LEABHARLANN CHOLÁISTE NA TRÍONÓIDE, BAILE ÁTHA CLIATH | TRINITY COLLEGE LIBRARY DUBLIN
Ollscoil Átha Cliath | The University of Dublin

Terms and Conditions of Use of Digitised Theses from Trinity College Library Dublin

Copyright statement

All material supplied by Trinity College Library is protected by copyright (under the Copyright and Related Rights Act, 2000 as amended) and other relevant Intellectual Property Rights. By accessing and using a Digitised Thesis from Trinity College Library you acknowledge that all Intellectual Property Rights in any Works supplied are the sole and exclusive property of the copyright and/or other IPR holder. Specific copyright holders may not be explicitly identified. Use of materials from other sources within a thesis should not be construed as a claim over them.

A non-exclusive, non-transferable licence is hereby granted to those using or reproducing, in whole or in part, the material for valid purposes, providing the copyright owners are acknowledged using the normal conventions. Where specific permission to use material is required, this is identified and such permission must be sought from the copyright holder or agency cited.

Liability statement

By using a Digitised Thesis, I accept that Trinity College Dublin bears no legal responsibility for the accuracy, legality or comprehensiveness of materials contained within the thesis, and that Trinity College Dublin accepts no liability for indirect, consequential, or incidental, damages or losses arising from use of the thesis for whatever reason. Information located in a thesis may be subject to specific use constraints, details of which may not be explicitly described. It is the responsibility of potential and actual users to be aware of such constraints and to abide by them. By making use of material from a digitised thesis, you accept these copyright and disclaimer provisions. Where it is brought to the attention of Trinity College Library that there may be a breach of copyright or other restraint, it is the policy to withdraw or take down access to a thesis while the issue is being resolved.

Access Agreement

By using a Digitised Thesis from Trinity College Library you are bound by the following Terms & Conditions. Please read them carefully.

I have read and I understand the following statement: All material supplied via a Digitised Thesis from Trinity College Library is protected by copyright and other intellectual property rights, and duplication or sale of all or part of any of a thesis is not permitted, except that material may be duplicated by you for your research use or for educational purposes in electronic or print form providing the copyright owners are acknowledged using the normal conventions. You must obtain permission for any other use. Electronic or print copies may not be offered, whether for sale or otherwise to anyone. This copy has been supplied on the understanding that it is copyright material and that no quotation from the thesis may be published without proper acknowledgement.

IMPAIRED AWARENESS OF DEFICITS AND NEUROPSYCHOLOGICAL FUNCTIONING FOLLOWING TRAUMATIC BRAIN INJURY, FRONTOTEMPORAL DEMENTIA, CORTICOBASAL DEGENERATION AND PROGRESSIVE SUPRANUCLEAR PALSY

By

Fiadhnait O'Keeffe

A dissertation submitted for the degree of Doctor of Philosophy of the University of Dublin, Trinity College, Dublin 2, Ireland.

This research was conducted in the Department of Psychology and Trinity College Institute of Neuroscience.

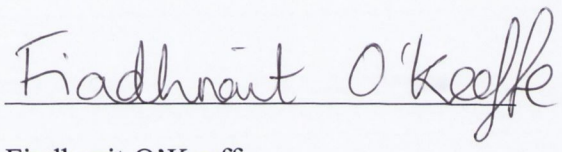
October 2005.

TRINITY COLLEGE
15 MAY 2006
LIBRARY DUBLIN
THESIS
7777

Declaration

I declare that this work has not been submitted previously as an exercise for a degree at this or any other university and that it is entirely my own work. The Trinity College Library may lend or copy this thesis upon request.

Signed



Fiadhnait O'Keeffe

Acknowledgements

To my supervisor Professor Ian Robertson for his constant support, enthusiasm, optimism, encouragement and positive attitude in every aspect of my research over the past 3 years. To Dr. Paul Dockree, for his never-ending help with theoretical, conceptual, methodological, statistical, practical and hypothetical questions. To all those without whom this research would not have been possible: Headway Ireland, The National Head Injuries Association; Dr. Simone Carton and all those from the Clinical Neuropsychology Department at the National Rehabilitation Hospital, Co. Dublin; Dr. Brian Murray and Dr. Tim Lynch from the Neurology Department of the Mater Misericordiae Hospital Dublin; Dr. Robert Coen, The Mercer Institute for Research on Aging, St. James's Hospital, Dublin. A special thanks to all participants from Headway Ireland, National Rehabilitiation Hospital, Mater Neurology and all referred patients and their families- this research relied on your generosity of time and energy. To others from Trinity College who helped with various aspects of the research, most especially to Pauline Moloney, Research Assistant. Also Michelle McCartney, Doreen Hoereld, Claire Waters, Helen Bates, Maria Pertl. To The Irish Research Council for the Humanities and Social Sciences for their financial support through the Government of Ireland Scholarship. To all my friends for the support, laughs and encouragement over the last few years, especially Trich, Clare K, Sarah, Paul, Redmond, Richard, Laura, Carol, Claire C. And to Glen, for making the last year infinitely more enjoyable. Finally, to all my amazing and wonderful family- especially to my parents, Maeve and Jim. Your love, support and encouragement in everything I do never ceases to astound me.

Thank you all for everything.

Summary

This dissertation consists of an investigation into the phenomenon of impaired self-awareness of deficits following brain damage. Despite a large interest in the area in the neuropsychological literature, there is a distinct lack of consensus regarding assessment and underlying neuropsychological correlates of impaired awareness. The three studies presented in this thesis attempted to elucidate the neuropsychological correlates of awareness by adopting a multidimensional approach to assessment in individuals with traumatic brain injury (TBI), frontotemporal dementia (FTD), corticobasal degeneration (CBD) and progressive supranuclear palsy (PSP).

The first study examines online error-awareness in a group of sixteen patients with TBI and compares their performance to sixteen neurologically-healthy matched controls. Participants were required to monitor their errors as they performed a simple task of sustained attention. Electrodermal activity was also measured during error-monitoring. This study found a significant impairment of online awareness of errors in TBI participants. TBI participants also showed an attenuated skin conductance response to making errors, which could not be explained by a general lack of arousal. This suggested an attenuated emotional response to error-processing by TBI participants.

The second study in this thesis examines impaired self-awareness in thirty-one TBI participants and thirty-one matched controls. This study investigated impaired self-awareness on three levels: metacognitive knowledge or intellectual awareness (knowledge of difficulties or deficits), online emergent awareness (monitoring of errors during task performance) and anticipatory awareness (ability to predict

performance on tasks accurately). Those TBI participants who demonstrated low self-awareness were significantly impaired across each of the awareness tasks measured, most notably on the online emergent awareness task. The TBI group with high self-awareness performed as well as, if not better than the neurologically-healthy controls on most of the awareness tasks. The low self-awareness TBI group did not differ significantly from the high self-awareness group on standard neuropsychological tests but were rated as significantly more impaired in terms of complex executive function behaviours and interpersonal relationships.

The third and final study of this dissertation examines impaired self-awareness on the three levels outline above in three groups of patients with atypical dementias, namely fourteen individuals with FTD, eleven individuals with CBD, ten individuals with PSP and twenty age and education matched controls. This study clearly showed that impaired self-awareness was not only a feature of FTD, but also of CBD and PSP.

Overall, this dissertation has clearly shown that impaired self-awareness in clinical populations can and should be assessed on three levels to gain a comprehensive and rich picture of this multifaceted phenomenon. It also shows that distinctive behavioural difficulties indicative of executive dysfunction and difficulties with interpersonal relationships are associated with impaired self-awareness in individuals with TBI, FTD, CBD and PSP.

Publications Resulting from the Present Work

Article:

O'Keeffe, F, Dockree, P., Robertson, IH. (2004). Poor insight in traumatic brain injury mediated by impaired error processing? Evidence from electrodermal activity. *Cognitive Brain Research,* **22**(1): 101-12.

Published Abstracts:

O'Keeffe, F, Dockree, P. & Robertson, IH. (2004) Awareness Deficits in Traumatic Brain Injury mediated by Impaired Error Processing? Evidence from Electrodermal Activity. *Proceedings of the Cognitive Neuroscience Society,* A65, 31.

O'Keeffe, F. Dockree,P., Moloney, P., Carton, S, and Robertson IH (2005). Impaired Error-Monitoring in Traumatic Brain Injury. *Proceedings of the Cognitive Neuroscience Society,* C18, 85.

O'Keeffe, F. & Robertson, IH (2005). Awareness of Deficits and neuropsychological impairment in clinical patients with frontal lobe abnormalities. *JINS,* 11, S52, 55.

O'Keeffe, F., Dockree, P., Moloney, P., Carton, S., Robertson, I. (2005) Awareness Deficits and Neuropsychological Impairment after Traumatic Brain Injury, *JINS,* 11, S52, 7.

O'Keeffe, F., Murray, B.,Coen, R, Hoerld, D., Garavan, H., Lynch, T. Robertson, IH. (2005). Impaired Error-Monitoring and Insight in tauopathies : Evidence from Frontotemporal Dementia, Corticobasal Degeneration and Progressive Supranuclear Palsy. *Acta Neurobiologiae Experimentalis,* 65, 73.

Murray B, O'Keeffe F, Coen R, Garavan H, Robertson I, Lynch T (2005). Insight and error monitoring in the tauopathies: use of a novel neuropsychology test battery in a prospective cohort-controlled study of frontotemporal dementia, corticobasal degeneration and progressive supranuclear palsy. *Proceedings of the American Academy of Neurology.*

O'Keeffe,F. & Robertson, IH (2005). Awareness of Deficits and Neuropsychological Impairment in Patients with Traumatic Brain Injury. *Brain Impairment,* 127.

O'Keeffe, F., Dockree, Moloney, Carton, Robertson (2005) Awareness of deficits following Traumatic Brain Injury. Proceedings of the Psychological Society of Ireland Annual Conference November 2005, *The Irish Psychologist.*

Other Publications :

McAvinue, L., O'Keeffe, F., McMackin, D., Robertson, I.H. (2005) Impaired sustainedattention and error awareness in Traumatic Brain Injury : Implications for Insight. *Neuropsychological Rehabilitation*, 15(5), 569-587.

Dockree PM, Bellgrove MA, O'Keeffe FM, Moloney P, Aimola L, Carton S, Robertson IH (2005). Sustained attention in traumatic brain injury (tbi) and healthy controls: enhanced sensitivity with dual-task load. *Experimental Brain Research*, 1-12.

Kidd, N.R., Garavan, H., O'Keeffe, F., Carton, S. (2005). The assessment of neuropsychological, cognitive and emotional variables of financial competency in acquired brain injury. *JINS,* 11, S52, 58.

Dockree PM, O'Keeffe FM, Moloney P, Bishara AJ, Carton S, Jacoby LL, Robertson IH (2005). Capture by misleading information and its false acceptance in patients with traumatic brain injury. *Brain.* In press.

Table of Contents

List of Abbreviations 5

List of Tables 6

List of Figures 8

Chapter 1

General Introduction

1.1 Introduction 10

1.2 Historical Perspective of Impaired Awareness 12

1.3 Definitions and Terminology of Self-Awareness 14

1.4 Clinical Populations Affected by Impaired Self-Awareness 16

1.5 Traumatic Brain Injury 26

1.6 Characteristics of Impaired Self-Awareness in TBI 32

1.7 Theories and Models of Impaired Self-Awareness 58

1.8 Measurement of Impaired Self-Awareness 76

1.9 Summary of Literature and Aims of the Present Thesis 84

Chapter 2

Impaired Online Monitoring in Traumatic Brain Injury (TBI) Patients: Evidence from an Error-Monitoring Task and Electrodermal Activity

Abstract 87

2.1 Introduction 88

2.2 Method 93

2.3 Results 101

2.4 Discussion 114

Chapter 3

Awareness of Deficits in Traumatic Brain Injury: A Multidimensional Approach to Assessing Metacognitive Knowledge and Online Awareness

Abstract 121

3.1 Introduction 122

3.2 Method 126

3.3 Results 137

3.4 Discussion 161

Chapter 4

Evidence of Impaired Metacognitive Knowledge and Online Awareness in Corticobasal Degeneration and Progressive Supranuclear Palsy Patients

Abstract 179

4.1 Introduction 180

4.2 Method 185

4.3 Results 194

4.4 Discussion 225

Chapter 5

General Discussion 234

5.1 Summary of Main Findings 235

5.2 Integration with Theoretical and Clinical Frameworks 238

5.3 Limitations of the Current Studies 240

5.4 Future Research 242

5.5 Conclusion 244

References 245

Appendices 308

List of Abbreviations Used

AACA: Aneurysm of the Anterior Communicating Artery

ACC: Anterior Cingulate Cortex

AD: Alzheimer's Disease

ADL: Activities of Daily Living

AQ: Awareness Questionnaire

CAM: Cognitive Awareness Model

CAS: Conscious Awareness System

CBD: Corticobasal Degeneration

CFQ: Cognitive Failures Questionnaire

CHI: Closed Head Injury

CT: Computerized Tomographic Imaging

CVA: Cerebrovascular accidents

DAI: Diffuse Axonal Injury

DICE: Dissociable Interactions and Conscious Experience

EDA: Electrodermal Activity

EEG: Electroencephalography

ERN: Error-Related Negativity

ERP: Event-Related Potentials

fMRI: Functional Magnetic Resonance Imaging (fMRI)

FTD: Frontotemporal Dementia

GCS: Glasgow Coma Scale

GSI: Global Severity Index

HADS: Hospital Anxiety and Depression Scale

HD: Huntington's Disease

ISA: Impaired Self awareness

MPAI: Mayo-Portland Adaptability Inventory

MRI: Magnetic Resonance Imaging

NART: National Adult Reading Test

NFI-R: Neurobehavioral Functioning Inventory-Revised

NPI: Neuropsychiatric Inventory

PCRS: Patient Competency Rating Scale

PD: Parkinson's Disease

PET: Positron Emission Tomography

PPA: Primary Progressive Aphasia

PSP: Progressive Supranuclear Palsy

R-SAT: Revised Strategy Application Task

RTA: Road Traffic Accidents

SART: Sustained Attention to Response Task

SAS: Supervisory Attentional System

SCR: Skin Conductance Response

SD: Semantic Dementia

SO: Significant Other

SPECT: Single Photon Emission Computer Tomography

TBI: Traumatic Brain Injury

TOM: Theory Of Mind

UMACL: The UWIST Mood Adjective Checklist

VOSP: Visual Object and Space Perception Battery

WAIS-R: Wechsler Adult Intelligence Scale-Revised

WMS: Wechsler Memory Scale

List of Tables

Table 2.1: Participant Characteristics for TBI and Control Participants 94

Table 2.2: Levels of Error Awareness (Proportion of Errors of which Participants were Aware) on Each SART Awareness Condition for TBI and Control Participants 102

Table 2.3: Comparison of Mean Errors of Commission Made by TBI and Control Participants on each SART Condition 104

Table 3.1: Clinical Data for the TBI Patients 127

Table 3.2: Demographic Details of TBI and Control Participants 129

Table 3.3: Clinical Data for TBI High SA and TBI Low SA Groups 140

Table 3.4: Neuropsychological Tests Scores for Each Participant Group 145

Table 3.5: Patient Competency Rating Scale (Self- and Other-Rated) Mean Scores and Standard Deviations for Each Group 148

Table 3.6: Patient Competency Rating Scale Discrepancy Mean scores, Standard Deviations, F Values, Significance Level and Post Hoc Comparisons for Each Group 149

Table 3.7: Mean and Standard Deviation Scores for Self- and Other-Rated Frontal Systems Behavioral Scale (Total and Subscales) 151

Table 3.8: Discrepancy Scores (Total and Subscale) on Frontal Systems Behavioral Scale for Each Group 152

Table 3.9: Mean and Standard Deviations (z-scores and Actual) Self- and Other-Rated CFQ Scores and Discrepancy Scores 155

Table 3.10: Proportion of Aware Errors on SART Conditions for Each Group 156

Table 3.11: Pearson Product-Moment Correlations between Awareness Category and Selected Measures for TBI Participants 160

Table 4.1: Demographic Data for Participants (N= 55) 195

Table 4.2: Neuropsychological Test Scores for Each Participant Group 197

Table 4.3: Awareness Interview Category Deviation Means, Standard Deviations and F-Values for Each Patient Group 201

Table 4.4: Patient Competency Rating Scale (Self- and Other-Rated) Mean Scores and Standard Deviations for Each Group 203

Table 4.5: Patient Competency Rating Scale Discrepancy Mean scores, Standard Deviations, F Values, Significance Level and Post Hoc Comparisons for Each Group 205

Table 4.6: Mean and Standard Deviation Scores for Self-and Other-Rated Frontal Systems Behavioral Scale (Total and Subscales). 208

Table 4.7: Discrepancy Scores (Total and Subscale) on Frontal Systems Behavioral Scale for Each Group 210

Table 4.8: Magnitude of Behaviour Change (FrSBe At Present Minus FrsBe Pre-illness) Self-Rated and Other-Rated for all Patient Groups 212

Table 4.9: Self- and Other-Rated Cognitive Failures Questionnaire Ratings and Discrepancy Scores Between Self- and Other-Ratings for All Participant Groups 215

Table 4.10: Mean Proportions of Error Awareness on SART and XY Tasks For All Groups 217

Table 4.11: Prediction-Performance Tasks for Each Participant Group: Mean Percentage Accuracy for Each Task and F-Values with Significance Levels 220

Table 4.12: Pearson Product-Moment Correlations Between Awareness Category and Selected Measures for Patient Participants 221

List of Figures

Figure 2.1: An example of the Fixed SART Sequence

Figure 2.2: Overall Proportion of Errors Reported by TBI and Control Participants for Each SART Awareness Condition 103

Figure 2.3: The Mean Skin Response Amplitude (μS) for Withholding a Response and Errors of Commission on Fixed-Baseline for TBI and Control Participants 107

Figure 2.4: The Mean Skin Conductance Response Amplitude (μS) for "Hold" on Withholds and for "Hit" on Errors of Commission on Fixed-Awareness2 for TBI and Control Participants 109

Figure 2.5: Example of SCR (μS) for TBI and Control Participant Following a Detected Error. The Arrow Indicates Stimulus Onset 110

Figure 2.6: The Mean Skin Conductance Response Amplitude (μS) for "Hold" on Withholds and for "Hit" on Errors of Commission on Standard-Awareness2 for TBI and Control Participants 112

Figure 3.1: Awareness Type by Group for TBI and Control Participants 139

Figure 3.2: Histograms for Each Awareness Measure for TBI Participants and Normality Tests 141

Figure 3.3: Interaction Between TBI High SA, TBI Low SA and Control Group on Three Composite Awareness Scores 143

Figure 3.4: Awareness Interview Awareness Index (0-16) Scores for Each Participant 146

Figure 3.5: PCRS Discrepancy Scores for TBI Low SA, TBI High SA and Controls 150

Figure 3.6: Self- and Other-Rated Behaviour Change (FrSBe At Present Minus FrSBe Pre-Injury) Since Brain Injury for TBI Participants 154

Figure 3.7: Error Awareness Proportion Scores on Each SART Task for Each Group 157

Figure 3.8: Percentage Differences Between Prediction and Performance on Digit Span and Verbal Fluency Task for Each Group 158

Figure 4.1: Example of the XY task 189

Figure 4.2: Awareness Type by Group for Each Participant Group 199

Figure 4.3: Awareness Interview Awareness Index (0-20) for Each Participant 200

Figure 4.4: Patient Competency Rating Scale Discrepancy Scores for Each Participant Group 206

Figure 4.5: FrSBe Total and Subscale Discrepancy Scores (Self-rated minus Other-rated) for Each Participant Group 211

Figure 4.6: Self- and Other-Rated Behaviour Change (FrSBe At Present Minus FrSBe Pre-Illness) Since diagnosis for FTD, CBD and PSP Patients 213

Figure 4.7: Error Monitoring on the SART and XY for Each Participant Group 218

Chapter 1

General Introduction

1.1 Introduction

A myriad of cognitive, behavioural, social, psychological, personality and physical changes can follow brain damage, acquired through either a traumatic impact, as in Traumatic Brain Injury, or through neurodegenerative disease, such as cortical and subcortical dementias (Lezak, Howieson, & Loring, 2004). The result of these sometimes subtle, often devastating changes are generally blatantly evident to family, caregivers and clinicians. Arguably one the most startling manifestations of brain damage is that the patients themselves frequently have distinctly inaccurate perceptions of these changes, how they impact on their daily lives, or how they may affect future activities and goals (Fleming, Strong, & Ashton, 1996; McGlynn & Schacter, 1989).

The study of impaired self-awareness is of enormous importance both clinically and theoretically. From a clinical perspective, impaired self-awareness of deficits can have a negative impact on functional, rehabilitative and vocational outcome (Sherer, Bergloff, Levin et al., 1998), and can cause increased caregiver stress and burden (DeBettignies, Mahurin, & Pirozzolo, 1990; Prigatano, Borgaro, Baker, & Wethe, 2005), thereby having obvious relevance for clinical psychologists, neuropsychologists, neurologists, medical and rehabilitation personnel.

Theoretically, self-awareness has been described as involving the integration of information from both external reality and inner experience (Fleming et al., 1996), and as the highest of all integrated cerebral functions (Stuss, 1991; Stuss & Benson, 1986). Therefore, investigations into self-awareness, both unimpaired and impaired, are of general interest to neuroscientists, philosophers, cognitive scientists, personality and social psychologists alike, who are in search of answers to the perennial question "What is human consciousness", by providing insight into the neuropsychological mechanisms that may underlie the conscious awareness of self.

The impairment of self-awareness or *anosognosia* of deficits will be the subject of this thesis, investigating its occurrence in individuals with Traumatic Brain Injury (TBI) and three atypical dementias and parkinsonian disorders, Frontotemporal Dementia (FTD), Corticobasal Degeneration (CBD) and Progressive Supranuclear Palsy (PSP). The first two of three studies of the thesis investigate this phenomenon in TBI. Considering the paucity of research regarding impaired self-awareness in the other three clinical syndromes investigated in the third study, there will be an emphasis placed on reviewing the literature relating to TBI and impaired awareness. The following review will briefly examine impaired awareness from a historical perspective and identify the several clinical populations affected. The review will then initially outline the prevalence, neuropathology and neuropsychological and behavioural correlates of TBI, before focussing on the characteristics of impaired self-awareness following TBI, with an emphasis on the clinical neuropsychological aspects, models and theories, its assessment, clinical issues and cognitive and neuropsychological correlates of this fascinating phenomenon.

1.2 Historical Perspective of Impaired Awareness

Towards the end of the 19th century, a number of prominent neurologists began to report clinical cases of disturbed awareness. However, as Bisiach and Geminiani (1991) point out, disorders of self-awareness were documented long before formal clinical neurological study began. Almost 2000 years prior to this, reports of loss of awareness appeared in letters from L.A. Seneca, writing about issues of self and morality:

"This foolish woman has suddenly lost her sight. Incredibly as it might appear, what I am going to tell you is true: She does not know she is blind...She claims that my home is dark...it is difficult to recover from illness just because we are unaware of it." (cited in Bisiach & Geminiani, 1991, p. 17).

The first official historical descriptions came from both von Monakow (1885) and Anton (1899) who reported patients lacking awareness for their cortical blindness, which resulted from diffuse brain injury (von Monakow's patient) and more focal brain dysfunction (Anton's patient). Although Arnold Pick is credited for describing the first case of impaired awareness of hemiplegia (paralysis of one side of the body) following brain damage (Gerstmann, 1942), Babinski (1914) was the first to introduce the term *anosognosia* (*a*, without; *noso*, disease; *gnosia,* knowledge) to describe several patients' loss of recognition or awareness of left hemiplegia after sustaining focal damage to the right parietal cortex. Despite their obvious paralysis, these patients insisted they could walk without difficulty. Further clinical cases were noted, indicating that patients with frontal lobe tumours had a "peculiar indifference" to their difficulties (Sachs, 1927). This indifference, or lack of genuine concern about illness or deficits was later termed *anosdiaphoria* (Critchley, 1953a). Freeman and

Watts (1950) described how following prefrontal lobotomy, their psychosurgical patients had a disturbance in the capacity of foresight in relation to the self and self-continuity.

