienced extreme trauma reactivity. Therefore, at a deeper level of analysis, this proposal implies that specific sub-cortical alterations are also involved during the activation of a sensori-motor circuit in a distributed system.
As previously discussed, a deficit in organisational patterns for the retrieval of a sequence of responses implicates spatial working memory and executive functions for the planning and initiation of tasks. The reciprocal links between the anterior cingulate and the prefrontal cortex have been associated with working memory performance (Bryant et al., 2005). An increase in mental effort may be associated with altered speed of processing in phase-locked evoked responses (Basar, 2004). Therefore, it is plausible that alterations in the functional role of the anterior cingulate may contribute to the development of behavioural changes, such as depression and fatigue, and to cognitive dysfunction (Botvinick et al., 2004). However, in examining the behavioural outcomes for a simple sensori-motor task, a similarity in performance scores between groups does not predict the neural circuitry underlying task execution and further investigation is warranted.

6.5.3.2. Selective Attention Bias in PTSD

In support of an emotional bias hypothesis, the slower tapping scores for both the PTSD group and the TEC group indicate a possible deficit in the planning and execution of task goals. This argument may be consistent with the idea that people with PTSD have a disruption to sequencing functions, and may be convergent with previous hypotheses of a disruption to left hemisphere functions in people with PTSD (Clark et al., 2003; Bremner et al., 1999). However, this interpretation does not account for the slower tapping scores in the TEC group and alternative plausible hypotheses can be offered.

One explanation of possible compensatory mechanisms in the TEC group may be associated with the initiation of goal-directed tasks. In other words, it is possible that the pattern of data for the TEC participants involves the suppression of early recognition of stimuli so that information is retrieved only once it is familiar. This explanation is based on the theory that conscious decisions associated with attentional scanning mechanisms inhibit the early recognition of sensory information (Aston-Jones et al., 1999; Svoboda et al., 2006). On the other hand, the current data lends support to the hypothesis that the PTSD participants use limited cues for recognition (Vasterling et al., 2006; McFarlane et al., 1993), resulting in an attentional bias and less efficient information processing (Vasterling et al., 1998; Bryant et al., 2005; Felmingham et al., 2002). Furthermore, to understand functional networks associated with emotional contexts in PTSD, Jatzko and colleagues (2006) found that emotional disruptions to processing trauma-neutral information were related to an initial activation of parietal pathways of the dorsal visual stream, implicating spatial working memory processing and increased attentional effort.
An alternative interpretation for the current results may suggest that right-hemisphere parietal interneuron signals to the prefrontal cortex interrupt feedback loops for endogenous spatial representations required in motor movements. The data support the view that the PTSD participants and the TEC participants have reduced scores suggesting a breakdown in functional connectivity between the executive processes required for initiating a task: that is, prefrontal cortex functions, and disruptions to parietal afferent processes that direct attention to the prefrontal cortex. This interpretation is based on the theory that interference between the shared resources of spatial rehearsal and attention mechanisms are associated with a right hemisphere disruption to psychological motor functions that require the realisation of specific goal-directed behaviours (Sakagami et al., 2006; Serrien et al., 2006). According to this model, the implementation of procedural knowledge for the execution of a specific sequential motor movement task is thought to involve precise functional circuitry associated with left hemisphere dominance (Boyer et al., 1998; Gazzaniga et al., 2002).

The above interpretation is indicative of a precise functional circuitry for long-range connections, suggestive of focused attention mechanisms for task execution (Aron et al., 2004; Serrien et al., 2006). Furthermore, an interpretation of the current data that accords with the proposal by Clark and colleagues (2003) is that hemispheric specialisation differences in people with PTSD result from interruptions to the storage and manipulation functions associated with working memory systems. In partial agreement with the above arguments, skills, such as motor programs, are generally defined as procedural memory (Serrien et al., 2006) and are not directly affected by a traumatic event but remain as programmed actions, automatically retrieved (Landau et al., 2004).

The automatic retrieval of information that is well practised and rehearsed has been linked to the parietal cortex rather than the prefrontal cortex (Landau et al., 2004). However, a change in this circuitry would suggest a change in the stable trait patterns for responding to information, rather than a phasic shift, which has been associated with the prefrontal cortex. Therefore, if changes to procedural memory can be shown, this finding may be in agreement with models proposing that neural plasticity mechanisms occur over time to alter mental algorithms for the functional manipulation of information across situations (McFarlane et al., 2002; Singer, 1999; Sejnowski and Paulsen, 2006). This finding might also be in agreement with hypotheses suggesting alterations associated with perceptual mechanisms in PTSD (Vasterling et al., 2004) and with altered tonic or structural patterns of knowledge that are linked to preparatory states (Neylan et al., 2003; Metzger et al., 2004).
6.5.4. Speed of Processing and Alpha Peak Frequency

The hypothesis of differences in the speed of processing between groups was not supported by the APF data. Therefore, in contrast to the above arguments for structural or local changes in people with PTSD, and a possible association with phase-locked motor frequency patterns, findings for APF rhythms in the current analysis indicated that all groups were oscillating within the normal range. However, reactivity to stimuli, as indicated by the non-significant shift in frontal and central APF in the PTSD group, with eyes open compared to the eyes closed condition, may warrant further investigation with a larger PTSD sample to assess the above proposal of alterations in voluntary control mechanisms for functional implementation. It is possible that the size of the groups in the current study may have contributed to the non-significant finding. Therefore, differences for APF require further investigation before conclusions can be drawn about the speed of neural activity in people with PTSD.

6.5.5. Resting EEG Patterns and Relationship to Psychological Functioning

The hypothesis of differences in amplitudes for all spectral frequency bands between groups was only partly supported by the current data. There were significant differences between groups for the alpha and beta frequency bandwidths. However, the hypothesis of a right hemisphere lateralisation in people with PTSD was not supported by the pattern of resting oscillations. On the other hand, there were significant interaction effects indicating differences between groups in lateralisation indices for frontal and posterior regions in the alpha frequency bandwidth.

Furthermore, the data demonstrated a significant difference between groups in frequency amplitude, particularly for higher amplitudes in the TEC group and lower amplitudes in the PTSD group. This finding was difficult to interpret using previous literature for EEG resting patterns in the PTSD literature. However, alpha power has been associated with attention mechanisms and there are proposals that cortical arousal is reduced while inhibitory control is increased (Klimesch, 1999). The current results converge with findings by Jokic-Begic and Begic (2003) for a pattern of lower alpha amplitudes (or high frequency low voltage EEG activity) in people with PTSD compared to controls. As with the discussion presented here, interpretations provided by Begic and colleagues (2001) also suggest that various factors contribute to baseline activation patterns.

An alternative explanation for the different patterns in amplitudes, reflected by the current data, may be consistent with hypotheses suggesting differences in EEG power may result
from genetic traits and/or environmental influences (Posthuma et al., 2001; Zietsch et al., 2007). Data for the current results indicate that both the PTSD group and the TEC group show altered amplitude patterns. Therefore, using a trait model, data for the PTSD group and the TEC group, may be interpreted to indicate a possible genetic predisposition to alterations in EEG power (Smit et al., 2007). The higher ratio of women in the TEC group might suggest that the results of higher amplitudes across all frequencies are related to a genetic predisposition. This interpretation would accord with findings suggesting that women have a higher risk of developing PTSD symptoms (Frans et al., 2005; Nemeroff et al., 2006).

However, this argument is not supported because, firstly, there were no indications of frontal EEG laterality effects, and, secondly, there were differences in frontal EEG alpha between groups. The results were more consistent with an environmental effect in the PTSD group (Zietsch et al., 2007) and consistent with a brain-behaviour model predicting alterations to neural organisation over time (McFarlane et al., 2002). Furthermore, data for the current study, indicating reduced alpha power for the PTSD participants, was also suggestive of an asymmetry in cortical arousal. But this finding was tapered by an increased amplitude pattern for the right hemisphere in all groups. The pattern of results presented in this study is consistent with earlier proposals, discussed above, for a difference between the PTSD group and the TEC group based on the age at which the trauma event was experienced. However, this question was not addressed by the current study.

Furthermore, resting EEG characteristics for the TEC group demonstrated a consistent difference for higher amplitudes in all frequencies compared to the NC group and the PTSD group. In the beta frequency, a consistently higher left hemisphere activation pattern was demonstrated for the TEC group compared to the NC group and the PTSD group, particularly for the eyes closed condition. This finding may be linked to models that propose the transitory maintenance of long-range cortical connections (Singer, 1999; Glassman, 1999). Therefore, supported by models proposing response grouping criteria, based on the precise timing between presynaptic and postsynaptic responses in distributed systems (Singer, 1999), it can be speculatively argued that the TEC group may represent a group that are resilient to the effects of extreme traumatic stress, and that compensatory mechanisms in this group may not be associated with high risk for PTSD, but with the maintenance of greater mental effort.

The finding of qualitative and quantitative differences between the PTSD group and the TEC group, particularly for alpha spectral frequency power, indicates that the functional characteristics of information processing differ, and, as discussed previously, may implicate
the ventral and dorsal streams of the prefrontal cortex for the active retrieval of information and interaction effects with the maintenance and monitoring of information. As discussed earlier, the implementation of automatic retrieval processes have been associated with precise functional circuitry associated with left hemisphere dominance (Boyer et al., 1998) and linked to the parietal cortex rather than the prefrontal cortex (Landau et al., 2004). This hypothesis is in agreement with models suggesting the mediation of these regions by attention mechanisms and reciprocal limbic connections (Jatzko et al., 2006; Bryant et al., 2005).

6.5.5.1. Alpha Frequency Patterns in Resting EEG

The pattern of resting frequency oscillations indicated significant differences between groups for amplitude and for asymmetry in the alpha bandwidth. Differences in frontal alpha are thought to represent emotional expressions (Blackhart et al., 2006; Bruder, 2004; Coan and Allen, 2004). Furthermore, the higher amplitudes, particularly for the alpha frequency, are thought to indicate a pattern of higher responsiveness to external stimuli (Basar, 2004; Klimesch, 1999). The consistently higher amplitudes for all frequencies in the TEC group, in this study, can be interpreted as being in agreement with the latter proposal, suggesting a different pattern for preparatory responses compared to those of the NC group and the PTSD group.

Differences in frontal EEG alpha power between the PTSD group and the TEC group were interpreted to mean emotional processing alterations and possibly an altered pattern of functional processing in cognitive performance tasks. Accordingly, differences in alpha amplitudes between the PTSD group and the TEC group in posterior regions were taken to indicate a unique pattern for neural activation in people with PTSD, possibly associated with affective attentional resources and transitory shifts in the integration of information in frontal-striatal circuitry, implicating alterations in ACC pathways. This interpretation suggests that compensatory mechanisms in the TEC group are associated with LTP alterations in neural circuitry and more stable patterns of functional implementation (McFarlane et al., 2002).

6.5.5.2. Rhythmic Oscillations in Resting EEG

Based on hypotheses that higher alpha power indicates a more strongly synchronised neural activity (Klimesch, 1999), and greater attentional scanning in preparation for external stimuli (Aston-Jones et al., 1999; Knyazev et al., 2005), it is possible that higher amplitudes represent a functional threshold for attentional control and self-regulation in the TEC group. In contrast, alterations toward lower frequency patterns in the PTSD group might indicate a disruption to
thresholds in attentional control that may represent a shift in neural circuitry for automatic retrieval of information. As previously discussed, disruptions in attention involving ventral frontal circuits may be associated with a shared function in spatial working memory rehearsal systems, in particular, the sequencing of response retrieval (Jatzko et al., 2006; Eichenbaum and Fortin, 2005). If this conclusion were correct, it would suggest a role for theta in functional connectivity patterns and disruptions to theta synchrony, specific to PTSD, for the encoding of new stimuli based on perceptual cues (Ward, 2003). This proposal is consistent with discussions following results presented in the previous chapter.

Thus, baseline resting EEG states may be a useful index for understanding the readiness of mental states (Basar, 2004), and particularly for interactions between high and low frequencies within the alpha rhythm (Cooper et al., 2003; Hanslmayr et al., 2005; Klimesch et al., 2005). The consistently lower amplitudes for the alpha bandwidth in the PTSD group, in the current study, represents a band-specific alteration and, as previously discussed, can be interpreted as an environmental influence, associated with altering a stable underlying genetic trait (Zietsch et al., 2007), consistent with a selective attention bias hypothesis (McFarlane et al., 1993; Bryant et al., 2005). Relevant to this argument, the consistently higher alpha amplitudes in the TEC group might also be interpreted as an environmental influence altering the stable EEG background state. On the other hand, this argument for the TEC group might not be tenable because amplitudes for all frequencies measured in the current analysis were consistently high in the TEC group, with less variation compared to those of the PTSD group.

In contrast, a different argument can be mounted by taking into consideration the interaction of psychological and physiological indices associated with behavioural performance. As proposed in earlier discussions, frequency signals may be altered by various factors in brain-behaviour models (McFarlane et al., 2002), including the shape of the neurons (Watson et al., 2006), the strength of the signal (Singer, 1999; Sejnowski and Tesauro, 1989), previous learning associated with predictions and appraisals (Dayan et al., 2000; Landau et al., 2004), and psychological factors associated with motor activity (Chemtob and Taylor, 2003; Teicher et al., 2003; Shelton and Gabrieli, 2004). Interactions have been theoretically demonstrated in models of response bias (Vasterling et al., 2004), where recognition thresholds and connotative meaning for perceived stimuli is influenced by emotional significance and may alter the statistical probability of response bias and selective attention mechanisms (Williams et al., 2006; Dayan and Yu, 2003).
Together with the profile of behavioural and electrocortical patterns presented in Chapter 5 of this study, the relationship between resting preparatory activation states and cognitive tasks may be in agreement with models that suggest the involvement of the hippocampus in the disruption of long-range neural signals, implicating the complex interaction between physiological and psychological processes in people with PTSD (Bremner et al., 1999). One hypothesis for the involvement of this region may be related to mechanisms of grouping patterns across task performance to identify the behavioural context required to integrate information (Singer, 1999; Seger and Cincotta, 2006) Another hypothesis is related to asymmetry for specialised processing and the reciprocal links between the PFC and hippocampal structures during retrieval of episodic memories (Hull, 2002; Nadel and Moscovitch, 1998).

However, the problem is one of assessing the role of each of the determinants in accounting for the lower or higher thresholds of alpha amplitudes. In the current analysis, the approach of using broad band frequency analyses for resting electrophysiological brain activity may not be appropriate for observing the frequency-specific effects required to elucidate the role of alpha and theta wave bands (Klimesch, 1999) in attention and memory processes. Thus, the role of the lower frequency amplitudes in the PTSD group and the higher frequency amplitudes in the TEC group remain to be clarified. In addition, the involvement of different mechanisms for working memory function in the TEC group cannot be interpreted using resting activation patterns and requires further investigation.

As previously stated, tentative conclusions for the higher amplitudes in resting states in the TEC group were interpreted as an alteration to the local or structural level of cognitive architecture. This change in knowledge structures might be associated with language rehearsal mechanisms and the articulatory loop of working memory systems. On the other hand, the argument supporting a disruption to structural or local level processing in PTSD participants was consistent with an altered mechanism in memory processing of spatial experiences associated with the activation of posterior and sensory-related brain regions. It was suggested, earlier, that a disruption to the sequencing of events, and also to the temporal organisation of events, might disrupt the flexibility of retrieval from long-term memory. Both arguments may be consistent with alterations to resting background oscillatory activity in the PTSD group and the TEC group and to the dysregulation of motor movement control. Thus, the current findings can also be interpreted as a disruption to the coordination of long-range neural signals, within the framework of a neural synchrony hypothesis (McFarlane et al., 2002; Singer, 1999).
Conclusions:

A large number of factors were identified, and discussed in this chapter, as plausible mechanisms contributing to altered psychological and physiological patterns of functioning, in PTSD participants as well as control participants reporting trauma exposure and reactivity to traumatic stressor events. However, there was a consistent trend in the data to conclude that there are common and plausible avenues for further investigation. These conclusions are related to how compensatory mechanisms in two groups with self-reported experiences of trauma reactivity might explain behavioural performance, particularly performance related to cognitive tasks involving verbal and nonverbal working memory processing.

This is one of the first studies in PTSD to explore the role of retrieval functions, associated with working memory processes of verbal and nonverbal information, and the correlates of spatial and temporal parameters in spontaneous neural oscillations. The results of this analysis suggest a disruption to the integration of information in memory systems at the encoding (or preparatory) stage that may be linked to two mechanisms. The data in this study have demonstrated asymmetry of tonic resting rhythmic patterns and the timing mechanisms associated with long-range coordination of information flow as plausible mechanisms for further considerations in understanding the loss of behavioural flexibility in people with strong reactivity to previous stressor events. Furthermore, the results are in agreement with previous research (Clark et al., 2003; Vasterling et al., 2006) and extend the findings to include community groups, with and without, previous trauma experiences. The data indicate a strong trend toward common mechanisms that include, firstly, posterior region abnormalities in frequency patterns associated with perception, and secondly, selective attention abnormalities associated with frontal regions, executive functions, and the speed of processing.

However, the role of altered perceptions in people with PTSD, who reported comorbid depression and anxiety, were not investigated. As discussed above, the interaction of mood with behavioural performance indices has been provided as evidence of hemispheric asymmetry in brain organisation patterns in the clinical literature (Hugdahl and Davidson, 2004) and has been a recent topic of investigation in the PTSD literature (Metzger et al., 2004).

Therefore, the results presented in this study indicate a trend toward a third possible conclusion. That is, altered perceptions in people with PTSD might stem from an interaction
effect in hemispheric asymmetry patterns, implicating comorbid depression, consistent with a role for secondary changes in working memory systems and alterations to the pattern of neural structure through synaptic plasticity (McFarlane et al., 2002). Extending this hypothesis, the possible disruption to the coordinated flow of information processing in people with PTSD is in agreement with previous findings of a disruption to long-range connectivity patterns (Shaw et al., 2002) and the timing of retrieval processes (Vasterling et al., 2006). Thus, a timing mechanism, associated with lower frequency patterns, may have particular relevance to the numbing characteristics of PTSD symptoms, as a unique pattern of functioning, that remains to be clarified (Kashdan et al., 2006; Metzger et al., 2004).
7. Neuropsychological and Electrophysiological Asymmetry Patterns in PTSD

7.1. Rationale

To extend the previous analysis, an investigation of reactions to stress is required for a clearer understanding of whether there is a common underlying mechanism associated with adaptive behaviour after perceived traumatic stressor events, and whether this can be inferred from a pattern of relationships between variables. The purpose of this chapter is to examine patterns of cortical asymmetry, cognitive performance, and the related symptom category responses of numbing and avoidance.

As previously reviewed, local networks have been defined as representations of simple sensory and motor features, while the contents of memory are complex associative networks related to self-organising probabilistic functions such as acquisition, storage, and retrieval based on selective attention, arousal or alertness (Fuster, 1997). Selective attention assumes a categorisation consistent with serial processing (Boyer et al., 1998; Ungerleider et al., 1998). However, stress dynamically alters the automatic retrieval of memories (Gold, 2005). In PTSD, symptoms are an adaptive strategy to reduce stress with avoidance behaviours or numbing responses the most commonly adopted strategies (Nemeroff et al., 2006; Yehuda, 2004; Ehlers and Clark, 2000). These behaviours have been shown to involve physiological and psychological disturbances in functioning, including irritability, impaired concentration, and poor memory performance (McFarlane et al., 2002). Therefore, to understand how PTSD symptoms are associated with altered cognitive functioning, through neural plasticity, or whether tonic characteristics are associated with a higher pattern of reactivity in PTSD participants, the self-reported indices of reactivity to traumatic exposure were examined for differences and similarities between groups in valance and cortical arousal.

Based on previous findings (Metzger et al., 2004), it was expected that stress and anxiety reaction symptoms would be associated with energy regulation and attention modulation at a local level of analysis, to indicate distinct neural pathways for information processing.

Therefore, three related analyses were conducted. The first study focused on specialised working memory processes for verbal and nonverbal tasks and the respective differences specific to anterior and posterior cortical regions and attention modulation, as indexed by frequency amplitude patterns in the alpha frequency for the eyes closed condition. The second study investigated hemispheric asymmetry indices of anterior and posterior regional patterns and their associations with behavioural performance measures correlated with mood and
specific numbing and avoidance reactions. From a pattern of simple correlations, the third study investigated the variance between the residuals as predictor variables associated with tonic resting states of cortical asymmetry, valence, and behavioural performance between groups.

However, this study does not examine the theoretical underpinnings of emotion regulation and the structural theory of emotions. The existing neuro-physiological research on activation and arousal has not been reviewed within the context of this study. The focus of this analysis is based on the relative contribution of different cognitive and emotional variables in an assessment of asymmetry in cortical tonic activation patterns at the local level of information processing.

7.2. **Study 1: Behavioural Tasks and Local Processing Networks**

7.2.1. **Introduction**

In PTSD, distinct circuits have been mapped for cortical arousal associated with affective states (Metzger et al., 2004). As right hemisphere processes, the functional role of lateralisation may be associated with self-registration rather than self-regulation (Schore, 2002; Litz et al., 2000; Lanius et al., 2002, 2005; Niebauer, 2004), and the dynamic alterations to neuronal pathways during automatic retrieval (Chemtob and Taylor, 2003; Saltzman et al., 2006; Vasterling et al., 2004). In the wider literature it has been shown that imagery associated with attentional scanning facilitates memory retrieval and is associated with reduced error rates (Fletcher et al., 1995; Ghatan et al., 1995). Conversely, it has been proposed in the PTSD literature that verbal working memory disturbances are associated with a right-hemisphere coding strategy (Clark et al., 2003). This raises the question of what specific mechanisms, associated with right hemisphere processing, facilitate or disrupt cognitive flexibility. A right hemisphere temporo-lateral PFC pathway has been implicated by the current pattern of results, and associated with an uncertainty hypothesis in people with PTSD.

At a mechanistic level of analysis, the relationship between alpha oscillation patterns and the associated functional processes of perception, attention, and semantic memory has not been fully clarified (Veltmeyer et al., 2006; Hanslmayr et al., 2005; Sauseng et al., 2005). However, various researchers have provided evidence to show differences in behavioural performance associated with alpha power based on frontal and regional activation patterns (Veltmeyer et al., 2006; Metzger et al., 2004; Bruder et al., 1997). One suggestion is that the
possible influences of alertness and emotional factors have a non-selective role in attention and contribute to a functional disturbance in memory processing (Williams et al., 2006; Bryant et al., 2005; Jatzko et al., 2006). This is consistent with a lateralisation hypothesis and complex interactions between specific spatial regions that have been mapped as altered neural circuits in PTSD (Nemeroff et al., 2006).

The aim of this first analysis was to investigate differences between groups using behavioural performance indices for an assessment of lateralised local processing, as well as differences in resting cortical patterns in the alpha frequency between frontal and posterior regions. Of interest was whether there was an efficiency difference in task performance for left or right hemisphere processing. It was hypothesised that, if selective attention mechanisms are associated with lower alpha amplitudes in people with PTSD, implicating a disruption to the rehearsal of content (manipulated) information in working memory systems, then the higher amplitudes in the alpha frequency for the TEC group would predict higher accuracy scores for the verbal fluency task in this group compared to the PTSD group and the NC group. From the pattern of cortical resting patterns in the previous chapter, it was expected that there would be hemispheric lateralisation differences between groups for resting alpha power.

On the other hand, in the previous analysis, the pattern of resting alpha frequency indicated a higher activation pattern in right hemisphere posterior regions for all three groups. Therefore, if anxiety reaction patterns are associated with higher right hemisphere posterior activation patterns, then it was also expected that an increased alert state would be predictive of an improvement in behavioural performance for visual tasks, but not for accuracy scores if those tasks required manipulation of information in frontal networks. Based on the current pattern of results so far, and previous PTSD evidence, it was expected that visual perception would be high in the PTSD group but efficiency associated with accuracy scores would be low.

7.2.2. Method

7.2.2.1. Participants

The current analysis extends the findings from Chapter 6 and accordingly includes only those participants meeting the added criteria of right-handedness and the control group stratified according to reactivity to traumatic stressor events. Participant data has been described in the previous chapter and the demographic characteristics of participants have been presented in Table 6.1 for (N=33) PTSD participants, (N=32) TEC participants, and (N=88) NC participants.
7.2.3. Instruments and Procedures

7.2.3.1. Modality-specific Cognitive Tasks

**Verbal Fluency:**

The „IntegNeuro” battery included, as a test from the language domain, the Letter fluency sub-test and the Animal fluency sub-test; reported in detail in Chapter 4. The Word Generation task was used in the current study as a measure of the ability to execute the retrieval of information from knowledge structures.

**Maze Task:**

The Maze task was included in the „IntegNeuro” battery as an executive function task. The task is described in Chapter 4. For the purposes of the present study, time taken to complete this task was used to index speed of processing. However, as an index of executive function, and specialised nonverbal working memory right hemisphere processing, the maintenance and manipulation of information (accuracy scores and overruns) were taken as an index of selective attention and efficiency.

7.2.3.2. Resting Electrophysiological Measures

Resting background EEG data were collected for the eyes closed condition according to procedures detailed in Chapter 4.