A conceptual shift from the neurological approach came with Weinstein and Kahn's redescription of anosognosia to *denial of illness* (Weinstein & Kahn, 1955), reflecting their psychodynamic view that pre-morbid personality factors were major determinants and that denial of illness in individuals was an "adaptive" defense mechanism in its attempt to structure traumatic experiences. Neurological and neuropsychological research into anosogonosia declined following Weinstein and Kahn's (1955) monograph, purported to reflect the dominance of behaviourism at that time and perhaps the discomfort psychologists and neuropsychologists with more "mentalistic" issues (Prigatano & Schacter, 1991).

Following a dearth of research lasting almost 25 years, interest in the area of anosognosia re-emerged for a number of reasons. Several neuropsychological syndromes demonstrated that specific disturbances of consciousness could indeed be associated with specific disturbances in brain function, such as prosopagnosic patients' ability to implicitly recognise facial familiarity (Tranel & Damasio, 1985), and patients who demonstrate "blindsight" following lesions to the striate cortex with no conscious perception of their environment (Weiskrantz, 1986). Research with split-brain or commissurotomy patients showed that unusual disorders of consciousness could be demonstrated indicating that conscious information processing does not rely on verbal reports alone (Sperry, 1974). The construct of consciousness also became the subject of investigation in academic psychology, playing a role in models of attention (Posner, 1978), memory (Schacter, 1990;

Tulving, 1985) and perception (Marcel, 1983), which provided methodological and theoretical grounding for empirical research into anosognosia (Prigatano & Schacter, 1991). Furthermore, following early attempts at neuropsychological rehabilitation of individuals with brain-damage, it became apparent that lack of insight into physical and cognitive deficits could cause significant barriers to the rehabilitative process (Prigatano, Fordyce, & Zeiner, 1986).

A flurry of research into awareness deficits followed but clear classification of these disturbances has been problematic, due to methodological and theoretical complications. It is clear from the brief historical overview that different terminology and perspectives can complicate concise descriptions of the disturbance. In more recent years, several definitions of impaired awareness have been proposed.

1.3 Definitions and Terminology of Self-Awareness

Terminology has developed over the years such that "anosognosia" has now become an umbrella term for all disturbances of awareness of deficits in clinical populations and no longer refers solely to unawareness of hemiplegia. The term "anosognosia" is frequently, and perhaps misleadingly, used interchangeably with other terms such as "loss of insight", "imperception of disease", "unawareness of deficits", "impaired metacognition", "error-monitoring difficulties", "deficits in self-regulation", and "impaired self-awareness" when referring to changes in self-awareness following neurological damage. "Denial of illness, "anosdiaphoria", "lack of concern" or "indifference" often imply a more psychological or psychodynamic explanation (Flashman, 2002; McGlynn & Schacter, 1989). For simplicity and clarity, this thesis will adopt the use of the term "impaired self-awareness" (ISA) when referring to the

general phenomenon of awareness deficits in clinical populations following neurological damage, consistent with recent reviews and research in the area (Hart, Whyte, Kim, & Vaccaro, 2005; Sherer, Hart, Whyte, Nick, & Yablon, 2005).

There has also been some lack of consensus with regard to definitions of self-awareness. Impaired self awareness has been described by Prigatano and Klonoff (1998): *"Impaired self awareness reflects impairments in the patient's ability to consciously represent (perceive and experience) a disturbance in higher cerebral functioning. That impairment appears to reflect a disruption of the integration of thinking and feeling.* (p.57)"

In order to broaden the scope of this definition it is also useful to explore the definitions for non-impaired self-awareness used in the self-awareness literature. Stuss and Benson (1986) refer to self-awareness as: *"... that attribute of the human which not only allows awareness of the self, but also realizes the position of the self in the social milieu."*. A similar definition comes from Prigatano and Schacter (1991) referring to self-awareness as "*the capacity to perceive the 'self' in relatively 'objective' terms while maintaining a sense of subjectivity...a natural paradox of human consciousness* (p.13)."

These definitions reflect the wide variety of processes potentially required for non-impaired self-awareness, such as functioning perceptual, memory and attention processes, emotional factors, effective higher cognitive functions such as social awareness, judgement, self-monitoring and self-regulation, decision-making and perhaps explain why Stuss and Benson (1986) referred to self-awareness as the highest of all integrated cerebral functions (Sawchyn, 2001).

1.4 Clinical populations affected by Impaired Self-Awareness: An overview

Considering these complex processes that self-awareness may involve, it is perhaps not surprising that patients with a vast array of clinical syndromes, not solely individuals with TBI, show inaccurate perception of their neuropsychological, behavioural, social, psychiatric, emotional and physical symptoms. Before reviewing the research on impaired awareness of deficits following TBI in detail, it is pertinent to briefly examine some of the relevant characteristics and central issues relating to other neuropsychological and neuropsychiatric syndromes that demonstrate impaired self-awareness.

Impaired self-awareness for hemiplegia

As outlined in the historical overview, impaired self-awareness for hemiplegia was one of the earliest clinical accounts of this phenomenon. Starkstein et al (Starkstein, Fedoroff, Price, Leiguarda, & Robinson, 1992) showed that 28% of their eighty patients with acute cerebrovascular lesions demonstrated impaired awareness for their hemiplegic limb, assessed by questionnaire relating to their stroke. Of those, 80% had right-hemisphere lesions and the remaining 20% had bilateral lesions. Measures of overall cognitive functioning did not distinguish between patients with impaired and unimpaired awareness. A review by Cocchini et al (Cocchini, Beschin, & Sala, 2002) indicated that the location of lesions in the vast majority of studies of impaired awareness for plegia were right hemisphere, frequently right parietal, though left hemisphere and bilateral lesions were also described. Hemispatial neglect (loss of awareness for visual events on the contralesional side of space) can be associated with impaired awareness, but it is neither necessary nor sufficient for

impaired awareness of plegia (Bisiach, Vallar, Perani, Papagno, & Berti, 1986; Marcel, Tegner, & Nimmo-Smith, 2004). Impaired awareness for plegia can manifest itself in several ways. Patients may deny outright that they have any paralysis, they may admit to some weakness but either downplay the extent of it, or ascribe it to a variety of other reasons such as arthritis or more external causes, or they may acknowledge the paralysis but continue to attempt activities involving the paralysed limb (Marcel et al., 2004).

Impaired Awareness for visual defects

Anton's syndrome, or unawareness of cortical blindness, is one of the most extreme examples of impaired awareness. Fascinating case reports exist such as that of a man who was rendered completely blind from a war injury who claimed to read the newspaper every day. He would report when asked that he was reading about the war and would come up with several excuses as to why he could not read aloud or give more detail, such as the room being too dark, or needing glasses. On attempting to walk alone and bumping into obstacles, he would again make excuses such as darkness. When pushed, he sometimes admitted his vision was poor, but consistently seemed completely unaware of the fact that he was totally blind (Bychowski, 1920, cited in McGlynn & Schacter, 1989). Heilman (1991) suggested that visual imagery and visual processing may compete for attention and representation and that destruction of visual processing can result in unimpeded visual imagery display, which might be misinterpreted by patients as the ability to see and may explain the confabulatory responses. McGlynn and Schacter (1989) point out that impaired awareness of visual defects is not limited to complete blindness and describe a number of studies that report impaired awareness of hemianopia (loss of vision for

one half of the visual field). Koehler et al (Koehler, Endtz, Te Velde, & Hekster, 1986) showed that of a group of 41 patients with hemianopia, 23 were classified as unaware of their deficit when asked in a clinical interview if they could a) see well, b) see as well on their left as on their right side and c) if their vision had changed. Lesions of the unaware group were more anterior and more extensive, involving the occipto-parietal and occipito-temporal regions, than the smaller lesions evident in the aware group which were more confined to the occipital lobe. Critchley (1953b) documented several characteristics relating to impaired awareness of hemianopia, including the spared integrity of patients' general mental processes and the presence of both cortical and subcortical lesions.

Impaired awareness in Aphasia

Patients with jargon aphasia, which is characterised by meaningless utterances, long and rambling sentences and neologisms, do not pause or monitor their utterances or self-correct (Lebrun, 1987; Prigatano, 1999b). Patients generally appear to be unaware that they do not comprehend what is said to them or that the listeners do not understand them, and have been found to have either bilateral or only left hemisphere damage (Gainotti, 1972; E. A. Weinstein, Lyerly, & Cole, 1966). However, accurate judgement of impaired awareness in aphasic patients is difficult considering the reliance on verbal reports for many assessments.

Impaired awareness in neuropsychiatric and neurodevelopmental conditions

Individuals with schizophrenia frequently demonstrate a lack of awareness. It has been reported that between 68-89% of patients demonstrate impaired awareness,

including misattributing symptoms to external causes, difficulty identifying their symptoms or recognising that they have a mental illness (Amador et al., 1994). Impaired awareness in schizophrenia does not appear to be consistently associated with demographic factors, epidemiological variables, neurological signs or positive and negative symptoms (Amador et al., 1993; Cuesta & Peralta, 1994; David et al., 1995). Weak relationships have been found with severity of illness and awareness deficits, but awareness does not appear to improve with symptomatic improvement (David, Buchanan, Reed, & Almeida, 1992; Markova & Berrios, 1992). The literature suggests that this lack of awareness is not necessarily a function of global cognitive deficits but more related to frontal-executive dysfunction (Lysaker & Bell, 1994; McEvoy et al., 1989). Impaired awareness in schizophrenia has been associated with selective structural brain changes including smaller brain sizes and selective atrophy of regions of the frontal lobes (Flashman, McAllister, Andreasen, & Saykin, 2000; Flashman et al., 2001).

Self-awareness deficits have also been noted in autism and Asperger's syndrome, manifested in impaired ability to maintain a stable concept of self over time, adapt this self-concept to different settings and make inferences about future behaviour (Kanner, 1973; Nair, 2004). Nair (2004) proposes that this is possibly related to studies showing changes in the orbitofrontal cortex in autism, an area of the cortex where damage is linked with insensitivity to social cues (Alexander, Benson, & Stuss, 1989). Theory-of-mind (TOM), the attribution of mental states to both oneself and others, is thought to be lacking in individuals with autism (Baron-Cohen, Tager-Flusberg, & Cohen, 2000; Premack & Woodruff, 1978). Vogeley et al (2001) relate this "mind-reading" ability or awareness of the mental state of others to self-consciousness and self-awareness, where the person is the agent (e.g. "I believe that

you/he thinks...). Increased left amygdala volume and reduced temporal blood flow has been shown in autistic adults (Abell, Krams, & Ashburner, 1999; George, Costa, Kouris, Ring, & Ell, 1992), a brain region purported to play a role in checking another person's gaze, reading their intent and drawing emotional inferences from the expression seen in another's eyes (Baron-Cohen et al., 1999; Nair, 2004).

Impaired awareness in amnesic syndromes

Impaired awareness into both memory and physical difficulties has been viewed as a primary feature of Korsakoff syndrome, and linked with abnormal reasoning and judgement in patients (Zangwill, 1966). Computerised tomographic (CT) and magnetic resonance imaging (MRI) have confirmed damage to the orbitofrontal and mediotemporal cortices, as well as damage to the thalamus and mamillary body in patients with Korsakoff syndrome (Shimamura, Jernigan, & Squire, 1988; Squire, Amaral, & Press, 1990). Korsakoff patients showed impairment at self-monitoring their memory performance and had poor predictive accuracy compared to controls using a feeling-of-knowing prediction memory task (Janowsky, Shimamura, & Squire, 1989; Shimamura & Squire, 1986). Amnesic patients who do not show frontally-related dysfunction are generally quite aware of their memory deficits (Kaszniak & Zak, 1996), thus, as McGlynn and Schacter (1989) point out, impaired awareness of memory deficits cannot be attributed solely to patient's "inability to remember that he cannot remember" (Whitlock, 1981, p.213). For example, Schacter et al (Schacter, Glisky, & McGlynn, 1990) administered subjective memory questionnaires to an encephalitic patient with severe amnesia but without frontal lobe involvement. This patient rated her memory very realistically, in contrast to highly unrealistic ratings by a patient with ruptured aneurysm of the anterior communicating

artery (AACA). The AACA patient substantially underrated the severity of his severe memory impairment. Similarly, Vilkki (1985) showed that of five patients examined with severe amnesic syndrome as a result of a ruptured AACA, the three who were unaware of their deficits had damage in the frontal lobe region, while the two with intact awareness did not. Patients with ruptured AACA are particularly prone to damage of the anterior cingulate and adjacent regions (Stemmer, Segalowitz, Witzke, & Schonle, 2004). A variety of behavioural and cognitive disturbances are associated with a ruptured ACAA including impaired self-awareness, apathy, confabulation, and control and error-monitoring problems (Ptak & Schnider, 1999; Schnider & Ptak, 1999; Shallice, 1999; Stemmer et al., 2004).

Impaired awareness in cortical and subcortical dementias

Dementia is referred to as the progressive impairment of multiple cognitive functions due to acquired brain disease or injury (Cummings & Benson, 1992; Kaszniak & Zak, 1996). Impaired self-awareness in one dementia syndrome, Alzheimer's Disease (AD), has been well-documented in recent years (Aalten, van Valen, Clare, Kenny, & Verhey, 2005; Agnew & Morris, 1998; Clare, 2004b; Seltzer, Vasterling, Hale, & Khurana, 1995). AD is a neurodegenerative disorder with neuropathological features of neuron loss most prominent in the mediotemporal, posterior temporal, parietal and frontal brain regions (Kaszniak & Zak, 1996). Specific domains of functioning in AD can be differentially affected by deficits in awareness, such as impaired awareness for daily functioning difficulties (DeBettignies et al., 1990) and behavioural and cognitive problems (Derouesne et al., 1999). Inconsistent and variable findings linking severity of AD and disease duration have been reported (Aalten et al., 2005; Clare, 2004a). Hypoperfusion in the frontal lobes has been

associated with impaired awareness in AD (Frackowiak et al., 1981), with two studies using Single Photon Emission Computer Tomography (SPECT) scans showing that lower awareness was associated with hypoperfusion of the right dorsolateral frontal lobe (Reed, Jagust, & Coulter, 1993; Starkstein et al., 1995). Derouesne et al (1999) reviewed 78 patient SPECT scans and also found that frontal or fronto-termporo-parietal hypoperfusion and predominantly right hemisphere deficits were more likely in those participants with lower awareness. Research has also shown correlations between impaired awareness in AD and measures of executive functioning (Lopez, Becker, Somsak, Dew, & DeKosky, 1994; Mangone et al., 1991; Michon, Deweer, Pillon, Agid, & Dubois, 1994), but others have failed to find associations between impaired awareness and neuropsychological or cognitive deficits (Derouesne et al., 1999; Seltzer, Vasterling, Mathias, & Brennan, 2001). Intact awareness of deficits in AD has been associated with higher depression, anxiety and hopelessness levels in some studies, (Harwood & Sultzer, 2002; Harwood, Sultzer, & Wheatley, 2000; Seltzer et al., 1995) whereas other studies have either not found this relationship or found contradictory results (Mangone et al., 1991; Migliorelli et al., 1995; Seltzer et al., 1995). Derousne et al (1999) found that increased apathy was associated with impaired awareness, referring to a link between unawareness and "emotional deficit". Other mood disturbances and psychiatric symptoms have also been associated with impaired awareness in AD, such as higher mania and more pathological laughing (Migliorelli et al., 1995) and the presence of delusional symptoms (Starkstein et al., 1997).

Despite a dearth of empirical evidence, impaired self awareness has been described as a feature of other dementias, both 'cortical' and 'subcortical' (McGlynn & Kaszniak, 1991). Early loss of insight is one of the core diagnostic criteria for

Frontotemporal Dementia (FTD) (Diehl & Kurz, 2002; Neary et al., 1998). It is frequently observed clinically that the majority of patients with FTD appear unaware of the pervasive changes in their personality, behaviour and social interactions (Gregory & Hodges, 1996). However, quantitative investigations into this core criterion are rare. Of those that have studied self-awareness in FTD patients explicitly, significant impairments have been found, even when using quite different methodologies to assess impaired awareness. Rankin et al (Rankin, Baldwin, Pace-Savitsky, Kramer, & Miller, 2005) used a questionnaire rated by both self and significant other (SO) describing the current personality of 12 FTD and 10 AD patients. They found that FTD patients showed significantly inaccurate self-awareness of their changes in personality compared to their SO ratings, whereas AD patients showed a mixture of both accurate and inaccurate self-assessments of personality. For impaired assessments, both FTD and AD patients described their personality as it had been prior to the onset of dementia. Another study investigated self-monitoring of memory performance in 6 patients with FTD and 16 with AD. The results indicated that FTD patients were more inaccurate at predicting their memory performance than the AD patients. FTD patients tended to overestimate their performance by approximately 35%, compared to 11% by the AD patients. This metamemory deficit was associated more with executive dysfunction than memory deficits (Souchay, Isingrini, Pillon, & Gil, 2003). Miller et al (2001) found from an analysis of FTD medical records, that those patients who exhibited the most dramatic changes in self as defined by changes in political, social or religious values, had more asymmetric loss of function in the right frontal lobe than left frontal or bilateral atrophy, evident from MRI and SPECT scans. A recent related study suggests that FTD patients are impaired on TOM tasks and are impaired on other aspects of social cognition such as moral reasoning, emotion processing and empathy. While

executive dysfunction appeared to underlie some of these elements, it did not adequately explain them all. The authors propose that some of these elements may provide explanations into the lack of social awareness frequently apparent in FTD patients (Lough et al., In Press). There has been a call for more systematic quantification of impaired awareness in FTD (Rankin et al., 2005).

Other more subcortical or frontal-subcortical dementias have also shown features of impaired awareness, including Huntington's Disease (HD) (Cummings & Benson, 1984; Deckel & Morrison, 1996), especially if the right hemisphere is involved (Aalten et al., 2005). HD is a genetic neurodegenerative disorder marked by motor, cognitive, and emotional dysfunction, that are associated with prominent caudate nucleus and frontal lobe atrophy (Kaszniak & Zak, 1996). McGlynn and Kaszniak (1991) showed that using a questionnaire discrepancy score measure, patients with HD significantly underestimated both their motor and cognitive impairments, but on a motor performance prediction task, patients were reasonably accurate. Parkinson's Disease (PD) is a disorder of the basal ganglia, associated with dopaminergic depletion of neurons in the substantia nigra (Cummings 1990). Loss of dopaminergic input to the striatum can subsequently alter efferent input to the globus pallidus, resulting in decreased activation of various cortical regions (Bergman & Deuschl, 2002; Cummings, 1990). It is characterised by classic motor symptoms of bradykinesia (slowness of movement), tremor, rigidity and is also associated with cognitive impairments such as bradyphrenia (slowness of thought processes), psychomotor slowing, memory deficits and difficulties with activities of daily living (ADL). In patients with PD, an early study found intact awareness using a clinical rating scale relative to patients with AD, vascular and multiple infarction dementia (Danielczyk, 1983). However, more recent studies using discrepancy scores between

patient and caregiver ratings as an indication of inaccurate perception of deficits have found significant impaired awareness in PD for both ADL difficulties (Leritz, Loftis, Crucian, Friedman, & Bowers, 2004) and cognitive deficits (Seltzer et al., 2001).

Summary of findings from clinical populations other than TBI affected by impaired self-awareness

The occurrence of impaired awareness in such diverse conditions as schizophrenia and cortical blindness underscores the pervasiveness of the phenomenon, the complexities in attempting to characterise it and the difficulties associated with assessment (McGlynn & Schacter, 1989). Nevertheless, several characteristics of impaired awareness in clinical populations can be observed. Neuroanatomically, many studies point to the association between damage to the frontal lobes and impaired self-awareness. Right hemisphere lesions appear to lead to increased risk of impaired awareness, at least in impaired self-awareness in hemiplegia, AD and FTD, although not exclusively, as both left hemisphere and bilateral lesions have also been implicated. Furthermore, both cortical and frontal-subcortical degeneration has been associated with impaired awareness. Neuropsychologically, some studies have shown associations between impaired awareness and deficits in executive functions, though this has been an inconsistent finding. Impaired awareness is not necessarily associated with a general cognitive impairment or severity of illness. Studies with AD have suggested associations with emotional distress and psychiatric disturbances and awareness, although the magnitude and directions are unclear. It is clear that there are different levels of impaired awareness, that it is not an all-or-nothing phenomenon. For example, dissociations between impaired awareness for physical/motor, behavioural and cognitive deficits have been reported. In terms of

measurement, the above overview highlights the wide range of methods used to assess impaired awareness, including clinical rating scales, questionnaire discrepancy scores, interviews, and performance prediction tasks, and that different findings can result from dissimilar methods of assessment. The lack of consensus about the correlates of impaired awareness can be partially explained by methodological issues, such as variety and accuracy of measures used. Heterogeneity at the metacognitive level has been purported to possibly explain some more of the variability between studies, at least in AD (Agnew & Morris, 1998). These observations will guide the review of the literature regarding impaired self-awareness following TBI, beginning with describing some common injury profiles following TBI.

1.5 Traumatic Brain Injury: Prevalence, Neuropathology and Cognitive and Behavioural Sequelae

Prevalence

Traumatic Brain Injury is described as "an insult to the brain caused by an external force that may produce diminished or altered states of consciousness, which results in impaired cognitive abilities or physical functioning" (NHIF, 1989, cited in Ponsford, 1995, p.1). It is the most common cause of brain injury in children and young adults (Grady & McIntosh, 2002) affecting approximately 200 - 220 individuals per 100,000 population (Granacher, 2004; Kraus, 1996) with the peak ages for TBI in the 15-24 year age range (Richardson, 2000). Road traffic accidents (RTA) represent the major cause of TBI (50-90%), followed by falls, assaults, occupational injuries and recreational accidents (Richardson, 2000). Modern medical

techniques are saving many accident victims who, as recently as ten or twenty years ago, may not have survived the metabolic and haemodynamic complications that follow TBI, resulting in an ever-increasing number of survivors, mostly young adults at the time of their injuries (Hsiang & Marshall, 1998; Lezak et al., 2004).

Neuropathology

The mechanics of head injury explain many of their common symptom patterns (Teasdale and Matthew, 1996). Head injury typically occurs in two stages: the primary injury is the acute physical insult that occurs at the moment of impact, caused directly by the blow, and the secondary injury consists of the effects of the physiological processes and systematic complications resulting from the primary injury. The predominant cause of brain damage from primary injury in static injuries, such as from blows to the head is contact force (force of impact). Inertial forces can involve translational acceleration (where the head moves in with the brain's centre of gravity) or rotational acceleration (where the brain rotates around its centre of gravity). Angular acceleration, a combination of translational and rotational acceleration, can result from movement of the head and neck on impact (Lezak et al., 2004).

Primary injury can result in cerebral contusions and/or diffuse axonal injury (DAI). Cerebral contusions consist of focal damage to brain tissue and vascular structure. They are usually found on the crests of the gyri of the cerebral hemispheres but can extend into the subcortical white matter (Adams et al., 1985; Halliday, 1999). The injury is referred to as a laceration when the lesions breach the fine tissue membrane that encloses the brain (the pia mater). The "coup" is the blow at the point of impact

and if local deformation has been sufficiently severe, a contusion will appear under this site of impact. "Contrecoup" lesions occur when the brain sustains a contusion in an area diametrically opposite the point of impact. Contrecoup lesions most frequently occur in the frontal and temporal lobes and lateral fissure/Sylvian fissure regions. Bruising occurs following rapid deceleration of the brain, such as when a vehicle comes to a sudden stop. A direct blow to the head is not necessary for bruising to occur as it can occur with rapid deceleration solely, resulting in the brain being slammed against the skull's bony protuberances in response to translational forces. These types of bruises are usually most pronounced at the frontal and temporal poles and their undersides (Halliday, 1999). When trauma has occurred with momentum on impact, such as with RTAs, victims can present with multiple-focal or bilateral damage, regardless of the site of impact, without clear-cut lateralisation (Ponsford, 1995).

Rapid acceleration, deceleration and rotational forces can result in damage to axons in cerebral and brainstem white matter. This damage is called diffuse axonal injury (DAI). DAI can be caused by torn axons, shearing of axon clusters, retraction balls of axonal substance and reactive swelling of strained and damaged axons (Graham, 1996). The shearing effects, although diffuse, tend to be concentrated in the form of microscopic lesions in the frontal and temporal lobes, the grey-white matter around the basal ganglia, the periventricular area of the hypothalamus, the superior cerebellar peduncles, the fornices, the corpus callosum and its fibre tracts (Groswasser, Reider-Groswasser, Soroker, & Machtey, 1987; Halliday, 1999; Pang, 1989). Severe DAI has been reported in nearly 50% of severe injuries (Graham, 1996).