A Fast Fourier Transformation was performed, as described in Chapter 4, to obtain estimates of absolute spectral power. Data from the alpha band of frequencies (8-13 Hz) were utilised. To reduce the EEG data for statistical analysis, laterality was examined by using leads based on regions of interest for inter-hemispheric and intra-hemispheric derivations. Electrode leads corresponded to anterior left hemisphere (Fp1, F3, F7), anterior right hemisphere (Fp2, F4, F8), posterior left hemisphere (P3, T3, T5), and posterior right hemisphere (P4, T4, T6) sites.

7.2.4. Data Analysis

All analyses were computed using SPSS (version 14) and results have been presented as absolute numbers, means and standard deviations or percentages. Significance levels for all analyses were set at the p = 0.05 alpha level (two-tailed). Bonferroni correction methods were used for post hoc analyses to correct for multiple testing.
Behavioural results were analysed by comparing means for each of the sub-tests as separate analyses using Univariate ANOVA or independent samples t-test.

Regions of interest for EEG data were compared as differences in log amplitude power. For the anterior analysis, a repeated measures ANOVA was conducted using Hemisphere (left, right) by Site (Fp1, F3, F7; Fp2, F4, F8) as the within-subjects factors and Group (PTSD: N=27; TEC: N=31; NC: N=83) as the between-subjects factor. A repeated measures ANOVA was conducted for the posterior regions using Hemisphere (left, right) by Site (P3, T3, T5; P4, T4, T6) as the within-subjects factors and Group (PTSD: N=32; TEC: N=31; NC: N=83) as the between-subjects factor. Post hoc analyses were conducted using one-way ANOVAs to investigate significant interaction effects. Bonferroni adjustments and Greenhouse-Geisser epsilon corrections were used where appropriate.

7.2.5. Results

7.2.5.1. Verbal Performance Measures

A one-way ANOVA revealed that there were no significant differences between groups for the production and recall of animal words \([F(2,134) = 1.62, p = 0.20]\). There were no significant differences between groups in oral production and recall of FAS words \([F(2,134) = 2.98, p = 0.054]\).

Multiple comparison tests for the FAS task indicated a significant difference between the PTSD group and the TEC group (Bonferroni tests indicating a mean difference of -2.76 fewer words \((SE = 1.13, p = 0.05)\) but not between the PTSD group and the NC group (Bonferroni: mean difference = -1.40, \(p = 0.29\)). There were fewer words produced by the NC group compared to the TEC group, but this was not significantly different (Bonferroni: mean difference = -1.36, \(p = 0.46\)). As shown by the descriptive data in Table 7.1, for both categories of words, the PTSD group produced fewer words than the two control subgroups.
### Table 7.1. Verbal fluency descriptive data

<table>
<thead>
<tr>
<th>Measure</th>
<th>FAS</th>
<th>Animal</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTSD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>M</td>
<td>14.43</td>
<td>22.23</td>
</tr>
<tr>
<td>sd</td>
<td>4.26</td>
<td>5.19</td>
</tr>
<tr>
<td>N</td>
<td>29</td>
<td>29</td>
</tr>
<tr>
<td>TEC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>17.20</td>
<td>24.66</td>
</tr>
<tr>
<td>sd</td>
<td>3.42</td>
<td>5.23</td>
</tr>
<tr>
<td>N</td>
<td>78</td>
<td>78</td>
</tr>
<tr>
<td>NC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>15.84</td>
<td>23.54</td>
</tr>
<tr>
<td>sd</td>
<td>4.67</td>
<td>5.16</td>
</tr>
</tbody>
</table>

#### 7.2.5.2. Nonverbal Performance Measures

Table 7.2 shows minimal differences between group means for each of the subtasks in the maze paradigm. There were no significant differences between groups on any of the subtasks. The only task in which differences almost reached statistical significance was time to complete the maze \([F(2,143) = 3.02, p = 0.052]\).

Bonferroni post hoc tests revealed that this difference was between the two control groups, the NC group time was the slowest compared to the TEC group, but could not be confirmed statistically. The PTSD group time to complete the maze was mid-way between the two control groups. There were no significant differences between groups for accuracy scores based on average number of errors \((p = 0.09)\) and average number of overruns \((p = 0.33)\).

### Table 7.2. Executive maze task descriptive data

<table>
<thead>
<tr>
<th>Measure</th>
<th>MCT</th>
<th>MAv E</th>
<th>MAv O</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTSD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>30</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>M</td>
<td>4.34</td>
<td>4.59</td>
<td>1.94</td>
</tr>
<tr>
<td>sd</td>
<td>1.31</td>
<td>2.50</td>
<td>0.78</td>
</tr>
<tr>
<td>N</td>
<td>31</td>
<td>31</td>
<td>31</td>
</tr>
<tr>
<td>TEC</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>3.98</td>
<td>4.80</td>
<td>2.12</td>
</tr>
<tr>
<td>sd</td>
<td>1.09</td>
<td>1.89</td>
<td>0.92</td>
</tr>
<tr>
<td>N</td>
<td>85</td>
<td>84</td>
<td>84</td>
</tr>
<tr>
<td>NC</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>4.65</td>
<td>4.08</td>
<td>1.88</td>
</tr>
<tr>
<td>sd</td>
<td>1.42</td>
<td>1.27</td>
<td>0.67</td>
</tr>
</tbody>
</table>

*Note: MCT = maze time to completion; MAvE = maze number of errors; MAvO = maze number of overruns*
7.2.5.3. Region-specific Spectral Alpha Amplitude Activity

Alpha Power - Anterior regions:

The ANOVA results, as revealed in Table 7.3, show a significant interaction of Hemisphere by Site by Group \([F(GG: 3,226) = 3.14, p = 0.02]\). A main effect for Site \([F(GG: 2,264) = 437.36, p < 0.001]\) revealed that each site was oscillating at a different amplitude. A main effect for Group \([F(2,138) = 5.15, p < 0.01]\) revealed differences in alpha amplitude between groups.

### Table 7.3. Results for ANOVA assessing anterior eyes closed alpha power hemisphere and electrode site effects between groups

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main Effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisphere</td>
<td>1,138 (G-G)</td>
<td>0.23</td>
</tr>
<tr>
<td>Site</td>
<td>2,264 (G-G)</td>
<td>437.36**</td>
</tr>
<tr>
<td>Group</td>
<td>2,138</td>
<td>5.15**</td>
</tr>
<tr>
<td><strong>Interaction Effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisphere x Site</td>
<td>2,226 (G-G)</td>
<td>1.28</td>
</tr>
<tr>
<td>Site x Group</td>
<td>4,264 (G-G)</td>
<td>0.57</td>
</tr>
<tr>
<td>Hemisphere x Group</td>
<td>2,138 (G-G)</td>
<td>2.91</td>
</tr>
<tr>
<td>Hemisphere x Site x Group</td>
<td>3,226 (G-G)</td>
<td>3.14*</td>
</tr>
</tbody>
</table>

*Note: *\(p < 0.05\); **\(p < 0.01\)

*Post hoc analysis: Anterior Alpha Frequency Hemisphere by Site by Group Interaction*

Post hoc analyses were conducted separately for the left and right hemispheres using one-way ANOVA to explore the simple effects for the interaction of Hemisphere by Site by Group.

For the left hemisphere there were significant differences in alpha amplitudes between groups for the three sites measured; Fp1 \([F(2,146) = 4.91, p < 0.01]\), F3 \([F(2,147) = 5.69, p < 0.01]\), and F7 \([F(2,142) = 5.25, p < 0.01]\).

Bonferroni post hoc comparisons (at the \(p = 0.025\) level; 0.5/2 to adjust for multiple tests) revealed significant differences for lower alpha amplitudes at Fp1 in the NC group compared to the TEC group (MD = -.49, SE = .16, \(p = 0.01\)). Differences between the PTSD group and the TEC group at Fp1 were not significant, \(p > 0.03\). There were significant differences for lower alpha amplitudes at the midfrontal left hemisphere F3 site between the PTSD group and
the TEC group (MD = -.60, SE = .20, p < 0.01), and lower amplitudes in the NC group compared to the TEC group (MD = -.50, SE = .17, p < 0.01). However, at the F7 site, Bonferroni post hoc comparisons revealed that the only differences were between the TEC group and the NC group (MD = -.54, SE = .17, p < 0.01), indicating an increase in alpha amplitudes for the PTSD group.

For the right hemisphere, there were significant differences in alpha amplitudes between groups at each of the three leads measured; Fp2 [F(2,147) = 4.74, p = 0.01], F4 [F(2,148) = 4.06, p = 0.02], F8 [F(2,147) = 4.09, p = 0.02], for consistently lower amplitudes in the PTSD group and the NC group compared to the TEC group.

Post hoc Bonferroni corrections indicated a significant difference for lower amplitudes at the Fp2 site between the PTSD group and the TEC group (MD = -.55, SE = .20, p = 0.02) and between the TEC group and the NC group (MD = -.46, SE = .17, p = 0.02). However, Bonferroni corrections at the p = 0.025 level were not met at the F4 site or the F8 site for between group differences in the right hemisphere.

**Alpha Power - Posterior regions:**

At posterior regions, the only significant interaction effect was for Hemisphere by Site [F(GG: 1,193) = 18.13, p = < 0.001]. Table 7.4 shows the main effects for Hemisphere [F(G-G: 1,143) = 24.21, p = < 0.001], Site [F(GG: 1,214) = 319.88, p = < 0.001], and Group [F(2,143) = 4.62, p = 0.01].

Bonferroni post hoc multiple comparisons revealed a difference between the PTSD group and the TEC group (MD = -.59, SE = .22, p = 0.02) and between the TEC group and the NC group (MD = -.50, SE = .18, p = 0.02) in alpha power at posterior regions, for higher alpha amplitudes in right hemisphere regions compared to left hemisphere regions.
Table 7.4. Results for ANOVA assessing posterior eyes closed alpha power hemisphere and electrode site effects between groups

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hemisphere</td>
<td>df</td>
</tr>
<tr>
<td></td>
<td>1,143 (G-G)</td>
<td>24.21**</td>
</tr>
<tr>
<td></td>
<td>Site</td>
<td>1,214 (G-G)</td>
</tr>
<tr>
<td></td>
<td>Group</td>
<td>2,143</td>
</tr>
<tr>
<td></td>
<td>Interaction Effect</td>
<td>df</td>
</tr>
<tr>
<td>Hemisphere x Site</td>
<td>1,193 (G-G)</td>
<td>18.13**</td>
</tr>
<tr>
<td>Hemisphere x Group</td>
<td>2,143 (G-G)</td>
<td>0.82</td>
</tr>
<tr>
<td>Site x Group</td>
<td>3,214 (G-G)</td>
<td>1.21</td>
</tr>
<tr>
<td>Hemisphere x Site x Group</td>
<td>3,193 (G-G)</td>
<td>0.68</td>
</tr>
</tbody>
</table>

Note: **p ≤ 0.01

Post hoc analysis: Posterior Alpha Frequency Hemisphere by Site Interaction

To explore differences between groups for each site a post hoc analysis was conducted using separate one-way ANOVAs for left and right hemisphere regions. At left hemisphere leads, there were significant differences between groups for all sites: P3 [F(2,148) = 4.26, p = 0.02], T3 [F(2,145) = 5.06, p < 0.01], T5 [F(2,147) = 4.56, p = 0.01].

Bonferroni comparisons for significance at the p = 0.025 level revealed that differences were for lower amplitudes in the NC group compared to the TEC group (MD = -.54, SE = .20, p = 0.02). At the T3 site an interaction effect was revealed for lower amplitudes between the PTSD group and the TEC group (MD = -.52, SE = .19, p = 0.02) and between the NC group and the TEC group (MD = -.46, SE = .16, p = 0.01). Similarly at the T5 site an interaction effect for lower amplitudes were indicated between the PTSD group and the TEC group (MD = -.70, SE = .26, p = 0.02) and for lower amplitudes in the NC group compared to the TEC group (MD = .60, SE = .22, p = 0.02).

At right hemisphere leads, the T4 site did not reach statistical significance for a difference between groups [F(2,147) = 2.90, p < 0.06], indicating slightly lower alpha amplitudes in the TEC group for this site. There were statistically significant differences at the P4 site [F(2,148) = 3.65, p = 0.03] and at the T6 site [F(2,147) = 4.22, p = 0.02] between groups for differences in alpha amplitude.

Bonferroni comparisons for significance at the p = 0.025 level (0.05/4) indicated that differences in amplitudes at the P4 site between the PTSD group and the TEC group failed to
reach statistical significance after correction for multiple testing \((p > 0.03)\). An interaction effect at the T6 site was significant between the PTSD group and the TEC group \((\text{MD} = -0.74, \text{SE} = 0.28, p = 0.03)\), but also failed to reach statistical significance between the NC group and the TEC group after correction \((p > 0.03)\).

7.2.5.4 Summary: Cognitive Performance and Alpha Power

Hypotheses in relation to differences between groups in cognitive performance were only partly supported by the current data. As expected, and as a replication of the results presented in Chapter 5, there were no significant differences between groups in the subtask animal words. Unexpectedly, there were no significant differences between groups in the subtask FAS. There was, however, a strong trend toward differences between groups, FAS word production just reaching statistical significance, when comparing the PTSD participants with TEC participants. As predicted, differences in the TEC group were for higher accuracy scores on both verbal subtests, while the PTSD group scored the lowest. This finding may be in partial support of predictions that higher alpha power is associated with higher accuracy scores.

On the other hand, predictions for efficiency and speed of processing associated with a nonverbal task were not supported by the data. The results indicated a trend for the speed of information processing, associated with the encoding and subsequent retrieval of visual information, to be faster for the TEC group compared to the NC group, but scores associated with accuracy and overruns, examining the maintenance and updating of information in the maze paradigm, did not reach statistical significance between groups. As predicted, therefore, this finding may imply differences between groups associated with higher right hemisphere posterior resting cortical activation patterns in specific regions, as suggested from the pattern of results in the previous chapter.

Differences between groups, in resting activation patterns for the alpha frequency, indicated left and right hemisphere amplitude shifts for frontal and posterior regions. The highest amplitude patterns were for the TEC group at all leads measured, in both anterior and posterior regions. All three groups indicated a higher pattern of alpha amplitudes in the right hemisphere compared to the left hemisphere.

Significant differences in alpha amplitudes between the two control groups (TEC and NC) were found at all left hemisphere frontal derivations but only at a specific frontal lead in the right hemisphere, indicating inferior frontal cortex regions. The lowest alpha amplitude means
were for the PTSD group compared to the TEC group at specific right hemisphere and left hemisphere frontal leads, indicating inferior frontal cortex, but also for a lead mapping onto left hemisphere mid-frontal sites. There was a significantly different pattern of higher amplitudes in the TEC group at right hemisphere leads compared to the PTSD group.

The results indicated a specific pattern of differences in right hemisphere posterior regions but only between TEC participants compared to PTSD participants. There were no significant differences between the two control groups in right hemisphere posterior derivations.

7.2.6. Discussion

The hypothesis of differences in behavioural performance, and theoretical notions of imagery associated with cognitive flexibility, was not supported by the data. However, there were indications in the verbal oral fluency task that there may be differences in the way imagery is used in neural architecture, particularly for categories of familiar words compared to non-category recall for letter fluency words. This finding was interpreted in the context of alpha activation differences between groups to suggest alterations to left-hemisphere processing during cognitive tasks and the possible interruption to rehearsal or maintenance mechanisms.

However, the lack of significant differences between groups, associated with the maze task for right hemisphere nonverbal working memory processing, were interpreted within the context of a pattern of increased activity for all three groups in resting cortical activation patterns in right hemisphere posterior regions. Higher amplitudes for all three groups in right hemisphere regions have been a consistent finding in this study. Therefore, it is inferred that coding for nonverbal working memory patterns has not altered between groups. This conclusion suggests that other factors associated with right hemisphere posterior cortical arousal, or a pattern of specific interactions with left hemisphere regions, are involved in decision processes, which influence behavioural performance or the transformation of input signals to motor output.

The hypothesis of lateralised local resting activity in people with PTSD was supported by the data. Significant differences between groups in alpha power indicated a specific left hemisphere mid-frontal alteration in the PTSD group compared to the TEC group and the NC group. This finding is in agreement with previous research suggesting cortical and subcortical differences in people with PTSD (Clark et al., 2003; Vasterling et al., 1998), and with possible alterations to hippocampal functioning (Bremner et al., 1999). The left hemisphere difference in cortical oscillation patterns in the resting state in people with PTSD can
interpreted as a disruption to the local processing of information, for encoding and updating, involving executive processes. Therefore, in people with PTSD, a pattern of selective attention bias can be speculatively associated with hippocampal temporal coding for endogenous stimuli rather than imagery associated with sensory stimuli. This interpretation is in agreement with models proposing that selective attention can be interrupted by spatial attention (Jatzko et al., 2006) and that stress dynamically alters the automatic retrieval of memories (Aston-Jones et al., 2005; Fan and Posner, 2004; Fox et al., 2001; Knyazev et al., 2005, 2006a).

In addition, the data for a different pattern of posterior region resting activation states was unexpected, but specific for a lateralised difference between the PTSD participants and the TEC participants. The consistent pattern of high amplitudes in people with previous trauma reactivity (TEC group) indicates alterations to background rhythmic activity in this group of people. In addition, the specific pattern of differences at temporal right hemisphere regions suggests alterations at the local level of cognitive architecture and a different pattern of network associations compared to the PTSD group and the control group. Whether this pattern of increased right hemisphere posterior alpha power in the TEC participants is functional for a predisposition to psychopathology, or, is associated with resilience, warrants further investigation. However, the interaction between right hemisphere activation patterns and left hemisphere activation patterns suggests alterations to the pattern of information processing between the three groups, with specific regions indicating convergent zones and shared neurobiological substrates for the integration of information.

As mentioned above, the finding of non-significant differences in behavioural tasks between groups may result not from higher or lower amplitudes in resting cortical arousal, but from the pattern of lateralised changes in alpha power in both anterior and posterior regions. Results for this analysis, and the previous analyses in this study, are consistent with a selective attention bias in local processing, with indications that the PTSD participants have reduced attentional scanning capacity in resting preparatory states associated with region-specific asymmetry patterns of rhythmic oscillations. Based on theory and evidence that the cortex is involved in the integration of exogenous and endogenous information (Aston-Jones and Cohen, 2005; Spitzer et al., 2004), the current pattern of results implicate both afferent (input) and efferent (output) pathways are altered for component information processing in people with PTSD. A plausible mechanism, at the local level of information processing, suggests an alteration in lateralisation patterns and dissociation between components of dorsal and ventral cortical processing streams in early and late stages of verbal and nonverbal task processing.
However, it is also plausible that phasic changes, or state changes, in people with PTSD are associated with different symptoms that maintain high activation patterns in right hemisphere posterior regions. In distributed systems, the dynamic automatic retrieval of memories might depend on context information that may be more endogenously derived, leading to selective attention bias and interference with components of verbal and nonverbal rehearsal mechanisms. This interpretation of the current data draws on recent models and evidence from a variety of sources (Munk, 2001; Schwartz et al., 2005; Herrmann, 2003) and from neuroimaging data in PTSD research (Jatzko et al., 2006). As suggested by Clark and colleagues (2003), pathways disrupted in people with PTSD may be associated with specific regions of the PFC, particularly the DLPFC for a disruption to the integration of information associated with verbal working memory systems, and the posterior cortex, for a dissociation of visual stimulus into dorsal (occipitoparietal) stream for spatial information and ventral (occipitotemporal) stream for object characteristics, during encoding information. Based on the results of the current data, it is possible that transitory states of visual information might interfere with attentional scanning during uncertainty, with the consequent interruption to serial processing sequences. The mechanisms for this hypothesis are consistent with different functions in frontal cortex neurons (Belger et al., 1998; Fuster et al., 2000). This view may be a useful framework within which to interpret the current pattern of results.

On the other hand, compensatory mechanisms associated with uncertainty during encoding processes have not been clarified in the PTSD literature and might be related to specific symptom categories and the way information is integrated in frontal regions through the various cross-modal and cross-temporal associations. Differences in emotional regulation and resting cortical arousal patterns might also be asymmetrically involved in behavioural performance patterns in people with reactivity to stress.

7.3. **Study 2: Cortical Asymmetry Patterns and Traumatic Stressor Events**

7.3.1. **Introduction**

Linked to the characteristic profiles of resting cortical arousal, derived from the previous analyses, it was of interest to understand whether emotional factors, associated with hemispheric alteration patterns in cortical arousal at the local level of information processing, were different or similar in the PTSD group compared to the TEC group. Therefore, a question, in part derived from the current results, focuses on the pattern of resting cortical activation in people with previous traumatic stressor exposure and reactivity to stress.
As a manipulation check, the purpose of this analysis was to examine patterns of cortical asymmetry and the related symptom category responses for numbing and avoidance in PTSD participants and in two control groups. However, based on previous findings, it was expected that the lateralisation of current mood states in a pattern of resting cortical arousal would be relevant to hypotheses of reduced attentional scanning in people with PTSD (Metzger et al., 2004; Vasterling et al., 2004). Therefore, it was predicted that the pattern of relationships for criteria, representing reactivity to traumatic stressor events, mood states, and alpha asymmetry, would be indicated by a characteristically different profile in the PTSD group compared to the two control groups.

7.3.2. Method

7.3.2.1. Participants

Participant data has been described in the previous chapter. However, because of missing data, a reduced number of PTSD (N=22: F=12, M=10) participants are represented in this analysis. The TEC group included all participants who responded to all questions of the CIDI with a full dataset (N=31: F=21, M=10). In addition, those in the NC group (N=51: F=25, M=26) included only participants who responded to the CIDI questionnaire for traumatic exposure and reactivity to that event. The details for each group are presented in Table 7.5.

<p>| Table 7.5. Characteristic demographic and mood data for a reduced number of participants |
|---------------------------------|-------|-------|-------|-------|</p>
<table>
<thead>
<tr>
<th>Measure</th>
<th>Age</th>
<th>Education</th>
<th>Depression</th>
<th>Anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTSD</td>
<td>N</td>
<td>22</td>
<td>22</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>40.49</td>
<td>13.27</td>
<td>11.36</td>
</tr>
<tr>
<td></td>
<td>sd</td>
<td>10.95</td>
<td>2.95</td>
<td>5.32</td>
</tr>
<tr>
<td>TEC</td>
<td>N</td>
<td>31</td>
<td>31</td>
<td>31</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>41.77</td>
<td>14.06</td>
<td>1.72</td>
</tr>
<tr>
<td></td>
<td>sd</td>
<td>8.75</td>
<td>2.86</td>
<td>2.85</td>
</tr>
<tr>
<td>NC</td>
<td>N</td>
<td>51</td>
<td>51</td>
<td>51</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>39.34</td>
<td>15.52</td>
<td>1.71</td>
</tr>
<tr>
<td></td>
<td>sd</td>
<td>11.47</td>
<td>2.26</td>
<td>2.43</td>
</tr>
</tbody>
</table>
7.3.3. Instruments and Procedures

7.3.3.1. Responses to CIDI Trauma Scales

Table 7.6 shows the CIDI responses for each category of symptoms in PTSD, as well as the numbing and avoidance items that are used in this analysis. Derived from the CIDI questionnaire, and from the pattern of results in Chapter 6, items of interest to the current analysis are responses to criterion C symptoms – numbing and avoidance. To examine specific reaction patterns for numbing and avoidance, individual items in criterion C, were summed to include the behavioural characteristics measuring emotional numbing, anhedonia and avoidance responses. The items measured by criterion C, that were consistent with emotional numbing, included disinterest in activities, detachment from others, and diminished positive affect, as items different from anhedonia. All other items in criterion C were included as avoidance characteristics, following item analysis as suggested by Kashdan and colleagues (2006), rather than the full criterion C total severity score.

7.3.3.2. Current Mood Scores

For the current analysis, Table 7.5 shows the pattern of mood scores (depression and anxiety), as measured using the DASS-21 (Lovibond and Lovibond, 1995). This scale has been described in Chapter 4.

7.3.3.3. Asymmetry Scores

An asymmetry score for the alpha frequency was used to investigate relationships between mood states and cognitive functioning. The asymmetry metric was computed using methods following Pivik et al (1993), as explained in Chapter 4, and provides a measure of the degree of left or right brain activation. Separate anterior and posterior analyses were computed for comparison by aggregating homologous leads in each region (anterior: Fp2-1, F4-3, F8-7; posterior: P4-3, T4-3, T6-5). Because alpha power has been taken as an index of the inverse of cortical activity (Allen and Kline, 2004), positive scores are indicative of greater alpha power at the right compared to the left aggregated electrode sites, which is assumed to reflect greater left-sided brain activation. Conversely, negative scores are indicative of greater right-sided brain activation.
Table 7.6. Self-report responses to CIDI detailing previous trauma experiences assessed by criterion A2 and reaction patterns

<table>
<thead>
<tr>
<th>Measure</th>
<th>Criterion - A2</th>
<th>Re-experiencing</th>
<th>Numbing</th>
<th>Avoidance</th>
<th>Hyperarousal</th>
<th>Total severity</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTSD</td>
<td>N 22</td>
<td>22</td>
<td>22</td>
<td>22</td>
<td>22</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>M 1.91</td>
<td>4.64</td>
<td>2.59</td>
<td>3.23</td>
<td>4.68</td>
<td>26.05</td>
</tr>
<tr>
<td></td>
<td>sd 0.29</td>
<td>0.58</td>
<td>0.80</td>
<td>0.87</td>
<td>0.72</td>
<td>3.63</td>
</tr>
<tr>
<td>TEC</td>
<td>N 32</td>
<td>32</td>
<td>31</td>
<td>31</td>
<td>31</td>
<td>32</td>
</tr>
<tr>
<td></td>
<td>M 2</td>
<td>2.69</td>
<td>1.16</td>
<td>1.48</td>
<td>2.45</td>
<td>15.53</td>
</tr>
<tr>
<td></td>
<td>sd 0</td>
<td>1.66</td>
<td>1.24</td>
<td>1.03</td>
<td>1.71</td>
<td>6.03</td>
</tr>
<tr>
<td>NC</td>
<td>N 31</td>
<td>51</td>
<td>51</td>
<td>51</td>
<td>51</td>
<td>51</td>
</tr>
<tr>
<td></td>
<td>M 0.61</td>
<td>1.08</td>
<td>0.45</td>
<td>0.29</td>
<td>0.92</td>
<td>8.37</td>
</tr>
<tr>
<td></td>
<td>sd 0.49</td>
<td>1.34</td>
<td>0.88</td>
<td>0.54</td>
<td>1.16</td>
<td>4.77</td>
</tr>
</tbody>
</table>

Note: Responses = Criterion A2 reactivity; Re-experiencing, Numbing, Avoidance, Hyperarousal, and Total Severity are items from the CIDI (World Health Organization, 1993) and closely related to PTSD criteria as per DSM-IV (American Psychiatric Association, 1994).
7.3.4. Data Analysis

Zero-order correlation analyses were used to examine the pattern of relationships between (1) anterior alpha asymmetry, (2) posterior alpha asymmetry, (3) numbing, (4) avoidance, (5) depression and (6) anxiety.