Secondary brain damage due to the physiological processes and complications can be as destructive, if not more destructive, than the effects of immediate impact (Richardson, 2000). Haematomas (blood clots) can form as result of tearing of blood vessels leading to bleeding inside of the skull, causing compression of the brain. An extradural haematoma occurs from bleeding between the skull and the outer covering of the brain, the dura mater. A subdural haematoma is a collection of blood between the dura mater and the arachnoid, a translucent membrane that loosely envelops the brain and can result in ischaemic brain damage (insufficient blood supply). Other forms of secondary damage to the brain include elevated intracranial pressure, brain swelling/oedema, ischaemia as a result of inadequate blood flow and constant hypoxia (reduction of oxegen supply to tissues), fever, infection, respiratory failure and hypotension (Graham, 1996; Ponsford, 1995).

Neuroimaging in TBI

Initial scans following TBI are generally Computerised Tomography (CT) imaging. While CT imaging can detect haematomata, larger contusions and atrophy, it does not have sufficient resolution required for the detection of smaller areas of contusion or diffuse white matter lesions (Ponsford, 1995). Magnetic Resonance Imaging (MRI) is more sensitive to some aspects of neuropathology following TBI, in particular DAI and orbitofrontal lesions (Levin, Williams, Eisenberg, High, & Guinto, 1992). Traumatic pathology may develop over time and may not be visible on initial scans (MacKenzie et al., 2002). However, MRI also lacks the sensitivity to detect the microscopic lesions that are commonly associated with TBI (Ponsford, 1995). Even when neuropsychological findings indicate impairment supporting diagnosis of brain damage, MRI can still fail to detect tissue damage (Bigler &

Snyder, 1995). More recent imaging techniques such as Positron Emission Tomography (PET), Single Photon Emission Computer Tomography (SPECT) and Electroencephalography (EEG) can provide some evidence of cerebral damage not visible on structural imaging using CT or MRI (Bigler, 1999; Henry, Gross, Herndon, & Furst, 2000; Nedd et al., 1993). However, these techniques are not routinely available due to high costs and caution is recommended in their interpretation on the basis of their investigational status (Ponsford, 1995; Silver, Hales, & Yudofsky, 2002).

Despite the heterogeneity of TBI, and the fact that the routine structural imaging techniques may not detect smaller contusions, DAI or white matter damage, the neuropathological evidence available indicates that diffuse injury is common and that frontal and temporal lobes, the hippocampus and the basal ganglia are most vulnerable to damage (Ponsford, 1995).

Neuropsychological and behavioural sequelae of TBI

TBI has been referred to as a "hidden epidemic" due to the lack of overt physical signs of injury as the sequelae of TBI are more typically seen in changed cognitive and behavioural functioning (Flashman & McAllister, 2002; Kraus & McArthur, 1996). The diffuse damage leading to minute lesions and dispersed lacerations frequently associated with mild, moderate and severe TBI can compromise speed of information processing, cognitive efficiency, attentional functions and impaired complex reasoning, reflected in complaints of confusion, headache, irritability, and fatigue (Lezak et al., 2004). More localised brain lesions involving the frontal and temporal lobes, the areas most susceptible to damage following TBI, have been

associated with specific impairments including sensory/perceptual and motor functioning (Lezak et al., 2004). Selective, divided and sustained attentional deficits are common (Ponsford & Kinsella, 1992; Robertson, Manly, Andrade, Baddeley, & Yiend, 1997; vanZomeren & Brouwer, 1994). Persistent memory difficulties in the acquisition and retrieval of information are also evident, ranging from severe amnesic syndrome following bilateral hippocampal damage, to a more material-specific learning difficulty following, for example, unilateral temporal lobe injury (Ponsford, 1995). While short-term memory is less likely to be affected, working memory deficits are frequently present when patients attempt two tasks at a time (Lezak, 1979; McDowell, Whyte, & D'Esposito, 1997). Both recall and recognition on verbal and non-verbal material can be impaired (Baddeley, Harris, Sunderland, Watts, & Wilson, 1987). Damage to the frontal lobes can lead to prospective memory difficulties of "maintaining a stable intention to remember" (Kliegel, Eschen, & Thone-Otto, 2004; Walsh, 1991).

Symptoms of executive dysfunction are also associated with frontal lobe damage and include difficulties with a wide range of the most complex cognitive tasks including planning and problem-solving, abstract thinking, response inhibition, shifting task-set, mental flexibility error-monitoring and self-regulation (Lezak et al., 2004). Damage to the frontal lobes can also affect patient's personality, social adjustment and interpersonal behaviour. (Silver et al., 2002; Zhang & Sachdev, 2003). Changes in behaviour include various manifestations of executive dysfunction such as disinhibition, which can lead to impulsivity and inappropriate or irresponsible social behaviour, self-centredness leading to attention-seeking, manipulative or childish behaviour and poor empathy skills. Irritability, verbally threatening or physically aggressive behaviour and temper outburst are also common. At the other end of the

spectrum, changes in affect, reduced drive, initiative and motivation are also observed (Ponsford, Olver, & Curran, 1995). Reports have indicated that impaired self-awareness of deficits is as likely following TBI as memory and affective disturbances (Borgaro & Prigatano, 2002). The next section of this review will examine the phenomenon of impaired awareness following TBI.

1.6 Characteristics of Impaired Self-awareness following TBI

Since the early eighties, there has been a steady increase in the number of empirical investigations of impaired self-awareness (ISA) in Traumatic Brain Injury. Clinical reports have indicated that ISA is present in the majority of TBI patients (Prigatano & Fordyce, 1986), with empirical reports suggesting ranges from 25% to 97%, depending on the definition and measurement used (Freeland, 1996; Prigatano & Altman, 1990; Sherer, Bergloff, Levin et al., 1998). As will become increasingly evident, methodological issues hamper many firm conclusions about ISA in TBI. These issues will be discussed in Section 1.8. Despite inherent methodological complications, it is clear that ISA is very common following TBI. Characteristics of ISA have been examined, such as modalities/domains of ISA, injury, behavioural, cognitive correlates of ISA, as well as associations between ISA and emotional distress, outcome and longitudinal course in TBI. These characteristics will be discussed separately.

Neuroanatomical considerations of ISA following TBI

The neural substrate of ISA following TBI remains elusive. Many investigators have emphasised the role of the frontal lobes, prefrontal and frontal systems in ISA (Frith & Dolan, 1996; Stuss, 1991; Stuss & Benson, 1986), which has intuitive merit considering the vulnerability of the frontal systems following TBI. However, ISA has been attributed to several brain regions, including heteromodal functional areas of temporoparietal and prefrontal cortices (Prigatano, 1991), and paralimbic structures of the temporal pole, caudal orbitofrontal cortex, anterior insula, cingulate parahippocampal and retrosplenial gyri (Cicerone & Tanenbaum, 1997; Mesulam, 1985b; Prigatano & Schacter, 1991). Very few studies have explicitly examined the association between ISA and location of lesions following TBI. Ranseen et al (Ranseen, Bohaska, & Schmitt, 1990) assigned 32 TBI patients into groups based on their CT scans, resulting in 13 being assigned to the left hemisphere lesion group, 8 to the right hemisphere group and 11 to the diffuse or bilateral injury group. ISA was assessed using a discrepancy score between self-rated competencies and clinician rated competencies on the Patient Competency Rating Scale (PCRS). The right hemisphere injury group rated their competency as much better than the clinicians ratings of their functioning, indicating impaired ISA. This discrepancy score was much larger in the right hemisphere group compared to the left hemisphere injury group and the diffuse/bilateral injury group. Vilkki and Holst (1991) found that patients with anterior lesions overestimated their performance predictions compared to patients with more posterior lesions, but they found no difference in patients' estimates with right or left hemisphere lesions. Prigatano and Altman (1990) measured ISA in a group of 64 TBIs, dividing the patients into three groups representing those who significantly overestimated their competencies on the PCRS compared to relatives, those who had similar ratings to their relatives ratings, and those who underestimated their functioning compared to relatives ratings. The group

that overestimated their competencies did not differ significantly from the other two groups in terms of presence of frontal lesions, or right hemisphere lesions. However, the group with most impaired ISA did have a greater number of lesions than the other two groups. Prigatano (1996) also failed to find differences in ISA in patients with right versus left hemisphere lesions. In a more recent study, Sherer et al (2005) analysed CT scans of 91 TBI participants whose ISA was assessed using the Awareness Questionnaire, rated by each patient and a clinician (Sherer, Bergloff, Boake, High, & Levin, 1998). In their study, almost all patients rated their functioning as more intact than did their clinician. Their results showed that the greater the number of lesions (both contusions and subdurals) seen on the initial CT scan was significantly predictive of ISA, consistent with Prigatano and Altman's (1990) findings, despite disparate methodologies used in both studies. Neither lesion volume nor location had any significant associations with ISA. The authors suggest that the small lesions typical of DAI in TBI would contribute to lesion number but less to lesion volume. Based on their findings, Sherer et al (2005) propose that intact self-awareness requires the integrated operation of broadly distributed neural networks. They suggest that the burden of a lesion in any specific location may be less relevant than disruption across multiple regions.

Support for this proposal of multiple neural network involvement in self-awareness comes from functional imaging studies with neurologically-healthy individuals. For example, Johnson et al (2002) asked healthy individuals to make self-related judgements using a yes/no paradigm (e.g. "I forget things easily/ I'm good at my job") and compared these to statements about factual judgements (e.g. "Ten seconds is more than a minute/ you need water to live"). An equivalent proportion of "yes" answers were given in both the semantic knowledge condition and the self-reflection

condition. Functional magnetic resonance imaging (fMRI) scan data for the self-reflection conditions showed consistent activation of the anterior medial prefrontal region, posterior cingulate areas as well as in the thalamus area. Vogeley et al (2001) scanned individuals using fMRI while they answered questions based on short stories presented relating to mental states of others (theory-of-mind, TOM), or relating to themselves. While both TOM and self-reflection conditions activated the right prefrontal cortex and the anterior cingulate cortex, self-reflection conditions individually also activated the right temporoparietal junction and the medial aspects of the superior parietal lobe. Many other studies investigating aspects of self-reflection in healthy individuals have found increased activation of medial frontal lobe areas (Frith & Frith, 1999; Gusnard, Akbudak, Shulman, & Raichle, 2001; Kelley et al., 2002; Macrae, Moran, Heatherton, Banfield, & Kelley, 2004; Platek, Keenan, Gallup, & Mohamed, 2004). However, other areas have also shown increased activation in fMRI investigations of self-reflection, including the left orbitofrontal cortex, the precuneus in the posterior parietal lobe, bilateral temporoparietal regions, the anterior cingulate (Kjaer, Nowak, & Lou, 2002), and the posterior cingulate region (Gusnard et al., 2001; Kelley et al., 2002). Despite different types of introspection/self-reflection tasks used, there does appear to be some agreement that the medial prefrontal cortex plays a significant role, along with other distributed regions including both anterior and posterior cingulate areas, parietal, temporal and thalamic activations.

This was shown in a very recent study where fMRI activation on a self-appraisal task of one's traits and abilities was examined in 20 TBI patients and 20 controls. For both TBI and control participants, the medial prefrontal and retrosplenial cortical regions were shown to play a primary role in self-referential evaluative processes.

Interestingly, the TBI group as a whole appeared to demonstrate a compensatory neural response to metacognitive processes underlying self-evaluation in greater activation in the anterior cingulate, precuneous and right temporal pole. Decreased right anterior superior frontal gyrus neural functioning was also associated with reduced self-evaluative accuracy on the Patient Competency Rating Scale (PCRS) Schmitz, Rowley, Kawahara, Johnson, in press).

More localised frontal regions are likely to be involved in specific performance-monitoring capacities, as shown in numerous studies of error detection abilities in neurologically-healthy individuals. Stuss (1991) proposed that self-awareness in behavioural terms is reflected in error-monitoring and self-regulation. Event-related potential (ERP) studies of error-detection capacities have shown a specific error-related negativity (ERN) 80 to 150 ms after incorrect responses on tasks (Falkenstein, Hoorman, Christ, & Hohnsbein, 2000; Gehring, Gross, Coles, Meyer, & Donchin, 1993). Investigators propose that the ERN occurs as a result of a mismatch of comparison between an internal representation of the correct response and the actual response (Bernstein, Scheffers, & Coles, 1995). The ERN is thought to have a medial-frontal generator, with the anterior cingulate cortex (ACC) frequently implicated (Dehaene, Posner, & Tucker, 1994). The ACC, along with the prefrontal cortex, have also been implicated in error-related processes using fMRI techniques, in particular processes involved in error-detection (Garavan, Ross, Murphy, Roche, & Stein, 2002a; Kiehl, Liddle, & Hopfinger, 2000,Garavan, Ross, Kaufman, & Stein, 2003). Bush et al (Bush, Luu, & Posner, 2000) propose that the ACC is divided between a more rostral affective region and a more caudal cognitive region. However, a recent fMRI investigation has found that activation of the ACC was equivalent for both aware and unaware errors on a response inhibition task. Explicit

awareness of an error and subsequent post-error behaviour change was associated with bilateral prefrontal and inferior parietal brain activation, suggesting that the ACC may be necessary for detection of errors, but not for either conscious error awareness or post error behaviour (Hester, Foxe, Molholm, Shpaner, & Garavan, 2005).

The above findings from neurologically-healthy individuals provide support for Sherer et al's (2005) proposal that multiple neural network disruption following TBI, rather than a specific lesion location results in ISA, although it is clear that prefrontal areas may have particular importance in various aspects of monitoring and self-reflection, as proposed by Stuss et al (Stuss, 1991; Stuss, Picton, & Alexander, 2001). While focal lesions may explain some of the possible mechanisms that underscore some physical, sensory and language deficits in some clinical syndromes, it is proposed that focal lesions cannot account for all manifestations of ISA, in particular ISA for personality and behavioural changes (McGlynn & Schacter, 1989). Furthermore, as outlined in section 1.5 above, DAI and white matter damage is common following TBI with the frontal and temporal lobes, hippocampus and basal ganglia most vulnerable (Ponsford, 1995). Because of the likelihood of diffuse and non-specific damage in TBI, investigations regarding specific focal neurological injury to explain the diverse presentations of ISA are considered to be somewhat arbitrary (Sawchyn, Mateer, & Suffield, 2005). This has led several investigators to focus on exploring indices of more diffuse brain damage, such as severity of injury and general cognitive decline.

As ISA is generally considered to be mainly a neurological deficit, it would seem intuitive based on clinical anecdotes that more severe injuries would show worse ISA (Sherer, Oden, Bergloff, Levin, & High, 1998). Levin et al (1987) found that clinician ratings of patients' accuracy of self-appraisal was dependent on severity of injury, with more severely injured patients being rated as less accurate in their self-appraisal. Other studies have shown that underestimation of deficits was more pronounced in TBI patients with severe injuries than mild or moderate injuries (Leathem, Murphy, & Flett, 1998; Sunderland, Harris, & Gleave, 1984).

Severity classification is generally based on duration of post-traumatic amnesia (PTA) and depth and duration of coma using the Glasgow Coma Scale (GCS) (PTA: (Bigler, 1990; GCS: Jennett, Teasdale, & Knill-Jones, 1975; Russel & Nathan, 1946). It has been suggested that severity of injury, at least as measured by PTA, is a behavioural marker of DAI (Katz & Alexander, 1994; Ommaya & Gennarelli, 1974). Severity as measured by longer duration of PTA has been found to correlate with worse ISA in some studies (Noe et al., 2005; Prigatano, 1999a; Prigatano et al., 1998; Sherer et al., 2005; Trudel, Tryone, & Purdum, 1998), and lower GCS scores in other studies (Prigatano et al., 1998; Sherer et al., 1998). Prigatano (1999b) also provided some indirect evidence of the relationship between ISA and severity of injury using the Halstead Finger Oscillation Test (Reitan, 1955), as speed of finger tapping correlates with the amount of time it takes TBI patients to respond to commands in a meaningful way (Dikmen, Machamer, Winn, & Temkin, 1995). Worse ISA and slower speed of finger tapping was shown in two studies (Prigatano & Altman, 1990;

Prigatano, Ogano, & Amakusa, 1997). Prigatano concluded that ISA may represent a disturbance of conscious experience and indicate the presence of diffuse injury.

However, even where severity is found to be associated with ISA, the correlations are generally quite modest, for example .39, in Sherer et al (1998), and -.39 in Prigatano et al (1998). Furthermore, the majority of studies have found no such association between severity measures and ISA in TBI (Allen & Ruff, 1990; Anderson & Tranel, 1989; Fleming, Strong, & Ashton, 1998; Gasquoine, 1992; Godfrey, Partridge, Knight, & Bishara, 1993; Hart, Sherer, Whyte, Polansky, & Novack, 2004; Hart et al., 2003; Lanham, Weissenburger, Schwab, & Rosner, 2000; Port, Willmott, & Charlton, 2002; Prigatano & Altman, 1990; Sawchyn et al., 2005; Sbordone, Seyranian, & Ruff, 1998), or mixed findings, such that severity or ISA as measured using one particular measure is associated while the association is not significant using another measure in the same study (Noe et al., 2005; Prigatano et al., 1998; Sherer et al, 1998).

Similarly, studies investigating the relationship between general cognitive functioning and ISA have also yielded inconsistent results. While there has been some limited support for an association between general cognitive decline and ISA based on IQ tests in some studies (Anderson & Tranel, 1989; Bogod, Mateer, & MacDonald, 2003; Fischer, Gauggel, & Trexler, 2004; Noe et al., 2005; Prigatano & Fordyce, 1986), a similar number of studies have found no associations with basic measures of intellect (Burgess, Alderman, Evans, Emslie, & Wilson, 1998; Fischer, Trexler, & Gauggel, 2004; McKinlay & Brooks, 1984; Newman, Garmoe, Beatty, & Ziccardi, 2000; Prigatano & Altman, 1990). Furthermore, ISA has been observed in

individuals with intact intellectual functioning following brain injury (McGlynn & Schacter, 1989).

The general lack of consistent associations between severity and ISA have been hypothesised to be due to small sample sizes, restricted range of severities used in many studies and different research designs used (Crisp, 1992; Sherer et al. 1998). However, taking all the findings into consideration, it appears neuropsychological and behavioural aspects may play a more significant role in ISA than severity or general cognitive decline (Ownsworth, McFarland, & Young, 2002).

ISA and neuropsychological correlates

Early research into ISA frequently failed to find any associations with standard neuropsychological measures of memory, attention and basic executive functions (Prigatano & Altman, 1990; Prigatano, Altman, & O'Brien, 1990) and a specific neuropsychological profile of ISA failed to develop (Giacino & Cicerone, 1998). Prigatano and Altman (1990) explained these limited findings by proposing that ISA after TBI may be related to neuropsychological changes not measured by standard tests as they do not provide an adequate indication of frontotemporal damage proposed to underlie deficits in awareness. These authors proposed that the heteromodal cortex and the paralimbic belt may serve to integrate perceptual information about external reality with information about the internal state of the individual and that it may be this function integrating both cognitive and affective experiences that may be involved in self-awareness. Consequently, many standard neuropsychological tests did not adequately incorporate the integration of cognitive

and affective components leading to the limited findings with only basic cognitive, perceptual or memory dimensions.

More recent investigations have incorporated a vast array of diverse measures sensitive to executive dysfunction and subtle memory difficulties in order to address this issue. While some investigations of ISA still fail to find correlations between ISA and neuropsychological measures (Bogod et al., 2003; Lanham et al., 2000; Newman et al., 2000), many studies have since found that various measures of executive function and some measures of memory function do indeed correlate with ISA, such that individuals with more impaired executive functions are likely to show worse ISA (Hart et al., 2005; Noe et al., 2005; Ownsworth & Fleming, 2005; Ownsworth et al., 2002).

A series of studies found that specific aspects of executive functioning were associated with levels of awareness including capacities reflecting generation or fluency of ideas and concept formation (Burgess et al., 1998; Coben, Boksenbaum, & Kulberg, 1995; Ownsworth & Fleming, 2005), response inhibition (Bogod et al., 2003), ability to set realistic goals (Fischer, Gauggel et al., 2004), sustained attention and freedom from distractibility (McAvinue, O'Keeffe, McMackin, & Robertson, 2005; Trudel et al., 1998), abstract reasoning and problem-solving (Bohac, Malec, & Moessner, 1997; Campodonico, 1992; Fischer, Trexler et al., 2004), error behaviour and self-regulation (Bogod et al., 2003; Burgess et al., 1998; Ownsworth & Fleming, 2005), perseverative errors (Burgess et al., 1998; Noe et al., 2005; Trudel et al., 1998) and working memory abilities (Bogod et al., 2003). Other aspects of memory capacity have also been related to ISA, such as delayed recall (Noe et al., 2005) and overall memory score (Trudel et al., 1998).

Examining executive functions more specifically, Lezak et al (2004) conceptualises these complex processes as having four main components: volition, planning, purposive action and effective performance. Volition is described as the capacity of intentional behaviour, the extent to which individuals are in touch with their own needs and that of their environment in order to determine what one needs or wants, and a future realisation of those needs and wants. Planning is considered to involve the identification and organisation of the elements needed to achieve a goal, of which working memory, self-monitoring, decision-making and good impulse control are considered vital components. Purposive action is described as how the plan or intention is translated into productive activity, which requires the ability to initiate, maintain, self-regulate, switch and stop sequences of complex behaviour. Effective performance is the ability to monitor, self-correct and regulate the delivery of intentions and goals. Each of these components is proposed to be intimately related with self-awareness. A series of studies by Ownsworth and colleagues (Ownsworth & Fleming, 2005; Ownsworth et al., 2002; Ownsworth, McFarland, & Young, 2000) have examined the contribution of some of Lezak's proposed elements of executive function to ISA following TBI. These studies used Crosson et al's (1989) model of awareness, described below section 1.7, which differentiates between "intellectual awareness"- the capacity to recognise deficits exist, "emergent awareness"- the capacity to recognise errors in one's performance as they occur, and "anticipatory awareness"- the capacity to predict how specific deficits will impact on tasks. Impaired capacity for volition, as measured by the Health and Safety subtest of the Independent Living Scale (Loeb, 1986), was found to be associated with more impaired intellectual awareness, emergent and anticipatory awareness. Impaired purposive behaviour, as measured by the Tinker Toy Test (Lezak, 1995), was associated with both impaired emergent and anticipatory awareness. These findings

provided support for the view that in order for neuropsychological tests to reflect impaired awareness, they needed to measure both cognitive and affective components in self-perception, and in particular tests that integrate perceptual information about an individual's internal status with perceptual information about the external environment, as reflected in tests of volition, and purposive behaviour (Ownsworth et al., 2002; Ownsworth et al., 2000).

ISA and behavioural and psychiatric outcome

Recent studies have found evidence that poorer ISA following TBI is significantly related to higher psychiatric symptomatology (Hoofien, Gilboa, Vakil, & Barak, 2004; Noe et al., 2005), as measured by Neuropsychiatric Inventory (NPI) and the Global Severity Index (GSI) of Symptom Checklist-90 (NPI: (Vilalta-Franch et al., 1999); GSI: (Derogatis, 1977). Hoofien and colleagues (2004) also found that TBI patients with worse ISA showed greater behavioural disturbances, such as impulsiveness, rigidity, dullness, frustration, and aggression as rated by their families or caregivers. Trudel et al (1998) also found that ISA was a significant predictor of asocial, externalised and internalised maladaptive behaviour.