7.3.5. Results

Table 7.7 shows the characteristic profiles of each group and the relationships between alpha anterior and posterior asymmetry with avoidance, numbing, depression, and anxiety.

For the PTSD group there was a significant negative correlation between alpha anterior asymmetry and numbing ($r = -.49$, $p = 0.02$). There was also a significant positive relationship between alpha posterior asymmetry and anxiety ($r = .49$, $p = 0.03$). Table 7.7 shows that the depression items were positively and moderately correlated with all variables for mood (numbing, avoidance, anxiety) and Figures 7-1 and 7-3 indicate that differences in the PTSD group were for a relatively left-brain pattern of activation in anterior regions with anxiety and depression, but a relatively right-brain pattern of alpha activation in posterior regions with anxiety and depression (Figures 7-2 and 7-4).

There was a moderate positive correlation between alpha posterior asymmetry and avoidance in the TEC group ($r = .40$, $p = 0.03$). There were no other significant correlation patterns for this group. As depicted by the graphs (Figures 7-1, 7.3 and 7.4), a bilateral pattern of alpha asymmetry with mood states of anxiety and depression were indicated for depression, and for anterior regions with anxiety, but there was a shift toward a relatively left-brain pattern in posterior regions with anxiety (Figure 7-2).

The pattern of correlations for the NC group indicated a negative relationship between alpha anterior asymmetry and alpha posterior asymmetry ($r = -.33$, $p < 0.01$). There was also a strong positive correlation between numbing and avoidance ($r = .64$, $p < 0.01$) and between depression and anxiety ($r = .60$, $p < 0.01$). However, as previously discussed, results for this group may be spurious because of the heterogeneous nature of the group and any interpretation must be taken cautiously. Figure 7-4 suggests some change to the pattern of alpha asymmetry in posterior regions in the NC group, compared to the TEC group, associated with depression items that may be representative of the heterogeneity in this group.
Table 7.7. Correlation profile for each group between alpha anterior and posterior asymmetry, avoidance, numbing, and mood states

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PTSD</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Alpha anterior asymmetry</td>
<td>-</td>
<td>-.18</td>
<td>-.49(*)</td>
<td>-.35</td>
<td>-.33</td>
<td>-.33</td>
</tr>
<tr>
<td>2. Alpha posterior asymmetry</td>
<td>-</td>
<td>.09</td>
<td>.04</td>
<td>.27</td>
<td>.49(*)</td>
<td>.33</td>
</tr>
<tr>
<td>3. Numbing</td>
<td>-</td>
<td></td>
<td>.69(**)</td>
<td>.44(*)</td>
<td>.33</td>
<td></td>
</tr>
<tr>
<td>4. Avoidance</td>
<td>-</td>
<td></td>
<td>.49(*)</td>
<td>.57(**)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Depression scale</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Anxiety scale</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>T-E Control</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Alpha anterior asymmetry</td>
<td>-</td>
<td>.17</td>
<td>-.19</td>
<td>-.22</td>
<td>.04</td>
<td>.24</td>
</tr>
<tr>
<td>2. Alpha posterior asymmetry</td>
<td>-</td>
<td>.12</td>
<td>.40(*)</td>
<td>-.08</td>
<td>-.13</td>
<td></td>
</tr>
<tr>
<td>3. Numbing</td>
<td>-</td>
<td></td>
<td>.33</td>
<td>.12</td>
<td>.04</td>
<td></td>
</tr>
<tr>
<td>4. Avoidance</td>
<td>-</td>
<td></td>
<td></td>
<td>.00</td>
<td>-.12</td>
<td></td>
</tr>
<tr>
<td>5. Depression scale</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td>.37</td>
<td></td>
</tr>
<tr>
<td>6. Anxiety scale</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>N-Control</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Alpha anterior asymmetry</td>
<td>-</td>
<td>-.33(**)</td>
<td>-.21</td>
<td>.00</td>
<td>.23</td>
<td>.17</td>
</tr>
<tr>
<td>2. Alpha posterior asymmetry</td>
<td>-</td>
<td>.13</td>
<td>.06</td>
<td>-.08</td>
<td>-.04</td>
<td></td>
</tr>
<tr>
<td>3. Numbing</td>
<td>-</td>
<td></td>
<td>.64(**)</td>
<td>.12</td>
<td>.03</td>
<td></td>
</tr>
<tr>
<td>4. Avoidance</td>
<td>-</td>
<td></td>
<td></td>
<td>.19</td>
<td>-.09</td>
<td></td>
</tr>
<tr>
<td>5. Depression scale</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td>.60(**)</td>
<td></td>
</tr>
<tr>
<td>6. Anxiety scale</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note:  * Correlation is significant at the 0.05 level (2-tailed)  
** Correlation is significant at the 0.01 level (2-tailed)
Figure 7-1. Scatterplot of alpha asymmetry patterns with anxiety items.

Note: Graph shows a bilateral pattern for the control groups and an alpha asymmetry in PTSD participants for a relatively left-brain pattern in anterior regions.

Figure 7-2. Scatterplot of alpha asymmetry patterns with anxiety items in posterior regions.

Note: Graph shows a relatively left-brain asymmetry in the two control groups but a relatively right-brain activation pattern in the PTSD group.
Figure 7-3. Scatterplot of alpha asymmetry patterns in anterior regions with depression items.

Note: Graph shows a bilateral pattern in the two control groups and a relatively left-brain pattern in the PTSD group.

Figure 7-4. Scatterplot of alpha asymmetry posterior region patterns with depression items.

Note: Graph shows a bilateral pattern for the TEC group, indicative of no relationship with depression, a relatively left-brain pattern in the NC group and a relatively right-brain asymmetry in the PTSD group.
Summary: Alpha Asymmetry and Mood States

The hypotheses for this analysis were supported by the correlation patterns between anterior and posterior alpha asymmetry, which show clearly a different pattern between the PTSD group and the TEC group. This data suggests that numbing may be a variable unique to people with PTSD and that numbing and anxiety are lateralised to frontal and posterior regions respectively, only in the PTSD group. Numbing was negatively correlated with alpha anterior asymmetry indicating a relatively greater left hemisphere activation pattern in the PTSD group, but anxiety was positively correlated with alpha posterior asymmetry indicating a relatively greater right hemisphere activation pattern. There was a strong pattern of positive inter-correlations for each of the mood scales measured, indicating comorbid depression and anxiety in the PTSD group. The pattern of comorbidity was associated with the same lateralised patterns in anterior and posterior regions, without distinction for alpha asymmetry patterns in depression and anxiety items.

The only relationship with alpha asymmetry and emotional functioning was a positive association between avoidance criteria and alpha posterior asymmetry in the TEC group, indicating a relatively right posterior alpha asymmetry activation pattern. There were no other significant correlation patterns with mood scales in the TEC group.

As expected, there were no relationships between alpha asymmetry patterns and mood states in the NC group. Thus, the data were interpreted to suggest valence differences between the PTSD participants and the TEC participants and lend further support to earlier conclusions of structural compensatory mechanisms in both groups.

7.3.6. Discussion

The current pattern of findings, of differences between groups of high and low alpha power (TEC and PTSD respectively), together with lateralised differences in specific regions, indicate that interaction effects at the implementation, or functional, level of cognitive architecture accord with theories suggesting that component stages can be dissociated and driven by learning mechanisms in a dynamic distributed system (Basar, 2004; Singer, 1999). Earlier discussions in this study have proposed that alterations to learning parameters are theoretically linked to changes in neuromodulator systems for a role in motivational factors such as certainty and uncertainty and the associations between stimuli for error correction or inhibition (Litz et al., 2000; Dayan and Yu, 2003). Furthermore, altered states of neurotransmitter systems have been linked to the strength and efficacy of neural signals,
particularly ACh and the increased role of bottom-up processes compared to top-down processes in people with PTSD (Aston-Jones et al., 1994; Berntson et al., 2003; Southwick et al., 1999). In PTSD research, a compensatory mechanisms hypothesis forms the basis of a model proposing that neural plasticity alters the molecular mechanisms or interneurons that change the synapse and adjust tonic patterns of functioning (McFarlane et al., 2002). The current data provide support to this model, indicating that alterations through learning occur in tonic states at the local level of information processing.

The current pattern of results has been consistent in providing preliminary support for a compensatory mechanism view, using both psychological and physiological indices, in clinical and non-clinical participants with self-reported trauma reactivity experiences. Therefore, the subjective nature of reporting reactions to events, in a retrospective analysis might be a useful way of modelling how the sequencing of information into left-hemisphere event-specific categories is organised for association networks with mood states in people with PTSD.

In PTSD, symptoms have been linked to compensatory strategies for coping with events that have not been integrated into memory structures (McFarlane et al., 2002; Bremner et al., 1999; Lanius et al., 2004). Different models have been proposed for coping after stress, suggesting compensatory adaptive mechanisms after traumatic experiences may be characteristic of specific reaction patterns (Lanius et al., 2005; Litz et al.; 2000; Mineka and Zinbarg, 2006). However, one perspective for differences in compensatory mechanisms argues for differences between the numbing and avoidance category of symptoms (criterion C), and for a motivational distinction between cognitive functions of numbing responses as opposed to arousal and avoidance responses (Kashdan et al., 2006).

Different functional roles, associated with stress and arousal, have been demonstrated using EEG methodology (Davidson et al., 1990). In addition, asymmetrical electrical brain activity has been demonstrated for cognitive tasks (Davidson et al., 1996). However, more recently, a model of cortical arousal was extended by Metzger and colleagues (2004) to show that two distinct circuits underlie brain activity patterns in PTSD and is associated with affective states. As suggested by Kashdan and colleagues (2006), the responses of numbing and avoidance may be linked to depression or anxiety, or may progress to distinct patterns of comorbidity. The data in this chapter support the view that numbing and avoidance are associated with different pathways, with evidence supporting asymmetry in anterior and posterior resting states in PTSD participants. Furthermore, the current pattern of results
supports the view for distinct patterns of comorbidity, indicating asymmetry in anterior or posterior regions between depression and anxiety in PTSD participants. In addition, the valence model for differences in category C symptoms (Kashdan et al., 2006) was extended in this study by comparing clinical and non-clinical participants with trauma reactivity patterns to show that there is a characteristic profile of differences between the reaction items corresponding to numbing and avoidance that correspond to differences in low and high alpha power in resting or tonic states.

The current results suggest a pattern of low alpha power asymmetry associated with numbing and relatively left-hemisphere anterior activations and with anxiety and relatively right-hemisphere activations in PTSD participants. On the other hand, high alpha power asymmetry was associated with avoidance and relatively right-hemisphere posterior activation in the TEC group. To model this pattern of results, a framework for parameters associated with learning may be relevant to an explanation of the data. Specifically, Dayan and Yu (2003) provide a model suggesting three perspectives from which to view alterations in brain organisation patterns that include a link between learning and uncertainty. The three perspectives comprise selective attention biases, the speed of learning associated with biased perceptions, and the influences of neuromodulators ACh and NE on learning and inference processes (Dayan and Yu, 2003).

Based on the profile of correlations in this analysis, posterior right hemisphere alterations in cortical network patterns may be linked to the three theoretical parameters defined by the learning model above. That is, it is plausible that the characteristic correlation patterns for the PTSD group and the TEC group involve a shift to different neural structures when associations for classes of items or stimuli are certain. However, when associability of stimuli decreases, this would involve two pathways. According to Dayan and Yu (2003), the first is “error correction” and the second is “uncertainty”. The model, as specified by the authors, proposes that enhanced associability is linked to appetitive conditioning, but uncertainty can also involve the association of multiple stimuli and create “joint uncertainty”.

Applying this model to the current results of correlations with posterior alpha asymmetry, it is plausible that the depression, avoidance, and anxiety reactions in people with PTSD are linked to appetitive conditioning and aversive conditioning by enhanced associability of multiple stimuli, which are also linked to vigilance states and the inhibition, or blocking, of new learning. If this model is correct, then the correlation pattern between anterior alpha asymmetry and numbing responses, in this study, may represent an interaction effect between
acetylcholine and norepinephrine in expected and unexpected uncertainty or vigilance states in PTSD participants. Such mechanisms have also been implicated in the speed of learning and the amount of learning or pattern matching for auditory and visual stimuli (McFarlane et al., 2002; Dayan and Yu, 2003). On the other hand, using the same model to interpret the correlation pattern between avoidance reactions and posterior alpha asymmetry in TEC participants, it is plausible that two pathways are associated with this relationship. That is, results may be interpreted as either learning through error correction, which leads to new learning, or uncertainty, where learning is associated with error-correction and blocking.

In the current study, selective attention and the speed of learning indices indicated that compensatory mechanisms in PTSD are implemented as a result of structural or local-level alterations. Results suggesting that biased selective attention mechanisms are associated with alterations to prefrontal cortical regions have been discussed in the previous sections of this study. However, the pattern of right hemisphere alpha activation, as discussed in the previous analysis, together with the pattern of correlations, is suggestive of possible changes to neuromodulators, particularly for the PTSD group. Changes to the TEC group, as suggested in previous discussions, may be highly specific and represent a compartmentalisation of event-specific codes. As previously discussed, the rapid encoding and automatic retrieval of information, in mapping or transforming representations into neural codes through connectivity associated with long-range patterns or diffuse patterns, may be significant to functional reorganisation after trauma experiences.

One hypothesis posits that contextual information encoded at the time of the event, and associated with fear, remains as a linked sequence of coded representations (Ramos and Savage, 2003; Volmer and Sommer, 2001; Klein, 1998). Within the framework of parallel distributed processing, and notions derived from an optimisation model (Dayan et al., 2000), this hypothesis implies differences to the categorisation of information into long-term memory and the subsequent retrieval of information when primed or cued (Nadel and Moscovitch, 1998). Based on this argument, and the symptoms associated with category B (re-experiencing) in people with PTSD, the frequent retrieval of event-specific items from long-term memory systems implies an alteration to behavioural patterns, depending on the dynamic processes of automatic retrieval and the organisation of knowledge (Conway and Pleydell-Pearce, 2000; Vollmer and Sommer, 2001). In mapping perceptual and conceptual information processing to neural signal transmission, the finding of low alpha power in resting states in people with PTSD, in the current study, and as demonstrated independently in an earlier study by Jokic-Begic and Begic (2003), may be relevant to patterns of behavioural
responses associated with PTSD symptoms. This finding may be suggestive of
desynchronisation associated with perceived complexity and the focal allocation of resources
for task management in people with PTSD, and suggests a disruption to the coordinated long-
range connections of neural signals.

A similar interpretation of the data for this study was presented in the last chapter and is in
agreement with findings in the PTSD literature suggesting that processes involving inhibition
are disrupted in people with PTSD (Britton et al., 2005), and that an alert vigilant state is
maintained (Bryant et al., 2005; Vasterling et al., 1998). This hypothesis is also in accord with
models examining the functional significance of alpha synchronisation for inhibition and
alertness (Cooper et al., 2003; Knyazev et al., 2006a). In addition, different mood states and
symptoms may be responsible for the maintenance of higher or lower power of specific
oscillations in the ongoing EEG, preparing the brain for a specific pattern of responses
(Felmingham et al., 2002; Basar, 2004), which may be in agreement with a compensatory
mechanism hypothesis for lateralised processing in people with previous trauma reactivity
(Clark et al., 2003; Lanius et al., 2004; Bremner et al., 1999).

The correlation patterns suggesting a link between comorbid depression and anxiety states,
and for comorbid numbing and avoidance in the PTSD group, but not the TEC group, may be
highly relevant to understanding the neural patterns for the anticipatory processing of sensory
information. On the other hand, a pattern of simple correlations does not provide indications
of the variance associated with differences between valence and arousal. Similarly, indicators
of valence states in cognitive task performance have not been previously investigated with
asymmetry in arousal patterns in the PTSD literature. The finding in this study of a different
pattern of relationships between valance and arousal variables between the PTSD group and
the TEC group may be reflective of compensatory mechanisms in both groups, aimed at
reducing responses to external sensory signals and to aversive stimuli. However, further
investigation into the subtypes associated with reaction to stress is required for a clearer
understanding of whether there is a common underlying mechanism associated with adaptive
behaviour after traumatic stressor events, and whether this can be inferred from a pattern of
relationships between variables.
7.4. **Study 3: Predictor Variables Associated with Cortical Asymmetry Patterns**

7.4.1. **Introduction**

The aim of this analysis was to investigate whether characteristic patterns in the symptom category for numbing and avoidance behaviours are predictive of cognitive functioning differences, and whether preparatory cortical resting rhythmic oscillation patterns are lateralised in PTSD participants for avoidance and numbing reactivity patterns. Two related parts to the question are addressed. The first part considers whether numbing or avoidance reactivity levels predict alpha asymmetry in resting cortical states. The second part examines whether different patterns of learning associated with uncertainty (that is, numbing or avoidance) predict performance in cognitive tasks for verbal and nonverbal information processing.

From the previous analysis, showing a characteristic pattern of correlations between emotional factors and alpha asymmetry in people with PTSD, the question was aimed at understanding how well numbing or avoidance items predict alpha asymmetry patterns in anterior and posterior regions. There is growing empirical support for a four-factor diagnostic model of PTSD (Asmundson et al., 2004). Therefore, it was inferred that numbing and avoidance reactions are associated with distinct neural pathways, as suggested by Kashdan and colleagues (2006). Furthermore, at a local level of information processing, it has been argued that an optimisation model can accommodate patterns of learning associated with certainty and uncertainty of sensory stimuli to account for spatial and temporal parameters of neural coding (Dayan and Yu, 2003). Thus, it was hypothesised that different patterns of learning are associated with numbing and avoidance responses and it would be possible to predict cognitive performance and working memory processes associated with verbal and nonverbal tasks.

Based on the above theoretical models, and the results so far, it was hypothesised that numbing symptoms would be conscious and voluntary, and cause a disruption to the timing of information flow and selective attention, associated with characteristic tonic states of responding to stimuli in high states of arousal. Numbing was expected to be associated with frontal patterns of cortical activation and relatively left hemisphere activation patterns in the PTSD group, and a bilateral association in the control groups. It was also expected that high alert states would be associated with posterior cortical regions and joint uncertainty of multiple stimuli, which would be predictive of poor cognitive performance.
On the other hand, avoidance symptoms were expected to be associated with a phasic pattern of remitted and active symptoms, based on conditioning and external or internal cues or priming mechanisms, associated with cholinergic pathways and instability of cortical resting activation patterns, rather than tonic differences. Furthermore, based on the above theoretical models, it was expected that avoidance would be associated with a relatively right-brain posterior activation pattern in all groups. It was expected that avoidance would be indicative of two pathways associated with certainty or error correction and uncertainty or blocking, which would be predictive of efficient task performance (verbal and nonverbal).

7.4.2. Method

7.4.2.1. Participants

Participant data has been described in the previous analysis and the demographic characteristics of participants have been presented in the previous chapter for (N=33) PTSD participants, (N=32) TEC participants, and (N=88) NC participants. However, a reduced number of participants answered all items in the CIDI and numbers for analyses vary according to respondents.

7.4.3. Instruments and Procedures

Full details of all measures are provided in Chapter 4.

7.4.3.1. Self-report Responses to Trauma Events

From the pattern of results in Table 7.6, items of interest to the current analysis are responses to criterion C symptoms – numbing and avoidance. As described in the previous analysis, the total severity score for category C symptoms, from the CIDI self-report questionnaire, was split to examine numbing criteria, which included disinterest in activities, detachment from others, and diminished positive affect, as items different from anhedonia. All other items relevant to category C symptoms were summed as avoidance criteria (following Kashdan et al., 2006).
7.4.3.2. Modality-specific Cognitive Tasks

Verbal Fluency:

From the results of the first study in this chapter, the sub-task, phonemic oral fluency (FAS), was the dependent variable for the current analysis.

Maze Task:

From the results of the first study in this chapter, the sub-component, time to complete the maze, was the dependent variable for the current analysis.

7.4.3.3. Alpha Asymmetry Index

As described in the previous analysis, an asymmetry index provides a measure of the degree of left or right brain activation. Separate anterior and posterior analyses were computed for comparison by aggregating homologous leads in each region (anterior: Fp2-1, F4-3, F8-7; posterior: P4-3, T4-3, T6-5) to obtain an index of the inverse of cortical activity. Positive scores are indicative of greater alpha power at the right compared to the left aggregated electrode sites, which is assumed to reflect greater left-sided brain activation. Conversely, negative scores are indicative of greater right-sided brain activation (following methods by Pivik et al., 1993).

7.4.4. Data Analysis

Hierarchical linear regression was used in separate analyses to examine the independent variables of numbing, avoidance, group membership, oral fluency (FAS), maze time to completion (MCT), and their interactions as predictors of alpha asymmetry for anterior and posterior regions. Numbing and avoidance were examined separately in standard regression analyses with group membership and their interactions as predictors of cognitive scores (FAS and MCT).

For the purposes of this analysis, only the interaction effects for the PTSD group versus the TEC group, and the PTSD group versus the NC group, were examined as predictors of hemispheric asymmetry. The interaction between the PTSD group and the TEC group and between the PTSD group and the NC group in the regression analyses were examined by taking the high and low scores at one standard deviation from the mean for each independent
variable. Based on previous results in this study, the interaction for the TEC group and the NC group was not examined.

7.4.5. Results

7.4.5.1. Numbing and Avoidance as Predictors of Alpha Asymmetry

Numbing as a Predictor Variable:

Numbing, group membership and their interactions were examined as predictors of anterior alpha asymmetry using hierarchical linear regression. In combination, numbing, group membership, and the interactions for PTSD v TEC and PTSD v NC, accounted for 10% of the variance in anterior alpha asymmetry scores; $R^2 = 0.10$, $F(5,111) = 2.41$, $p < 0.05$, as a model. With adjusted scores for group membership, numbing continued to be associated with alpha anterior asymmetry; $\Delta R^2 = 0.45$, $t(111) = -2.69$, $p < 0.01$ and was associated with a strong right-sided asymmetry pattern with high numbing for all three groups. Table 7.8 indicates that there was an interaction effect for the TEC vs PTSD group with anterior asymmetry, $\Delta R^2 = 0.38$, $t(111) = -2.14$, $p < 0.05$. The direction of the t-test for numbing and the interaction between TEC and PTSD indicated that numbing in the TEC group is associated with a relatively right-sided anterior activation pattern but in the PTSD group low numbing is associated with a relatively left-sided anterior asymmetry pattern and changes to a right-sided pattern with high numbing. This interaction is represented diagrammatically (Figure 7-5). A similar pattern was predicted for the relationship between numbing and alpha anterior asymmetry between the PTSD group and the NC group but did not reach statistical significance ($p = 0.07$).