ISA and outcome for individuals with TBI – Functional, Rehabilitation, Vocational

Early neuropsychological interest in the concept of ISA was initially based on the clinical impression that poor self-awareness and unrealistic expectations were primary reasons for failure to gain employment following rehabilitation (Ben-Yishay, Rattok, & Piasetsky, 1985; Ben-Yishay, Silver, Piasetsky, & Rattok, 1987). It has been consistently proposed that patients with ISA may be indifferent or even

resistant towards rehabilitation and treatment (Port et al., 2002; Sohlberg, Mateer, Penkman, Glang, & Todis, 1998). The relationship between worse ISA and poorer outcome, which has intuitive merit, has had strong empirical support in the literature. A number of studies have found that TBI patients in post-acute rehabilitation with worse ISA have shown poorer compliance and participation in treatment (Fleming et al., 1998; Lam, McMahon, Priddy, & Gehred-Schultz, 1988; Malec, Smigielski, & DePompolo, 1991). Patients with worse ISA have also been found to require longer lengths of stay in postacute rehabilitation before work placements (Malec, Buffington, Moessner, & Degiorgio, 2000). Poorer levels of ISA during acute inpatient rehabilitation has also been associated with poorer functional status at discharge (Sherer, Hart, Nick et al., 2003). Poorer self-awareness in post-acute rehabilitation has also been frequently associated with a poorer employment outcome (Ezrachi et al, 1991; Fischer et al., 2004; Ownsworth & Fleming, 2005; Sherer et al., 1998; Wise, Ownsworth, & Fleming, 2005). Sherer et al (2003) have shown that TBI patients who showed better self-awareness of deficits were almost three times more likely to be employable at the end of a rehabilitation programme. Better self-awareness has also been significantly associated with greater functional independence, independent living skills and better residential status (Hoofien et al., 2004; Malec & Moessner, 2000; Noe et al., 2005; Ownsworth & Fleming, 2005; Trudel et al., 1998). Marcia (1991) found that greater self-awareness was a positive significant predictor of the individual's marital consensus and adjustment, the spouses psychological well being, marital satisfaction, and affectional expression.

However, there has also been mixed findings in relation to outcome. For example, some studies have found that ISA is related to worse behavioural or functional outcome but not to vocational status (Hoofien et al., 2004; Malec & Moessner,

2000). It was suggested following both these studies that the commonly accepted association between outcome and ISA is revealed more strongly when outcome is measured by subjective ratings of behaviour or functional outcome relative to more objective measures of outcome, such as actual vocational status. Furthermore, other studies have also failed to find any significant differences between TBI patients with high self-awareness and low self-awareness in terms of vocational status and rehabilitation outcome and adjustment to disability (Cavallo, Kay, & Ezrachi, 1992; Malia, Torode, & Powell, 1993). In another study, although TBI with low self-awareness showed significantly lower motivation for rehabilitation participation than those with high self-awareness, there was no significant differences in either high or low self-awareness outcome in terms of perceived disability or community re-integration (Fleming et al., 1998). Sherer et al (1998) proposed that outcome is not necessarily directly affected by awareness but it may be mediated by cognitive factors and readiness for change, or as can be seen in Fleming et al's (1998) study, the influence of emotional distress.

While the above review highlights that ISA is generally considered to be a significant barrier to successful rehabilitation, functional, behavioural and vocational outcome, it is clear that there are many inconsistencies. Yet again, a wide variety of different methodologies have been employed in the assessment of ISA in particular but also in the assessment of outcome. For example, Sherer et al (2003) use larger discrepancy scores on a questionnaire (Awareness Questionnaire) to indicate ISA, and employability scale from the Disability Rating Scale. In contrast, Cavallo et al (1992) used an interview (NYU Head Injury Family Interview) to assess vocational and academic status and differences in frequency of reported problems by patient and significant others as an indicator of ISA. Methodological issues in the assessment of

ISA are discussed further in section 1.8. Overall, it has been suggested that better self-awareness, as manifested in greater capacity to self-monitor, recognise errors and adjust performance to an adequate level of functioning may lead to greater success in a range of productive activities. It may be also be the case that successful reintegration into work and recreational activities may aid the development of self-awareness and self-regulation skills (Ownsworth & Fleming, 2005).

ISA and relationship with emotional distress

As described, while self-awareness of competencies and deficits is generally thought to be beneficial or even a pre-requisite for successful rehabilitation, better vocational outcome and independent living status (Sherer et al., 1998; Trudel et al., 1998), research has shown that as self-awareness improves, emotional reactions can also increase, including depression, anxiety, grief and catastrophic reactions. In a cross-sectional study, TBI patients in the acute stages of recovery whose ratings of competency were closer to staff ratings also rated themselves as having more depressive symptoms (Ranseen et al., 1990). Similar findings were shown in a study by Heilbronner et al (Heilbronner, Roueche, Everson, & Epler, 1989) who showed that TBI patients with good insight were rated significantly more depressed than those who demonstrated poorer insight. In a longitudinal study, Godfrey et al (1993) followed a group of TBI patients at 6 months post-injury to 3 years post-injury. They found that awareness improvement in the post-acute phase was associated with an increase in emotional distress. Fordyce and colleagues (Fordyce & Roueche, 1986; Fordyce, Roueche, & Prigatano, 1983) made similar observations. A recent study by Sawchyn et al (2005) also found that emotional adjustment rated by a significant other is a stronger predictor of a patients' awareness than the severity of their injury,

such that patients' acknowledgements of neurobehavioural problems was positively associated with ratings of their emotional adjustment. Fleming, Strong and Ashton (Fleming et al., 1998) also found that TBI patients who showed higher levels of self-awareness showed significantly higher levels of emotional distress. Interestingly, their high self-awareness group also had higher levels of motivation to change behaviour. TBI patients in the low self-awareness group displayed a lower level of motivation to change but also lower levels of emotional distress. The authors proposed that depression formed a feedback loop for the patients with high self-awareness such that their increased motivational state was influenced and led to a reduction in positive coping behaviours, as the groups did not differ in terms of overall outcome variables. They suggest, considering the patients in this study were only 1 year post-injury, that given time, patients with high self-awareness may be able to work through their emotional distress which in turn might lead to better outcomes. Prigatano (1988) also proposed that a feedback system exists where emotion and motivation contribute to self-awareness and vice versa.

Some argument exists regarding the direction of the relationship between emotional distress and greater awareness of deficits. It has been argued that symptoms of depression may increase the likelihood of reporting more deficits for example (Malec, Machulda, & Moessner, 1997; Wallace & Bogner, 2000). Furthermore, a number of studies have not found a significant relationship between awareness ratings and reported levels of depression and anxiety (Fischer, Trexler et al., 2004; Noe et al., 2005; Wallace & Bogner, 2000) and some have even found that increased awareness is associated with *better* emotional outcome (Ownsworth & Fleming, 2005).

A possible reason for these inconsistent findings is that in the acute stages post-injury increased self-awareness following TBI is associated with greatest emotional distress (Fleming et al., 1998; Godfrey et al., 1993), while in the later years post-injury individuals increased awareness, manifested in self-efficacy, strategy behaviour, coping skills and improved error-monitoring is related to increased hope for the future, while all these factors interact to influence individual's vulnerability to emotional distress. These interactions were found in a group of patients with relatively long-term TBI (Ownsworth & Fleming, 2005). It is also possible that lower levels of awareness or "denial" in acute stages post-injury may act as a useful coping mechanism to preserve self-esteem and maintain hope (Fleming & Strong, 1995; Nockelby & Deaton, 1987). Some authors proposed that the use of coping strategies such as denial may act as a buffer for emotional distress, thereby giving individuals some time to develop more effective coping strategies (Fleming & Strong, 1995; Gainotti, 1993; Moore, Stambrook, & Peters, 1989). While the role of psychodynamic explanations of ISA has received much criticism in the literature from many fronts, as will be discussed in section 1.7, there has been some evidence of an indirect relationship between coping-related and personality-related denial and impaired awareness (Ownsworth et al., 2002).

Another aspect of emotional distress relates to the caregivers of TBI survivors, who have frequently been shown to experience long-term psychological consequences following brain injury of a close relative (Brooks, 1984; Brooks, Campsie, symington, Beattie, & McKinlay, 1986; Cavallo et al., 1992; Douglas & Spellacy, 1996). McKinlay et al (McKinlay, Brooks, Bond, Martinage, & Marshall, 1981) found that almost one third of family members of patients reported severe stress levels with no change up to 12 months post-injury, while up to 47% of one sample of

families met diagnostic criteria for a major depressive episode (Gillen, Tennen, Affleck, & Steinpreis, 1998). A number of authors have found that high caregiver distress was related to the behavioural and personality changes of TBI patients (Brooks et al., 1986; Kreutzer, Marwitz, & Kepler, 1992; Livingston, Brooks, & Bond, 1985). Caregivers perception of increased problems in the individuals with TBI has been significantly related to poorer quality of life and psychosocial outcome (Hickey, O'Boyle, McGee, & McDonald, 1997) and an increase in carer's reported symptoms of depression (Wallace & Bogner, 2000). In AD, as mentioned above, greater perceived caregiver distress has been related to ISA (DeBettignies et al., 1990). Similar associations have been recently found in the TBI literature, such that family members' level of distress was significantly correlated with their perception of the patients' ISA (Prigatano et al., 2005). ISA was also shown to be one of the most significant correlates of caregiver life satisfaction and strain in a study of quality of life 10 years after TBI (Koskinen, 1998). Similarly, another study showed that in the absence of adequate social support, caregiver distress significantly increased with worse ISA in TBI patients (Ergh, Rapport, Coleman, & Hanks, 2002).

It is clear that further research is required to elucidate the interplay of emotional distress and ISA in individuals with TBI in both the acute and long-term post-injury stages, as well as the proposed association between increased ISA and increased caregiver burden and distress. It is of particular importance during the rehabilitative process considering that emotional distress may have a negative impact on positive rehabilitation outcome in the early stages post-injury (Fleming et al., 1998).

The term *specificity* has been used to describe the degree to which lack of awareness is restricted to a particular deficit, or domain of functioning (Schacter & Prigatano, 1991). It has been frequently suggested that awareness is not a unitary, all-or-nothing concept, such that ISA can differ across various areas of functioning (Toglia & Kirk, 2000). Early observations found high agreement between TBI patients and relatives on motor and sensory impairments (for example 85% agreement for hearing impairment and 77% for visual impairment), moderate agreement on memory and attention impairments (65% and 63% respectively) and least agreement on emotional and behavioural changes (60% bad temper and 52% for anxiety) (McKinlay & Brooks, 1984). Similar findings using item analysis of the Patient Competency Rating Scale (PCRS) have found that discrepancy between self and SO ratings is greater for items of an interpersonal or emotional nature with TBI patients consistently underreporting their difficulties on these items compared to relatives or clinicians, whereas no such underestimation is frequently found on items primarily related to self-care and basic ADLs (Fischer, Trexler et al., 2004; Fleming & Strong, 1999; Prigatano, 1996; Prigatano et al., 1990). This distinction between TBI patients showing greater accuracy at reporting physical impairments than non-physical (cognitive and emotional) impairments has been demonstrated in several other studies using different methodologies. Other discrepancy ratings have found similar physical/non-physical distinctions, including the Awareness Questionnaire (AQ) with both clinician and relative ratings (Hart et al., 2004; Sherer, Boake et al., 1998; Sherer, Hart, Nick et al., 2003), the Mayo-Portland Adaptability Inventory (MPAI) study that found TBI patients (n=43) underreported difficulties in the areas of communication, emotional, independent living skills and relationships compared to

relatives, but high concordance was found for the mobility category (Murrey, Hale, & Williams, 2005), and a self-other discrepancy scale the Neurobehavioral Functioning Inventory-Revised (NFI-R) found that where there was disagreement, TBI patients (n=267) tended to underreport aggression and depression levels, and memory and attention difficulties, rather than physical or somatic symptoms (Hart et al., 2003). Similar findings have been shown using an awareness interview that compares self-reported difficulties to actual neuropsychological and neurological ratings, with only 2 of 19 head trauma patients showing ISA of motor defects compared to 68% showing ISA on cognitive defects (Anderson & Tranel, 1989). In a study that compared the frequency of subjective problems reported on a general interview by patient and SO, patients (n=50) significantly underreported emotional and cognitive complaints compared to SO, while frequency of reported somatic complaints did not differ (Sbordone et al., 1998). In a task of naturalistic actions, Hart and colleagues (Hart, Giovannetti, Montgomery, & Schwartz, 1998) demonstrated that TBI patients (n=18) did not rate themselves as performing more poorly than controls on the cognitive elements of the task, despite making more errors, but were more accurate at rating the physical elements of their errors.

A number of suggestions have been put forward to explain the generally more accurate reporting of physical/adls/somatic/motor difficulties found compared to more inaccurate cognitive/interpersonal/behavioural/personality changes. Hillier and Metzer (Hillier & Metzer, 1997) discussed awareness across different domains in terms of Maslow's (1954) Hierarchy of Needs. They propose that patients may be more aware of physical/self-care difficulties because these needs have to be first met in order to address higher-order needs such as socialisation, self-actualization etc. Another proposal relates to the degree of concreteness or abstraction of the different

functions, with physical and motor items considered to be more concrete, observable deficits to which individuals may be more frequently exposed to in daily functioning than more abstract cognitive and socioemotional deficits. However, the one study that examined this concrete-abstract continuum hypothesis found that at least for awareness of cognitive deficits (attention, memory and verbal comprehension) awareness was not differentially distributed along that continuum (Hoofien et al., 2004). Further evidence against either the concrete-abstract hypothesis or the Hierarchy of Needs hypothesis has come from studies that have found TBI patients reporting less physical deficits than cognitive/social/emotional difficulties compared to SO/clinician (Hillier & Metzer, 1997). In a study with 87 acquired brain injury patients, mainly from CVAs, most disagreement was found between staff and patients with regard to physical items of the PCRS, with patients rating themselves as far less impaired than staff. In contrast, patients rated themselves as more severely impaired on items of cognitive and emotional difficulties than staff members rated them (Gauggel, Peleska, & Bode, 2000).

Other studies that have shown considerably high agreement in most domains of functioning with large TBI samples, with the exception of those relating to home integration or self-care, which showed significantly different perspectives from patient and SO, with TBI patients considering themselves as more active in completion of household responsibilities in areas such as meal preparation, housekeeping and basic self-care. It was suggested that discrepancies in home integration may be due to the prevalence of males in the TBI study samples and that 50% of caregivers were mothers and 25% were spouses of the patients and that disagreement about household responsibilities may also be common among a non brain-injured population (Sander et al., 1997; Tepper, Beatty, & DeJong, 1996).

Greater perceived difficulty found on mobility and self-care tasks could be due to the close involvement of the SOs in the daily care of the individual with brain injury (Hillier & Metzer, 1997) or that specific underreporting of self-care/adls may vary according to cultural values. For example, while Japanese TBI patients tended to underreport their difficulties overall on the PCRS, they showed accurate reporting of emotional and behavioural items, but significantly overestimated their ability to perform self-care activities, considered important socially in Japanese culture (Prigatano et al., 1997). Similarly, the importance of the wider socio-cultural context was purported to explain differences found in a study of New Zealand TBI patients with English ancestry and Maori ancestry. Non-Maori TBI patients overestimated their competencies particularly in social and emotional functioning using the PCRS, similar to the many studies outlined that demonstrated these findings in American and Australian participants. However, the Maori TBI patients responded differently, reporting a significantly lower level of competencies compared to their SO ratings or the non-Maori TBIs. There was a confounding variable that the Maori TBIs had a significantly higher incidence of left hemisphere dysfunction compared to the non-Maori TBIs, limiting the cross-validity and cultural interpretations of the study (Prigatano & Leathem, 1993).

Psychosocial factors have therefore been put forward as an alternative to the concrete-abstract explanation as to what may influence which symptoms are more readily reported. In a longitudinal study, Powell et al (Powell, Machamer, Temkin, & Dikmen, 2001) found that TBI patients perceived that physical impairments were the primary barrier to recovery within 1 year post-injury, while over time, physical problems decreased and concerns about cognitive problems increased. It was proposed that TBI patients may place greater importance on the role of physical

difficulties in recovery, reflecting priorities of young individuals who may have led very active lives prior to their injury in terms of employment and recreation, and that TBI patients may see physical impairments as the main block to returning to community living. It may not be until they return to familiar settings and situations post-discharge that they recognise their cognitive difficulties more readily as they experience problems in activities that they were able to accomplish prior to their injury. On the other hand, clinicians and families may be aware that it is possible to compensate for physical deficits with good cognition and social skills but the opposite is not so easily achievable. The authors propose that such dimensions may not be evident in the many cross-sectional studies examining domains of ISA as many studies have a large range of chronicity in their TBI patient groups.

Schacter (1990) proposed a more neurologically based explanation, relating to the existence of a modular Conscious Awareness System. If the CAS itself is disrupted, this is hypothesised to lead to general, global ISA across all domains, whereas if the connection between the CAS and one of its knowledge modules (such as a cognitive or behavioural module) is disrupted due to brain injury, the TBI patient may have more limited access to this information compared to accurate access to information regarding other domains (Prigatano & Schacter, 1991). This may explain why not all studies have found the same physical/non-physical distinction. This theory will be expanded upon in more detail in section 1.7.

Methodological issues have yet again been purported to explain the inconsistencies in the findings, such as the frequently small sample sizes, ranging from 18 to 64 in many studies, before the large samples of TBI patients were recruited that included between 161 and 301 TBI patients (Hart et al., 2004; Seel, Kreutzer, & Sander,

1997). Secondly, with the exception of some studies (Leathem et al., 1998; Seel et al., 1997), severity groups were not always differentiated in studies examining ISA and domains of functioning, and many studies only included those with severe head injuries. In those that also examined patients with mild and moderate head injuries, high agreement in general was found between patients and SO, with no differences being found for cognitive and emotional items (Leathem et al., 1998) or patients reporting greater levels of dysfunction than their SO (Seel et al., 1997). Thirdly, inconsistent use of awareness measures across studies, and investigators depending on one discrepancy score from one single questionnaire given to patients and their SO/clinicians to indicate ISA has many limitations. A further methodological issue is that clinicians and SO do not always agree on TBI patient functioning, as found in a study that demonstrated a modest association on physical and community items between clinician and SO raters, but no association for ratings on cognitive and behavioural items (Sherer, Boake et al., 1998). This finding suggests that clinicians and SO may have different ideas as to what is considered impaired or unimpaired behavioural functioning and that different raters on ISA measures utilising the discrepancy score method may produce different results. Further methodological issues will be discussed in section 1.8.

Development of ISA over time

One final dimension of interest regarding ISA following TBI relates to the natural course of development of self-awareness over time. Significant ISA has been reported in individuals with TBI 7 years post-injury (Oddy, Coughlan, Tyerman, & Jenkins, 1985) and 10 years post-injury (Koskinen, 1998). Ranseen et al (1990) found very poor initial ISA in a group of TBI patients within the first week of

admission to an inpatient rehabilitation unit and no significant change one month later at discharge. Prigatano and Klonoff (1988) suggested that patients with brain injury may take 2 to 4 years to develop the realistic awareness of deficits necessary for employment.

There have been some limited studies into the development of better self-awareness as time progresses. Newman et al (2000) examined a group 37 of mixed aetiology brain injury patients, some with TBI, others with AACA, though the numbers in each group were not specified. Initial ratings at 8 weeks post-injury indicated that 97% of patients underestimated their functional impairments compared to rehabilitation staff. By time of discharge, ratings of both staff and patients had converged, suggesting a decrease in ISA. However, limiting factors in this interpretation include lack of details regarding patient injuries, the time period between the two assessments, and details as to why half the patients did not complete the second assessment. Furthermore, the authors themselves claim that while the staff ratings changed between Time 1 and Time 2, the patient ratings did not, suggesting the greater convergence was more reflective of improved performance on the functional tasks at Time 2 and lack of sufficient sensitivity of their measurement, the Functional Self-Appraisal Scale. Another longitudinal study of ISA examined 55 TBI participants at 1 month post-injury as a baseline measure, followed by assessments at 6 months and 12 months. In this study, awareness was measured using discrepancy scores on the Katz Adjustment Scale (KAS) between the patient and significant other. The TBI group was divided into thirds based on division of the discrepancy scores. The "Unaware" group significantly underestimated their difficulties on the KAS, the "Hyperaware" group significantly overreported their difficulties, while the middle third were considered the "aware" group, whose reports were similar to their SOs.

The agreement between TBI reports and their SO reports significantly increased from the 6 month to the 12 month evaluation. Nevertheless, the authors cautioned interpretation of their findings, as the discrepancy measure did not correlate with any baseline measure or clinician ratings of ISA. They also commented on the limitations of using items from just one scale to reflect ISA (Lanham et al., 2000).

Two other studies have also shown improvements in ISA in longitudinal studies. Godfrey et al (1993) examined level of insight at six months, one year and between two and three years post-injury. They found that ISA for behavioural impairments did improve, but was accompanied by emotional dysfunction. Fleming and Strong (1999) used discrepancy scores on the PCRS to examine development of ISA in 55 TBI individuals at 3 months post injury and again 12 months post injury. They found that ISA did improve in most areas, as measured by greater agreement across items between self and SO. The authors propose that the development of self-awareness is strongly related with personal experience, such that awareness of deficits cannot begin until the individual experiences difficulty performing activities compared to pre-morbid abilities. However, significant differences persisted on 3 of 14 items that had shown discrepancy at 3 months. These were related to managing finances, driving a car, and recognising if something one has said has upset someone. The persistent disagreement on these items may be a consequence of the first two of these items being key areas of independence and so non-neurological factors, such as denial or bias towards making a good impression, may play a role. Alternatively, as a more neurological explanation, the individual with TBI may be focussing on the physical elements required for driving for example, whereas the SO may be focussing more on the complex cognitive components. However, a limitation of this

study includes the use of mainly therapists as informants at 3 months post-injury, but caregivers/relatives at 12 months post-injury.

As persistent ISA has been shown as many as 10 years post-injury, and considering its importance for positive outcome, it is clear that further studies are required to examine this important dimension of ISA following TBI.

1.7 Theories and Models of ISA

Considering the heterogeneity of ISA and the vast number of clinical syndromes that are affected with different manifestations, several theories and models have been developed over the years to account for the origin of impaired self-awareness. As pointed out by Giacino and Cicerone (1998), the variety of explanations of ISA relate to the disparate perspectives including neurological, neuropsychological, psychiatric and psychogenic backgrounds.

There is general agreement in the literature that the neuroanatomical bases for modality specific "classic anosognosia", or ISA for physical, sensory and language deficits can be understood with focal lesion explanations. ISA for left hemiplegia is generally associated with lesions of the right cerebral hemisphere, usually involving the parietal region and its connections, and ISA for cortical blindness is frequently associated with bilateral lesions in the distributions of the posterior cerebral arteries (Heilman, Barrett, & Adair, 1998; Sherer et al., 2005). However, focal lesion explanations do not account for ISA of cognitive and executive function deficits, or changes in behaviour and personality that are frequent sequelae of brain injury and neurodegenerative disorders. Diffuse damage explanations view ISA in terms of a

manifestation of a general intellectual impairment. However, as outlined in section 1.6, ISA following TBI is not consistently associated with severity of injury or general intellectual decline and ISA has been observed in many patients with intact intellectual functioning (McGlynn & Schacter, 1989). This review will now turn to theories and models that have been proposed to account for the heterogeneous forms of ISA that have been developed to explain ISA following TBI.

Neuroanatomical and Neuropsychological Theories

Several theories have been advanced that propose the existence of an executive or supervisory supraordinate neural system responsible for monitoring the performance of lower-level cognitive subsystems. It is purported that ISA can result from either damage to the lower-level cognitive modules, its connections or to the supraordinate monitoring system itself. These models would suggest that disruption to this executive control system in terms of monitoring and regulation would result in different forms of ISA (Mesulam, 1985a; Stuss, 1991).

The Hierarchy of Brain Function Model was proposed by Stuss and colleagues (Stuss, 1991; Stuss & Benson, 1986; Stuss et al., 2001) and consists of a hierarchical integrated organisation of brain function that encompasses four levels. Awareness, according to Stuss and colleagues, results from mental processes that construct models of the world at each of the four levels of processing. Within this model, the frontal lobes and the frontal systems are considered to be the site of the highest level of cerebral processing, for the interpretation of and response to incoming information. The four interrelated, interactive hierarchical levels function via a feedback loop that allows for feedback and feedforward systems. These processes

allow for both top-down control of lower levels and bottom-up flow of sensory information and general arousal to activate the higher levels. Different levels within the hierarchy construct different representations of the self. The first level involves the brainstem reticular system and mediates basic arousal. At the second level, processing of sensory/perceptual information leads to the emergence of a model of the self and environs. This level is thought to involve the association cortices of the posterior cortex. The third level is responsible for executive functions, mediated by the frontal lobes, adjusting and directing outputs of the posterior processes by integrating the information provided by the sensory systems and organising goal-directed behaviour. Executive control is specifically required for novel, complex or non-routine situations. At the final level of integration, the highly processed information is elaborated by associative processes such as personal experience and expectation which transforms the objective experience into a subjective personal experience, and can be used to guide future behaviour. This self-awareness at the final level is related to the frontal lobes and its limbic connections, with the right frontal lobe playing a crucial role. This theoretical construct of self-awareness, self-reflection or self-consciousness engages and interacts with other brain processes in order to achieve the overall goal, that is *"developing an aware individual interacting with the environment in a personal and socially appropriate manner*" (Stuss, 1991, p.67). Damage at different levels results in different manifestations of ISA. Damage at the lowest level of the hierarchy would result in diminished general arousal and at the most extreme, coma. Damage at the second level would lead to "classic anosognosia" outlined above, such as ISA for hemiplegia or Jargon aphasia. Damage at the third level leads to classic executive dysfunction, which can include disorganised behaviour, poor self-monitoring and self-regulation, disinhibition and impaired judgement and reasoning skills. Stuss and Alexander (2000) propose that

damage at the highest level to the prefrontal cortex and the frontal poles leads to a disturbance in this subjective sense of personal continuity or extended temporality, the ability to draw events from the past and use them to guide future behaviour.

This model has merit due to its basis in clinical observations of different manifestations of ISA and for a variety of neurological disorders. It has further value in that it is one of the few neuropsychological frameworks that integrates both psychological and neuropsychological processes. Based on William James discussions of the self and consciousness (James, 1890), Stuss and colleagues (1991) also highlighted the importance of incorporating warmth, immediacy, continuity of memories, choice and selectivity and self-reflectiveness into models of ISA. Emphasis on the frontal lobe and prefrontal cortex were extended to include frontal systems in later formulations of the model because as pointed out by Turner and Levine (2004), posterior, subcortical and diffuse damage can mimic prefrontal cortical damage through disconnection with frontal systems, as the prefrontal cortex is widely and reciprocally interconnected with the rest of the brain.

Other models propose a tripartite loop to explain the basis of ISA based on monitoring and feedback mechanisms (Goldberg & Barr, 1991; Heilman, 1991; Heilman et al., 1998). According to these frameworks, an intact error-monitoring system is essential for intact awareness of deficits. Monitoring involves three components, an internal representation of the desired cognitive output, feedback regarding this cognitive output and an intact mechanism which compares the output with the internal representation. Failure of any of these elements in the mechanism can cause absence of or false feedback or a dysfunction of the comparator. Heilman's theory (Heilman 1991; Heilman et al., 1998) is limited in that it only relates to ISA

of hemiplegia and does not explain the more complex forms of ISA for behavioural and social deficits. Goldberg and Barr (1991), however, describe how damage to the comparator mechanism in the prefrontal cortex can lead to a breakdown in "local" or "global" error detection (Zaidel, 1987). A local error monitoring deficit is lack of awareness of specific errors in one's performance, such as seen in frontal syndrome patients with perseverative or field-dependent behaviour. These types of error-monitoring deficits cannot be explained by lack of knowledge, the internal representation, of the correct response or faulty feedback, as patients can frequently verbalise the correct responses in field dependent behaviour, as described by Luria (1980). Damage to sensory connections, sensory feedback, or degradation of internal representations can lead to ISA for sensory deficits, such as cortical blindness or aphasia. While this model emphasises the heterogeneity of the mechanisms that can lead to ISA, and propose that diverse lesions can relate to breakdown at different parts of the mechanism, it is a macroscopic neuroantomical model and does not explain how different ISA can correspond to distinct neural processes.

Because of the heterogeneous nature of ISA, other theories have postulated that widely distributed networks support awareness (Mesulam, 1985, 2000; Prigatano, 1991, 1999b). Based on his definition that ISA reflects a disruption of the integration of cognitive and affective components, Prigatano (1999) identified areas described by Mesulam (1985) as the "heteromodal cortex" as a likely substrate of self-awareness. The heteromodal cortex is thought to involve large areas of the prefrontal and frontal cortex, the inferior parietal lobe, the supramarginal and angular gyri and the anterior tips of the temporal lobes. These areas are thought to be involved in the integration of information processed by the primary sensorimotor cortex about the external world with information processed in the limbic regions about the inner

world. Prigatano (1991) observes that the frontal lobes and paralimbic areas are particularly vulnerable to the types of damage that results from acceleration/deceleration injuries following TBI and describes how different syndromes of ISA can be conceptualised based on which region of the heteromodal cortex is damaged. Frontal heteromodal disruption would theoretically involve bilateral or unilateral prefrontal and frontal damage and diffuse involvement of frontal and cingulate connections. This would result in poor awareness as a social being and ISA for socially appropriate behaviour, reduced capacity for empathy in interpersonal situations and impaired planning and anticipation. Parietal heteromodal damage, involving bilateral or unilateral parietal cortex, particularly the interior parietal and cingulate connections, would result in ISA for impaired sensorimotor function, including hemiplegia and its consequences on functional independence from reduced mobility. Temporal heteromodal damage to either bilateral or unilateral superior and mesial temporal cortex and its limbic connections involving the amygdala and hippocampal regions could lead to ISA for memory impairment or visual and auditory deficits. ISA for visual and auditory deficits is commonly manifested in patients giving explanations based on external causes such as poor lighting for sensory deficits. Occipital heteromodal damage would lead to ISA for blindness or hemianopia or poor awareness of the consequences for the patients' safety. This model has value in that it incorporates many possible clinical presentations of ISA and reflects ISA as a disturbance of the integration both cognitive and affective components, not just as a purely cognitive dysfunction.

Prigatano and Johnson (2003) developed another related hierarchical model to account for disturbances in awareness by expanding on a model conceptualised by Zeman (2001) regarding human consciousness. This model is based on three "vectors" of consciousness which interact and are subserved by overlapping

neurocircuit in the brain, and evolve to aid survival of the organism. Vector 1 is the wakefulness dimension of consciousness and involves the upper brainstem, the posterior hypothalamus and the thalamus. Vector 2 of consciousness depends on the heteromodal regions outlined above and subserves the experiential sense of "me" in the real world here and now, or consciousness as a phenomenological state. The third vector is involved in theory of mind, or consciousness as a mental state. It is thought to be a natural refinement of Vector 2 and dependent on an enhanced frontal heteromodal cortex. It is proposed that self-knowledge is necessary in order to have knowledge of another and point out that patients with frontal lobe injury as well as individuals with autism and schizophrenia have difficulty with TOM tasks that require the patient to make inferences about another person's intentions (Frith & Frith, 1999; Stuss, Gallup, & Alexander, 2001). Neuroimaging studies can provide insight into each of these vectors, including PET studies of coma, anaesthesia and stages of sleep (Vector 1) that show activation of posterior cingulate and retrosplenial regions as well as dorsal medial prefrontal and anterior cingulate regions (Gusnard & Raichle, 2001; Raichle et al., 2001). Vector 2 can be examined by asking subjects to monitor their own mental states in the "here and now", showing activation of the anterior cingulate and paracingulate regions (Blakemore, Wolpert, & Frith, 2000; Gusnard et al., 2001). Studies examining TOM have shown activation of the anterior medial prefrontal and posterior cingulate regions by requiring participants to predict the behaviour of others by mentalising about their beliefs and desires (Fletcher et al., 1995; Goel, Grafman, Sadato, & Hallett, 1995). Merits of this theoretical model are that disturbances at each vector can cause different manifestations of ISA, such as coma (vector 1), poor social awareness and many of the manifestations of ISA following TBI (vector 2) or impaired ability for TOM and interpreting the actions of others (vector 3). A further advantage of this model is that

it allows for the development of tasks suitable for neuroimaging with clinical populations that may be affected at the different levels.

McGlynn and Schacter (McGlynn & Schacter, 1989; Schacter, 1990) proposed an interactional hierarchical model known as the Dissociable Interactions and Conscious Experience (DICE) which includes the existence of a system called the Conscious Awareness System (CAS). The CAS is responsible for monitoring changes in baseline states of individual cognitive modules, such as sensory/perception, memory, and language. McGlynn and Schacter (1989) propose that the CAS is a posterior system involving the inferior parietal lobes and connections, particularly with the cingulate areas. The CAS has a proposed output link to an executive system in the frontal regions involved in the organisation and monitoring of complex behaviour, although it is emphasised that CAS and executive system are separate entities. Different types of ISA are proposed to result from damage to the parietal CAS, the cognitive modules or the frontal executive system. Domain-specific ISA could occur if the central CAS is selectively disconnected from a specific lower-order cognitive module, such as occurs in ISA for a language or visual impairment, resulting in information regarding the activation in the disconnected module not reaching conscious awareness. Damage to the CAS itself could lead to a global awareness deficit which would lead to ISA for all neuropsychological impairments as CAS could not respond to inputs from the cognitive modules. Disruption to the frontal executive system or its connections to CAS would lead to ISA for complex deficits such as reasoning, self-monitoring, social behaviour and personality changes. Merits of the DICE model include its ability to account for many heterogeneous clinical manifestations of ISA. It is has been related to findings of several studies of ISA in AD in particular (Graham, Kunik, Doody, & Snow, 2005; Green, Goldstein,

Sirockman, & Green, 1993). However, it does not account for all clinical syndromes of ISA and cannot explain the predominance of right hemisphere damage in ISA. Like many of the neuropsychological models, it does not provide a role for possible nonneurological elements of ISA such as defensive denial. Specifically in relation to ISA in AD, Agnew and Morris (1998) note that the DICE model does not describe mechanisms for metacognitive output or for the involvement of memory in metacognitive processes.

In order to address some of these limitations and to further explain ISA in AD, the DICE model was reformulated by Agnew and Morris (1998) and later Morris and Hannesdottir (2004) to the Cognitive Awareness Model (CAM) and the CAS to the MAS-Metacognitive Awareness System. The CAM describes three types of ISA in AD. Mnemonic Anosognosia occurs when the memory impairment means that the Personal Data Base of semantic memory is not updated, leading to the individual believing memory functioning is unimpaired. Patients could display implicit knowledge however, as the comparator mechanism in the executive system would remain intact, which could explain behavioural change without explicit knowledge. Executive anosognosia would result from an impaired executive system affecting the comparator mechanism. As patients would have intact personal knowledge about the actual state of their memory, Agnew and Morris (1998) propose that confabulation may be likely in an attempt to process the failure experienced. Primary anosognosia would result from damage to the MAS leading to global ISA and only implicit knowledge of memory failure, despite a functioning comparator mechanism. Morris and Hannesdottir (2004) propose that damage to the frontal lobes or its connections might result in executive anosognosia, while damage to the temporo-parietal region would result in the PDB failing to be updated and damage to fronto-parietal regions,

in particular the right hemisphere could lead to an impairment of the MAS, or primary anosognosia. The CAM model is useful in that it allows for predictions to be made about cognitive functioning and their association with ISA in AD and so is open to empirical research (Clare, 2004b). Also, it incorporates both implicit and explicit forms of ISA, an area of the awareness literature that has been generally neglected. There has been some empirical support for the model (Graham et al., 2005; McGlynn & Kaszniak, 1991). The CAM provides a detailed account of the heterogeneity of ISA for memory impairments in AD and could potentially be extended to explain ISA of other cognitive or behavioural changes in AD and ISA in brain injury (Ownsworth, Clare, & Morris, In Press).

Overall the neurological and neuropsychological models have developed and reformulated to account for the vast heterogeneity of ISA following TBI and other neurodegenerative disorders such as AD. Common to the hierarchical theories is that each conceptualises a supraordinate system and/or an executive system for monitoring the state of the subordinate cognitive subsystems. The monitoring component described as an aspect of many of these models has been developed in models of executive functioning. Shallice and Burgess (1988; 1996) proposed the Supervisory Attentional System (SAS) of executive control, used in novel, nonroutine or complex tasks to direct attentional resources. Self-monitoring of behaviour would involve an active, multi-stage process of maintaining vigilant attention, problem orientation, checking solutions through the SAS problem-solving operations, assessment and verification of schema and comparisons to intentions. Damage to the SAS could lead to failure of this complex process, leading to self-monitoring impairments that could manifest in a variety of different ways at different stages. Similarly, Baddley's (1986) model of working memory includes a central

executive (CE) that is proposed to direct attentional resources to aspects of information processing. Failure of the CE to direct resources effectively would lead to impaired self-monitoring.

Despite the development of more complex and detailed models of ISA and its components, most of these models, with the exception of Stuss's model, still neglect to account for the nonneurological components of ISA. Clinicians have criticised their utility for not addressing the psychological and psychosocial factors, such as the emotional impact of injury from TBI (Prigatano, 1999b).

Psychological, Sociological and Integrated Models

As mentioned in section 1.6, the development of awareness has been associated with an increase in emotional distress at least in early stages of recovery (Godfrey et al., 1993). It has been proposed that ISA in early stages may reflect psychological processes, such as denial as a coping mechanism, to preserve self-image and prevent unnecessary psychological distress (Godfrey, Knight, & Partridge, 1996). Denial has been described as an unconscious defense mobilised as a buffer from emotional distress against the painful realisation of one's condition and its potential consequences, to give individuals time to impose personal meaning onto the unfamiliar experience of neurological disability and to possibly develop more effective coping strategies (Gainotti, 1993; Kortte, Wegener, & Chwalisz, 2003; Prigatano & Weinstein, 1996; Weinstein & Kahn, 1955). During the initial period following injury, denial may serve the purpose of maintaining hope and motivation. It has been proposed that pushing a patient to know what is being minimised may be emotionally destructive at that stage and potentially even counterproductive for

rehabilitation (Deaton, 1986; Langer & Padrone, 1992; Prigatano & Weinstein, 1996; Sohlberg et al., 1998).

It has been suggested that the use of defensive denial is influenced by pre-morbid personality traits (Gainotti, 1993; Prigatano & Weinstein, 1996; Weinstein, 1991). For example, patients who pre-morbidly regarded illness as an imperfection or weakness, had a history of denying their perceived inadequacies, had compulsive drives with a strong work ethic or had a need to be held in high regard by others were reportedly more frequently seen to present with denial (McGlynn & Schacter, 1989; Weinstein & Kahn, 1955). Seiffer et al (Seiffer, Clare, & Harvey, In Press) found that the personality trait of conscientiousness was associated with level of ISA in AD. Post-injury personality changes have been implicated as types of deficits in awareness by some researchers (Prigatano, 1992; Varney & Medefee, 1993). For example, Prigatano (1992) proposed that post-injury psychosocial difficulties might be a continuation or exacerbation of pre-morbid disturbances. Prigatano (1999b) proposed a distinction between defensive and nondefensive denial. Defensive denial would reflect at least partial knowledge of the existence of a problem and an attempt to protect oneself against emotional distress, whereas nondefensive denial would reflect pre-existing personality characteristics and strategies to cope with partial knowledge. Empirical research conducted by Kortte et al (2003) found that defensive denial was associated with increased use of avoidant coping strategies, which was in turn associated with higher levels of clinical depression, suggesting that persistent denial could be maladaptive.

McGlynn and Schacter (1989) observe that psychological and psychodynamic explanations for ISA have been subject to several criticisms. Denial as a defense

mechanism does not explain specificity of ISA, as denial should theoretically apply to all deficits. Denial also cannot explain how frontal and parietal lesion locations have been associated with ISA. If ISA was purely a psychological defense such as a coping mechanism or related to pre-morbid personality, there would be no associations with lesion location. Sohlberg et al (1998) propose that the stability of ISA over long periods of time suggests a neurological substrate, rather than an emotional or psychological one, as this would be more likely to fluctuate. Patients who demonstrate ISA for deficits for many years after their initial injuries would not necessarily have any obvious reason for motivated denial. Stuss and Benson (1986) argue that psychodynamic factors are prevalent in the general population also, yet such dramatic ISA as seen in the clinical populations affected are not observed in either general population or many other medical conditions.

Ownsworth et al (2002) conducted an empirical investigation of the relative contribution of psychological factors and neuropsychological factors to ISA following TBI. The findings showed that neuropsychological factors had a more direct relationship with ISA, whereas psychological factors, in particular coping related denial had a more indirect association with ISA.

Socio-cultural factors have been purported to influence ISA, as described in section 1.6, with regard to differing trends in reporting of deficits in Japanese culture (Prigatano et al., 1997) and Maori culture (Prigatano & Leathem, 1993). Prigatano related specific neural areas that may be vulnerable following TBI to these context-dependent judgements. He proposed that more severe injuries leading to diffuse brain damage affecting the fronto-temporal regions might reduce individual's ability to make context-dependent judgements and to be aware of what is culturally and

socially sensitive (Prigatano, 1999b). However, this prediction has not been empirically investigated.

Clare (2004b) reviews models of the self from a social cognitive perspective and from a social constructionist perspective. Viewing the self from a social cognitive perspective may explain how neurologically-healthy individuals also engage in inaccurate self-representation, by using self-serving biases in appraisals of behaviour and outcome (Flashman, 2002). Clare (2004b) proposes that neuropsychological factors may contribute to the development of self-serving biases in information processing. A social constructionist perspective would suggest that the interactions between patient and clinicians or patients and caregivers impact on the social presentation of the self, which highlights some of the complications inherent in measurement of ISA (Clare, 2004b). Marcel et al (2004) showed that ISA for hemiplegia was more accurate in patients when they were framed in the third person, rather than to the patient themselves. Similarly, Tyerman and Humphrey (1984) found that when asked to rate changes in themselves and in a "Typical Head-Injured Person", TBI patients rated themselves more positively than the typical head-injured person, while their ratings of themselves did not differ significantly from their ratings of a "Typical Person". Sherer et al (1998) found that TBI patients responded in a more consistent fashion with their relatives when asked specific questions about their deficits, rather than general, global questions. The methodological implications of the possible influence of interactions between patient and clinician or caregiver and the different responses depending on the method used will be discussed in section 1.8.

An integrated biopsychosocial model was developed in order to account for psychological and psychosocial factors as well as neurological and

neuropsychological factors in ISA. Developed initially with regard to ISA in AD (Clare, 2004b), it has since been expanded to possibly explain other forms of ISA, such as following brain injury (Ownsworth et al., In Press). It integrates neurocognitive factors to explain domain-specific awareness, global ISA and impaired self-monitoring, with psychological factors including defensive and nondefensive denial with the socio-environmental context such as methodological considerations involving social interactions between patient and clinicians. This new integrated model is built on developments of the CAM model (Agnew & Morris, 1998; Morris & Hannesdottir, 2004) and the biopsychosocial framework (Clare, 2004b) and demonstrates a comprehensive picture of clinical presentations of ISA. However, it requires empirical research, as it has of yet only been supported with a case study.

Clinical Models of ISA

A number of clinically-based models from a rehabilitation perspective have been developed to guide assessment and interventions of ISA. Fleming and Strong (1995) propose a three-level model of ISA in the context of head-injury that includes self-awareness of deficits at the first level, awareness of the functional implications of deficits for independent living at the second level and ability to set realistic goals and predict one's future accurately at the third level. Birnboim (1995) also presents a three-level model that includes understanding one's deficits, understanding characteristics of the tasks and understanding how to use strategies appropriate to each task. While descriptively useful for rehabilitation, these models lack the complexity to explain the multifaceted nature of ISA. Knowledge of deficits form the

basis for each of these models, a theme evident in an influential model, the Pyramid Model.

The Pyramid Model was an early framework proposed by Crosson and colleagues (Barco, Crosson, Bolesta, Werts, & Stout, 1991; Crosson et al., 1989) who classified ISA into three hierarchical and independent levels. *Intellectual awareness* reflects a basic knowledge of impairment or difficulty and the knowledge that a deficit may have implications on functional performance. This basic intellectual awareness was thought to provide the prerequisite foundation for *emergent awareness* described as the ability to recognise difficulties as they occur. *Anticipatory awareness* refers to the ability to predict that these difficulties may result in poor performance on tasks, and to reflect on future consequences of the deficits. Emergent and anticipatory awareness are referred to as *online awareness* (Crosson et al., 1989; Toglia & Kirk, 2000). The authors propose that severe deficits in abstract reasoning, which may accompany dorsolateral, frontal or diffuse brain injury and memory, associated with diencephalic, basal forebrain or bilateral mesial temporal lesions may impair intellectual awareness and anticipatory awareness, while injury to the right hemisphere may impair emergent awareness. However, this model was proposed as a clinical model and was not intended to be a neurological model and so these are not formal links with neurological substrates, rather clinical observations and predictions. The pyramid model has logical appeal and was widely referred to clinically as the authors provided clear and detailed recommendations for rehabilitation of each level. Malia (1997) proposed an extension of the model to include cognitive, metacognitive, executive, psychosocial and acceptance issues. However, its hierarchical, linear nature has not been empirically supported, suggesting that self-awareness at an intellectual level is not required before emergent

or anticipatory awareness (Abreu et al., 2001). The pyramid model was also criticised for not explaining how the different levels work, why some levels can be observed in some situations but not in others, for failing to account for discrepancies in behaviour across skill domains and situations and for being too simplified (Toglia & Kirk, 2000).

In an attempt to address some of these limitations, Toglia and Kirk (2000) proposed the Dynamic Comprehensive Model of Awareness in a framework based on the concept of metacognition. Metacognition has been defined as the conscious knowledge of cognitive processes, the ability to consciously monitor and regulate one's ongoing activities or processes while engaging in a task (Hacker, 1998). Toglia and Kirk (2000) elaborate from this definition to describe two interrelated but distinct processes: Metacognitive Knowledge and Online Awareness. Metacognitive Knowledge includes declarative knowledge of cognitive processes, task characteristics and knowledge of one's own capabilities, while online awareness involves monitoring (ongoing evaluation of performance within the context of a task) and self-regulation (the ability to change strategies and adjust performance in response to changing task demands) (Stuss, 1991). Metacognitive knowledge incorporates Crosson et al's concept of intellectual awareness, while online awareness incorporates emergent and anticipatory awareness. However, their model is viewed as a dynamic, interactional process between knowledge, beliefs, task demands, contextual factors and other influences (cognitive, emotional, motivational) rather than a linear, hierarchical one. The model suggests for example that online experiences may feed back to improve intellectual awareness. The online process is proposed to be a multifaceted process of feedback, and the authors suggest that a variety of deficits in executive function as a result of damage to the prefrontal

regions, such as reasoning, impulsivity, poor attention and flexibility and impaired strategy application, could compromise various stages of the process. They propose that knowledge of specific facts may be associated with more posterior brain functions. The model also provides an explanation for differing domains of awareness, as online awareness is dependent of the context of the task or situation, so emergent or anticipatory awareness may be evident in some situations but not others. It also suggests how internal influences, such as patients' perceptions of their capabilities may interact with task performance. Despite its many advantages and its emphasis on how both internal and external factors can influence ISA, the complexity of the overall model suggests that empirical studies and predictions may be difficult if one were to attempt to include every aspect and variable. Nevertheless, the differentiation between metacognitive knowledge and online awareness has taken the understanding of ISA forward and explains some of the inconsistencies found in previous research, as the vast majority of studies have examined ISA solely in terms of metacognitive knowledge or intellectual awareness.

In general, the clinically based models do not attempt to clearly identify neurological, neuropsychological or psychological substrates of ISA in detail. However, they do provide descriptive detail into the complexity and heterogeneity of ISA and the implications on the quality of life of the patients. Perhaps within the neuropsychological domain, their greatest advantage is in providing a practical framework for both assessment and rehabilitation.

1.8 Measurement of ISA

Schacter (1990) described the problem of ISA as "one of the enduring mysteries of the human mind" (p.174). Perhaps part of its enduring mystery is that awareness cannot be measured directly, but must be inferred through the interpretation of indirect measures (Sohlberg et al., 1998). As has been evident throughout this review, methodological issues have plagued progress in understanding neurological, neuropsychological and psychological substrates of ISA and may explain the numerous inconsistencies reported. Several different methodologies have been used as indices of ISA and no established method or "gold standard" exists for accurate and comprehensive assessment. The vast majority of investigations into ISA have adopted one of three main methods to assess awareness of deficits in TBI and other neurological disorders:

1) Comparison between the patients report and that of a significant other (SO) or clinician. The most common form of this comparison is the use of questionnaires, rated by the patient and SO or clinician. Examples include The Patient Competency Rating Scale (PCRS) (Prigatano, 1991), The Awareness Questionnaire (Sherer et al., 1998) or Dysexecutive Questionnaire (Wilson, Alderman, Burgess, Emslie, & Evans, 1996).
2) Comparison between the self-report of the patient with objective measures. This approach generally involves performance on neuropsychological tests, and has been used in some studies of ISA (Allen & Ruff, 1990; Anderson & Tranel, 1989; Fischer, Trexler et al., 2004; Trosset & Kaszniak, 1996)
3) Clinician Ratings. This method can involve rating scales, such as the Clinician's Rating Scale for Evaluating Impaired Self-Awareness and Denial

of Disability (Prigatano & Klonoff, 1998), or ratings based on structured interviews (Fleming et al., 1996).