Numbing, group membership and their interactions were also examined as predictors of posterior alpha asymmetry. In combination, numbing, group membership, and the interactions for PTSD v TEC and PTSD v NC, accounted for only 1.5% of the variance in posterior alpha asymmetry scores and the model was not significant.
### Table 7.8. Regression coefficients: numbing as a predictor of alpha anterior asymmetry

<table>
<thead>
<tr>
<th>Model</th>
<th>Unstandardized Coefficients</th>
<th>Standardized Coefficients</th>
<th>t</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>Std. Error</td>
<td>Beta</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>(Constant)</td>
<td>0.039</td>
<td>0.017</td>
<td>2.324</td>
</tr>
<tr>
<td></td>
<td>grp2v1</td>
<td>-0.036</td>
<td>0.018</td>
<td>-0.233</td>
</tr>
<tr>
<td></td>
<td>grp3v1</td>
<td>-0.029</td>
<td>0.018</td>
<td>-0.202</td>
</tr>
<tr>
<td></td>
<td>Numbing</td>
<td>-0.017</td>
<td>0.006</td>
<td>-0.316</td>
</tr>
<tr>
<td>2</td>
<td>(Constant)</td>
<td>0.053</td>
<td>0.022</td>
<td>2.437</td>
</tr>
<tr>
<td></td>
<td>Numbing</td>
<td>-0.025</td>
<td>0.009</td>
<td>-0.450</td>
</tr>
<tr>
<td></td>
<td>grp2v1</td>
<td>-0.059</td>
<td>0.028</td>
<td>-0.377</td>
</tr>
<tr>
<td></td>
<td>grp3v1</td>
<td>-0.044</td>
<td>0.024</td>
<td>-0.312</td>
</tr>
<tr>
<td></td>
<td>grp2numb</td>
<td>0.014</td>
<td>0.014</td>
<td>0.168</td>
</tr>
<tr>
<td></td>
<td>grp3numb</td>
<td>0.010</td>
<td>0.015</td>
<td>0.084</td>
</tr>
</tbody>
</table>

Note: (AAA) = Dependent Variable: Alpha anterior asymmetry; grp1 = PTSD; grp2 = TEC; grp3 = NC

![Alpha asymmetry index with high and low numbing regression lines.](image)

**Figure 7-5.** Alpha asymmetry index with high and low numbing regression lines.

Note: (Positive alpha anterior asymmetry (AAA) scores represent relatively more left-brain activation and negative scores represent relatively more right-brain activation).
Avoidance, group membership, and their interactions were examined as predictors of anterior alpha asymmetry using hierarchical linear regression. In combination, avoidance, group membership, and their interactions, accounted for 7% of the variance in anterior alpha asymmetry scores but the model was not significant ($R^2 = 0.07, F(5,111) = 1.53, p = 0.19$). Table 7.9 shows that with adjusted scores for group membership and their interactions, avoidance continued to be associated with alpha anterior asymmetry; $\Delta R^2 = 0.35, t(111) = -2.04, p < 0.05$ and was associated with relatively left-sided anterior activation for both high and low avoidance scores in the PTSD group and relatively right-sided anterior activation with both low and high avoidance scores for the TEC group. Diagrammatically (Figure 7-6), it is shown that there was no association for the NC group between avoidance and alpha anterior asymmetry index.

Table 7.9. Regression coefficients: avoidance as a predictor of alpha anterior asymmetry

<table>
<thead>
<tr>
<th>Model</th>
<th>Unstandardized Coefficients</th>
<th>Standardized Coefficients</th>
<th>t</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>Std. Error</td>
<td>Beta</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Constant)</td>
<td>0.042</td>
<td>0.021</td>
<td>-0.245</td>
<td>2.009</td>
</tr>
<tr>
<td>grp2v1</td>
<td>-0.038</td>
<td>0.019</td>
<td>-0.251</td>
<td>-2.041</td>
</tr>
<tr>
<td>grp3v1</td>
<td>-0.036</td>
<td>0.022</td>
<td>-0.290</td>
<td>-2.215</td>
</tr>
<tr>
<td>Avoidance</td>
<td>-0.015</td>
<td>0.007</td>
<td>-2.215</td>
<td>-2.215</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Constant)</td>
<td>0.050</td>
<td>0.026</td>
<td>1.957</td>
<td>0.053</td>
</tr>
<tr>
<td>Avoidance</td>
<td>-0.018</td>
<td>0.009</td>
<td>-0.351</td>
<td>-2.039</td>
</tr>
<tr>
<td>grp2v1</td>
<td>-0.046</td>
<td>0.034</td>
<td>-0.294</td>
<td>-1.358</td>
</tr>
<tr>
<td>grp3v1</td>
<td>-0.048</td>
<td>0.028</td>
<td>-0.339</td>
<td>-1.706</td>
</tr>
<tr>
<td>grp2avoid</td>
<td>0.003</td>
<td>0.015</td>
<td>0.034</td>
<td>0.188</td>
</tr>
<tr>
<td>grp3avoid</td>
<td>0.018</td>
<td>0.021</td>
<td>0.098</td>
<td>0.874</td>
</tr>
</tbody>
</table>

Note: (AAA) = Dependent Variable: Alpha anterior asymmetry; grp1 = PTSD; grp2 = TEC; grp3 = NC
**Figure 7-6.** Anterior alpha asymmetry (AAA) index with high and low avoidance regression lines.

*Note:* PTSD avoidance is associated with relatively left-brain activation, but TEC avoidance is associated with relatively right-brain activation.

As predictors of posterior asymmetry, avoidance, group membership and their interactions accounted for only 6% of the variance and the model was not significant ($R^2 = 0.06$, $F(5,111) = 1.42$, $p = 0.22$). Table 7.10 shows that with adjusted scores for group membership and their interactions, only TEC avoidance indices continued to be associated with alpha posterior asymmetry; $\Delta R^2 = 0.46$, $t(111) = 2.49$, $p < 0.05$. The TEC vs PTSD interaction was also significantly associated with posterior asymmetry, $\Delta R^2 = 0.45$, $t(111) = -2.07$, $p < 0.05$. The direction of the t-test was reversed for these associations, indicating that with low avoidance scores the TEC group was associated with relatively right-sided posterior activation but with high avoidance scores the TEC group was associated with relatively left-sided posterior activation. Diagrammatically (Figure 7-7), the model indicates that both the PTSD group and the NC group show an association of avoidance with posterior asymmetry as relatively left-sided activation patterns, the interaction between these groups was not significant.
Table 7.10. Regression coefficients: avoidance as a predictor of posterior anterior asymmetry

<table>
<thead>
<tr>
<th>Model</th>
<th>Unstandardized Coefficients</th>
<th>Standardized Coefficients</th>
<th>t</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>Std. Error</td>
<td>Beta</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>(Constant)</td>
<td>0.008</td>
<td>0.05</td>
<td>0.161</td>
</tr>
<tr>
<td></td>
<td>grp2v1</td>
<td>-0.001</td>
<td>0.045</td>
<td>-0.001</td>
</tr>
<tr>
<td></td>
<td>grp3v1</td>
<td>0.029</td>
<td>0.052</td>
<td>0.088</td>
</tr>
<tr>
<td></td>
<td>Avoidance</td>
<td>0.013</td>
<td>0.016</td>
<td>0.112</td>
</tr>
<tr>
<td>2</td>
<td>(Constant)</td>
<td>0.085</td>
<td>0.060</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Avoidance</td>
<td>-0.016</td>
<td>0.020</td>
<td>-0.140</td>
</tr>
<tr>
<td></td>
<td>grp2v1</td>
<td>-0.163</td>
<td>0.079</td>
<td>-0.449</td>
</tr>
<tr>
<td></td>
<td>grp3v1</td>
<td>-0.049</td>
<td>0.065</td>
<td>-0.150</td>
</tr>
<tr>
<td></td>
<td>grp2avoid</td>
<td>0.087</td>
<td>0.035</td>
<td>0.457</td>
</tr>
<tr>
<td></td>
<td>grp3avoid</td>
<td>0.034</td>
<td>0.048</td>
<td>0.079</td>
</tr>
</tbody>
</table>

Note: (APA) = Dependent Variable: Alpha posterior asymmetry; grp1 = PTSD; grp2 = TEC; grp3 = NC

Figure 7-7. Alpha posterior asymmetry with high and low avoidance regression lines.

Note: The PTSD group asymmetry indicates a pattern of relatively left-brain activation with avoidance. The TEC group asymmetry indicates a pattern of low avoidance with relatively right-brain activation but relatively left-brain activation for high avoidance.
7.4.5.2. Numbing-avoidance and Cognitive Performance

Numbing as Predictor Variable:

Numbing, group membership, and their interactions were examined as predictors of FAS scores using standard multiple regression. In combination, numbing, group membership, and their interactions, accounted for 11% of the variance in FAS scores; $R^2 = 0.11$, $F(5,103) = 2.41$, $p < 0.05$, as a model. However, standardised coefficients indicated that there were no unique contributions to the prediction of FAS with the pattern of numbing or group membership, indicating a possible overlap in the independent variables.

In a separate standard multiple regression analysis, numbing, group membership and their interactions were examined as predictors of MCT scores. In combination, numbing, group membership and their interactions, accounted for 7% of the variance in MCT scores; $R^2 = 0.07$, $F(5,110) = 1.64$, $p = 0.16$, and the model was not significant. There were no unique contributions to the model by any of the independent variables.

Avoidance as Predictor Variable:

Avoidance, group membership and their interactions were examined as predictors of FAS scores using standard multiple regression. In combination, avoidance, group membership and their interactions, accounted for 13% of the variance in FAS scores; $R^2 = 0.13$, $F(5,103) = 3.02$, $p = 0.01$, as a model. Table 7.11 shows that with adjusted scores for group membership and their interactions, avoidance was still the best predictor for the FAS scores model, with the strongest unique contribution: $\Delta R^2 = 0.37$, $t(103) = -2.09$, $p < 0.05$. There were no further contributions to the model by adding group membership. Diagrammatically (Figure 7-8), the model predicts that, in contrast to the NC group, both the PTSD group and the TEC group show a relationship between low FAS scores and high avoidance reactivity.
<table>
<thead>
<tr>
<th>Model</th>
<th>Unstandardized Coefficients</th>
<th>Standardized Coefficients</th>
<th>t</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>Std. Error</td>
<td>Beta</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>(Constant)</td>
<td>17.22</td>
<td>1.739</td>
<td>9.903</td>
</tr>
<tr>
<td></td>
<td>Avoidance</td>
<td>-1.246</td>
<td>0.595</td>
<td>-0.374</td>
</tr>
<tr>
<td></td>
<td>grp2v1</td>
<td>1.358</td>
<td>2.330</td>
<td>0.133</td>
</tr>
<tr>
<td></td>
<td>grp3v1</td>
<td>-1.163</td>
<td>1.887</td>
<td>-0.128</td>
</tr>
<tr>
<td></td>
<td>grp2avoid</td>
<td>0.441</td>
<td>1.028</td>
<td>0.084</td>
</tr>
<tr>
<td></td>
<td>grp3avoid</td>
<td>2.093</td>
<td>1.314</td>
<td>0.182</td>
</tr>
</tbody>
</table>

Note: (FAS) = Dependent Variable: FAS (average number of phonemic words); grp1 = PTSD; grp2 = TEC; grp3 = NC

Table 7.11. Regression coefficients: avoidance as a predictor of verbal fluency (FAS)

![Figure 7-8. Avoidance as a predictor of verbal fluency (FAS).](image)

In a separate standard multiple regression analysis, avoidance, group membership and their interactions were examined as predictors of MTC scores. In combination, avoidance, group membership and their interactions, accounted for 6% of the variance in MTC scores; $R^2 = 0.06$, $F(5,110) = 1.39$, $p = 0.24$, and the model was not significant. There were no unique contributions to the model by any of the independent variables.
Summary: Characteristic profiles derived from the data:

The patterns revealed by the current data indicate two separate pathways for information processing in people with PTSD and in those control participants described as having experienced severe trauma reactivity. These are presented here as plausible mechanisms for understanding the complex reaction patterns and the overlap in symptom categories in people with PTSD. They also suggest different patterns of cognitive functioning at the local level of information processing. The models explore predictive relationships and suggest that the PTSD group variables can be interpreted as a reliance on perceptual cues associated with right hemisphere local storage processes. On the other hand, the TEC group model indicates a pattern that is suggestive of relationships reliant on interpretative language functions for updating or blocking unwanted stimuli. The direction of the arrows in Figures 7-9 and 7-10 represent the predicted direction of information flow between cortical regions.

The models have not been derived using statistical modelling techniques but are presented as an example for possible future investigation. Furthermore, the theoretical assumptions of the models, particularly for concepts such as backward blocking, and learning associations involving complex inferential processes based on propositional knowledge (Dayan and Yu, 2003; David et al., 2004), including a review of the separate mechanisms associated with preparatory states, will not be addressed further in this study. However, a characteristic profile of the results is presented as a baseline for continued investigations in understanding the psychological and biological mechanisms in memory phenomena and behavioural functioning in people with PTSD.

PTSD and the unique pattern of numbing associated with left hemisphere frontal regions:

Figure 7-9 plots numbing in people with PTSD as low in the left hemisphere but the direction of the arrow suggests higher numbing levels are associated with right hemisphere activation patterns. Numbing in the current regression analyses was associated with relatively left hemisphere anterior alpha asymmetry (AA) and anxiety was positively correlated with relatively right-hemisphere posterior alpha asymmetry (PA). The zero-order correlation patterns (in this chapter) indicated a unique association of numbing with relatively left-brain alpha asymmetry in the PTSD group.

The correlation patterns also suggest a plausible pathway for preparatory states in PTSD and the initial stages of information processing associated with local networks. The pattern of
differences in resting alpha power, discussed in the first study in this chapter, indicated that the left hemisphere frontal regions were oscillating with lower alpha power compared to the two control groups. This was interpreted as a disruption to attention mechanisms and a selective attention bias. In addition, there were differences in the activation patterns in right hemisphere anterior and posterior regions. These findings have been represented diagrammatically (Figure 7-9) as a plausible model proposing an inability in people with PTSD to switch off high alert states. It is reasonable to suggest that asymmetry in resting cerebral activity patterns are a mechanism for the altered functional pathways associated with information processing in people with PTSD. These activation patterns were interpreted as deficits in retrieval from modality-specific memory systems. As demonstrated by the model, information processing in people with PTSD is associated with a “joint uncertainty” (Dayan and Yu, 2003) linked to attention mechanisms that may be a plausible mechanism for the loss of inhibitory executive functioning and the loss of integration in the coordinated flow of information involving verbal working memory processing and specialised left hemisphere domain-specific patterns of functional integration.
Figure 7-9. PTSD model based on numbing as a predictor of alpha anterior asymmetry and the interaction with anxiety for a loss of inhibitory functions associated with incoming stimuli and loss of integrative functions in posterior left hemisphere regions.

TEC group patterns of preparatory states associated with avoidance and right hemisphere posterior regions:

The characteristic pattern of results for the TEC group revealed a different network for information processing in local structures. Based on the data in this chapter, the characteristic model in Figure 7-10 represents the predicted low avoidance with right hemisphere activation patterns and high avoidance levels with left hemisphere activation patterns. Unique to this group were the associations of avoidance with right hemisphere posterior alpha asymmetry (PA), and the lack of significant relationships with other mood variables, as discussed in the previous analysis.

If the pattern of results is modelled within the optimisation or learning framework (Dayan and Yu, 2003), then, diagrammatically (Figure 7-10), two pathways are predicted to be associated...
with preparatory information processing in the TEC group. These include a “certainty” for the prediction of anticipated stimuli, which is matched with content information, or structural knowledge and then updated via modality-specific left hemisphere processes. Alternatively, if a match to a negative stimulus is detected, this is represented by an interaction with right hemisphere functions and the possibility of a block in communication channels, compartmentalisation of event-specific memories, and/or dissociation, with the implementation of decisions involving aversive conditioning. At a local level of processing, the current pattern of results for the TEC group were interpreted as indicating alterations to inter-hemispheric transfer channels, associated with speed of processing, and modality-specific processing systems.

Figure 7-10. Model of TEC group data representing avoidance as a predictor of posterior alpha asymmetry and plausible pathways for processing incoming stimuli - emotionally valent and neutral.
7.4.6. Discussion

Hypotheses that numbing and avoidance reactions are associated with hemispheric asymmetry patterns in different regions were only partly supported by the data. Hypotheses that numbing and avoidance are predictive of working memory functions in verbal and nonverbal behavioural tasks were also partly supported by the data. The hypotheses for differences between groups, and predictions that an interaction between groups would be associated with separate or different neural pathways in people with PTSD were supported by the data. However, the variance accounted for by each variable in the regression models was low, suggesting other factors accounted for the asymmetry in anterior and posterior alpha patterns and in the behavioural performance scores in the current samples under investigation. The current investigation was a novel approach to differentiate emotional reaction indices as predictive variables for alpha asymmetry patterns in anterior and posterior regions, and to correlate these with cognitive task performance for verbal and nonverbal working memory.

The prediction that numbing and avoidance is associated with two distinct pathways of information processing in PTSD participants was not fully supported by the data using the current methodology. In support of the hypothesis, findings indicated a left hemisphere anterior overlap in the PTSD participants between numbing and avoidance, and a pattern of low and high avoidance in left hemisphere posterior regions. This was an unexpected finding and plausible hypotheses for interpretation, to tease apart the functional roles of this result, were not advanced here but require further analysis and investigation. Overall, the current pattern of results indicates that numbing is unique to PTSD participants. It was also demonstrated that numbing is associated with frontal regions and with relatively left hemisphere alpha activation patterns.

In contrast to predictions of separate pathways for numbing and avoidance in PTSD, the current data indicated that avoidance is associated with a model predictive of relatively left-hemisphere asymmetry patterns in both anterior and posterior regions. Therefore, there is an overlap in symptoms of avoidance and numbing associated with specific left hemisphere regions. However, the posterior left hemisphere association with avoidance suggests a different pattern to the right hemisphere association in the TEC group and may be indicative of disruption to the retrieval of content information or interpretative functions in people with PTSD. In previous studies, findings have also indicated a different pattern of attention and memory in task activation paradigms (Britton et al., 2005; Lindauer et al., 2005).
The findings of this study were interpreted as being in agreement with models proposing executive function disruptions for the manipulation of information in cognitive-emotional interaction patterns (Clark et al., 2003; Felmingham et al., 2002). Furthermore, this pattern of results may be interpreted as consistent with perspectives positing a selective attention bias in people with PTSD (Jatzko et al., 2006) and a disruption to inhibitory mechanisms associated with specific regions of the ACC (Bryant et al., 2005). The finding of avoidance contributing to alpha posterior asymmetry, as well as anterior asymmetry in PTSD participants, suggests that different pathways may be involved for the transfer of information, thus contributing to symptom overlap (Kashdan et al., 2006), depending on retrieval from memory, associated with vigilance states (Vasterling et al., 1998), or symptoms of hyperarousal (Lindauer et al., 2005).

Numbing as a predictor variable for alpha anterior asymmetry was, as expected, associated with a relatively left-brain pattern for low numbing scores but high numbing scores were modelled as a relatively right-brain pattern. On the other hand, an interaction effect between groups predicted a relatively right-sided activation pattern in anterior regions for both low and high levels of numbing in the TEC group. This was characteristically modelled as a blocking mechanism (Figure 7-10) in the TEC group and might be interpreted as a compensatory mechanism. There was no contribution of numbing to alpha anterior asymmetry in the NC group. In addition, there was no contribution of numbing, or group variables, to alpha posterior asymmetry. The models were consistent with previous discussions that joint uncertainty of stimuli in high alert states is associated with high anxiety in PTSD, which was indicated by a moderate positive correlation with alpha anterior asymmetry indices in the PTSD group.

Contrary to expectations, avoidance as a predictor variable was associated with frontal alpha asymmetry patterns. However, when group variables were held constant, avoidance predicted an interaction effect between the PTSD group and the TEC group in anterior regions; but when scores were adjusted, avoidance in anterior alpha asymmetry was the only variable to make a unique contribution, possibly indicative of the positive correlation between avoidance and numbing found in the PTSD group. In posterior regions, there was a unique contribution to the model of avoidance and the TEC group, and an interaction effect between the PTSD group and the TEC group in the alpha asymmetry model. The association between avoidance and posterior alpha asymmetry suggested a pattern of relatively left-hemisphere activation for the PTSD group for both low and high avoidance. However, in the TEC group there was a relatively right-hemisphere alpha asymmetry pattern with low avoidance and a relatively left-
hemisphere pattern with high avoidance. This was represented in Figure 7-10 as the loss of interpretative functions with high avoidance in the TEC group and a shift to blocking mechanisms. The model did not predict an association between avoidance and posterior activation patterns for the PTSD group and NC group interactions. This finding may be relevant to the hypothesis for alterations in posterior frequency patterns associated with phasic symptoms and instability in cortical arousal, which has been linked to hyperarousal states (Lindauer et al., 2005). In addition, avoidance as a predictive variable, suggesting specific patterns of cortical activation in posterior regions between groups, may be plausibly associated with interaction effects in left hemisphere regions that alter the synapse during information processing, as proposed in Study 1 of this chapter.

The predicted pattern of cortical asymmetry between the PTSD group and the TEC group is consistent with results of the various analyses in this chapter investigating resting cortical alpha power. Based on previous discussions, the interaction of a relatively left-hemisphere high avoidance association with posterior alpha asymmetry in the TEC group may be plausibly linked to a blocking mechanism associated with the retrieval of information from memory and the compartmentalisation of information associated with aversive stimuli (as suggested in Figure 7-10).

On the other hand, the prediction of a relatively left-hemisphere low or high avoidance association with both anterior and posterior alpha asymmetry in the PTSD group may be speculatively linked to higher-order processing involving joint uncertainty and the phenomenon of backwards blocking (Dayan and Yu, 2003) or failure to inhibit irrelevant stimuli (Bryant et al., 2005), associated with attention mechanisms (Felmingham et al., 2002). It can be inferred that a disruption to left hemisphere processing, for the two executive processes of selective attention and shifting attention, involves a reduced ability for task management and implicates the role of the hippocampus in retention and retrieval processes.

In contrast, the pattern indicative of an association between avoidance and posterior alpha asymmetry in the TEC group may be interpreted as an alteration to memory mechanisms in people with previous reactivity to traumatic stressor events, compared to those with no pattern of stress reactivity. This pattern of results may be congruent with data presented by Vasterling and colleagues (2006), providing evidence of compensatory strategies and alterations to reaction time speeds in people experiencing situations where learning involves error correction but also uncertainty toward stimuli. This explanation is also in agreement with proposals by Banich (2004), that emotional stimuli arrive within a different time window,
thus causing interference and disruption to the coordinated flow of information. It is reasonable to infer that avoidance as a mechanism for reducing stress is linked to selective attention and is associated with self-referential codes (Svoboda et al., 2006).

Numbing and avoidance reactivity indices, as predictors of cognitive scores, were significant only in a model with avoidance and FAS. Models were not significant for an association with numbing and avoidance and maze completion time. Further investigation is required to understand how perceptual tasks may differ from language tasks and the unique contribution of valence and mood on cognitive tasks. A unique contribution of avoidance to FAS scores was significant, but there were no contributions to this model for group membership. The model is represented in Figure 7-8 and indicates overlap between the variables, but also a pattern of low avoidance and high FAS scores indicated in both the PTSD group and the TEC group. This pattern was congruent with the first analysis in this chapter showing a significant difference in FAS scores between these two groups, but not for the NC group.

7.5. General Discussion and Conclusions

The aim of this chapter was to understand how emotional reaction patterns influence the lateralisation of cognitive functions in people with PTSD. A three-group design was used to elucidate the asymmetry patterns in alpha power for both frontal and posterior regions in resting EEG eyes closed states. A characteristic pattern of low alpha power in the PTSD group and high alpha power in the TEC group was supported by the data. Furthermore, the data indicated a characteristic pattern of high cortical oscillatory activation in right hemisphere posterior regions in all groups. These patterns were correlated with mood states for an understanding of how compensatory mechanisms might alter tonic states after extreme traumatic stressor events. The results were consistent with previous research proposing two pathways for energy regulation and attention modulation in people with PTSD (Metzger et al., 2004), but did not support views that numbing and avoidance items were lateralised as distinct patterns associated with cortical arousal in PTSD participants (Kashdan et al., 2006). In contrast, there was an overlap between numbing and avoidance in left anterior regions in PTSD participants and a distinct pattern of avoidance in left posterior regions. This was interpreted as an alteration in retrieval patterns in people with PTSD, and associated with hyperarousal states for memory of specific classes of stimuli.

To understand how retrieval mechanisms might be associated with resting preparatory rhythmic oscillations and lateralised functioning, verbal and nonverbal task performance was investigated as differences between groups. Behavioural measures for the current study
indicated that there were no differences between groups for the animal fluency task. This finding is consistent with suggestions that an association network for the familiar recall of words is similar between the three groups and is plausibly linked with non-manipulated items and automatic retrieval processes (Landau et al., 2004). On the other hand, the trend toward a significant difference between PTSD participants and TEC participants in the letter fluency task (FAS) may be consistent with the hypothesis that differences in alpha power are associated with the modulation of attention (Jokic-Begic and Begic, 2003; Sauseng et al., 2005) and the lateralisation of cortical processing involving different stages of recognition and transformation (Bremner et al., 1999; Protopopescu et al., 2005; Williams et al., 2006).

Based on the current results, one hypothesis is that for some participants, the way visual imagery is used is not information-general but event-specific, and stylistic strategies for the recall of information may have altered through experience and learning (Britton et al., 2005; Seger and Cincotta, 2006). Event-specific knowledge has been described as self-referential (Conway and Pleydell-Pearce, 2000; Wagner et al., 1999). Thus, memory of an item will be facilitated or inhibited depending on previous experiences and knowledge structures. At a neural representation level, differences in alpha power indicate that a plausible mechanism for alterations to information retrieval may be linked to phase-locked frequency patterns in preparatory states (Sauseng et al., 2005; Schack et al., 2001). Therefore, the low values for alpha power in people with PTSD may represent an interaction between spatial and temporal parameters of information processing, depending on the way information is encoded, stored, and retrieved for each set of computations involving the mapping of perceptual representations or conceptual representations. Similar proposals have suggested that separate spatial parameters (Lanius et al., 2002; Britton et al., 2005; Bremner et al., 1999), or timing parameters (Williams et al., 2006; Protopopescu et al., 2005), are involved in PTSD functioning.