ISA has most commonly been measured in terms of discrepancy between the patients self-report and that of a SO or clinician. These methods assume that the reports of an objective rater, either the SO or clinician, is more reliable than the patients self-report. However, the use of relatives or clinicians as objective standards has its limitations. Reliability of the objectivity of these raters has been called into question, due to frequent lack of concordance or only weak agreement between relative and clinician perceptions (Lanham et al., 2000; Malec et al., 1997; Sherer et al., 1998; Sunderland et al., 1984). Fordyce and Roueche (1986) found that relatives ratings fell between patient and clinician ratings. Some evidence suggests that relatives may deny or minimise deficits (McKinlay & Brooks, 1984; Romano, 1974). Factors such as the dependency level of the patient, stress, fatigue and personality characteristics of the SO, as well as length of time since injury may influence the accuracy of relative ratings (Brooks & McKinlay, 1983; McKinlay & Brooks, 1984). Cavallo et al (1992) studied 34 families of people with TBI who were between 1 and 3 years post-injury. They noted that not all families respond in the same manner to the effect of a traumatic injury within their family. Relative ratings may also be influenced by the quality of the relationship with the patient (Clare, Wilson, Carter, Roth, & Hodges, 2002). Nevertheless, Fleming et al (1996) point out that the relative's experience of living with the person with TBI or other disabilities remains a vast source of information to researchers and clinicians, due to their unique insight into the abilities of the patient on a daily basis and their pre-morbid personality characteristics and abilities. Ponsford and Kinsella (1991) highlight that for these

very reasons relatives may be in the best position to observe and judge a patients performance.

The use of clinicians as objective raters for questionnaires of rating scales is also limited in that they have no pre-morbid experience of the patient, and the validity of their ratings can be influenced by their emotional response to the patient, mood, attitude, and expectations of performance as well as subjectivity of interpretation from some scales (Fleming et al., 1996; Heilbronner et al., 1989; Ponsford, 1988). The Self-Awareness of Deficits Interview (SADI) was developed as a clinician rating scale based on a structured interview by Fleming et al (1996) in order to overcome some of the limitations of comparing self-ratings with other sources. However, limitations of this and similar approaches are that a clinician needs to have specific knowledge about the patient and trained interviewing skills for accurate administration. Also this approach relies on clinician judgements and inter-rater reliability (Simmond & Fleming, 2003). Furthermore, it has been pointed out that a clinical interview is a social interaction, potentially resulting in patients desire to present themselves in the best possible light, as it has been shown that patients who may deny their difficulties in formal clinical interviews may demonstrate awareness in other contexts (Clare et al., 2002; Weinstein, Friedland, & Wagner, 1994).

The limitations associated with using a relative or clinician as an objective standard may be overcome when the patients' reports are compared to test performance. Difficulties with this approach include the fact that administering a neuropsychological test battery can be time consuming, costly and less accessible than the use of questionnaires or rating scales (Simmond & Fleming, 2003). Subtle changes in behaviour and personality that are common following TBI and

neurodegenerative disorders may be difficult to objectively measure using standard neuropsychological assessments (Sherer et al., 1998). Specific neuropsychological functions that are frequently measured in standard batteries have been criticised for poorly reflecting difficulties experienced in the everyday life of the patient (Ponsford, 1988). Also, Trosset and Kazniak (1996) suggested that use of test performance as an objective standard may have its limitations as it is unclear that patients necessarily understand what abilities are required for each particular test.

The use of patient self-reports for ISA investigations in general has its limitations, including the fact that they rely predominantly on patients verbal comprehension and verbalisation of their responses (Flashman, Amador, & McAllister, 1998). Also, it has been observed that different styles of questioning can lead to more or less accurate answers. Gasquoine (1992) found that patients were more likely to report deficits when asked specific questions about their abilities than when asked more general, open-ended questions, a finding replicated by Sherer et al (1998).

Investigation into the convergent validity of different measures of ISA has been limited. The few studies that have explicitly measured this aspect have frequently only found weak correlations between the measures, suggesting that measures may be assessing different constructs of awareness (Bogod et al., 2003; Fischer, Gauggel et al., 2004; Fischer, Trexler et al., 2004; Sherer, 1998). This is a particularly important issue considering the vast majority of investigations into ISA adopt one single method to assess whether a patient displays ISA or not. Each approach to assessment reflects a conceptual framework and underlying theoretical perspectives and consequently it is not surprising that the methods are frequently found to be measuring different constructs of awareness. ISA is proposed to be assessed in

relation to many different "objects" of functioning, such as memory, attention, behaviour, personality, basic ADL functioning or executive functioning. Therefore, the "object of insight" that is assessed will influence the phenomenon of ISA that is elicited, regardless of the approach used (Clare, Markova, Verhey, & Kenny, 2005; Markova & Berrios, 2001).

It is therefore reasonable to assume that the inconsistencies found in the literature regarding the characteristics of ISA exist due to the frequent approach to ISA as a unimodal concept and use of one single method to assess a specific object of insight, perhaps with inherent limitations such as rater biases (Ownsworth et al., In Press; Toglia & Kirk, 2000). Markova et al (Markova, Clare, Wang, Romero, & Kenny, 2005) point out that any correlates of ISA can only be a partial reflection of the multifaceted phenomenon of awareness. In recent years, many investigators have called for the use of multiple measures to assess ISA, as no one perspective can be considered a reliable indicator of awareness (Clare et al., 2005; Fischer, Trexler et al., 2004; Fleming et al., 1996; Katz & Hartman-Maeir, 2005; Ownsworth et al., 2002; Simmond & Fleming, 2003a; Sohlberg, 2000; Toglia & Kirk, 2000). Researchers have since begun to employ a richer approach to assessment of ISA by using two or more indicators of awareness in order to capture the nature and complexity of the concept (Ownsworth et al., In Press).

Adopting Toglia and Kirk's (2000) model, questionnaire discrepancy scores, clinician rating scales and structured interviews generally reflect assessment of metacognitive knowledge or intellectual awareness of deficits. There has been a serious dearth in the TBI literature in the assessment of local or online awareness, either emergent or anticipatory (McAvinue et al, 2005).

Online awareness and self-monitoring in brain injury and neurodegenerative disorders has operationalised metamemory protocols, such as asking individuals to make judgements or self-assessments based on internal feedback, typically through global, or item-by-item predictions of performance, judgement of learning and feeling of knowing tasks (Kennedy & Yorkston, 2004; Schmitter-Edgecombe & Woo, 2004; Souchay et al., 2003). Fischer et al (2004) adopted a prediction-performance method with a group of patients with brain injuries (TBI and CVA) and orthopaedic patients on a cognitive and a motor task, where ISA is indicated by a higher prediction than actual performance. The brain injury patients significantly overestimated their competency on the memory task compared to the orthopaedic controls, whereas both groups accurately predicted their performance on the motor task. Unfortunately, the participants in this study were of mixed aetiology, with only 11 being TBI patients. This study also lacked measures of executive function. However, this study confirmed the usefulness of predicted-performance experiments with TBI patients and may represent a useful operationalisation of the concept of anticipatory awareness, the ability to predict how deficits may interfere with performance on tasks. Future work could investigate this by adopting the methodology used here with a larger group of TBI patients and more detailed neuropsychological profiles, within a larger framework of assessing ISA.

The development of the Self-Regulation Skills Interview (SRSI) (Ownsworth et al., 2000) reflects one of the few attempts in the literature to explicitly assess both emergent and anticipatory aspects of online awareness. This semi-structured interview consists of six questions relating to a specific difficulty nominated by the patient. Two questions are thought to reflect emergent awareness ("Can you tell me how you know that you experience difficulty; that is, what do you notice about

yourself?") and anticipatory awareness ("When are you most likely to experience the difficulty, or in which situations does it mainly occur?"). The SRSI has been used effectively to demonstrate differential correlations between both neuropsychological and psychological correlates to intellectual, emergent and anticipatory awareness, strongly suggesting these constructs are separate entities and should be assessed as such (Ownsworth & Fleming, 2005; Ownsworth et al., 2002). Limitations of this method are that assessment of high or low ISA is based on one score for each of the single questions related to emergent and anticipatory awareness. The fact that both these assessments are based on memory of events in the past that require intact episodic memory may also be a limitation, especially considering that Barco et al (1991) recommended the assessment of online awareness should involve online awareness of actual task performance as the patient is engaged in the task. Toglia (1998) also proposes that online awareness be assessed in terms of patients error detection and correction abilities.

Online awareness was explicitly assessed in three studies using error-monitoring on performance tasks. Hart et al (1998) observed 18 TBI patients and matched controls perform a task requiring naturalistic actions, such as making toast, a cup of coffee, wrapping a present and preparing a child's lunch and schoolbag. Error awareness was assessed by observing the behaviour of the TBI patients as they performed each task, noting their detection and correction of errors. TBI patients detected and corrected proportionally fewer errors when compared to controls and despite making more errors than controls on the more cognitively-demanding tasks, they rated themselves as less competent with the physical tasks than the cognitive ones. This study represents a significant advancement in the assessment of online awareness. While having the advantage of not depending on verbal self-reports, the limitations

to this type of assessment of online awareness are that it depends on subjective ratings of assessors based on video scoring, which may not be entirely reliable. This method is also time-consuming in its administration and scoring. A simple, easy to administer approach to assessing error awareness was developed by McAvinue et al (2005). A group of TBI patients and matched controls were required to monitor their errors by saying "hit" when they were aware that they had made an error as they performed a simple task of sustained attention. The TBI participants showed significantly reduced error awareness on this task compared to controls. This degree of error awareness was correlated with sustained attention capacity, suggesting that sustained attention may be one of the executive processes involved in the maintenance of error awareness. This error awareness task allows for adaptation of a simple laboratory task to operationalise emergent awareness, the ability to monitor errors during task performance. Abreu et al (2001) attempted to operationalise intellectual awareness, emergent awareness and anticipatory awareness while patients performed four naturalistic tasks (dressing, meal planning, simple maths, and basic accounting). Participants were asked if they had noticed any changes in their ability to perform specific functions, to predict how well they would perform the tasks, rate how they did after the task, and anticipate how performance might affect ability to live independently. While establishing the value in assessing online-awareness using behavioural activities, the authors recognise this task as a pilot study with narrow focus and subsequently call for additional means to operationalise intellectual, emergent and anticipatory awareness (Abreu et al., 2001).

Broadening investigations of ISA from single measures of metacognitive knowledge or intellectual awareness not only has the clear advantage of providing a much richer approach to assessment of ISA in clinical populations, but also has the advantage of

developing tasks that neurologically-healthy population do not perform at ceiling and so advance the investigation of emergent and anticipatory awareness in general. In a recent study of error-awareness within healthy individuals, subjects reported being aware of 70% of their errors, with an awareness range of 15% to 93% (Hester et al., 2005). Following another recent study examining ISA in AD patients, it was unexpectantly observed that while 64% of mild AD patients overestimated their performance on tasks, 31% of healthy controls also overestimated compared to how they actually performed (Graham et al., 2005). This development allows for more accurately controlled clinical studies that include tasks that challenge not only the clinical populations under investigation but also show the variability evident within the neurologically-healthy controls, therefore providing an indices of "normal" online awareness.

1.9 Summary of Literature Review and Aim of Present thesis

A patient's inability to accurately assess their abilities and disabilities has been strongly associated with a variety of negative outcomes in terms of maladaptive behaviour, rehabilitation success and poor vocational outcome. From the patient's perspective, impaired self awareness can therefore add to the already devastating consequences of brain injury or degeneration. The theoretical models described all purport impaired self-awareness to be a multifaceted phenomenon, possibly resulting from disturbance of multiple neural networks. Despite the growing literature on metacognitive knowledge or intellectual awareness of deficits following TBI and AD in particular, there is a distinct dearth of research regarding online awareness, both emergent and anticipatory. There is also very limited literature that approaches the assessment of ISA from a multidimensional perspective, despite many calls from

investigators to adopt such an approach in order to gain a better insight into the complexities of deficits in awareness. While recent studies have begun to focus on the specific executive function and behavioural correlates of ISA based on some of the theoretical models proposed, due to the aforementioned limited unimodal approach to assessment that has dominated until now, it is strongly felt that there is much more to learn regarding intellectual awareness, emergent awareness and anticipatory awareness. It is evident from the review of the literature that despite over twenty years of fervent neuropsychological research in the area, there is also a lack of consensus regarding the characteristics of impaired awareness. Clearly, if some of the methodological issues could be even partially resolved, a broader understanding of the substrates of impaired awareness could be achieved.

Aim of Present Thesis

This thesis will adopt a multidimensional approach to the assessment of impaired awareness. Initially, a task of online awareness, specifically emergent awareness, will be further developed and related to psychophysiological elements in a group of TBI patients. This will be followed by a large study operationalising each component of Toglia and Kirk's (2000) model by examining metacognitive knowledge, emergent awareness and anticipatory awareness in TBI patients. An investigation into specific neuropsychological profiles of poor self-awareness will also be conducted. Following development of this multidimensional approach with TBI participants, aspects will be adopted to comprehensively assess impaired self-awareness in FTD patients, considered to be a core criteria of this disorder, yet lacking systematic studies. CBD and PSP patients share several clinical and pathological features with FTD and PD. To the best knowledge of this researcher,

impaired self awareness has never been investigated in CBD or PSP before. It was therefore hypothesised that these two patient groups may also exhibit features of impaired awareness.

Chapter 2

Study 1

Impaired Online Monitoring in Traumatic Brain Injury (TBI) Patients: Evidence from an Error-Monitoring Task and Electrodermal Activity

Abstract

Impaired deficit awareness is common following Traumatic Brain Injury (TBI) and is a major obstacle to rehabilitation. In the present study, online error-monitoring abilities on a simple laboratory task are examined. Sixteen participants with TBI and sixteen age, sex and education matched controls performed the Sustained Attention to Response Task (SART) while monitoring their errors. Electrodermal Activity (EDA) was recorded while performing the error-monitoring task. TBI participants detected significantly fewer errors compared to controls. EDA was significantly attenuated for TBI participants, even to errors of which they were aware; error detection rates and EDA amplitude were also correlated. These findings suggest that poor insight following TBI may be a result in part from impaired error processing abilities.

2.1 Introduction

As mentioned, following TBI, survivors with diffuse axonal damage, more focal lesions or haemorrhages often demonstrate similar patterns of executive function deficits to patients with focal prefrontal lesions (Adams, 1984; Cooke & Kausler, 1995; Fontaine, Azouvi, Remy, Bussel, & Samson, 1999; Mattson & Levin, 1990; McAllister et al., 1999; Stuss, 1987; Stuss & Gow, 1992; Umilta & Stablum, 1998; Vilkki, 1992). The white matter of the prefrontal cortex is known to be particularly vulnerable to the effects of TBI (Mattson & Levin, 1990; Stuss & Gow, 1992). These impairments in executive control often evident in TBI patients can manifest themselves in numerous ways, including difficulties in self-direction, planning, organisation, problem-solving, sustaining attention, disinhibition and self-monitoring. Goldberg and Barr (1991) implicate the executive functions of cognitive self-monitoring and the comparison of the outcome of one's cognitive operations with self-awareness.

Self-awareness and realistic self-appraisal following TBI are recognised as significant factors in predicting potential benefits from treatments, overall long-term outcome, return to community living and productive lifestyle (Ben-Yishay et al., 1985; Ezrachi et al., 1991; Prigatano & Altman, 1990; Prigatano & Fordyce, 1986; Prigatano & Schacter, 1991). Toglia and Kirk (2000) have differentiated between metacognitive knowledge of deficits, or global awareness and online monitoring, or local awareness. Many studies investigating aspects of awareness in brain injury populations have focussed on the more general global awareness of their deficits, or metacognitive knowledge, reflected in the vast number of methods developed to assess this type of awareness, such as the Awareness Questionnaire (Sherer et al, 1998), the Self-Awareness of Deficits Interview (Fleming et al., 1996), the

Awareness Interview (Anderson & Tranel, 1989), and the Patient Competency Rating Scale (Prigatano & Altman, 1990). There are a number of methodological difficulties involved in assessing global awareness deficits in TBI patients, including accuracy and subjectivity of self, significant other and clinician report (Brooks, 1984; Sherer et al., 1998). It has been proposed that investigations into error processing, detection and correction, i.e. investigations into "local awareness" or online monitoring, may provide insights and explanations into the more general phenomena of unawareness (Bisiach & Berti, 1985; Hart et al., 1998).

In comparison to the extensive research in brain injury and global awareness, there is a very limited number of studies that have investigated error awareness, or local awareness in TBI and focal lesions. Though not conclusive, ERP studies have shown disrupted or unusual Ne and Pe production in patients with lateral and medial prefrontal lesions, and lesions of the anterior cingulate region (Gehring & Knight, 2000; Stemmer et al., 2004; Ullsperger, von Cramon, & Muller, 2002). McAvinue et al (2005) demonstrated that TBI participants showed reduced error awareness on a task of sustained attention. Hart and colleagues (1998) studied awareness of errors among people with TBI in naturalistic everyday situations. TBI patients showed less awareness and corrected fewer errors than controls. It was suggested that head injury may result in impairment in the allocation of resources that are needed for effective execution and monitoring of routine tasks. Stemmer et al (2004) recorded error awareness behaviour on a flanker task trial based on overt behaviour recorded by the researchers by documenting behavioural signs, such as exclamations, whispered swearing and grimaces during an ERP study of patients with lesions to the prefrontal cortex and anterior cingulated regions following ruptured aneurysm of the anterior communicating artery (AACA). In their study, three of the 5 patients showed

inconsistent or mixed error awareness behaviour. It is clear from the limited work in the area that further investigations into local or online awareness and TBI is required.

Electrodermal activity (EDA) measures reflect the activity of the sympathetic nervous system and are commonly used as an indirect measure of emotional arousal, attention and cognitive effort, an index of psychological processing properties of stimuli, such as significance, novelty or emotional relevance and effortful processing (Critchley, 2002; Zahn, Grafman, & Tranel, 1999). EDA has been reported to be one of the most sensitive physiological indicators of psychological phenomena (Hugdahl, 1995). There are a number of advantages in using EDA in cognitive research. Unlike other physiological measures, such as heart rate, EDA provides an undiluted and direct representation of sympathetic activity, as the neural control of the eccrine sweat glands is entirely under sympathetic control. EDA response is generally readily discriminable, again in contrast to a heart rate response, which might be difficult to distinguish from ongoing heart-rate. Furthermore, of all varieties of ANS activity, it has been shown that individual differences in EDA are most reliably associated with psychopathological states (Dawson, Schell, & Filion, 2000). Practically, EDA is harmless, non-intrusive and relatively inexpensive to record in a variety of settings, particularly when compared to brain imaging techniques, such as ERP, fMRI and PET. A number of studies implicate the role of the right hemisphere in the EDA response, as it has been shown that right hemisphere lesions can reduce or abolish EDA responses, whereas left hemisphere lesions have not shown consistent abnormality (Critchley, 2002; Tranel & Damasio, 1994; Zahn et al., 1999; Zahn & Mirsky, 1999). Boucsein (1992) proposed that there are at least two independent CNS pathways involved in the control of EDA response production. One is proposed to be mainly responsible for the EDA response in emotional contexts and involves the hypothalamus, the anterior thalamus and the cingulate

gyrus. The other control system is proposed to control EDA responses in more cognitive contexts and involves the lateral frontal cortex, including the premotor cortex and the basal ganglia.

Lehrer, Groveman, Randolph, Miller & Pollack (Lehrer, Groveman, Randolph, Miller, & Pollack, 1989) showed that a group of closed head injury patients showed less physiological reactivity, including attenuated Skin Conductance Response (SCR), during cognitive tasks, compared to controls. They suggested that this may have been a result of a deficit in situation-appropriate modulation of physiological activity in that group. The pattern of physiological reactivity found in the control group was interpreted as adaptive responses to specific tasks, whereas patients failed to respond similarly. Zahn and Mirsky (1999) have shown that persons with Closed Head Injury (CHI) show marked deficits in both tonic and phasic electrodermal responding to meaningful, significant or demanding situations or stimuli. Their results suggest that CHI has long-lasting effects on the ability to mobilize activation during effortful, controlled information processing. Patients with frontal lobe damage also tend to display attenuated EDA responses to stimuli of emotional or psychological significance (Zahn et al., 1999). They can quite often recall disturbing images in detail but with a distinct absence of feeling. Their lack of affective response has been interpreted as their descriptions representing semantic, rather than emotional, associations (Damasio, Tranel, & Damasio, 1990; Tranel & Damasio, 1994). Damasio refers to this distinction between factual knowledge and emotional response as "To know does not necessarily mean to feel" (Damasio, 1994).

Based on previous studies showing that TBI patients have poor error processing abilities (Hart et al., 1998; McAvinue et al., 2005) this study aims to replicate

previous findings that TBI patients are impaired in monitoring their errors on a simple laboratory task. As there is evidence that TBI and frontal lesion patients tend to show decreased physiological response as measured by EDA to emotional significant stimuli, this study also focuses on the emotional response to making an error in a paradigm that imposes cognitive significance to a rare no-go target. This may aid understanding of error-monitoring in TBI patients, as it is possible that the impairment of emotional processing in TBI may be linked to their awareness deficits in error detection and correction. This study hopes to identify a specific error processing deficit in response to making an error on a simple continuous performance task (the SART) and compare the emotional responses of TBIs to controls on making such an error.

The aim of the present study was to investigate the performance of 16 TBI patients on a task of sustained attention (SART), monitor their detection of errors while they performed the task, and analyse their EDA response to errors and correct responses. TBI participants' performance and EDA responses were compared to neurologically healthy controls in order to determine whether error awareness deficits were evident in impaired psychophysiological responsiveness to errors.

2.2 Method

Participants

Sixteen Traumatically Brain-Injured (TBI) participants were recruited from Headway Ireland, the National Head Injuries Association of Ireland.

Exclusion criteria for TBI participants included a pre-trauma history of epilepsy or other neurological condition, a history of major psychiatric disorder, or a history of drug or alcohol problems. The mean age of the TBI group was 32.13 (SD 10.60) and included twelve men and four women. An estimate of Post-Traumatic Amnesia (PTA) duration was used as a measure of severity of head-injury and was available for fourteen TBI participants (PTA: Teasdale, 1995). By this classification, the TBI group consisted of three severe (PTA 1-7 days), six very severe (PTA 7-28 days), and five extremely severe (PTA >28 days) cases. The mean length of time since injury to the time of testing was 5.51 years (SD 5.16).

The comparison group of sixteen neurologically healthy individuals consisted of eleven men and five women, recruited to match the TBI group on sex, age and education-level variables. Their mean age was 29.81 (SD 10.58). The same exclusion criteria applied to the comparison control group, with the additional criteria that they had never suffered a loss of consciousness from a head injury.

The local research ethics committee approved the study and all participants gave informed consent prior to participation. The demographic and self-report measures for both TBI and control participants are displayed in Table 2.1.

Table 2.1: Participant Characteristics for TBI and Control Participants

	TBI	CONTROL
N	16	16
Sex (m, f)	12, 4	11, 5
Mean Age	32.13 (SD 10.60)	29.81 (SD 10.58)
Education Level	2: 7: 7	0: 6:10
CFQ	43.06 (SD 20.79)	35.44 (SD 7.75)
HADS- Anxiety	7.56 (S.D. 5.81)	7.88 (SD 3.48)
HADS-Depression	5.81 (SD 3.04)	3.62 (SD 2.47)
UMACL-Energetic Arousal	24.33 (SD 3.66)	21.31 (SD 4.64)
UMACL-Tense Arousal	15.87 (SD 4.64)	14.63 (SD 3.54)
UMACL-Hedonic Tone	27.60 (SD 3.83)	25.44 (SD 4.73)
UMACL- Anger/ Frustration	7.73 (SD 2.74)	6.25 (SD 1.18)

Education Level: No School-Leaving: School-Leaving : Third Level

CFQ: Cognitive Failures Questionnaire (Broadbent, Cooper, FitzGerald, & Parkes, 1982); HADS: Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983); UMACL: The UWIST Mood Adjective Checklist (Matthews, Jones, & Chamberlain, 1990)

Measures

Self-Report Measures

The following questionnaires were used to provide a profile of everyday cognitive and affective functioning of each participant:

The UWIST Mood Adjective Checklist (UMACL) (Matthews et al., 1990).

This checklist comprises 29 mood descriptors, each rated on a 4-point Likert scale. It is used to measure dimensions of participants' current mood in terms of energetic arousal, tense arousal, hedonic tone and anger and frustration

Cognitive Failure Questionnaires (CFQ) (Broadbent et al., 1982)

The CFQ consists of 25 questions, rated on a 5-point Likert scale, measuring slips of action, failures in everyday memory and absentmindedness.

The Hospital Anxiety and Depression Scale (HADS) (Zigmond & Snaith, 1983)

The HADS scale is comprised of 14 items from which participants choose the response which most accurately reflects their feelings. Seven statements reflect anxiety levels and seven relate to depression levels.

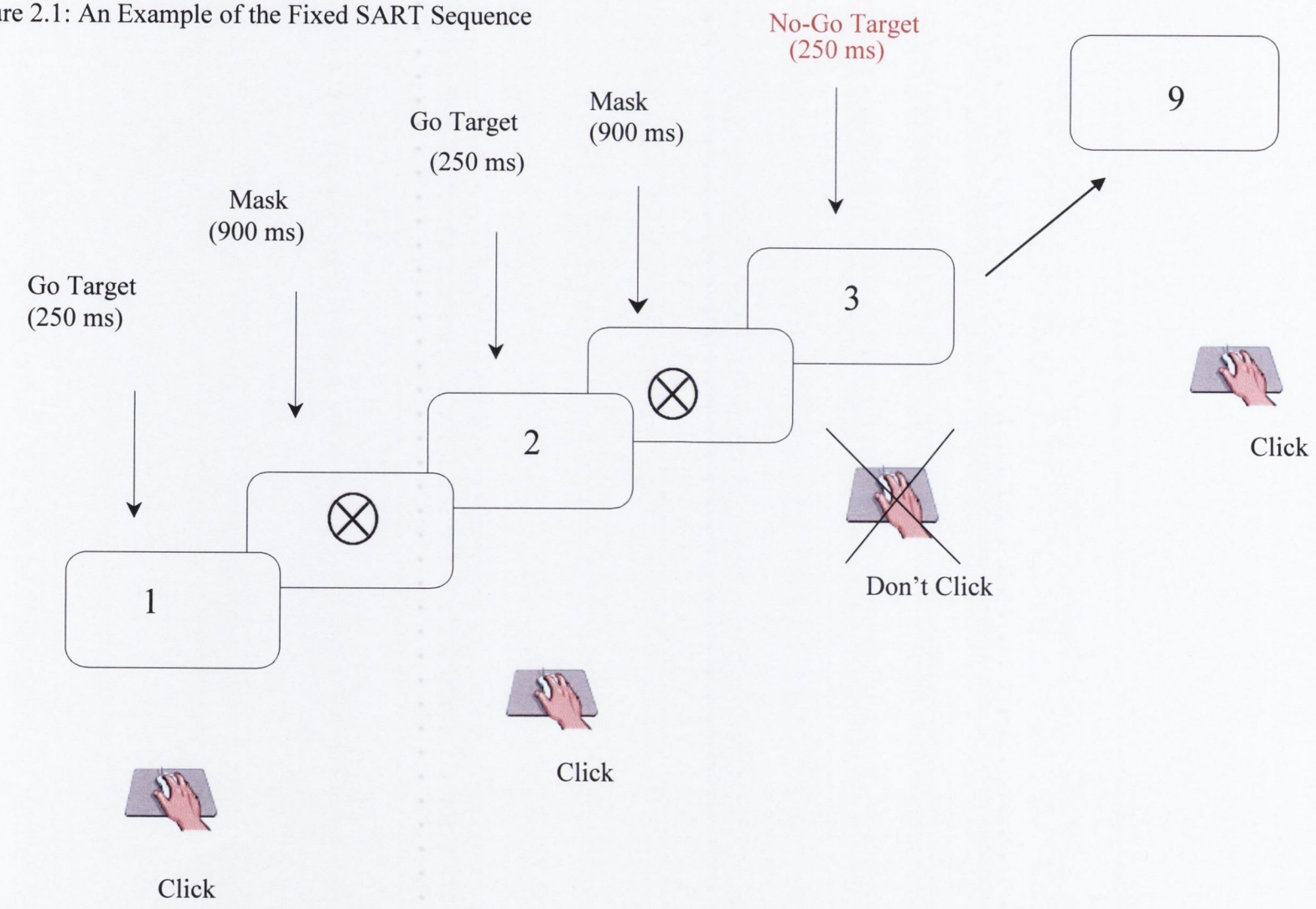
Sustained Attention to Response Task (SART)

The reliability and validity of the SART, as a measure of executive control of behaviour and its sensitivity to frontal damage, has been demonstrated in previous studies (Dockree et al., 2004; Manly et al., 2003; McAvinue et al., 2005; Robertson et al., 1997). The SART was programmed on E-Prime software and administered on

a laptop computer, with screen dimensions of 23cm X 30cm. Over a 4.3-minute period, 225 single digits from one to nine were presented centrally on the computer screen. Each digit was presented 25 times and was displayed for 250 milliseconds. A 900-millisecond mask of an X within a circle followed each digit. This meant that there was a regularly paced 1150 millisecond period from digit onset to the next digit onset. Digits were presented in one of five randomly assigned font sizes, corresponding with digit heights of between 12 and 29 mm. This was deemed necessary to ensure that each number was evaluated for its specific value as a go or no-go target and to avoid peripheral perceptual processing of the numbers. The digits and masks were centrally displayed in black on a white background. Two versions of the SART were used. The Fixed SART consisted of digits from 1 to 9 being presented in a fixed order from 1 to 9 (1, 2, 3, 4, 5, 6, 7, 8, 9, 1, 2, 3...) for 25 presentations of this sequence. The no-go target of the number 3 was thus presented 25 times. Figure 2.1 depicts an example of the Fixed SART. In the Random SART, numbers from 1 to 9 are presented in a pre-fixed quasi-random order (Manly et al., 2003; Manly et al., 1999; Robertson et al., 1997). The no-go target, the number 3, had 0.11 probability of appearing and was presented a total of 25 times. No-go targets were never presented in direct succession.

Participants performed the Fixed SART once at the beginning and once at the end of the experimental procedure as a baseline measure of executive control of behaviour and to ensure that no practice effects could account for between-condition differences.

Figure 2.1: An Example of the Fixed SART Sequence



Error Awareness

In the first error-awareness condition (Awareness1) participants were asked to verbally indicate their errors of commission on the no-go target (the number 3) by saying "hit". This was a measure of the participant's awareness of having made an error and was performed on both the Fixed SART and Random SART. A second awareness condition (Awareness2) required participants to say "hit" on making an error as well as additionally saying "hold" when they correctly withheld on the no-go target. The additional "hold" requirement was introduced as a control measure to ensure that any skin responses elicited from the EDA were due to a response to either correctly withholding or to making an error of commission following the presentation of the no-go target and not due to the actual verbalisation on the awareness condition.

There were therefore 4 main experimental conditions – the two versions of SART, Fixed and Random, each with two awareness conditions. The order of the 4 main experimental conditions was counterbalanced across subjects. As both the baseline and experimental measures utilized two versions of the same task and in order to simplify labelling, the baseline and experimental conditions are referred to as follows: Fixed-Baseline, Fixed-Awareness1, Random-Awareness1, Fixed-Awareness2, Random-Awareness2.

Error awareness was calculated by dividing the number of errors reported by the number of errors made on each of the awareness conditions, which represented an 'error awareness' score.

Electrodermal Activity

Electrodermal activity (EDA) was measured using the Biopac Student Lab system (Biopac Systems, Inc, Version 3.6.6.1). EDA was continuously recorded from the distal phalanges of the index and middle fingers of the non-dominant hand. Two Ag/AgCl electrodes were mounted in individual housings and shielded to minimise noise interference. They were attached to the distal phalanges by a velcro strap. Each electrode was 16 X 17 X 8 mm. The electrodes used had a 6mm contact area with a 1.6mm cavity for accommodating the electrode gel. Multi-purpose gel, "Signa Gel", was used as the electrolyte. The incoming skin response signals were converted to digital signals via an MP30 data acquisition unit and processed with BIOPAC Student Lab PRO for off-line analysis.

Electrodermal responses were analysed in the same manner as described in previous studies (Tranel & Damasio, 1994; Zahn et al., 1999). The rate of spontaneous, Non-Specific Skin Responses per Minute (NS-SCR/Min) of at least 0.02 μS in amplitude was recorded. Mean Skin Conductance Level (SCL) in μS was measured from SCL measurements at 30 and 60 seconds during the rest period. For each no-go target ("3") presentation, a latency window of 1 to 4 seconds after stimulus onset was specified for elicited SCR measurement. Measurement of the amplitude of SCR was taken as the conductance at the peak minus the conductance prior to the response, as defined by Stern, Ray and Quigley (2001). The amplitude of the largest Skin Conductance Response (SCR) that had an onset within this latency window was measured. The criterion for the smallest scorable SCR was set at 0.02 μS. Amplitude measurements were thus recorded for SCR to correct withholds, errors of commission (on Fixed-Baseline) and SCR for saying "hit" and "hold" (on Fixed-

Awareness2 and Standard-Awareness2), as these were directly comparable to each other.

Procedure

Participants were seated with the laptop on a table in front of them. The participants indicated with which hand they preferred to make responses with during the SART. The other hand (generally the non-dominant hand) was used to measure EDA. The electrodermal recordings took place at least five minutes after the electrodes were applied to allow for hydration. Participants were next asked to sit quietly while recording skin responses for a one-minute rest period.

The computer screen was approximately 40cm away from the eyes of the participants, with no restrictions on head movements. The experimenter explained that equal weight should be given to accuracy and speed of responses on both the Fixed and Random versions of the SART. A practice trial of 30 digits preceded each condition, which included 4 no-go targets for the fixed conditions and 5 no-go targets on the standard conditions. After the practice trials on each condition, the testing began.

Testing sessions lasted no more than one hour for all participants. This included consenting, questionnaire administration, calibration, hydration and rest period recording of EDA, SART practice trials, SART baseline conditions and experimental conditions. Rests were given to all participants upon request.

2.3 Results

Section A: Demographic and Screening Data

Demographic data and scores of self-report measures for the TBI group and control group are detailed in Table 2.1. Matching was successful as the TBI group and control group did not significantly differ in terms of age ($t(30)= 0.617, p > .05$), sex ($\chi^2 = .155, df = 1, p > .05$), education ($\chi^2 = 2.606, df = 2, p > .05$), CFQ ($t(30)=1.375, p > .05$), HADS anxiety/depression scores (HADS-Anxiety: $U = 111.0, n_1 = 16, n_2 = 16, p > .05$; HADS- Depression: $U = 78.0, n_1 = 16, n_2 = 16, p > .05$), UMACL (UMACL-Energetic arousal: $U = 74.0, n_1 = 15, n_2 = 16, p > .05$; UMACL- Tense arousal: $U = 90.0, n_1 = 15, n_2 = 16, p > .05$; UMACL-Hedonic tone: $U = 89.5, n_1 = 15, n_2 = 16, p > .05$; UMACL-Anger-frustration: $U = 80.5, n_1 = 15, n_2 = 16, p > .05$).

Section B: Do TBI participants differ from control participants on Online Error-Monitoring?

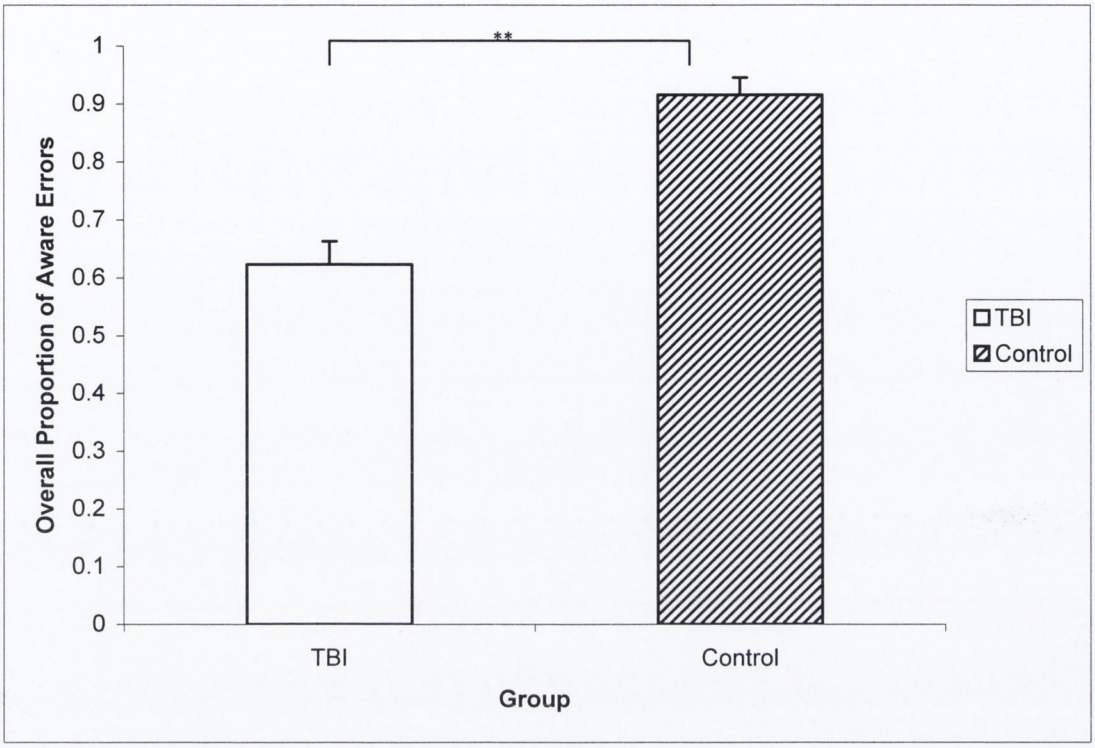
TBI participants showed a significantly lower level of awareness of errors than control participants on all SART awareness conditions, as depicted in Table 2.2.

Table 2.2: Levels of Error Awareness (Proportion of Errors of which Participants were Aware) on Each SART Awareness Condition for TBI and Control Participants

Awareness Condition	**TBI**	**CON**	**t value**	***p***
Fixed-Awareness1	.7492 (.25)	1.0 (0.0)	-3.955	.001
Random-Awareness1	.7690 (.21)	.9625 (.15)	-2.894	.01
Fixed-Awareness2	.2442 (.24)	.7188(.45)	-3.747	.001
Random-Awareness2	.7609 (.29)	.9807(.01)	-2.98	.01

An overall error awareness score was calculated by getting the mean error awareness score of the four SART awareness conditions. There was a significant difference between the overall error awareness score between TBI participants and controls ($t(30) = -6.302$, $p < .001$), as can be seen in Figure 2.2 below.

Figure 2.2: Overall Proportion of Errors Reported by TBI and Control participants for Each SART Awareness Condition



**Significant at $p<.01$ level

Section C: Do TBI participants differ from control participants on sustained attention performance?

To ensure there were no practice effects, the Fixed-Baseline was presented in two blocks, one at the beginning and one at the end of testing. As there was no significant difference between mean errors of commission made on the first and last Fixed-Baseline for either TBI or controls (TBI: $t(15) = .78$, $p>.05$/ CON: $t(15) = 1.00$, $p>.05$), for all subsequent analyses, the scores from these two identical conditions were combined, forming an overall Fixed-Baseline score.

Errors of commission were compared between TBI and control participants. To examine if TBI participants made significantly more errors of commission on each SART condition, a mixed ANOVA was run. Between-subjects variable was Group (2

levels: TBI and control) and the within-subjects variable was SART Condition (5 levels: Fixed-Baseline, Fixed-Awareness1, Random-Awareness1, Fixed-Awareness2, Random-Awareness2). A significant main effect of SART condition was found [F (2.189, 63.472) = 54.636.13, p< .001]. There was also a significant main effect of group [F(1,29)= 39.514, p<.001]. The interaction between SART condition and group was also significant [F(2.189, 63.472) =3.619, p <.05].

Post-hoc t-tests (independent samples) showed that on each SART condition, the TBI participants made significantly more errors of commission than the control participants. Both TBI and controls made significantly fewer errors on the fixed conditions than the random conditions. Table 2.3 shows that the SART significantly discriminates between TBI and control groups on each SART condition.

Table 2.3: Comparison of Mean Errors of Commission Made by TBI and Control Participants on Each SART Condition.

SART Condition	TBI	CON	*t*	*p*<
Fixed-Baseline	3.84 (2.13)	1.09(0.68)	4.904	.001
Fixed-Awareness1	3.88(3.38)	1.06(1.48)	3.045	.01
Random-Awareness1	14.47(5.0)	7.63(3.34)	4.506	.01
Fixed-Awareness2	8.56(6.55)	1.25(1.00)	4.413	.001
Random-Awareness2	15.50(4.43)	8.38(5.83)	3.893	.01

Paired sample post-hoc t-tests showed that TBI participants made significantly more errors on Fixed-Awareness2 than Fixed-Baseline (t= 2.764, df = 15, p< .05) and Fixed-Awareness1 ($t(15)$= -2.937, p< .05), all fixed SART conditions. Controls did not make significantly more errors on Fixed-Awareness2 than the other fixed

conditions, Fixed-Baseline ($t(15)$= .675, p> .05) or Fixed-Awareness1 ($t(15)$= -.481, p> .05).

Section D: Do TBI participants differ from control participants on Electrodermal Activity (EDA) on baseline measures?

a) EDA at rest

In order to investigate whether non-specific skin conductance responses (NS-SCR) and mean skin conductance level (SCL) over the one-minute rest period differed between TBI and control participants, two independent sample t-tests were run. The difference between the mean frequency of NS-SCR of TBI participants (M=10.63, SD= 4.46) and controls (M=12.19, SD= 5.44) did not reach statistical significance (t (30) = -0.889, p>.05). The mean SCL of TBI participants (M=.009μS, SD = 0.05) was not significantly different from that of control participants (M=.07μS, SD= 0.25) (t (30) = .348, p> .05). There were therefore no significant differences in the base level measurements of skin conductance between the two groups.

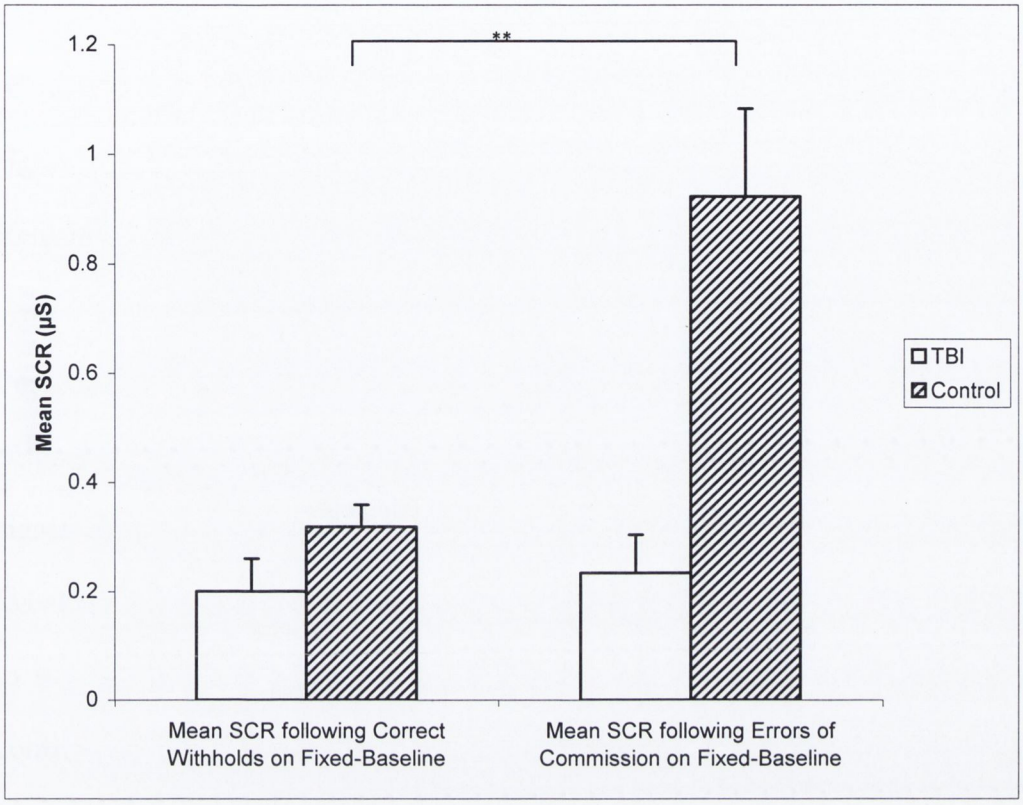
b) EDA on Fixed-Baseline

Skin conductance responses (SCR) following presentation of no-go targets were examined. In order to investigate if there was a significant difference between the mean amplitude of SCR to correct withholds and incorrect errors of commission between TBI and control participants on Fixed-baseline, a mixed ANOVA was performed. Group was the between-subjects variable (Two levels: TBI and controls) and Response type (Two levels: correct withholds and errors of commission) was the

within-subjects variable. There was a significant main effect of Response type [$F(1,26) = 17.075$, $p<.001$], a significant main effect of Group [$F(1, 26) = 16.538$, $p< .001$] and a significant interaction between Response type and Group [$F(1, 26) = 13.674$, $p < .01$].

Post-hoc independent samples t-tests showed that the mean amplitude of SCRs to a correct withhold on Fixed-Baseline made by TBI participants (M=0.2 μS, SD=.22) was not significantly different from the mean SCR amplitude to a correct withhold made by controls [M= 0.35μS, SD =0.2; t (29)= -1.883, $p>.05$). The mean skin response to errors of commission on Fixed-Baseline by TBI (M=.23 μS, SD= .269) was significantly lower than the mean SCR amplitude of controls (M=.92 μS, SD= .56; t (26) = -4.22, $p <.001$). Paired t-tests showed that the difference between mean skin response amplitude to correct withholds and to errors of commission was not significant for TBI participants (t (14) = -.472, $p> .05$). Control participants showed significantly higher SCR amplitudes to errors of commission than to correct withholds on Fixed-Baseline (t (12)= -4.184, $p< .01$). The mean SCR amplitudes following correct withholds and errors of commission by TBI and control participants are portrayed in Figure 2.3.

Figure 2.3: The Mean Skin Response Amplitude (μS) for Withholding a Response and Errors of Commission on Fixed-Baseline for TBI and Control Participants.



** Significant at $p<.01$

Section E: Do TBI participants differ from control participants on Electrodermal Activity (EDA) on aware errors on the Fixed SART?

As it is possible that the reduced SCR to errors may have been due to a lack of awareness of some of these errors, a second EDA analysis was conducted, restricted to those responses where both errors ('hit') and correct withholds ('hold') had been signalled by participants, and of which they were therefore fully aware.

A mixed ANOVA was therefore performed in order to compare Fixed-Awareness2 EDA response for TBI and control participants. Group (Two levels: TBI and control)

was the between-subjects variable and Response type (Two levels: indicating "hold" to correctly withholding to no-go targets and indicating "hit" to making an error of commission) were the within-subjects variables. There was a significant main effect of response type [$F(1,16) = 12.494$, $p < .01$]. The main effect for Group was also significant [$F(1,16) = 4.75$, $p < .05$]. There was also a significant interaction between Response and Group [$F(1,16) = 1.745$, $p < .01$].

Post-hoc (Independent and Paired samples) t-tests showed that following the detection of an error on Fixed-awareness2, the mean SCR amplitude was significantly lower for TBI participants (M=0.481 μS, SD = .59) than controls [M=1.68 μS, SD= 1.2; $t(16) = -2.76$, $p < .05$]. The difference between skin responses to saying "hold" for correct withholds between TBI (M=0.336 μS, SD= .37) and controls (M=0.651, SD = .72) did not reach statistical significance (t (30) = -1.568, $p > .05$). Control participants showed a significantly larger skin response to awareness of making an error of commission than to correctly withholding on the no-go target ($t(7) = -3.571$, $p < .01$). TBI participants' skin responses to awareness of error of commission were not significantly different from their skin responses to correct withholds (t (9)= -.547, $p > .05$). This analysis confirmed that non-awareness of errors did not account for the reduced SCR to errors in the TBI group.