The current results for the FAS task may also be consistent with predictions that slower information processing in PTSD is associated with an asymmetry in the way items are linked to long-term memory stores and with stylistic retrieval strategies. In other words, the frequent use of event-specific (non-manipulated) information may be influenced by priming and temporal contiguity factors and the consequent alteration in association networks (Protopopescu et al., 2005; Glassman, 2000; Howard et al., 2006) or in specific differences between semantic and episodic codes related to retrieval mechanisms and hippocampal structures (Hoge and Kesner, 2007). Overall, the results indicated a lower performance outcome for the PTSD group, suggesting increased effort and reduced efficiency in cognitive
flexibility consistent with disruptions to executive working memory processes. This may be linked to the unique pattern of numbing reactivity with left hemisphere anterior regions in PTSD participants. The current results are also in agreement with previous findings suggesting that parallel distributed functioning is altered in PTSD (Shaw et al., 2002) and that working memory, for the coding of verbal information, has been disrupted (Clark et al., 2003; Bremner et al., 1999). The consistent finding that a narrow focus of attention in PTSD reduces the ability for flexible behaviour may be linked to numbing responses involving ventral and dorsal pathways of the mPFC (Lanius et al., 2002) and a shift between these pathways (Knyazev et al., 2006b), depending on context information (Neylan et al., 2003).

On the other hand, hypotheses of differences in alpha activation patterns in anterior and posterior regions were only partly supported by the current data. Resting alpha rhythms have been used to index cognitive and affective alterations among various population samples (Hugdahl and Davidson, 2004; Davidson et al., 1990; Coan and Allen 2004). The data for this study were indicative of inter-group differences in qualitative and quantitative changes, predominantly in the TEC group for consistently higher amplitudes in the alpha frequency, but also for an interaction effect at midfrontal and temporal left hemisphere regions. The PTSD data indicated a consistently lower pattern of alpha amplitudes in these regions. However, a hypothesised selective attention bias associated with posterior alpha asymmetry in PTSD participants was not supported by the data. On the other hand, the various analyses demonstrated that the TEC group data was reflective of alterations to patterns of cortical arousal in right hemisphere posterior regions, particularly temporal regions. Thus the results were interpreted as a loss of inhibitory functions in PTSD participants, in accord with previous evidence of a disruption resulting from high alert and vigilance states (Vasterling et al., 1998; Bryant et al., 2005; Felmingham et al., 2002).

PTSD symptoms associated with category C (DSM-IV, American Psychiatric Association, 1994), for numbing and avoidance characteristics, have been linked to high-risk populations and resistance to improvement with treatment (Nemeroff et al., 2006). Hypotheses of distinct neural pathways associated with numbing and avoidance reaction patterns in people with traumatic stressor experiences (clinical and non-clinical) were supported by the data, (Kashdan et al., 2006). However, correlation patterns for mood states and PTSD category C symptoms of numbing and avoidance with alpha anterior and posterior asymmetry index indicated differences between groups. This finding supports neuroimaging studies suggesting there is no pattern of overall right-lateralisation associated with emotional functioning (Wager et al., 2003). A unique pattern of numbing for the PTSD group was characterised by a
negative correlation pattern between numbing and alpha asymmetry in anterior regions and a positive correlation between anxiety and alpha asymmetry in posterior regions. This finding, together with the correlation patterns reported in Chapter 5, may be in agreement with views that propose alterations to cognitive functioning in people with apprehensive arousal are different to functioning with anxiety (Metzger et al., 2004; Knyazev et al., 2006b).

A positive correlation pattern for posterior alpha asymmetry and avoidance reactivity was significant for the TEC group. This result provided evidence of valence differences in the lateralised patterns of synchronous oscillatory activity of the PTSD participants and the TEC participants associated with reaction to extreme stress. The proposal of a unique association between numbing and PTSD symptoms (Kashdan et al., 2006) was congruent with the current results. Different pathways were predicted using regression models for an interaction effect between the PTSD group and control groups and the associated numbing and avoidance reactivity variables. However, the data did not support distinct pathways of numbing and avoidance in anterior left-brain regions in the PTSD group, or a lateralised effect for the separate numbing and avoidance items.

The relevance of the current findings for understanding brain organisation patterns and memory processes in PTSD was indicated by data supporting a hypothesis for the dysregulation in higher-order cognitive functioning in PTSD. This was revealed by symptoms of numbing and avoidance uniquely associated with PTSD in anterior and posterior regions, and predictive of relatively left-brain anterior alpha asymmetry. This finding supports previous views by Metzger and colleagues (2004) for a different pattern of arousal compared to anxiety, and one that is consistent with uncertainty, depending on symptom subtypes. As hypothesised in previous discussions, joint uncertainty of stimuli may lead to particular pathways in people with PTSD that are compensatory and disrupt verbal working memory processes (Clark et al., 2003). A switch between high and low levels of reactivity, and between numbing and anxiety reactions in people with PTSD, linked to hemispheric asymmetry patterns of cortical activation, may suggest a mechanism to account for failure to inhibit irrelevant information during the encoding stages of information processing. Thus, the data lend support to models suggesting that PTSD is a secondary characteristic of traumatic experiences and is associated with poor integration of the event in memory systems (Britton et al., 2005; McFarlane et al., 2002).

Based on this argument, the current data demonstrated that disruptions to executive functions are associated with a selective attention bias in people with PTSD, resulting in poor
performance on cognitive tasks. In addition, the current pattern of results demonstrated that cognitive tasks, involving left- and right-hemisphere specialised working memory processing, are disrupted by mood states that are associated with emotion regulation as a top-down thalamocortical function. The models derived from the data suggest a plausible pattern of information processing alterations in PTSD consistent with the disruption to verbal working memory and the retrieval of information associated with content information or interpretative functions. This interpretation of the results is in agreement with findings and proposals by Clark and colleagues (2003) and the current pattern of results extends previous research to demonstrate a plausible mechanism for the disruption to inhibitory functions. That is, as a conscious and deliberate attempt to reduce high arousal states during retrieval processes; numbing associated with left hemisphere anterior functions, implicates alterations to working memory feedback loops, uncertainty in decision processes, and interactions between inter-hemispheric communication channels, that is indicative of abnormality.

Furthermore, the results of this study indicated that verbal working memory processes were most affected by emotional reactions of anxiety and numbing in people with PTSD. The interaction between posterior right hemisphere activation patterns and left hemisphere anterior activation patterns provided a plausible pathway, or channel for cortical communication, thus implicating asymmetry as a likely mechanism for compensation. Avoidance was predictive of cognitive performance on the FAS task in people with PTSD, and was specifically associated with asymmetry and previous trauma reactivity in non-clinical participants (TEC). However, numbing reactivity indicated no association with FAS scores in people with PTSD and may be plausibly interpreted as a disruption to executive functioning and the loss of a verbal working memory network that is not accessed due to compensatory mechanisms. This preliminary evidence for altered distributed neural synchrony patterns, associated with working memory and attention processes, provides support to current PTSD theories (McFarlane et al., 2002; Clark et al., 2003) positing that the coordination of neural activity between and within functionally specialised brain regions is a mechanism related to the structural or tonic states of information processing and can be detected in resting cortical activation patterns, as suggested by Begic and colleagues (2001).

However, the link between female gender and language skills may have contributed to the results found in this study. Thus, as suggested by different groups of researchers (Metzger et al., 2004; Kashdan et al., 2006; Protopopescu et al., 2005), understanding reaction patterns associated with PTSD may require a detailed analysis of specific subtypes and the unique contribution of symptoms in associative networks or the overlap in network paths that might
be altered by spike neurons (Singer, 1999; Munk, 2001). Furthermore, psychological states may be influenced by a number of different parameters. The higher proportion of women in the TEC group, compared to the PTSD group and the NC group, may be relevant to priming effects and stylistic strategies for the retrieval of information involving association networks based on socialisation skills for the recall of words (Frans et al., 2005; Kline et al., 1998). According to Fullerton and colleagues (2001) women are more likely than men to meet tonic symptoms of avoidance and hyperarousal. Speculatively, the increased ratio of females in the TEC group might be a contributing factor related to reactivity patterns such as dissociation. Such effects at the time of the event may have a likely consequence on voluntary or cognitive blocks in communication channels for certain classes of information, without interrupting the flexibility for other types of information. However, gender effects were not controlled in the current study and further research is warranted to examine the validity of this argument.

7.5.1. Behavioural Performance for Specialised Modality-specific Processes

The organisation of information for verbal and nonverbal tasks in this study has been modelled using the theoretical framework of working memory processes for an executive function system and two specialised rehearsal systems (Baddeley, 2003). Using this framework, the behavioural performance indices indicated a trend toward differences in functional organisation between the PTSD group and the two control groups.

While there were no significant differences between groups for accuracy scores and the retrieval of semantic information from long-term memory for a category of familiar words (animals), there were indications that tasks requiring increased levels of attention were compromised in the PTSD group compared to performance levels of the two control groups. The subtasks, phonemic verbal fluency production (FAS) and time taken to complete the maze (MCT), indicated a strong trend toward significant differences between groups in behavioural performance measures. The pattern of results implied a greater degree of executive functioning is associated with a shift in endogenous selective attention processes and higher-order frontal system processes (Williams et al., 2006; Seger and Cincotta, 2006).

The hypothesis that spatial working memory is a shared resource with attention processes (Jatzko et al., 2006; Awh and Jonides, 2001; Miyake et al., 2001; Gallese and Lakoff, 2005) might be relevant to the findings for the performance measures in the PTSD group. The finding of a trend toward differences, only in sub-processes thought to share neurobiological substrates, raises further questions about how information is organised in PTSD - firstly, about the role of spatial working memory in encoding new information in people with trauma.
experiences; and secondly, whether a reliance on over-learned information is a preferred style for optimal performance in familiar contexts when confidence in the prediction of anticipated stimuli is low.

The results of the behavioural measures did not support a hypothesis of a shift in local processing toward modality-specific processes and right hemisphere spatial lateralisation in people with PTSD. In contrast, the data suggest that temporal alterations associated with the retrieval of salient information interrupt the coordinated flow of information for the type of processing required (verbal and nonverbal). In support of this argument, the idea that more recent trauma experiences may be a causal factor in altering knowledge structures is plausible (Vasterling et al., 2006; McFarlane et al., 2002), and in agreement with models suggesting that attention modulation is based on right-hemisphere specialisation processes within posterior visual cortex ventral streams for directing perception (Lanius et al., 2002; Mesulam, 1998; Sakagami et al., 2006). In addition, emotion regulation and valence factors have been linked to the timing of information flow (Protopopescu et al., 2005; Williams et al., 2006), with disruptions to the coordination of information when items arrive outside a specific time window (Singer, 1999; Banich, 2004). Therefore further investigation is required for an understanding of the subjective role of context information in cognitive paradigms (Jatzko et al., 2006) and the role of recognition and familiarity in processing cognitive tasks associated with efficiency and speed.

Mixed results have been reported in the literature for cognitive functioning between PTSD participants and control participants, with a number of studies reporting no differences in cognitive functioning in people diagnosed with PTSD (Lindauer et al., 2005; Stein et al., 2002; Twamley et al., 2004). However, the organisation of knowledge is not reflected in accuracy scores. That is, a focus on the more qualitative aspects of performance proficiency in the clinical literature has not clarified the role of context information in decision processes and the transfer time of information between hemispheres associated with binding and phase-locked cycles of temporal coding (Protopopescu et al., 2005; Schack and Weiss, 2005). Further investigation to investigate this proposal may provide a useful candidate mechanism for examining whether compensatory strategies at the implementation level for the coordination and flow of information occur without alterations at the structural or local level of information processing. This proposal may be consistent with distinguishing processes for semantic memory and episodic memory in understanding how the speed of processing is associated with retention and retrieval mechanisms.
Various models of executive function and working memory have been proposed (Miyake and Shah, 1999). Accordingly, Chein and colleagues (2003) speculate that rehearsal mechanisms are attention focusing, and remain as speech based rehearsals for phonological processing or as reactivated decaying activity to support the maintenance of information in certain contexts; with a second mechanism that focuses attention on stored memories for covert reactivation in a domain general processing system for cognitive flexibility. This may be associated with the role of the hippocampus in episodic memories and linked to retention and retrieval processes (Nadel and Moscovitch, 1998; Eichenbaum and Fortin, 2005; Sega and Cincotta, 2006). There have been numerous studies validating various sub-components associated with attention modulation in the PTSD literature (Felmingham et al., 2002; Vasterling et al., 1998; McFarlane et al., 1993), and it is plausible that the results for the current study are indicative of specific mechanisms for the organisation of context-specific information in PTSD participants that alter pathways for the automatic retrieval of information. Processes discussed in the previous chapters that may differ between individuals, were response selection, response inhibition, and conflict monitoring, which can be influenced by a number of factors in functional control. Furthermore, the differences implicate specific spatial and temporal dynamics associated with altered functioning.

Of particular interest to this study was the role of imagery in memory retrieval. According to Fletcher and colleagues (1995), imagery is not a unitary process and the authors posit that selective attention is required for the activation of functionally specialised cortical regions. Thus, depending on whether an image is associated with episodic memory and is implemented as a knowledge structure, which is modulated by attention, or is associated with an event-specific experience, which is automatically retrieved as a memory structure, the speed at which these processes are implemented will be defined by individual differences or possibly a common underlying mechanism. The results of the current study indicated a slower speed of processing in the PTSD group, suggesting a disruption to the timing and coordination of information flow and a common mechanism of trait characteristics or as an environmental influence.

Furthermore, functional neuroimaging studies have demonstrated that working memory involves the coordinated activity of multiple cortical regions (Clark et al., 2000). Regions most consistently activated during working memory functions include the middle frontal gyrus (MFG) of the dorsolateral prefrontal cortex (DLPFC) and the supramarginal gyrus (SMG) of the inferior parietal lobule (IPL) (Clark et al., 2000). The MFG has been consistently involved when active manipulation and monitoring of information is required for
the purposes of planned actions (Clark et al., 2003). The left IPL has been implicated in the updating of verbal perceptual working memory and has been associated with phonological storage (Clark et al., 2003).

It is possible that the current finding of numbing, associated with PTSD as a relatively left-brain pattern of anterior activation, may inhibit the functional pathways associated with the IPL because of right hemisphere activation patterns and anxiety reactions. As suggested by Lanius and colleagues (2002), two pathways are associated with acute hyperarousal states and implicate different reaction patterns. This interpretation implies a disruption to long-range connectivity patterns for information binding and integration. This proposal warrants further investigation to understand the nature of memory deficits in people with PTSD. In particular, the role of hippocampal function and associated functions in the medial temporal cortices for transient memories (Lindauer et al., 2005; Glassman, 1999) and episodic memory function (Lanius et al., 2003; Eichenbaum and Fortin, 2005) that may be linked to altered frequency patterns.

Evidence has been growing for the functional role of specialised anatomical regions and selective response patterns associated with the PFC (Goldman-Rakic, 1999; Williams et al., 2006). Two regions that have been investigated in the wider literature related to the integration of new information and in control mechanisms for the monitoring and manipulation of cognitive information, have also been the focus of recent investigations in the PTSD literature. These include the DLPFC and the anterior cingulate cortex (ACC). Both regions have been consistently reported as mediating components of cognitive control in PTSD activation studies (Bryant et al., 2005; Clark et al., 2003; Jatzko et al., 2006; Williams et al., 2006). However, the precise mechanisms for information processing associated with these regions remain to be clarified.

In addition, the functional role of the cerebellum in cognitive and affective processing has received little attention in the PTSD literature to date. Of particular interest, for an understanding of the current pattern of results, are the neural connections and interactions associated with parietal cortex, prefrontal cortex and cerebellar activity. Previous findings have revealed that cerebellar activation contributes to the limbic circuitry, including septal nuclei and hippocampus (Schutter and van Honk, 2006). Models of regional specialisation have linked the cerebellum to an association with cognitive and affective abnormalities (Teicher et al., 2003; Schmahmann and Sherman, 1998). A specific role for the cerebellum in higher order processing has been postulated (Shaw et al., 2002), but as yet not clearly defined in the PTSD literature.
It was postulated that rehearsal mechanisms contributed to the pattern of results in the current study. If this interpretation is correct, then rehearsal mechanisms associated with cerebellar activity and linked to right hemisphere prefrontal cortical activity may suggest a separate pathway for information processing that is secondary to PFC executive functioning. This notion represents a proposal for further research into how compensatory mechanisms might limit cognitive and behavioural flexibility in people with extreme reactivity to traumatic stressor events by considering the role of medial temporal lobe structures in memory performance. It is possible that an over-reliance on right hemisphere processing in PTSD may be associated with the automatic retrieval of new structural patterns that are encoded over time to match adaptive response sequences (Lanius et al., 2005; McFarlane et al., 2002; Nadel and Moscovitch, 1998). Thus, it is reasonable that self-referential processing leads to bias in overlapping bilateral cortical networks that orient spatial attention and disrupt the speed of learning and control mechanisms for emotion regulation (Protopopescu et al., 2005; Sakagami et al., 2006; Wilson et al., 2005).

Areas of the middle temporal cortex, particularly the dorsal pathway, have been implicated in motion detection (Linden et al., 2003) and more recently have been demonstrated in studies emphasising the identification of emotions (Britton et al., 2005; Lanius et al., 2002; Williams et al., 2006). In addition these regions have been described as supramodal regions exhibiting modality-dependent coupling with higher sensory cortices (Mesulam, 1998; Thiel and Fink, 2007). Taken together with the consistent finding of altered hippocampal function in PTSD (Bremner et al., 1999; Hull, 2002; Nutt and Malizia, 2004), these findings may be significant in understanding how visual pattern matching is used to maintain an alert state for a response to warning cues and for a shift in attention processes. The above views also implicate different patterns for synchronisation, with focal and diffuse patterns representing differences in the coordination and flow of information processing (Banich, 2004; Singer, 1999) that have been associated with early and late stages of information processing (Protopopescu et al., 2005; Vasterling et al., 1998; Williams et al., 2006). Altered patterns of rhythmic oscillation frequencies, in people with PTSD, may be a useful way to understand how different cortical regions are connected via temporal channels of communication to rehearsal mechanisms associated with semantic information and to the preparation and execution of motor acts.

7.5.2. Alpha Asymmetry Patterns

Results of this study indicated that resting alpha amplitudes were consistent with higher activation patterns to right hemisphere posterior regions among all three groups. The
suggestion is that increased right parietal activation in PTSD is associated with high arousal or anxiety states (Metzger et al., 2004) and, as discussed, the current results concur with this finding. However, low alpha amplitudes were consistently reported in the PTSD group, for both anterior and posterior regions, raising the possibility that the interaction of alpha frequency signals with other frequencies in cross-frequency phase synchrony is required for the performance of specific cognitive control functions (Veltmeyer et al., 2006; Ward, 2003). This interpretation applies to the PTSD group compared to the control groups for the regulation of adaptive emotional strategies or compensation and functional adaptation. Thus, it is plausible that participants with high numbing responses to stress might present with a different pattern of self-regulation, as indicated by the current results, for disruptions to frontal regions and to executive functioning.

The data in this study indicated significant differences between groups in the pattern of frequency oscillations in different regions, localised to different hemispheres. The hypothesis that differences between groups can be characterised by resting cortical arousal patterns, with PTSD showing instability in preparatory activation states, is in agreement with previous evidence (Neylan et al., 2003) and converges with findings by Jockic-Begic and Begic (2003) for lower alpha power in people with PTSD. Furthermore, these findings were extended to demonstrate that specific regional changes in anterior and posterior regions implicate lateralisation as a plausible compensatory mechanism for brain reorganisation patterns.

Despite the non-significant differences between groups in the higher beta frequency, there were indications in the current study that interaction effects warrant further investigation, particularly for the link between speed of processing and long-range synchrony patterns. Furthermore, there were significant differences in behavioural indices of motor function, suggesting alterations to the timing of information processing between groups, which also implicate differences in neural connectivity for long-range synchronisation and binding mechanisms. Derived mainly from behavioural indices of speed of processing, the current pattern of results indicates some convergence with proposals by Begic and colleagues (2001) that alterations to the beta frequency is a plausible characterisation of cortical arousal in PTSD participants.

The significant difference for higher alpha amplitudes in all analyses in the TEC group was interpreted to mean a pattern of cognitive control, more consistent with better memory performance measures, whereas the lower alpha amplitudes in the PTSD group were interpreted to mean a pattern more consistent with focused attention mechanisms, as
suggested in previous studies (Hanslmayr et al., 2005). The clinical relevance of these findings is in relation to understanding how the encoding of stimuli alters patterns of memory for a selective attention bias or for attentional scanning and cognitive flexibility (Bryant et al., 2005; Felmingham et al., 2002).

Both the PTSD group and the NC group data were characteristically similar and results were taken to suggest a greater reliance on perceptual mechanisms compared to the TEC group. Anticipatory response patterns, as indexed by resting EEG, may suggest localised regions for abnormality in the pattern of alpha activation in eyes closed states (Niedermeyer, 2005). However, inconsistencies in resting EEG patterns have been reported in the wider literature (Basar, 2004; Jausovec and Jausovec, 2000) and in the PTSD literature (Begic et al., 2001; Jokic-Begic and Begic, 2003). It is possible that the mixed nature of the control group in this study may have biased results. In explaining the lack of a significant difference between the PTSD group and the NC group on behavioural performance indices, and in preparatory resting EEG states, a number of factors might apply.

Firstly, it is plausible that the lack of differences in synchrony values, in specific anterior and/or posterior regions, may have contributed to the dynamic capacity to automatically retrieve information, based on familiar tasks using associative networks for over-learned material, in both groups. This may be particularly relevant to the increased alpha activation patterns in right hemisphere posterior regions and an association with increased alertness or vigilance.

Secondly, the mixed nature of the control group, with some of the participants in this group having reported previous traumatic stressors, as well as strong reactivity to those stressors, may have contributed to a similar pattern of attention in preparatory states for both groups, thus indicating lower scores compared to the TEC group. Differences in frontal and posterior activation patterns have been shown with vulnerability for the development of depression and anxiety (Blackhart et al., 2006; Bruder et al., 1997).

Thirdly, familiar tasks may retain the same response networks, once items are recognised, even though encoding networks for new information may have altered. This argument is consistent with models indicating spike neurons can dynamically change working memory feedback loop connections, without altering association networks (Munk, 2001). Accordingly, data for the TEC group was interpreted as an alteration at the structural level of analysis, and associated with compensatory mechanisms at the functional or implementation level that
change with task demands. This interpretation offers one explanation for the similarity in the EEG resting patterns for the PTSD group and the NC group. Other research groups have also suggested a compensatory mechanism hypothesis for non-PTSD people with previous trauma exposure and reactivity.

On the other hand, the limited capacity working memory model (Baddeley, 1986) for both verbal and nonverbal information processing, has led to assumptions of a single flexible resource (or neural substrate) in working memory that underlies EEG frequencies (Ward, 2003). Therefore, one suggestion proposed in the clinical literature is that asymmetries in anterior cortical regions are associated with different emotional behavioural responses and psychopathologies (Bruder, 2004; Jackson et al., 2003). Another suggestion is that resting anterior asymmetry relates to a stylistic tendency toward emotional reaction that is present during infancy and early childhood (Blackhart et al., 2006; Elzinga and Bremner, 2002; Nemeroff et al., 2006; Smit et al., 2005). Accordingly, resting frequency patterns are generally thought to represent more stable or tonic states of functioning (Jokic-Begic and Begic, 2003; Galletly et al., 2001; Neylan et al., 2003) and can indicate altered patterns of preparatory states at the local level of information processing.

Thus, in addressing the question of whether the resting cortical patterns in the current study were associated with structural or functional changes, it was not possible to determine a pattern of structural alteration in the PTSD data compared to the NC data without adding emotional factors that might contribute to the implementation of information processing pathways. The pattern of numbing and anxiety responses in the PTSD group added another level of interpretation to the asymmetry patterns in resting conditions. Together with the valence variables for the TEC group, it was possible to surmise that both groups with strong reactivity to traumatic stressors may have demonstrated a pattern of structural alterations at the local level of information processing, resulting from environmental factors, in the case of PTSD participants, and from long-term plasticity in the TEC group.

As discussed previously (in the literature review chapters), theories of cognitive architecture propose that stylistic or tonic patterns of information processing operate at the functional implementation level for the combinatorial manipulation of information (Anderson, 1987; David et al., 2004). However, recent studies investigating the genetic effects on EEG band-specificity, have suggested that psychiatric disorders are characterised by band-specific alterations to the EEG (Zietsch et al., 2007). The current data for the alpha band of frequencies revealed frontal differences between the PTSD group and the TEC group,
implicating affective alterations and an altered pattern of functional processing mechanisms in cognitive performance tasks, consistent with previous reports of disrupted executive working memory functions (Clark et al., 2003; Vasterling et al., 1998). Consequently, the current finding of qualitative and quantitative differences between the PTSD group and the TEC group, particularly for the alpha frequency in spectral power, was interpreted as a structural characteristic of altered information processing in both groups.

However, as discussed previously, these differences between the two groups may be consistent with differences in the ventral and dorsal streams of the PFC for the active retrieval of information at the functional level of information processing (Clark et al., 2003; Jatzko et al., 2006; Lanius et al., 2002; Williams et al., 2006). Furthermore, any differences between the two groups indicated interaction effects, which were associated with the maintenance and monitoring of information, possibly mediated by PFC attention mechanisms and limbic reciprocal connections (Williams et al., 2006). Accordingly, in posterior regions, differences in alpha amplitudes between the PTSD group and the TEC group may indicate unique patterns for PTSD neural activation patterns, associated with affective attentional resources and alterations for integration in frontal-striatal circuitry influenced by inhibitory GABAergic connectivity (Wang et al., 2004). Thus, an assessment about whether alterations occur at the structural level or functional level, for information processing in the TEC group, remains open to further investigation and clarification.

As previously stated, the problem is one of assessing the role of each of the causal elements in accounting for the lower thresholds of alpha amplitudes in the PTSD group, compared to the higher amplitudes in the TEC group. Previous research evidence has suggested that imagery associated with episodic memory and with the precuneus region, is distinct from the neural architecture for visual imagery and visual perception (Fletcher et al., 1995; Caplan et al., 2001). Thus, it is plausible that the results for the PTSD participants are suggestive of automatic retrieval of information from long-term memory, which is associated with a failure to maintain incoming information by the consequent shift in attention and interneuron activity (Corbetta et al., 2002; Linden et al., 2003; Sakagami et al., 2006). This explanation may be consistent with the results for lower amplitudes in the left hemisphere posterior regions and with the interaction between the PTSD group and the TEC group for alpha activity in temporal regions.

Taken together, the results of the current analyses suggest alterations to both structure and function in people with PTSD. The results support the idea that neural codes, as structural
abnormalities, are associated with asymmetry patterns of cortical activation and functional abnormalities are associated with timing mechanisms linking the different stages of processing (Singer, 1999). This notion is supported by findings in previous research indicating both structural and functional abnormalities in PTSD (Vasterling et al., 1998; Protopopescu et al., 2005; Lindauer et al., 2005). Further clarification is required to understand how learning and neural plasticity alters structural parameters. This may be linked to retrieval processes in acute hyperarousal states (Lanius et al., 2002). Furthermore, the results for the TEC group, using both psychological and physiological parameters, were indicative of an altered pattern of functioning, suggesting different mechanisms in the retrieval of information from memory. The higher resting cortical activation amplitudes may be suggestive of structural alterations in this group of people, involving mechanisms such as LTP for the alterations to knowledge structures. The data in this study indicated differences in cortical rhythmic patterns, and behavioural performance between groups and supported an association between compensatory mechanisms and asymmetry in neural networks for both the PTSD group and the TEC group.

7.5.3. Local Differences for the Organisation of Knowledge Structures

Significant differences in resting EEG rhythms were found between frontal regions compared to activity in posterior regions, and differences in the left hemisphere, for a laterality effect in people with PTSD compared to the TEC group. On the other hand, the differences between PTSD participants and TEC participants in posterior regions, associated with leads corresponding to temporal sites, may be a significant finding for understanding perceptual mechanisms in preparatory resting states for the dynamic integration of sensory and motor stimuli. This interpretation of the data implicates a role for the hippocampus and medial temporal cortices and interaction effects with striatal circuitry, which may correspond to a lateralisation of function or a compensatory pathway for response selection. Furthermore, this interpretation of the data may be in agreement with theoretical models of attention proposing that an interaction effect between hemispheres is necessary for the integration of information (Banich, 2004).

It is also possible that higher amplitudes represent a functional threshold for attentional control and self-regulation in the TEC group, in line with hypotheses that higher alpha power indicates a more strongly synchronised neural activity (Knyazev et al., 2005) and greater attentional scanning in preparation for external stimuli (Aston-Jones et al., 1999; Knyazev et al., 2006a). However, the consistently lower alpha amplitudes demonstrated by the PTSD data suggests a marker for a common environmental factor in this group of participants.
Speculatively, alterations toward lower frequency patterns in the PTSD group may indicate a disruption to thresholds in attentional control. This pattern may represent a shift in the neural circuitry for the automatic retrieval of information involving ventral frontal circuits, and involving the disruption to item specificity in short-term storage based on the spatial sequencing of responses, as represented in the characteristic pathways formulated using the current pattern of results.

Alternatively, disruptions in attention may be associated with a shared function in spatial working memory rehearsal systems, in particular the sequencing of response retrieval. If this conclusion were correct, it would implicate the role of theta in functional connectivity patterns, and disruptions to theta synchrony specific to PTSD for the encoding of new stimuli based on perceptual cues. This proposal is in agreement with previously reported alterations to an alpha-theta network in people with PTSD during working memory processing (Veltmeyer et al., 2006). It has been documented that evidence of EEG alpha activity and associations with cognitive performance vary according to different parameters, but pre-stimulus alpha power has been shown to have a strong influence on ERP components, supporting the view that low alpha activity reflects excitation of the cortex and good perceptual performance (Hanslmayr et al., 2005).

The interaction effects indicated by the current data, between the PTSD participants compared to the two control groups, for mental preparation states associated with the active processing of incoming stimuli, suggest altered mechanisms of cortical neural synchronisation, particularly in temporal and frontal regions. Therefore, a focus on the role of frequency amplitudes between groups may provide a plausible explanation for the results. Frequencies mediating synaptic transmission have been shown to alter receptor binding, with intracellular effects of hormones released as noradrenalin and acetylcholine associated with consequent inhibitory and excitatory responses (Nelson et al., 2005; Schutter and van Honk, 2006), and implicate a role for hyperarousal in models of PTSD (Lindauer et al., 2005; McFarlane et al., 2002). The allocation of attention for structural and functional connectivity between the prefrontal cortex and the parietal cortex has been widely reported (Awh and Jonides, 2001; Ungerleider et al., 1998) and implicates alterations to neurochemical systems and the role of acetylcholine in learning (McFarlane et al., 2002; Aston-Jones et al., 1994; Dayan and Yu, 2003; Nieuwenhuis et al., 2005).

Thus the findings in this study of lower frequency amplitudes in the PTSD group can be interpreted to suggest a possible role for dysfunction associated with the synchronisation of
theta frequency and the speed of information processing, implicating frontal-striatal circuitry. Theta rhythm has been associated with hippocampal function and memory demands (Klimesch, 2003). This interpretation has clinical implications in people with PTSD for the allocation of attention and control in the prefrontal cortex. The current data provide evidence of disruption to executive functions in people with PTSD that are related to disturbances in memory, concentration, and the implementation of plans or goals. This interpretation is in agreement with previous findings (Galletly et al., 2001; Clark et al., 2003).

A review of previous studies suggests that a temporal channel for the transfer of information is a plausible mechanism for information that is encoded and retrieved (Banich, 2004; Protopopescu et al., 2005). This hypothesis carries implications for the encoding of stimuli and the depth of encoding (Singer, 1999; Vasterling et al., 1998; Wagner et al., 1999). An alteration to temporal parameters has also been linked to the shared neurobiological substrates for attention and spatial working memory (Awh and Jonides, 2001), and may be a plausible explanation for the proposed shifts to compensatory right hemisphere mechanisms in PTSD (Clark et al., 2003; Bremner et al., 1999; Lanius et al., 2002; Britton et al., 2005).

Furthermore, the importance of theta activity in synchrony with alpha frequency, for a coherent long-range network associated with executive cognitive functions during task processing, has been repeatedly demonstrated (Ward, 2003; Klimesch et al., 2005; Kahana et al., 2001; Sauseng et al., 2002). The posterior association cortex is reportedly involved in the storage of sensory information, and the prefrontal cortex has been implicated in updating information (Clark et al., 2004; Klimesch, 1999). Therefore sensory information linked to a pacemaker model that drives firing at altered frequencies for receiving incoming stimuli (Rowe, 2005; Jones and Wilson, 2005), is likely to be associated with differences in functional and clinical outcomes. That is, the timing window within which information arrives is likely to lateralise processing functions and result in either confident decisions or interrupt the flow of information through an endogenous attention circuit (Felmingham et al., 2002; Dayan et al., 2000), which is a shared resource with spatial attention (Linden et al., 2003; Awh and Jonides, 2001; Callejas et al., 2004). Retrieval networks in such a model would be more consistent with the maintenance of information rather than the manipulation of information, and with spatial working memory in mPFC structures for the retrieval of context cues that may be linked to a pattern of avoidance behaviours based on previous experiences (Conway and Pleydell-Pearce, 2000; Britton et al., 2005; Schutter and van Honk, 2006).
If the above argument is correct, then it may be in agreement with models suggesting that internal neural afferent connections from the parietal cortex to the frontal cortex are responsible for the alteration in neural pathways (Basar, 1980; Nobre et al., 2000; Thomsen et al., 2004; Woldorff et al., 2004) as specialised processes in different functions of alertness and awareness (Callejas et al., 2004; Fan et al., 2005; Woldorff et al., 2004; Knyazev et al., 2006b). One plausible hypothesis is that the disruption of integrated information, and long-range coordination of neural signals, from posterior to anterior regions, may be speculatively related to “spindle” cells (von Economo neurons (VENs)) in the anterior ACC and fronto-insula (Watson et al., 2006). These regions are commonly implicated in PTSD dysfunction (Bryant et al., 2005; Lanius et al., 2005). Speculatively, the functional role of the PFC may be disrupted by the localisation of specific neurons for emotion regulation, thought to be projections from the ACC and fronto-insular cortex, which may be involved in the control of complex integration involving emotions (Bryant et al., 2005; Lanius et al., 2003). Further evidence associated with an early switch in processing, based on emotional cues, is presented in a model by Jatzko and colleagues (2006) suggesting altered positive affect and an increased perception of aversion in people with PTSD. Jatzko and colleagues argue that over time, a disruption to emotional stimuli may be associated with parietal pathways and visuospatial processing rather than prefrontal cortex pathways associated with executive control functions.

In theories proposing a functional role for cerebral rhythmic oscillation patterns, the low delta frequency has been associated with the consolidation of memories (Klimesch, 2003), whereas the theta frequency has been associated with the encoding of new memories (Ward, 2003). The structural abnormality of higher amplitudes in the delta frequency for the PTSD group, but also for groups with trauma experience, as revealed by the previous chapter in this study, may be a plausible explanation for the mediating effects of neuromodulation from feed-forward to feed-back loops to selectively enhance intra-cortical synaptic transmission and selectively suppress thalamo-cortical synapses (Palva and Palva, 2007).

Therefore, it is reasonable that resting EEG alpha patterns associated with low alpha amplitudes in the left hemisphere, and higher activation patterns in right hemisphere, reduce the capacity at the implementation level for an analytic propositional representation of information in people with PTSD. This proposal is in agreement with theories that suggest alterations to the interpretation of incoming stimuli is limited to visuospatial processes that infer meaning from the relative position of stimulus elements (Nobre et al., 2000; Umita, 2001; Shelton and Gabrieli, 2004; Nadel and Hardt, 2004; Woldorff et al., 2004). In addition,
this proposal may be relevant to theories proposing alterations in structural connectivity patterns with mixed handedness (Chemtob and Taylor, 2003; Saltzman et al., 2005).

The competition for attention with visuospatial processing, to monitor and anticipate external stimuli that serve as the basis for response activation, may be a plausible factor contributing to the high anxiety states reported in the PTSD group in this study, and to the correlation of anxiety indices with the alpha posterior asymmetry index. This interpretation is also in agreement with suggestions by Metzger and colleagues (2004) of differences between cognitive states in people with PTSD, suggesting that apprehensive arousal is a key feature of comorbidity in the disorder.

In addition, the coordination of distributed neural activity has been functionally related to high-frequency oscillations, including the beta frequency (Rodriguez et al., 1999; Roelfsema et al., 1999; Ward, 2003). Therefore, abnormality in electrophysiological frequency patterns in PTSD participant data lends support to the earlier findings of a decoupling of fronto-parietal regions in PTSD by Shaw and others (2002) and later by Clark and colleagues (2003) using neuroimaging data. As discussed, differences in the beta frequency between groups were not supported by the current data, but there were indications that the beta frequency patterns were altered in all three groups. In contrast, Begic and colleagues (2001) propose that their finding of increased beta activation in the resting EEG in Croatian war veterans is associated with attentional disturbances and possibly medication effects. However, in the current study, the inclusion of a mixed gender control group from community samples, with high reactivity to self-report traumatic events, may have contributed to the non-significant result for the beta frequency in the PTSD group.

On the other hand, one possibility for the low alpha amplitudes in the current PTSD group may be due to medication effects, as suggested by previous researchers (Begic et al., 2001; Jokic-Begic and Begic, 2003; Veltmeyer et al., 2006), that involve the normalisation of alpha frequencies in tonic resting states but not in phasic states.

Responsiveness to stimulus type is not implied in the differences between high and low amplitudes in the respective groups. However, learning in the wider literature has been associated with the updating of information on a temporal gradient (Sejnowski and Tesauro, 1989; Howard et al., 2006; Hoge and Kesner, 2007), involving phase synchrony between rhythmic oscillations (Ward, 2003; Palva and Palva, 2007; Knyazev et al., 2006b). This model of learning is in agreement with findings in the PTSD literature (Protopopescu et al., 2005;
Vasterling et al., 2006; Williams et al., 2006). It has also been demonstrated that integrated, cooperative, synchronised assemblies of neurons enhance orientation and discrimination (Basar, 2004; Singer, 1999), and that synchrony is dependent on temporal patterns, correlated with the orientation and coherence of visual stimuli (Singer, 1999; Munk, 2001).

In this study, a plausible hypothesis is that the type of trauma experienced may have biased perception to a more alert state in the PTSD group and in those who reported traumatic stressor events in the NC group, resulting in similarities in the activation pattern for resting alpha frequency in these two groups. On the other hand, early experiences with trauma in the TEC group may have altered cognitive control mechanisms for a greater reliance on memory structures rather than visual structures for the encoding of new information associated with compensatory mechanisms for processing information. Alternatively, it is possible that the results are due to other factors related to brain activity and preparatory states for stimulus processing, and that there is no relationship between perceptual discrimination in the PTSD group and the NC group.

Optimal performance has also been modelled using statistical probability as an attention bias (Dayan et al., 2000), and such arguments, taken together, may be consistent with a plausible explanation for the alterations to cognitive control mechanisms in PTSD. This interpretation of the results also poses the question of whether numbing is a control mechanism in people with PTSD. Differences between automatic retrieval and conscious, voluntary retrieval, might suggest differences between mastery, or procedural skill, and conditioned associative links between stimuli, consistent with individual differences (Van Horn et al., 1998; Eichenbaum and Fortin, 2005; Niebauer, 2004; Chemtob and Taylor, 2003). Conditioned anticipatory mediating states are functionally different from recalling a past event and reward expectancies have been associated with processes that activate a behavioural system that is different from that which is activated when recalling information of a general nature (Britton et al., 2005; Ramos and Savage, 2003).

Furthermore, recent proposals have been made that “mirror” neurons, found in parietal and pre-motor cortical networks, can be activated or deactivated in compensatory strategies for monitoring and predicting information of a social nature (Gallese and Lakoff, 2005). Mirror neurons, or VENs, in the ACC and fronto-insula cortex for the control of vocalisation, visceral functions and emotion integration (Watson et al., 2006), may prove to be useful mechanisms for interpreting specific symptom types such as numbing and avoidance behaviours in PTSD that have been shown to be resistant to treatment (Nemeroff et al., 2006).
Uncertainty in stimulus discrimination has been a key finding in cognitive abnormality in PTSD research (Felmingham et al., 2002; McFarlane et al., 1993) and may be functionally related to cognitive control mechanisms for emotion regulation implicated in a number of psychiatric presentations (David et al., 2004; Salustri et al., 2007; Sotres-Bayon et al., 2006; Williams and Moulds, 2007). The data from the current study provide support for the idea that numbing and avoidance are associated with different pathways in anterior and posterior regions between groups, and are associated with different mechanisms for attention modulation. This interpretation of the findings converges with suggestions by Vasterling and colleagues (2006) that, from an evolutionary perspective, attention mechanisms modulate the response patterns of people faced with extreme stress.

Furthermore, the current findings may offer an explanation for resistance to treatment. That is, the current data predicted a strong relationship between numbing and anterior cortex activation patterns, associated with alpha asymmetry in the PTSD group, while predicting a strong relationship with avoidance and posterior region activation in the TEC group. Clark and colleagues (2003) proposed that the ineffective use of executive control in working memory systems, together with reduced dependence on symbolic processing of perceptual and conceptual material, leads to information processing biases in PTSD. The current pattern of results, indicating numbing associated with relatively left-brain anterior alpha asymmetry and anxiety associated with relatively right-brain alpha asymmetry, in PTSD participants, supports a model of inefficient executive control, which implicates a resistance to change.

Focused attention, rather than attentional scanning, has been proposed as a mechanism for the facilitation of certain classes of information by numbing feelings and thereby reducing distress (Felmingham et al., 2002; Keller et al., 2000; Smith et al., 2003). According to Banich (2004), a numbing of attention would produce a block in cortical or subcortical communication channels, which would be linked to memory disturbances or dissociative states (Lanius et al., 2002). An alternative hypothesis is that biased attention toward threatening stimuli activates association networks for similar memories and alters interneuron feedback loops for top-down processes (Williams et al., 2006). However, it is also possible that learning-induced changes of neuronal representations may leave the response properties of synchronously firing neurons unaffected (Munk, 2001). This idea implies an involuntary response to stimuli, often reported in valence-arousal models (Kensinger and Corkin, 2004; Lanius et al., 2003), and involving the recognition of reward or aversive states and failure of extinction (Kirsch et al., 2004; Ramos and Savage, 2003), thus activating association
networks and bottom-up responses (McFarlane et al., 2002; Yu and Dayan, 2002; Compte et al., 2000).

Thus, PTSD symptoms associated with criterion C numbing and avoidance characteristics may activate different pathways for processes associated with executive functions, such as attention modulation, self-regulation, and mental flexibility. The results of this study indicated a strong relationship between high numbing and avoidance scores in the PTSD participants and reduced cognitive flexibility. However, the specific compensatory pathways were not indicated by the current data. Furthermore, numbing associations with alpha frequency patterns were not evident in the two control groups, even though there was a strong association between avoidance reactivity and posterior alpha asymmetry index in the TEC group. These results may provide a useful avenue for further investigation, particularly for a more detailed examination of the mechanisms associated with numbing response patterns and cross-frequency adrenergic receptor binding.

Well-rehearsed responses to anticipated stress stimuli might inhibit prefrontal cortex functions and shift information processing to alternative pathways. That is, thresholds for perceptual recognition have empirically been shown to be attributable to the higher probability of previous responses (Glassman, 2000; Hartikainen and Knight, 2003; Howard et al., 2006; Nadel and Moscovitch, 1998); and have been demonstrated in theoretical models of response bias (Dayan et al 2000). One hypothesis applicable to the altered patterns of functioning in people with PTSD is that recognition thresholds and connotative meaning for perceived stimuli, influenced by emotional significance, may alter the statistical probability of response bias and selective attention mechanisms (Dayan and Yu, 2003; David et al., 2004; Singer, 1999). This view is in agreement with models proposing that increased distractibility in people with PTSD is associated with altered attentional processes and response inhibition, not for target selection but for target response (McFarlane et al., 1993), and is congruent with the findings presented in this study for altered attention mechanisms associated with preparatory information processing states.

The unique pattern of numbing relationships with alpha activation patterns, and the interaction between the PTSD group and the TEC group for avoidance reactivity, may suggest mechanisms for specific patterns of spatially and temporally altered functioning in people with PTSD. One plausible argument is that the strength and form of synaptic interactions between inhibitory interneurons in the TEC group compared to the PTSD group operate in a functionally distinct way, modulated by synaptic changes as a result of learning. If the
argument for visual perception and the organisation of information is one of response selection, then current results suggest differences in the activation of anterior cingulate cortices, as modelled by Bryant and colleagues (2005). That is, perception that does not match existing response selection sequences will activate regions of the anterior cingulate cortex, likely to result in emotional states when the automatic retrieval of sensory information does not conform to the relative position of the elements in visually perceived structures (Botvinick et al., 2004; van Veen and Carter, 2002; Sakagami et al., 2006). On the other hand, the recognition of different emotions, perceived as “egocentric” and “allocentric” knowledge structures in the coding and retrieval of context information (Nadel and Hardt, 2004), has been linked to the sensory-motor system model for spatial information processing and defined as critical in automated behavioural responses for survival (Sakagami et al., 2006).

Thus, data in this study supporting the dysregulation of motor movement control in people with PTSD may also be interpreted as evidence for a temporal alteration to mechanisms associated with memory processing of spatial experiences as sequences of events, and for the temporal organisation of events that consequently disrupt the flexibility of retrieval from long-term memory associated with the activation of posterior and sensori-related brain regions. This interpretation is in agreement with models proposing that a common mechanism and shared neural substrates coordinate the activity of sensory-motor and affective neural systems, involving subcortical structures such as the caudate and cerebellum (Fuster, 1997; Schutter and van Honk, 2006; Gallese, 2003).

Extending this proposal, neuroimaging studies have provided evidence that it is possible to generate perceptual images without sensory stimulation (Hasegawa et al., 1998). This finding has supported proposals for a link between spatial processing, or sensory encoding, and episodic memory retrieval (Fletcher et al., 1995; Nadel and Hardt, 2004). As previously discussed, models of recency memory suggest that the ability to organise and segregate events in memory includes the sequencing of action and temporal tags for events, so that past knowledge influences and constrains current behaviour (Eichenbaum and Fortin, 2005; Gazzaniga et al., 2002). This interpretation may be consistent with models for a sensory-motor memory capacity (Fuster, 1995), positing the ability to predict upcoming sensory events and to predict the expected consequences of action (Gallese, 2003). The above interpretation implies the conscious or compensatory involvement of self-regulatory mechanisms in people with PTSD and a dysregulation of automatic voluntary control associated with parietal regions and inhibitory mechanisms, which was indicated by the current pattern of results.
The hippocampus has been shown to play an important role in short-term storage and learning of new information (Lindauer et al., 2005). Early studies documenting poor memory function in PTSD patients, as evidenced by reduced verbal memory and visuospatial recall, have supported the theory of hippocampal damage in PTSD (Bremner et al., 1993, 1999). More recent evidence in animal studies has implicated the regional specificity of hippocampal regions and receptor-dependent synaptic plasticity mechanisms (Fuster et al., 2000; Sakagami et al., 2006). Models of brain receptor-dependent synaptic plasticity function are in accord with anatomical specialisation views, arguing that subregional hippocampal specificity plays an essential role in learning and memory, particularly for temporal information (Hoge and Kesner, 2007).

The current results converge with findings of altered frequency patterns in PTSD (Begic et al., 2001; Jokic-Begic and Begic, 2003; Veltmeyer et al., 2006) and disruption to working memory systems (Clark et al., 2003; Vasterling et al., 1998; Attias et al., 1996; Galletly et al., 2001; Veltmeyer et al., 2006). Furthermore, the current pattern of result may also be in agreement with an interpretation that the role of the right hemisphere in specialised spatial working memory function in people with PTSD is related to language-based interpretations of certain patterns of familiar stimuli (Lanius et al., 2004, 2005; Nadel and Moscovitch, 1998). Symptom patterns in PTSD may be associated with activation involving neuromodulatory processes influencing the build up of patterns and of competition between patterns (Lanius et al., 2002; Britton et al., 2005). This proposal is reasonably associated with rehearsal systems for the short-term storage of knowledge representations as semantic codes and thus information processing may become distorted by recency effects and altered hippocampal functioning (Lindauer et al., 2005; McFarlane et al., 2002; Nadel and Moscovitch, 1998).

Of particular significance, the present research identified the possibility that the lower performance scores for phonemic categories of word production in the PTSD group might be speculatively associated with a similar finding by McFarlane and colleagues (1993) for the poor discrimination of auditory signals in PTSD and a disruption to attention mechanisms associated with numbing responses (Felmingham et al., 2002). That is, the oral production of phonemic categories of words has been associated with left-hemisphere modality-specific working memory processes (Spreen and Strauss, 1998) for the discrimination of sequences of auditory information. However, it can be inferred that the discrimination of items and the labelling of those items are pertinent to confidence in decision processes and to the reliability of prediction for anticipated stimuli in optimising behavioural functioning (Dayan et al., 2005).
An inability to discriminate items for optimal decisions would also speculatively be associated with irritability, confusion, and poor memory and a tendency toward familiarity (subsumed under theories of appetitive functioning), involving recognition or alertness (Cooper et al., 2004; Kensinger and Corkin, 2004); in contrast to processing new information modulated by attentional scanning strategies and processes of awareness for self-regulation (Aston-Jones et al., 1999). This proposal may be applicable to mechanisms associated with detecting any incongruence in frequency patterns associated with cortico-thalamic loops involving the mPFC and cerebellar activity. The hypothesis that the sequencing of information, as a left-lateralised function, becomes dysregulated in people with PTSD, may be an important proposal for future research investigating the dynamic relationship between structural and temporal channels of information processing.

Based on the above models, an abnormality in processing and encoding novel stimuli may result in dysfunction in people with PTSD, through mechanisms associated with the failure to update memory stores, particularly for threat-related stimuli. However, the capacity for self-regulation may be intact and compensatory mechanisms may indicate that a processing system is organised to process stimuli through extended long-term memory stores rather than working memory short-term stores for the maintenance of information (Chein et al., 2003; Glassman, 1999). Furthermore, the oscillation patterns between frontal and posterior regions have been implicated by the current results as altered in people with PTSD. Based on results showing that EEG activity in resting states is altered for the preparation of mental activity, and on results showing an asymmetry in relationships with numbing and avoidance responses in PTSD participants, there may be benefits in extending these results to provide a deeper understanding of how phase-locked connectivity between regions is altered in PTSD. In addition, as a plausible mechanism for alterations to information processing, on a moment-by-moment basis in people with PTSD, temporal contiguity as a function of altered frequency signals may be relevant to the phasic and remitted symptom characteristics of PTSD.

The disruption to functioning, as assessed by neuropsychological performance indices in this study, was interpreted within the framework of parallel distributed processing and the loss of cognitive flexibility in people with PTSD. However, questions addressing differences between the maintenance of semantic rehearsal processes and context information related to a specific event associated with self-registration were not examined by this study. From a psychological perspective, the above hypothesis implies processes associated with the medial prefrontal cortex, particularly the role of the hippocampus in the temporal dynamics of
information processing and emotion-regulation, which was not measured by the current pattern of results.

Limitations:

There were various methodological limitations pertinent to this study. One limitation of the current study was the difficulty in isolating and holding constant any compounding factors associated with the construct of laterality. As indicated by various models presented in the literature review, neural network patterns can be altered through probabilistic parameters of functioning, and the various measures adopted by the current analyses do not support a modality-specific hemispheric localised pattern of information processing. This is particularly relevant for tasks such as the maze, used to assess right hemisphere nonverbal processing. Previous studies have shown that the maze task is multifactorial in processing demands and involves many regions of the cortex (Caplan et al., 2001; Fletcher et al., 1995; Van Horn et al., 1998). In addition, in more recent neuroimaging studies of emotion, empirical evidence has supported a complex interaction of regions associated with emotional activity (Wager et al., 2003). The role of stress in cognitive functioning suggests further implications for assessing modalities of information processing underpinning neural functions.

Another limitation of the current study was related to the nature of the samples. Subtypes for the PTSD group and the NC group were not controlled in the current analysis, and as has been suggested in previous research (Metzger et al., 2004; Begic et al., 2001), this factor may lead to diverse interpretations associated with the pattern of interneuronal connections for excitatory and inhibitory functions and the synchronisation of large groups of neurons in resting EEG states. This may be a relevant consideration when conducting analyses using a mixed gender sample and also for any differences in reporting of time since the stressor event.

Furthermore, the sample-dependent limitations relevant to this study are also germane to conclusions drawn from self-report measures using control participant data. The number of responses for each of the CIDI scales did not meet the full symptom criteria for PTSD in the two control groups and the results presented must be accepted cautiously. As previously discussed, medication effects in the PTSD participants may have influenced the results associated with frequency amplitudes and these were not controlled in the current analyses.
**Conclusion:**

The results of this study demonstrated a pattern of cognitive deficits in the PTSD group related to verbal working memory and relationships between comorbid mood states and prefrontal cortex asymmetry patterns. Thus, the current data extend earlier research findings that suggest compensatory mechanisms are a system of altered functioning in psychopathology, by demonstrating that there are differences in the pattern of modality-specific working memory systems, which are associated with specific resting cortical power values in people with PTSD and in people with strong reactivity to previous perceived traumatic stressor events.

Two elements of functioning, that are fundamental to an altered pattern of working memory processes in people with PTSD, were linked to preparatory resting states. The first was inferred as an alteration in neural connectivity patterns in people with PTSD, indicated by slowed information processing and altered amplitude patterns in the resting EEG. The second was indicated by rhythmic asymmetry patterns associated with a shift in brain organisation pathways, for information processing and an association with symptom patterns for higher-order or lower-order neural activation. These were demonstrated for numbing and avoidance reactions (category C symptoms) and correlated with specific brain regions and modality-specific cognitive processes.

A key finding from the pattern of results indicated that alpha power asymmetry in preparatory states is a common variable in a compensatory mechanisms hypothesis for brain reorganisation after severe psychological distress. The data demonstrated preliminary, compensatory pathways associated with alpha asymmetry patterns and the unique contribution of numbing and anxiety associated with resting states in PTSD participants. A different pattern of avoidance and alpha asymmetry in resting states was demonstrated for normal participants with previous trauma experiences. The findings were suggestive of neural plasticity and structural alterations to the pattern of cortical resting states in people with previous strong reactivity to traumatic stressor events and may be linked to a mechanism maintaining PTSD symptoms.

In addition, by examining the relationship between alpha asymmetry in frontal and posterior regions, and symptom characteristics of numbing and avoidance, the current study extended prior research findings to reveal that there are specific patterns of brain organisation associated with numbing and avoidance as separate, and definite sub-processes in
compensatory mechanisms, and in subtypes associated with hypervigilant or alert states (category D symptoms). The role of hyperarousal in cognitive processes is implicated in these findings.

Given the limitations of the data, and the above arguments, this then raises the question of how automatic retrieval of information is best compared to the controlled, voluntary retrieval in conscious processing. The models presented in this study are useful in suggesting that a common variable in brain organisation patterns, distinguishing pathways for information processing, can be plausibly identified as alpha power (low for PTSD and high for TEC) and the asymmetry of relative left- and right-brain activation patterns associated with emotional response patterns. Further investigations using statistical modelling techniques may be helpful in future research to detect predicted patterns of information transfer in cortical and subcortical structures associated with pattern matching for memory retrieval processes and emotion regulation.
8. Overview and General Conclusions

8.1. Complex Neural Interactions in PTSD

The conclusions derived from the pattern of results in this study were framed within a parallel-distributed model of brain organisation and a compensatory mechanism hypothesis, proposing neural reorganisation patterns in PTSD. This proposal suggests that arousal patterns vary between stress and motivation (Bremner et al., 1999; Lanius et al., 2005), and that switching attention recruits and modulates the entire working memory network for local networks of verbal and visuospatial modalities (Clark et al., 2003). This study is a first approach investigating how spatial information might be related to symptom categories for reduced verbal working memory in PTSD, and for compensatory shifts in resting EEG patterns as an adaptive strategy associated with traumatic events.

The discussion for each of the analyses presented in this study was drawn from a wide and extensive body of literature, thus synthesising findings from the general literature and from empirical research in PTSD. The current pattern of results also indicated and provided further support to previous hypotheses and models proposing structural and functional alterations of brain organisation in people with previous traumatic experiences. The study was a novel attempt to move beyond traditional information processing models of computational internal representations, by examining, and considering, the functional role of neural synchrony patterns associated with working memory and attention systems.

At a general level of analysis, a consistent finding in this study was for lower frequency patterns in PTSD participants, which implied specific neural alterations at the cellular level. As a mechanism for structural reorganisation, lateralisation associated with timing mechanisms, for the coordinated flow of information, is fundamental to memory functioning at the neural level of analysis (Basar, 2004; Elzinga and Bremner, 2002; Protopopescu et al., 2005). In an attempt to understand the complex relationships of brain organisation patterns associated with trauma reactivity this study demonstrated that a common mechanism is involved in alterations to psychological and physiological indices of functioning. Thus, the results and subsequent interpretations provide numerous avenues for future investigation, both at the mechanistic level and also for intervention strategies aimed at normalising the functional level, and understanding the complex syndrome of PTSD.

From a psychological perspective, the demonstrated differences in resting cortical patterns in the alpha frequency, between different sample populations, suggests a common mechanism
for altered functional patterns in learning and memory. The focus on cortical asymmetry patterns, as a mechanism for altered neural pathways, is relevant to memory functioning associated with modality-specific alterations in working memory systems, and may be a useful area of investigation in understanding the fundamental differences in psychopathology and, in particular, psychiatric disorders with complex aetiologies and symptom overlap.

Specialised verbal and non-verbal processing tasks were not found to be significantly different between groups. However, the data for the retrieval of information from long-term memory and episodic memory supported an altered pattern of functioning associated with verbal working memory in people with PTSD. The altered pattern of behavioural functioning was interpreted as converging with evidence of a disruption to left-hemisphere circuits and disruption to the sequencing and flow of information in people with PTSD (Clark et al., 2003). This interpretation was consistent with models suggesting an abnormality to attention (Felmingham et al., 2002), and also to retrieval of memory associated with learning processes in people with PTSD (Vasterling et al., 1998, 2006). The current findings are also in accord with models arguing for abnormal executive functioning in people with PTSD, and with deficits to working memory storage functions at the tonic or baseline level of information processing (Clark et al., 2003; Galletly et al., 2001; McFarlane et al., 2002); implicating a compensatory shift in parallel distributed processes for the coordination and flow of information.

At the structural level, localisation differences in EEG resting patterns, correlated with behavioural indices, and as reported in this study, also supported models of PTSD that suggest a disruption to information processing as a secondary characteristic of trauma experiences (McFarlane et al., 2002). The current results indicated a consistent pattern of reduced speed of processing and functioning in people with PTSD. An altered pattern of functioning was also indicated in people in the control group who reported a pattern of strong reactivity to previous traumatic stressor events. Hypotheses for mechanisms involving abnormal working memory systems, particularly verbal working memory in PTSD, and the role of compensatory mechanisms for altered functioning after traumatic experiences (Clark et al., 2003; Bremner et al., 1999), were supported or partly supported by the data. The current pattern of results suggested asymmetry patterns at local or structural levels of brain organisation might be a plausible mechanism for brain reorganisation involving working memory systems associated with timing mechanisms that change behavioural functioning.
On the other hand, without an analysis of emotional reactivity patterns, the preliminary results of this study provided only partial support for a shift to right hemisphere coding and to neural plasticity at the local level of cognitive architecture in people with PTSD compared to a normal control group. Two reasons for this result might apply. Firstly, an overall analysis of global cortical power does not account for region-specific differences in brain organisation patterns. Secondly, an examination of power differences with the exclusion of other variables does not account for the distribution of variance in each sample group. Therefore, an assessment of how variance between different variables is distributed within and between groups has demonstrated the importance of individual difference factors, and the need to address subtypes in future research, as has been suggested previously (Metzger et al., 2004; Begic et al., 2001; Kashdan et al., 2006; Lanius et al., 2002). Exploring differences associated with hemispheric asymmetry of psychological and physiological phenomena indicated a deeper level of understanding associated with PTSD functioning. By integrating different perspectives about how localised patterns can be altered in brain organisation enabled certain factors to be associated with variance structures in the coordination and integration of information processing at the implementation or global level.

8.2. Methodological Limitations of the Study

A number of methodological limitations in this study are outlined for an understanding of why the preliminary analyses did not suggest a pattern of differences between groups. Furthermore, for a clinical application of this study, one question relevant to the data was whether a modularity thesis, for differences in functioning between higher-order and lower-order levels of functioning, is observable in psychopathology. In view of the conclusions drawn, an analysis of how the study might have been improved focuses on epistemological and epidemiological parameters, which are considered below.

One difficulty with understanding PTSD phenomena, from a mechanistic perspective, is that each module can be studied by how a task is performed, thus creating problems for the identification of the boundaries of each component, particularly for behaviour associated with cognition and emotion (Schutter and van Honk, 2006). Therefore, as previously mentioned, and based on a modularity assumption, an analysis of right hemisphere processing in people with PTSD does not provide evidence of the mechanisms that produce behaviour, but can only identify a subsystem that is part of how the system works. At a local level of investigation this may be useful in providing future research endeavours with a guide to baseline patterns in the characterisation of the functional organisation of behaviour in people.
with PTSD. However, it does not provide details of causal properties or learning mechanisms for symptom manifestation.

*Implied lateralisation and task demands:*

The current analyses were based on the assumption that behavioural indices of lateralisation can be measured by tasks performed using standardised average scores collected from a computerized neuropsychological battery of tests. However, as has been previously discussed, the construct of laterality is not modality-specific and is difficult to define using traditional neuropsychological measures. Therefore, holding all task parameters constant, to define laterality measured by the selected verbal and nonverbal tasks, was limited to a localisation of structural parameters focusing primarily on cortical asymmetry. Hence, the methodology adopted by this research does not reflect a functional analysis of information processing in distributed cortical systems and consequently cannot accurately reflect the extent of dysfunction in working memory systems associated with modality-specific processes. A different approach to using the current neuropsychological indices of lateralisation would have been to investigate more closely the extent of similarities across tasks as differences between groups. Further research would benefit from the design of parameters that allow for a componential examination of modality-specific tasks, as separate constructs, in different domains of information processing.

Another consideration in understanding a lack of differences between groups is the question of whether all people with PTSD have right-hemisphere coding patterns. An examination of the complex overlap in symptom categories and the difficulty in objectively classifying information as threat-related has led to inconsistencies in the literature and poses a methodological limitation in the current study. Furthermore, and as previously mentioned from findings utilising neuroimaging methodology (Wager et al., 2003), the difficulty of identifying boundaries for each of the component modules in a mechanistic perspective, leads to difficulties in understanding PTSD phenomena.

One element of functional analysis that was considered in this study, and related to the query of whether people with PTSD have a preference for right hemisphere coding, questions the purpose that symptom categories serve in goal-directed behaviour. Goal-serving mechanisms can be shown to be products of selection processes, and the interaction of expressions of psychological activity that fulfils the needs of an individual, based on their cognitive capacities (McFarlane et al., 2002; Lanius et al., 2005). Hence, numbing and avoidance
behaviours can be considered from the perspective of theories of memory that postulate that
how information is organised is a key to retrieval processes, with automatic retrieval serving
as a functional adaptation mechanism (Fuster, 1995; Nadel and Moscovitch, 1998). Furthemore,
language-based knowledge is a mechanism for the organisation and integration
of different brain functions in different contexts (David et al., 2004; Seger and Cincotta,
2006). From this perspective, core meaning consists of references or schema, through which
words are an important link between the external world and the rules for behaviour, and from
which expectations for a particular task can emerge as required by the goals of a task (Ehlers
and Clark, 2000; Conway and Pleydell-Pearce, 2000).

However, the complexity of understanding symptom categories in brain organisation is
related to the different levels of integration of psychological and physiological functions.
Therefore, another limitation of the current study is related to how the investigation of the
relationship between symptom items and resting neural states was conducted. The lower
performance in cognitive tasks in participants with PTSD were taken as correlates of
synchronous oscillatory activity and the relationships between frequency patterns and
amplitude differences were investigated for structural differences in working memory by
investigating self-reported perception of traumatic experience and reactivity to those
experiences. As a methodological shortcoming of the current study, an analysis based on
simple correlation patterns does not provide evidence that such a relationship contributes to
symptoms or their maintenance. In particular, while a relationship between variables can be
established, the causal nature of the relationship cannot be provided.

*Small group sizes and uneven group sizes:*

However, fundamental to an examination of whether neural patterns associated with
information processing in people with PTSD are right-hemisphere biased, it is also necessary
to address the question of whether all PTSD sufferers have altered right-hemisphere
functioning. One of the limitations of the current study was the small clinical sample for
PTSD. Based on a small sample size, it was not possible to examine distinct symptom clusters
for a comparison of functional relationships using an independent-subjects design. Therefore,
a comparison of self-report measures was used to examine responses associated with previous
perceptions of traumatic stressors in a matched-samples design. In many of the analyses,
effect sizes were small, and possibly associated with a type II error (false negative). In
addition, the repeated measures design may have biased results for a type I error (false
positive). Therefore, the current analyses and results, for a right-lateralised pattern of functioning at the local level in people with PTSD, require replication.

Furthermore, alterations in amplitudes and interactions associated with resting EEG asymmetry patterns, between groups in this study, may have been related to the size of the samples and the methods used; thus, failing to detect a clear laterality bias in people with PTSD. It has been proposed that different factors influence the EEG, such as genetic and environmental factors (Zietsch et al., 2007; Niedermeyer and Lopes da Silva, 2005). Therefore, based on earlier suggestions that sub-typing might provide a useful methodological consideration in PTSD research (Begic et al., 2001; Metzger et al., 2004), further investigation is required with larger groups for an understanding of the various functional roles of frequency patterns in people with PTSD.

However, the lower performances on neuropsychological tests, together with indices of desynchronised arousal states in people with PTSD, implicate differences in specialised hemispheric local interactions. The current results suggest specific functional roles for electrocortical rhythms, particularly in the lower frequencies for rehearsal processes and in the higher frequencies for the recognition of familiar items, implicating confidence in decision processes.

The analyses conducted in this study do not account for the numerous variables that may influence interactions between the perceptual-motor dichotomies of memory retrieval. A limitation of this study was that statistical analyses were low in power, and failure to detect differences may have been avoided using more powerful statistical techniques and methods (Bagiella et al., 2000; Smith et al., 2003). The analysis of complex interactions involving multiple variables requires specialised techniques and algorithms to understand how computationally complex problems are solved in distributed brain systems that may be subject to neural plasticity (Cacioppo et al., 2000; Robinson, 2003).

Statistical artefacts and the problem of false positives:

As a comparison study between two groups, the results for this study demonstrated little variation between the sample distributions. However, as a three-group design, there was a tendency for statistical artefacts to be associated with the mixed nature of the normal control group. This study identified differences in physiological indices between age-matched groups. However, drawing samples from a standardised database for the comparison of retrospective
self-reported experiences associated with previous trauma introduces problems associated with screening techniques for normal participants compared with clinical participants. In particular, objective indices associated with previous trauma experiences were not administered to the normal group. Therefore, it is possible that a true indication of trauma reactivity in community samples was not portrayed by this study because a full set of minimum criteria defining trauma reactivity patterns using the CIDI (World Health Organization, 1993) was not obtained for the control group. A true indication of reactivity to traumatic stressor events may have been confounded further by the administration of computerised and highly standardised interviews rather than clinician administered interviews (Komiti et al., 2001). As discussed in Chapter 6 of this study, statistical artefacts may have also arisen from the formation of subgroups based on self-reported trauma experiences.

After early childhood traumatic experiences, people with altered psychological functioning may adopt various stylistic strategies for coping with stressor events (Schore, 2002; Teicher et al., 2003). As mentioned above, one limitation of the current study was the investigation of a mixed pattern of self-reported stressor events. Only patterns of adult trauma were examined in people with PTSD, while a mix of self-reported childhood and adult traumatic stressor events were examined in the control group. Therefore, it was difficult to compare results with previous studies. The interpretation of resting EEG patterns, particularly for higher amplitudes in those reporting strong patterns of trauma reactivity (TEC group), were not clearly identified as premorbid or related to trauma reactivity variables. Thus, the pattern of tonic cortical alterations for the TEC group were interpreted as consistent with neural plasticity (McFarlane et al., 2002) and supported the idea that alterations at the tonic or local level occur as a result of reactivity patterns to experiences of perceived traumatic stressor events (Vasterling et al., 2006). However, this interpretation was based on results from predictive models and for a limited number of variables and may be subject to false positives and/or statistical artefacts.

Furthermore, relevant to a knowledge base or structural level of analysis, semantic patterns for classifying environmental stimuli are an important attentional scanning mechanism for cognitive flexibility and self-regulation (McFarlane et al., 2002; Svoboda et al., 2006). However, the possibility that time since trauma may represent an ontogenetic variable contributing to high stress reactivity in some people after traumatic stressor events could not be easily identified using the current methodological techniques. In addition, the finding of a higher proportion of women in the high reactivity normal control group also raises questions about the validity of the methods used for subgroup formation in the current study.
Mixed gender and mixed event variables:

The finding of a gender difference in the control group with high reactivity to perceived stressor events is an interesting result, and may be pertinent to an understanding of the way information is organised by the visual system and then categorised for identification and for responding based on temporal parameters (Protopopescu et al., 2005). This interpretation might be in agreement with attention modulation based on a two-stage model of spatial working memory processing within the lateral frontal cortex (Petrides, 2000). The organisation of information changes as contexts change or when attention shifts (Vasterling et al., 2006; Ribary, 2005). Thus, patterns are internally derived by context and the discrimination of stimuli is based on past experiences (McFarlane et al., 1993; Lanius et al., 2003). The current findings, of differences in the TEC group, may be in agreement with models proposing that retrieval based on pattern matching of features is less effortful than pattern matching for function (Klein, 1998; Seger and Cincotta, 2006). On the other hand, the finding may have been an artefact of group selection processes and requires further investigation.

Furthermore, the finding of a different pattern of resting states and functioning, in the high-reacting control group, raises another set of questions; in particular, implicating gender differences and the reorganisation of brain patterns after stressful events. Region-specific differences in brain organisation patterns have been associated with gender and the lateralisation of emotional activity (Wager et al., 2003). Therefore, important to the pattern of current findings, is the question of differences between content and context information and whether there is a gender-bias associated with language-based meanings. Consequently further enquiries probing the role of dissociative symptoms, as a gender-bias for coping with overwhelming events, may be useful to understanding the neurobiological mechanisms of memory in people with PTSD and psychological disruptions to self-regulation. Theories suggesting a compartmentalisation of events, as a self-protective mechanism (Lanius et al., 2002, 2005; Spitzer et al., 2004; Klein, 1998), may be relevant to the findings reported in this study for the trauma-exposed control group. Based on these theories, inconsistencies in beliefs can be supported by memory structures because different contexts enable the myriad permutations of how a memory can be accessed.

Analyses conducted with a mixed gender group represented an important element of the design of this study, but also suggested methodological limitations in an examination of memory functions associated with trauma reactivity. Linked to a possible gender-bias for
content and context information is the characterisation of patterns of reported reactivity to stress. This notion is important for understanding differences in tonic alpha resting patterns. Differences in gender have been repeatedly reported in analyses of subjective reactivity to threat (Nemeroff et al., 2006; Fullerton et al., 2001; Veltmeyer et al., 2006) and associated with alterations to right hemisphere processing (Foa and Street, 2001; Lanius et al., 2002; Protopopescu et al., 2005). As a consequence, the findings of the current study may have been masked by the differences in gender and warrant further investigation, particularly for a replication of right hemisphere posterior resting EEG activation patterns and altered attentional scanning mechanisms.

The self-report measures for the three groups assessed in the current study suggested that a continuum of events might be instrumental in a threshold of learning and in behavioural responses. On the other hand, mechanisms relevant to the pattern of results in the PTSD group may be associated with temporal alterations in the flow of information processing and may be consistent with phasic shifts in working memory processes and consistent with an uncertainty of appraisal hypothesis (Metzger et al., 2004; Felmingham et al., 2002; McFarlane et al., 1993).

This study examined aspects of learning (by using a maze paradigm), where learning may depend on the conditions under which an event occurs. Thus, learning associated with encoding and retrieval mechanisms may complicate an analysis of psychological functioning by the threshold for asymptotic reactivity. In other words, differences in function may depend, not on the number of events, but on the temporal precision of the interaction between a stimulus and the response (Ward, 2003; Ramos and Savage, 2003; Hoge and Kesner, 2007). Delineating these ideas by examining patterns of self-report appraisals for trauma events, the current study predicted that likely differences in the local specialised processing of verbal and nonverbal cognitive tasks would be difficult to detect using the chosen design of measuring accuracy scores in neuropsychological tests.

A further difficulty in understanding lateralised patterns of functioning, when using a mechanistic approach, is the limitation of not establishing the conditions under which learning occurs. As previously discussed, establishing the time elapsed since a traumatic experience occurred may be a useful variable for understanding appraisal mechanisms and learning processes in people with PTSD. In addition to verbal and spatial processing mechanisms, the role of time as a dimension in working memory processes, and in stress reactivity patterns, has received little investigation and requires further clarification.
Results of this study were in agreement with views that differences in the types of traumatic stressor events may be indicative of an atypical stress response that alters catecholamine systems selectively (McFarlane et al., 2002). In addition, the current findings are in support of previous findings that alterations to functioning occur as a result of learning (Vasterling et al., 1998). Therefore, higher-order cognitive processes are implicated, as appraisals involve consequent changes to rules for predicting stimuli (Lanius et al., 2005). However, a full examination of these processes was not undertaken and the current study did not examine age-related differences by splitting groups into younger and older participants. Thus, an assessment of age-related differences may be of value in future research to examine underlying mechanisms of spatial working memory sub-processes associated with hippocampal function and retrieval mechanisms.

In addition, age-related differences in contextual binding of information, particularly in older people, might imply a role for the hippocampus and the substructures involving episodic memories for the maintenance and coordinated flow of information (Howard et al., 2006). A decrease in the ability of older people to recover temporal contexts and to time-tag item specificity may be related to lateralisation of functioning after traumatic stressor events and compensatory mechanisms to reduce distress.

The preliminary current analyses indicated that time elapsed since the event was an important variable associated with symptom severity. Because of the experimental design, and the selection of participants, this variable was not examined in detail in this study. However, results indicated that it contributed significantly to differences between groups and may be a variable associated with learning and the updating of memories. The implications of this finding are relevant to whether early intervention prevents chronic maladaptive behaviours, or whether specific patterns for resilience are implemented by changes in cortical structure. This may be a useful hypothesis for understanding the functional role of cortical frequency patterns and pathways for information processing.

Age is also a relevant consideration for when the traumatic event first occurs (Teicher et al., 2006) and how long after the event the memory remains active by continued retrieval processes, stimulus generalisation and sensitivity to specific classes of contextual cues (Ehlers and Clark, 2000). An understanding of brain lateralisation patterns associated with appraisal mechanisms must also include different dimensions of time. Firstly, for the invariance of an object, but more importantly, for the experiential processing of that object. Secondly, the
space that an object occupies is relative to other objects and underscores the importance of context information in interpretative functions. Therefore, as previously discussed, meanings and appraisals can alter for the same class of stimuli depending on context information and the speed with which fragments of information are matched to events that carry a high probability of predicted certainty (David et al., 2004).

*Lack of intermediary processes examined in behavioural variables and component processes in task execution:*

A plausible hypothesis concluded by this study is that left-hemisphere language-based processes and rehearsal mechanisms mediate the switch from visual or perceptual cues to psychological functions depending on familiar context information. By conceptualising local connectivity patterns, not as dichotomous verbal and nonverbal specialised patterns but rather as perceptual-motor patterns influenced by goal-directed optimisation in computational-representational systems, the complexity of the cognitive processing systems may be influential at a tonic or phasic level. This hypothesis may offer an explanation for the current pattern of differences between people with PTSD and people in the control group with strong stress reactivity patterns.

However, learning changes over time and the intermediary processes associated with learning are not detected at the behavioural level of functioning. Investigating psychological functioning from retrieval processes, and correlating these to preparatory tonic or resting states, leaves numerous sub-process and stages of information processing unaccounted for. Both at the behavioural level, and at the physiological level, further research is warranted to understand how preparatory states determine retrieval processes for behavioural functioning.

As a retrospective study, the current data are relevant to a mechanistic orientation. This perspective focuses on the underlying question of what function the PTSD symptoms serve in the adaptive response to previous events and to the reduction of stress. An analysis of goal-directed activity must account for the selection processes of past behaviour and involves recognising processes at different levels of organisation and the interactions between the different levels. Using this argument, Bernsten and Rubin (2006) posit that in explaining teleological phenomena, it is important to show how a phenomenon at one level is situated in a higher-level context and is present because of what it contributes to that context. In PTSD research, Lanius and colleagues (2005) also suggest that a goal serving function is present in a current situation because its presence in a previous situation aided survival. However, Lanius
and colleagues propose that what is important in the new situation is that selection is operating on a level higher than that of the goal-serving device. In the study of PTSD, an underlying theory to support these ideas corresponds to a threat expectancy model (Litz et al., 2000; Ehlers and Clark, 2000), where current behaviour is constrained through past experience.

Incorporating the above theoretical notions, the analyses presented in this study considered how the different elements of functioning influenced neural connectivity. However, the various connectivity patterns and pathways for information processing were not investigated as dynamic indices associated with task processing in this study.

_Pre-synaptic potentials rather than post-synaptic followed by pre-synaptic for an assessment of entrainment:_

As a main focus, this study examined structural, local level processing patterns, to assess how attention mechanisms in anticipatory states differ between people with different perceptions of traumatic stress. The focus of this research was not on identifying the parts of the system as performing component cognitive tasks, but the neural structures that the tasks may be related to. That is, a beginning state in information processing, assessed as preparatory states for sensory stimulus processing in brain organisational patterns, and an end state, or response stage, that includes behavioural performance indicators (symptoms or accuracy scores). Hemispheric specialisation is a structural component that aids adaptive function (Gazzaniga et al., 2002). Therefore, identifying a component part of the system that is disrupted in people with PTSD may reveal how the system is inter-related at a functional level of analysis. The first, or base level, element of functioning examined in this study was an analysis of whether people with PTSD have prevalence for right hemisphere coding. However, a specific limitation of the methodology was the assumption that elements in a system are related in a specific way.

_The temporal parameters of learning as a change processes were not examined:_

Differences in EEG parameters were indicated by the data for this study in people with PTSD and in those in the control group reporting strong reactivity to previous stressor events. The findings were consistent with models of PTSD positing a disruption to frontal systems and to executive control mechanisms for attentional scanning and behavioural flexibility in people with PTSD (Clark et al., 2003; McFarlane et al., 1993). However, alterations to the speed of
processing and to power differences in spectral frequencies, for those in the control group reporting strong reactivity to traumatic stressor events, were tentatively interpreted as compensatory mechanisms associated with neural plasticity and a reorganisation of neural circuits at the structural level. This finding is congruent with previous studies indicating altered information processing in people with previous traumatic experiences but without a clinical diagnosis (Britton et al., 2005; Saltzman et al., 2006; Vasterling et al., 2006).

Findings of functional differences in people with PTSD for speed of processing were supported at the biological level by a significant difference in alpha peak frequency. Furthermore, a desynchronisation in cortical activation patterns, evidenced by lower alpha amplitudes, also implicated alterations in the speed of cortical processing. Resting EEG patterns for the PTSD participants were suggestive of a difference in perceptual mechanisms for attention selection and functional memory processes. However, what the current data does not explain are alterations to resting cortical patterns in the control group who reported strong reactivity to traumatic stressor events.

As indicated in the review chapters, various modes of memory organisation for specialised functions can influence how information is integrated and rehearsed for later retrieval. Therefore, the question of whether people with PTSD have a bias toward right-hemisphere processing is a complex one. There are a number of considerations that precede an analysis of how phenomena interact in systems where objects can be defined as having a natural time dependency. In essence, the answer to the question will depend on whether investigations are directed at a goal-oriented level, aiming at understanding the functional analysis of behaviour, or whether a mechanistic orientation is pursued, and phenomena are explained in terms of past events.

The results of the current study identified alterations to the pattern of alpha frequency, possibly associated with attentional disturbances and a disruption to cognitive flexibility through the alteration of synchrony patterns and synaptic plasticity. Furthermore, there was partial support for a disruption to the higher beta frequency, which has been associated with the binding of information and with long-range connectivity (Rodriguez et al., 1999; Ward, 2003). The results for the tapping task in the current analysis were interpreted as suggestive of a diffuse pattern of neural connectivity for those in the PTSD group and also those in the control group reporting strong reactivity to traumatic stress. This interpretation may be consistent with altered patterns of beta frequency, implying reduced flexibility in people with previous traumatic stressor reactivity, and therefore warrants further investigation. As
previously reported by Begic and colleagues (2001), the beta frequency may be a potential biological marker for tonic alterations in baseline functioning in people with PTSD. Beta frequency has been associated with the implementation of information processing or the higher-level of cognitive architecture (Kopell et al., 2000; Roelfsema et al., 1999).

However, to understand how the storage components of working memory are disrupted, it is also important to understand how executive function processes operate on the system through mechanisms of attention, thought to be associated with the alpha frequency (Klimesch, 1999; Ward, 2003). In systems that activate or inhibit each other (Fuster, 1997), local operations for a stable pattern of energy regulation, can be measured in tonic characteristics as genetic predispositions or as environmental influences defined as phenotypic adjustments (Posner, 2005; Zietsch et al., 2007). Together with disruptions to procedural memory, in people with PTSD and in people with strong stressor reactivity, the differences between groups found in this study in specialised hemispheric local interactions, as indexed by electrocortical signals, suggest a distortion of context information that may be related to mechanisms of retrieval and faulty gating mechanisms. However, a comprehensive evaluation of these mechanisms was outside the scope of this study and remains to be clarified.

Furthermore, an examination of lateral dominance was not conducted in this study but may be associated with diffuse connectivity patterns in people with a mixed handedness preference, as reported in previous PTSD studies (Chemtob and Taylor, 2003; Saltzman et al., 2006). Diffuse connectivity patterns might be relevant to the pattern of high-low or low-high frequency patterns in baseline states (Veltmeyer et al., 2006) and to the subsequent retrieval of memories in specific neural pathways. Therefore, the question of attentional scanning in people with mixed laterality may be a useful avenue for further research aimed at understanding the loss of inhibitory functions in PTSD.

The hypotheses for increased reliance on nonverbal working memory processing in people with PTSD were only partially supported, and the current results remain to be clarified. The design of this study and the data indicated that there were no significant differences between groups using a maze paradigm. Therefore, as a plausible mechanism for disrupted psychological function in people with PTSD, the results do not clarify whether different aspects of spatial working memory, that are obligatory in planning functions are disrupted, or, whether faulty gating mechanisms interrupt the maintenance of information. A spatial working memory overlap with attention processes remains to be clarified. A clearer understanding of the functional role of spatial working memory processing in specific
contexts would be useful to an understanding of neurobiological pathways associated with a decoupling in frontal and parietal cortices in people with PTSD.

8.3. Implications and Relevance of Findings

An examination of behavioural indices, involving retrieval processes in this study, has provided an argument that working memory systems alter patterns of integration for content and context information. Models of working memory have been a useful framework within which to investigate decision processes and compensatory mechanisms involving neural synchrony values and cortical asymmetry patterns. The notion that attention modulates parallel distributed processing (McFarlane et al., 1993; Galletly et al., 2001; Vasterling et al., 2006), and disrupts the coordinated flow of information, was investigated in the current study by examining the qualitative and quantitative aspects of electrocortical activation patterns in resting states between PTSD participants and control participants. By examining the relationship between alpha asymmetry in frontal and posterior regions, and between symptom characteristics of numbing and avoidance, this study has extended previous findings of asymmetry in cortical arousal and brain organisation patterns in people with PTSD to show how cognitive performance indices might be disrupted.

The role of selective attention in goal-directed behaviour was explored as a primary argument for the advancement of the hypothesis that there would be differences between the PTSD group and the control group in EEG resting activation patterns and in neuropsychological tasks of verbal and nonverbal performance. This argument was central to previous findings of psychological disturbances in PTSD, as reported in the clinical literature and as postulated by vigilance theories (Felmingham et al., 2002; Ehlers and Clark, 2000). The present study supported a comorbid profile of depression and anxiety in PTSD participants compared to control participants, which is suggestive of structural and functional alterations in working memory, and convergent with findings in previous studies (Clark et al., 2003; Bremner et al., 1999).

Conclusions also speculatively supported models proposing that cellular bistability mechanisms can alter memory fields (Munk, 2001; Howard et al., 2006; Gallese, 2003) in brain reorganisation or in compensatory mechanisms associated with working memory systems. These ideas may be helpful to future research endeavours. Such models posit that spike trains induce a random drift over time in encoded cue positions when interacting with sustained synaptic inputs from excitatory feedback circuits and recurrent synaptic inhibition to produce a loss of stored spatial information (Compte et al., 2000; Holscher, 2001). To date,
only preliminary empirical evidence has been provided in the PTSD literature, but suggests that this theory is a plausible mechanism for neural compensation patterns (Jatzko et al., 2006). However, the current pattern of results indicate findings that are consistent with a reorganisation of integrative functions, which might be dependent on preparatory states and the correlates of reaction patterns associated with stress.

The organising principles of how cognition fits within brain structure were examined from a conceptualisation of working memory processing, with component rehearsal parts (or “slave systems”), related to brain structures by an executive function (Baddeley, 1986). The data for the present study primarily focused on baseline measures of functioning utilising a cross-sectional design. In this study two elements of functioning were fundamental to an investigation of working memory processes in people with PTSD. The first considered a shift in neural information processing in people with PTSD and the knowledge structures or stable units that connect to each other in the role of activation or inhibition. The second focused on the specific patterns associated with the shift in brain organisation and the role of numbing and avoidance reactions. This level of analysis included the selection processes and the emerging properties for task performance in different contexts.

At a local level of investigation, the pattern of anticipatory electrocortical resting states enables the characterisation of the stable patterns underlying cognitive operations that will determine connectivity between neural regions (Basar, 2004). Various interaction effects provided preliminary evidence of lateralised functioning in people with previous traumatic stressor experiences, including plausible pathways for inter- or intra-hemispheric asymmetry. Results suggested a contra-lateral alteration in the PTSD group for a left hemisphere prefrontal interaction with a right hemisphere posterior alteration. On the other hand, the non-clinical trauma exposed control group data confirmed a right posterior alteration to information processing mechanisms, which may also plausibly be associated with a contra-lateral interaction effect involving left hemisphere verbal working memory systems and right hemisphere functions associated with problem-solving strategies (Jausovec and Jausovec, 2000) or with blocking mechanisms (Lanius et al., 2002).

Of interest to the current study was the theory that through dendritic pruning, or synaptic plasticity, new pathways access selective memories faster than old memories, resulting in learning and alterations to association networks (McFarlane et al., 2002). Anticipatory responses and the appraisal of situations will vary depending on various dimensions of information categorisation. Therefore, the suggestion is that the selective probability of
attention mechanisms will be associated with patterns that are recognised and easily discriminated between the different elements that serve as schema for goal-directed behaviour (Dayan et al., 2000; Seger and Cincotta, 2006).

The patterns of alpha frequency in the current study differed between groups, with the PTSD group showing low amplitudes and the TEC group showing high amplitudes compared to the normal control group. This finding implicates theoretical models of LTP, proposing that memories will be strengthened through repetition, requiring only a relatively short interval intervening between the onset of activity and the pre- and post-synaptic neurons (Matzel and Shors, 2001; Klein, 1998). However, to examine this theory, and to fully understand the pattern of results in this study, a future research consideration might be to focus on the post-synaptic signals in resting states, after task completion and prior to a new stimulus onset, rather than the pre-synaptic spontaneous oscillations. This would involve an examination of where, when, and how fast information processing occurs (Duzel et al., 1999; Schack and Weiss, 2005) and be useful in understanding how different patterns of oscillations may be associated with distinct memory functions for manipulated and non-manipulated items.

Thus, in future research, better use of time-series data might identify the local, as well, as functional processes that occur over time in a distributed system. In addition, EEG data can be enhanced with the use of neuroimaging data to provide a finer detailed analysis of temporal as well as spatial characteristics of neural phenomena in brain organisation, and may provide a useful consideration for an understanding of differences between resilience and psychopathology in trauma reactivity.

The current study examined self-report appraisals of traumatic stressor events and the reactivity to those events by choosing a three-group design to understand the nature of differences between groups. The study examined structural or local level processing patterns to assess how attention mechanisms in anticipatory states differ between people with different perceptions of traumatic stress. The role of attention as a modulator in memory structures has been discussed in the review chapters, and demonstrated by the current data. Thus, a novel consideration of how spatial information is related to symptom categories for reduced verbal working memory in PTSD and for the optimal retrieval of information as an adaptive compensatory strategy for coping was formulated. In a three-group design, it was possible to show that attention mechanisms are disrupted in people with previous trauma reactivity (clinical and non-clinical participants).
Furthermore, the advantages of using standardised database techniques for the assessment of physiological indices in cortical arousal, and psychometric indices of psychological functioning, have been previously discussed (Gordon et al., 2005), and add to the strengths of the current study. The results of this study were drawn from a wide cross-section of the population, which contributed to the randomness of the sample and the generalisation of the results.

The strengths of this study also rest on findings associated with the integration of psychological and physiological indices and parameters, in an understanding of the complex interaction of neural processes, and a delineation of functional mechanisms associated with the heterogeneous reaction patterns after severe traumatic events. In examining the role of stress in memory function, the current study focused on differences between groups in mood states, their relationship with trauma reactivity patterns, and the pattern of cortical resting asymmetry patterns. The current results were in agreement with earlier findings indicating that people with PTSD have disruptions to working memory processes and cognitive functioning (Clark et al., 2003; McFarlane et al., 1993; Vasterling et al., 2006). In addition, laterised synchrony patterns in specific regions, and associated with specific reaction patterns, were proposed as a common mechanism for the compensatory transfer of information at the implementation or functional level.

Brain-behaviour theories (McFarlane et al., 2002) posit that over time there is an asymmetrical drift in the pattern of retrieval, implicating hippocampal structures and alterations to memory. An important contribution of this study, implying a timing mechanism is associated with the retrieval of information and with specific regions of the PFC and the hippocampus, is based on theoretical assumptions (Howard et al., 2006), which suggest that the frequent retrieval of intrusive phenomena may be associated with the loss of a temporal-tag for specific (non-manipulated) events in people with PTSD.

A timing mechanism, as suggested by various researchers (Rowe, 2005; Banich, 2004; Ward, 2003; Singer, 1999; Thatcher, 1997), might be fundamental to an understanding of inhibition and increased facilitation of certain categories of information, influencing the build up of patterns and setting up competition between patterns. This mechanism may be suggestive of a possible disruption to short-term storage at the local or structural level of information processing. Furthermore, this mechanism may be plausibly associated with alterations to neurochemical systems related to high anxiety when appraisal of information is uncertain (Litz et al., 2000) and linked to responses associated with right hemisphere processing.
An alteration to the memory of when information was first encoded will be associated with increasing levels of uncertainty and anxiety (Vasterling et al., 2006). Further investigation and the replication of this finding are warranted for an understanding of hippocampal function and episodic memory retrieval in people with PTSD or in those with extreme reactivity to traumatic stressor events (Lindauer et al., 2005).

The significance of the above conclusions is related to the practical implications of the functional role of asymmetry patterns in rhythmic tonic oscillations. Understanding asymmetry of synchronisation in task selection and processing can provide valuable information that might contribute to the design of screening tools for a deeper understanding of differences between psychopathology and resilience, and why symptoms are maintained in chronic states.

8.4. Future Directions

The hypothesis that the sequencing of information, as a left-lateralised function, becomes dysregulated in PTSD may be an important proposal for future research investigating the dynamic relationship between structural and temporal channels of information processing. In addition, by comparing clinical and non-clinical samples, and reactivity patterns associated with traumatic stressor events, the results of the current study may be relevant to an understanding of psychological and physiological differences between resilience and vulnerability of risk. This pattern of results requires replication for an understanding of a functional cerebral asymmetry favouring the right hemisphere in people with strong stress reactivity.

Together with altered temporal dynamics of information transfer, reduced language-based performance indices suggested a disruption in people with PTSD, involving mechanism associated with learning and attention that indicated blocking of inter-hemispheric and intra-hemispheric communication channels and reduced attentional scanning. In addition, the slower speed of processing found in people with PTSD in the current study may be in agreement with an “appraisal uncertainty” model (Metzger et al., 2004). Rehearsal mechanisms associated with lower frequency patterns and cerebellum activation for verbal and motor functions have received little investigation in the PTSD literature (Shaw et al., 2002; Teicher et al., 2006) but may warrant further investigation for an understanding of how timing mechanisms and changes in qEEG patterns interrupt the flow of coordinated information and the integration of traumatic experiences in memory functions.
Findings in this study indicated a less lateralised pattern of relationships between regions of interest in each frequency band and the performance scores for verbal and nonverbal domains in the PTSD group. The findings were interpreted as a more diffuse pattern of processing, possibly reflecting an inability to discriminate items within phase-locked temporal windows for the coherent transfer of information between hemispheres. The consistent central-temporal associations rather than anterior and posterior associations with cognitive scores in the PTSD group also suggested alterations to specific regions and processes. Thus, the possibility that the perisylvian fissure is a region corresponding to the shared resource of attentional control with spatial attention and a unilateral zone for disruption to incoming stimuli (Seger and Cincotta, 2006; Sandberg et al., 2003) may be a plausible hypothesis for the switching of attention in people with PTSD between specialised verbal and nonverbal processing. This hypothesis implicates a shift in people with PTSD in the recruitment of dorsal and ventral prefrontal areas (Clark et al., 2003; Jatzko et al., 2006) and a decoupling in frontoparietal circuits (Lanius et al., 2005; Shaw et al., 2002).

A simplified analysis of the multiple factors, that are related to the retrieval of items from memory, and the biological synergistic organisation of electrocortical and neurochemical processes, was presented in this study and may be a starting point from which to assess the relevance of asymmetry in brain organisation patterns in future research. Furthermore, the clinical significance of this finding is relevant to treatment outcomes, and may suggest alternative intervention strategies, depending on an objective analysis of dysfunctional behaviour. This study extended previous research (Felmingham et al., 2002; Metzger et al., 2004) by demonstrating how comorbid mood and volition states predict differences in information processing pathways. Thus, the originality of this study is in its elucidation of the integrative functions of psychological and physiological phenomena at the local level of processing and the relationship of these variables in high stress populations.

8.4.1. Learning as a Change Process

This study is a first attempt to examine spatial working memory as an orienting mechanism associated with preparatory resting oscillation patterns in memory function in people with PTSD. A unique common factor in people with PTSD may be an attempt to keep objects invariant in specific contexts. As suggested by McFarlane and colleagues (2002), PTSD becomes an atypical response to stress. Maintaining an invariant object in the space of changing contextual cues implies alterations to confidence in predictions for the anticipated stimuli and results in increasing errors of judgement (Bryant et al., 2005). This hypothesis is consistent with proposals that PTSD is a stress response that alters catecholamine systems and
changes the distribution of ACh in PFC regions (Aston-Jones et al., 1994). This is an important consideration for the allocation of attention, depending on the emotional significance based on fragments of information for features rather than function (Felmingham et al., 2002; McFarlane et al., 2002). Understanding the mechanisms associated with the ability to regulate attention in the face of uncertainty may be relevant in clinical settings and for a range of psychiatric disorders or during medical intervention procedures.

The separate aspects of information processing associated with spatial mechanisms, as a result of practice or learning, were not within the scope of the current study and warrant further investigation. In addition, perception is a multi-faceted variable and to formulate an operational construct for an objective evaluation, on the basis of knowledge structures, will require a deeper level of understanding of spatial working memory systems.

At a mechanistic level, the current results indicated a pattern of relationships between delta and beta frequencies in the PTSD group. This finding may be an important start to understanding signal transmission in neural circuits for verbal and nonverbal information in low and high frequency patterns. Speculatively, the idea of holding spatial information, or sensory information in rehearsal systems for fast recollection, may be an adaptive mechanism for maintaining patterns of the self in the world. This idea has not been adequately addressed in the literature.

8.4.2. The Role of Stress in Memory Structures

The role of stress in memory function was investigated in this study by examining the pattern of relationships with resting cortical activation patterns for an assessment of tonic states at the local level of information processing. Differences between groups suggested a distinct pattern of altered functioning in both the TEC group and the PTSD group. One interpretation for this finding was the proposal that memories may be compartmentalised or dissociated to suppress retrieval, based on the idea that memory retrieval is intentionally and voluntarily organised according to salient and temporally distant items from one another (Glassman, 2000; Eichenbaum and Fortin, 2005). Furthermore, various hypotheses have been postulated as to how this might occur. The idea that attention modulation is associated with asymmetry patterns of cortical activation has been one view proposed to account for the alterations in memory function in people with PTSD (McFarlane et al., 1993; Felmingham et al., 2002; Vasterling et al., 2006) and is convergent with the current pattern of results.
However, the mechanisms for the integration of cognitive and affective functions have not been clarified in the literature (Williams et al., 2006). As indicated in this study, lateralisation in people with PTSD is generally seen as a disruption to left hemisphere processes for the facilitation of serial or sequential processing. This function appears to be disrupted in a specific way in the PTSD group and affects frontal regions of the brain (Clark et al., 2003; Bremner et al., 1999). This model was supported by the distinct patterns for pre-stimulus background rhythms, in this study, and in performance indices on the motor memory task. In addition the left hemisphere was associated with a pattern of comorbid mood states in people with PTSD.

Conversely, the role of stress associated with different types of traumatic events reported was not investigated by this study. A higher reporting of traumatic stressor events by women (Rosemann, 2002; Nemeroff et al., 2006), and for sexual molestation as the most frequently reported traumatic stressor in the trauma exposed control group, was interpreted as a specific psychological and physiological compensatory adjustment that requires further investigation. The significance of this finding may be relevant to an understanding of gender differences and risk of developing PTSD or treatment outcomes, particularly during phases of remitted symptoms. The findings also demonstrate that techniques for the assessment of a number of variables, that can improve our understanding of how knowledge structures facilitate or block stressful events, and the subsequent assessment of integration of information for behavioural flexibility, are as important at the mechanistic level of analysis as at the functional level.

**General Conclusions:**

The results from the present study suggest a number of important conclusions:

1. Results of this study show that information processing is slowed in people with previous traumatic exposure and reactivity and that this reduced speed of processing is associated with distinct patterns of baseline EEG frequency patterns of cortical arousal in clinical and non-clinical participants.

2. The current results confirm the findings of previous studies in PTSD that demonstrated a disruption to left hemisphere processing in frontal regions. This finding was modelled to show dysregulation in PTSD is associated with the loss of inhibitory functions and uncertainty of appraisals for processing external stimuli. This conclusion is supported by the current finding of higher resting activation patterns in people with previous trauma exposure in right
hemisphere posterior regions, and reduced verbal working memory capacity in people with PTSD.

(3) The current results indicate a relationship with lower rhythmic frequency patterns and lateralised indices of cognitive performance, which are consistent with conclusions that long-range connectivity between fronto-parietal regions is disrupted in people with PTSD.

(4) The current results support and extend previous findings that trauma-reactivity linked to affective states are associated with two distinct pathways for information processing. Diffuse connectivity between frontal and posterior regions was implicated by the pattern of results.

(5) The current results provide partial support for a brain-behaviour model of compensation and neural plasticity in people with previous traumatic experiences and are consistent with views that structural and functional alterations occur in people with PTSD as secondary characteristics.

At the same time, the results raise the possibility that a lateralised pattern of cortical networks is not dependent on specialised verbal and nonverbal working memory functions but on a perceptual-motor dichotomy of brain activation.

One way of understanding this model in PTSD would be to investigate the degree of dominance in mixed-laterality handedness groups for an assessment of speed of processing, trauma reactivity, and qEEG frequency patterns. However, numerous alternative proposals have been put forward in this chapter that may lead to an understanding of compensatory mechanisms and lateralised characteristic profiles of functioning associated with resilience or psychopathology.

Of particular relevance are timing mechanisms and associations with context information. The current pattern of results implied a probable compensatory pathway in people with PTSD associated with the interaction of specific hippocampal functions, medial temporal cortices and striatal circuits that may be related to the retrieval of episodic memories and altered synchrony patterns in different oscillatory systems. Further replication of the current results is warranted to confirm the pathways for altered working memory functioning in people with PTSD and to understand the compensatory role of uncoupling fronto-parietal regions.
References


Sauseng, P., Klimesch, W., Gruber, W., Doppelmayr, M., Stadler, W., & Schabus, M. (2002). The interplay between theta and alpha oscillations in the human electroencephalogram reflects the transfer of information between memory systems. *Neuroscience Letters, 324*, 121-124.


van der Kolk, B. A. (2001). The psychobiology and psychopharmacology of PTSD. *Human Psychopharmacology, 16*(S1), S49-S64.