Figure 2.4 depicts the mean SCR amplitude (μS) following "hold" and "hit" on Fixed-Awareness2 in TBI and control participants. An example of a SCR following a detected error ("hit") for both a TBI and control participant is illustrated in Figure 2.5.

Figure 2.4: The Mean Skin Conductance Response Amplitude (μS) for "Hold" on Withholds and for "Hit" on Errors of Commission on Fixed-Awareness2 for TBI and Control Participants.

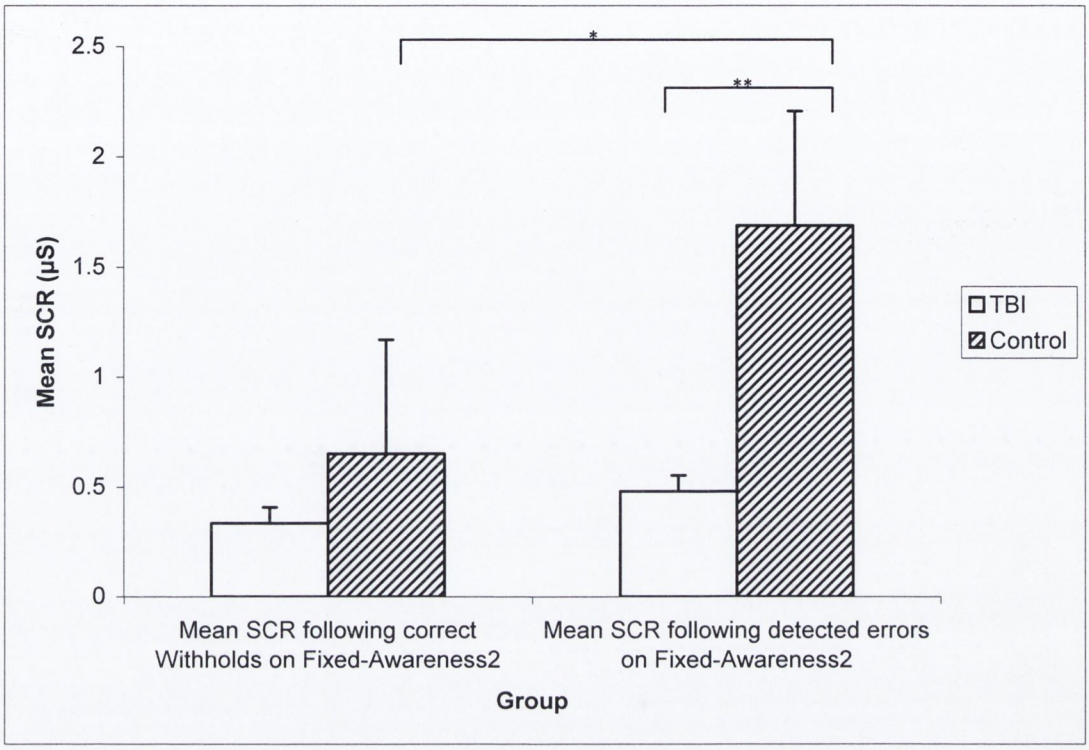
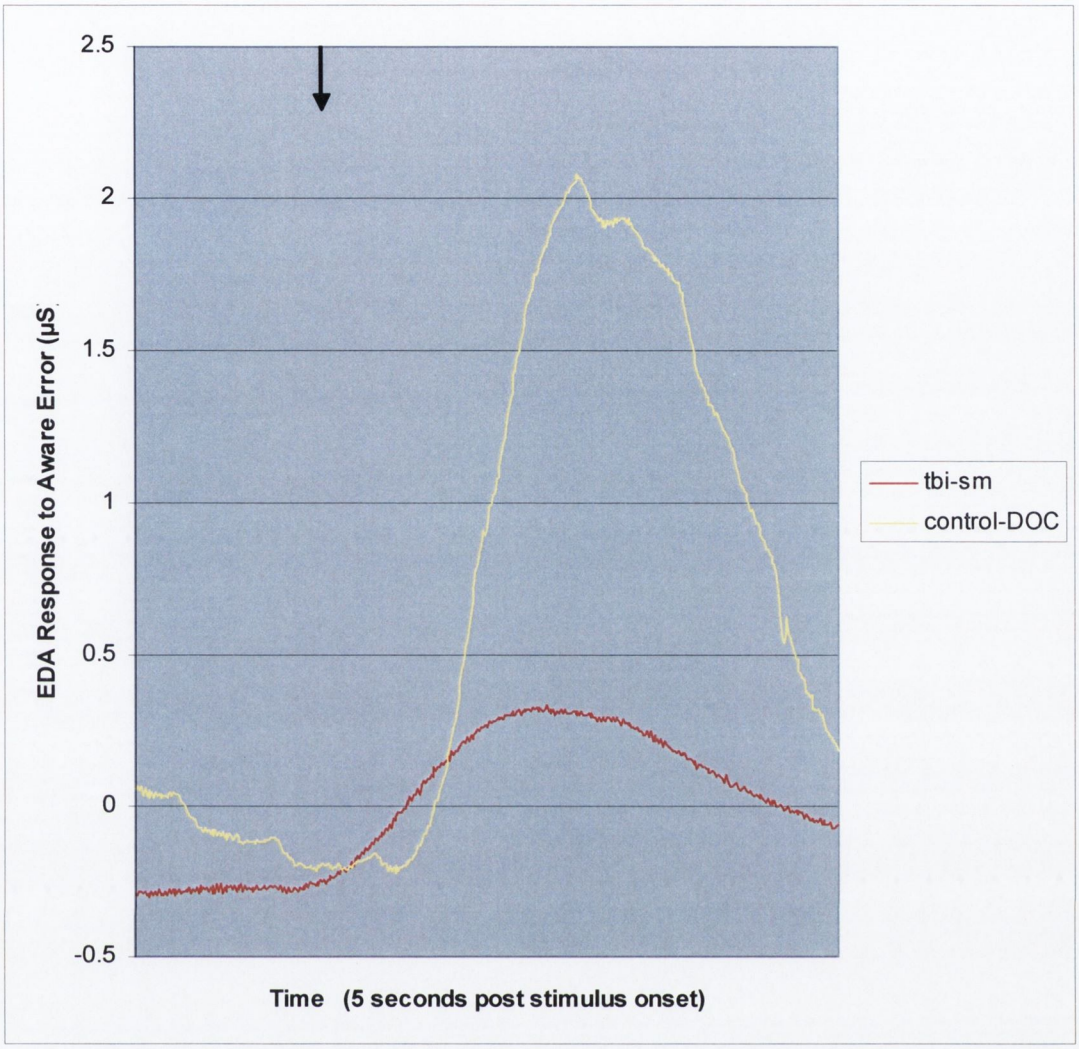


Figure 2.5: Example of SCR (μS) for TBI and Control Participant following a Detected Error. The Arrow Indicates Stimulus Onset.

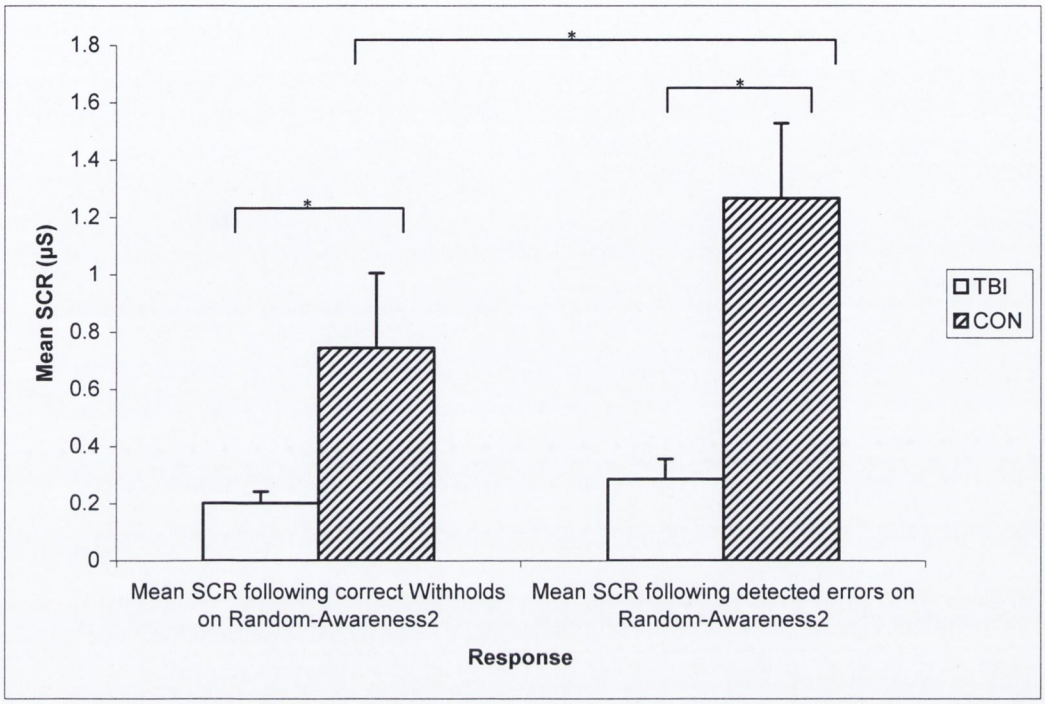


Section F: Do TBI participants differ from control participants on Electrodermal Activity (EDA) on aware errors on Random SART?

A mixed ANOVA was run to uncover any significant differences in skin responses on Random-Awareness2, with Groups (Two levels: TBI and control) as the between-subjects factor and Response type (Two levels: indicating "hold" to correctly withholding to no-go targets and indicating "hit" to making an error of commission) as the within-subjects factor. There was a main effect of response type [$F(1, 29) = 14.917$, $p<.01$], a main effect of Group [$F(1, 29) = 11.344$, $p<.01$] and a significant interaction between Response type and Group [$F(1, 29) = 11.707$, $p <.01$].

Post-hoc t-tests revealed that on Random-Awareness2 TBI participants showed a significantly lower SCR amplitude to indicating a correct withhold (M=.202 μS, SD = .16) than controls (M=.7451 μS, SD = .73; $t(16.5)= -2.922$, $p< .05$). On indicating awareness of error by saying "hit", TBI participants' mean SCR amplitude (M=.286 μS, SD = .29) was significantly lower than controls (M=1.2 μS, SD=1.07; t (17.21) = -3.545, $p< .05$). TBI participants' skin responses did not differ on saying "hold" or "hit" (t (14) = -1.442, $p> .05$). Control participants did have a much larger skin response to indicating awareness of error of commission by saying "hit" than to correctly withholding and saying "hold" (t (15) = -3.79, $p < .05$). Figure 2.6 shows the mean skin response amplitude of TBI and controls following "hold" or "hit" responses on Random-Awareness2.

Figure 2.6: The Mean Skin Conductance Response Amplitude (μS) for "Hold" on Withholds and for "Hit" on Errors of Commission on Standard-Awareness2 for TBI and Control Participants.



Significant at $p<.05$

Section G: Is there a relationship between sustained attention, error awareness and EDA measures for TBI participants?

For TBI participants, the degree of SCR amplitude following errors on Fixed-Awareness2 and Random-Awareness2 correlated significantly with the combined error awareness score from Fixed-Awareness1, Random-Awareness1, Fixed-Awareness2 and Random-Awareness2, the four SART error awareness conditions (r_s = .618; n = 16; $p<.05$). No significant association was found between error awareness and sustained attention score (r_s= 0.027; n = 16; $p>.05$) or between sustained attention and SCR amplitude (r_s = -0.057; n = 16; $p>.05$).

Self-reported CFQ score for TBI participants was not significantly correlated with overall sustained attention score (r_s= -.197; n= 16; $p < .05$), or with error awareness score (r_s= -.181; n= 16; $p > .05$). The correlation between Fixed SART and CFQ was approaching significance (r_s= .411; n= 16; $p = .057$).

Self-reported CFQ score for controls was significantly correlated with overall sustained attention score (r_s= .495; n= 16; $p < .05$) and Fixed SART (r_s= .542; n= 16; $p < .05$). CFQ was not significantly correlated with error awareness score (r_s= .055; n= 16; $p > .05$).

2.4 Discussion

TBI participants had a significantly reduced error awareness compared to control participants on all four SART awareness conditions. These results replicate previous findings of impaired error awareness on this task (McAvinue et al., 2005) and confirm, using a completely different method of assessment, that TBI patients have impairments in error detection (Hart et al., 1998; Sherer, Oden et al., 1998). As proposed by Hart et al (1998), tests of error detection may provide insights into more general phenomena of unawareness in TBI. General self-awareness is vital for positive rehabilitation outcome (Sherer et al, 1998).

TBI participants consistently displayed diminished EDA responses to making errors on this simple sustained attention task compared to neurologically healthy controls, but showed either no such differences or a much smaller difference in response to executing successful withholds. There were no significant differences between TBI and control participants' skin conductance levels and non-specific skin conductance responses while participants were at rest and not engaged in any task. This is only partially consistent with Zahn et al's (Zahn et al., 1999; Zahn & Mirsky, 1999) findings that frontal lesions do not intrinsically affect EDA but that the differences are on psychological response to significance or effortful processing, indexed by the EDA. Rather, this study indicates that the EDA deficit is *specific* to the processing of errors and is almost entirely absent to correct withholds. The one exception to this – Random SART in the second awareness condition – is likely due to dual task effects of this condition combined with greater difficulty of Random SART test. Furthermore, even in this condition, there was a significant interaction, with the EDA deficit for errors in TBI being significantly greater than that for correct withholds.

The magnitude of this EDA-error deficit predicts significantly the degree of unawareness of error that individuals show.

TBI participants performed significantly worse than controls on both the Fixed and Random SART. In keeping with previous studies (Chan, 2001; Manly et al., 2003; McAvinue et al., 2005; Robertson et al., 1997), this suggests that this clinical group also had marked deficits in the executive control of behaviour that could be discriminated from controls using the SART.

The SART paradigm imposes importance or cognitive significance to the presentation of the no-go target, the "3". This would suggest a certain emotional significance to making an error of commission and detecting such an error. Controls showed a significant increase in EDA amplitude following committing an error on Fixed-Baseline and following detection and reporting of an error on Fixed-Awareness2 and Random-Awareness2. These results suggest that following errors of commission and following detecting such errors, controls showed some significant emotional reaction to their mistakes. Alternatively, this increased amplitude may be indicative of some increased effortful processing following committing or detecting an error. TBI participants did not show differences in EDA amplitude between successfully withholding for a no-go target and making an error of commission on any of the SART conditions analysed. This study suggests that patients did not show the same emotional reaction to the significance of detecting an error, or did not show an increase in effortful cognitive processing compared to controls. These findings refine the results of previous studies showing diminished EDA in individuals with TBI to stimuli with emotional content or to the psychological processes involved in cognitive effortful processing (Lehrer et al., 1989; Tranel & Damasio, 1994; Zahn et

al., 1999; Zahn & Mirsky, 1999). These results now suggest that the emotional-cognitive deficit is linked specifically to error processing, more than to general cognitive processing.

The SCR amplitude of controls and TBI participants did not differ significantly following the correct withholding of a response on Fixed-Baseline and Fixed-Awareness2, indicating that TBI participants' general psychophysiological responsivity is not generally reduced compared to controls. On Random-Awareness2, controls' SCR to correctly withholding to a target was significantly higher than that of TBI participants. In terms of effortful processing, this may be due to the fact that the Fixed SART is a cognitively simpler, more predictable task (Manly et al., 2003). The Random SART may require a greater cognitive effort to withhold correctly, as the presentation of the no-go target is unpredictable. TBI participants did not show this same increased EDA response on this version of the task, indicating some impaired ability to adjust to increased task demands compared to controls. Again, this is consistent with previous findings which indicate that head injured patients show deficits in EDA responding to meaningful, significant, or demanding situations and stimuli (Zahn et al., 1999; Zahn & Mirsky, 1999).

For TBI participants, there was a significant positive correlation between overall error awareness score and overall EDA amplitude. There was no significant association between overall errors of commission, and amplitude, or errors of commission and error awareness of TBI participants. As error awareness increases, EDA amplitude increases. This indicates that the more aware an individual with TBI is aware of his or her errors overall, the greater EDA emotional response to errors. This suggests that as detection of errors increases, EDA amplitude increases. The

fact that there is no significant correlation between amplitudes and overall errors or between errors of commission and error awareness for TBIs may suggest that even TBI participants who are able to adequately detect errors may show diminished error correction capacities. This indicates that even if they can respond to errors, they may be unable to learn from their mistakes.

Consistent with previous findings (Robertson et al., 1997), control participants' degree of everyday absentmindedness as measured by the CFQ predicted their performance on the SART. There was no association with TBI performance on the SART and their CFQ scores. This may be another indication of their lack of insight into their everyday absentmindedness, as only informant reports of everyday absentmindedness correlated with TBI performance on the SART in Robertson et al (Robertson et al., 1997).

It is noteworthy that there was a significant increase in errors of commission and a decrease in error awareness for both TBI participants and controls on Fixed-Awareness2 where participants were not only asked to report awareness of their errors of commission, but also to say "hold" on correct withholds. This may be due to the monotonous and dull nature of the Fixed SART, combined with the routine of saying "hold" for withholding on presentation of the no-go target. The "hold" may thus become an additional prepotent response to the "3", and participants may attend less well to the withholding response because their attention is divided between a verbal and a motor response. The results indicate that both TBI and controls both detected fewer errors on this condition. However, there was no significant increase in actual errors of commission for controls, whereas TBI participants made a mean of 8.56 errors compared to only 3.88 on Fixed-Awareness1. It was noted that on the

Fixed-Awareness2, some TBI participants began to say "hold" *before* presentation of the 3. They made an error of commission nonetheless and were unaware they had done so. This may suggest a breakdown of error monitoring, even though it was clear that in these situations, TBI patients were aware of the correct response. This is also consistent with the example given by Luria (1980) of patients verbalising the correct responses but continuing to imitate the tester. It may be that this minor extra element of verbalising for a "hold" response taxes the ability to successfully allocate attentional resources.

This study has shown that error detection is impaired in TBI patients. There is also evidence in previous research that even when an error is detected, TBI patients show diminished capacity to correct their error (Hart et al., 1998). It has been suggested by numerous researchers that EDA amplitude may reflect the activity of an appraisal system which incorporates goals (Zimmer, 2000) or that EDA is an index of psychological and emotional response to significance (Zahn et al., 1999). In this study, TBI patients showed an attenuated EDA, possibly due to a diminished emotional response to making an error. It may be that this diminished emotional response affects error correction as well as error recognition mechanisms. Damasio's (1994) "somatic marker hypothesis" proposes that emotional evaluation of an action's consequences guides decision-making. He proposed that the interconnections between the ventromedial regions of the frontal lobes and the limbic structures associated with emotions act as the pathway through which somatic markers, or bodily sensations, mediate cognitive decisions. Damage to the ventromedial cortex may preserve the cognitive evaluations required in decision-making but these evaluations often lack emotional content and can be observed as inappropriate affect or inappropriate responses, typical in patients with frontal lobe

damage (Damasio, 1994). Theoretically, even if patients detect errors, their emotional responses to the significance of committing errors may be reduced. Luria (1980) demonstrated that even when frontal patients verbalised correct responses in a task of field-dependent behaviour, they still imitated the examiner's behaviour. This suggested a breakdown of error-monitoring, despite the knowledge of a correct response remaining intact.

Any strong inferences made from TBI patients must be speculative due to widespread diffuse axonal damage. However, it is widely acknowledged that diffuse axonal damage following TBI can affect the innervations of the prefrontal cortex, even if the injury itself does not impact these regions (Fontaine et al., 1999; Mattson & Levin, 1990; Stuss & Gow, 1992). A speculative link between diminished emotional response and deficits in error processing impairment could be made such that damaged frontal lobes results in lowered capacity to evaluate the emotional significance of an action, as described by Damasio (1994). TBI patients in this study showed a diminished EDA to the emotional significance of detecting an error, and it could be hypothesised that this in turn reduces the probability of that error accessing consciousness.

Future research could investigate further electrophysiological markers of impaired awareness in this patient group, such as ERP markers of aware versus unaware errors in patient and neurologically healthy control groups. Rehabilitation of online monitoring could be examined using such simple laboratory tasks such as used in this study. For example, feedback on error led to improved sustained attention performance on the SART for both TBI and control participants (McAvinue et al.,

2005) and this method of feedback could be examined in relation to improving error awareness performance also. It is also possible that this deficit in EDA responding found in the present study could be used in a type of self-alert training. Recent research with normal healthy adults who were trained to increase their EDA levels on cue has shown some positive findings. Sustained attention levels and arousal levels (as measured by EDA) improved following this self-alert training, using their EDA responses as cues (O'Connell, Bellgrove, Dockree, & Robertson, 2005). This rehabilitative route could be investigated with brain injury patients who show impaired EDA responses to making errors. It would also be beneficial to examine online-monitoring of errors in relation to the more general global awareness measures already used clinically, which is one of the purposes of the study in the next chapter. Furthermore, clinical and neuropsychological backgrounds to the patients in question would be beneficial to help elucidate the cognitive and neural correlates of online monitoring and global awareness, which will also be examined in the next study.

In conclusion, this study identified a specific deficit in error processing following TBI and showed that skin conductance markers of this deficit are correlated with poor overall error awareness. This error awareness deficit may be a significant factor in the observed insight deficits that are common following TBI. This study refines our understanding of the nature of error awareness deficits, a common and lasting consequence of TBI. In terms of promoting the development of better assessment, this study demonstrates a practical, non-intrusive and easy-to-administer clinical method of assessing online error-monitoring and diminished emotional response to error in TBI patients, deficits that are known to be major obstacles to successful rehabilitation.

Chapter 3

Study 2

Awareness of Deficits in Traumatic Brain Injury: A Multidimensional Approach to Assessing Metacognitive Knowledge and Online- Awareness

Abstract

Recent models of impaired awareness in brain injury have drawn a distinction between metacognitive knowledge of difficulties and online awareness of errors and ability to accurately anticipate performance on tasks. This study examines the performance of 31 Traumatic Brain Injury (TBI) participants and 31 controls using a three-strand approach to assessing awareness. Metacognitive Knowledge was assessed with an Awareness Interview and discrepancy scores on three questionnaires. Online Emergent Awareness was assessed using an online error-monitoring task while participants performed simple tasks of sustained attention. Online Anticipatory Awareness was examined using prediction performance tasks on two cognitive tasks. Results indicated that TBI participants with overall low self-awareness (SA) (n=15) were significantly impaired across each of the three levels compared to TBI with High SA (n=16), who performed equally as well as controls on most awareness tasks. The TBI Low SA and High SA group did not differ in terms of severity, chronicity or on standard neuropsychological task performance,

but those with Low SA were more likely to exhibit more disinhibition, interpersonal problems and more difficulties in total competency than TBI with High SA. Sustained Attention abilities were associated with both types of online awareness (emergent and anticipatory). There was a strong relationship between online emergent and online anticipatory awareness, but metacognitive knowledge did not show any such association with the other two measures, consistent with recent theoretical models. This study highlights the absolute necessity in adopting a multidimensional approach to assessing this multifaceted phenomenon.

3.1 Introduction

Impaired awareness of deficits following Traumatic Brain Injury (TBI) has been identified as a significant factor in determining successful outcome, in terms of rehabilitation (Lam et al., 1988), vocational status (Sherer, Bergloff, Levin et al., 1998; Wise et al., 2005), functional independence, maladaptive behaviour (Trudel et al., 1998) and caregiver distress (Prigatano et al., 2005), as described in chapter 1.

Previous research investigating impaired awareness of deficits in TBI has indicated that TBI patients are more accurate at assessing their strengths and limitations for concrete and observable deficits such as physical limitations or difficulties, activities of daily living and memory problems (Fleming & Strong, 1999), whereas impaired awareness of deficits becomes more evident for more abstract or higher level cognitive functions, personality changes, social interactions and emotional control (Fischer, Trexler et al., 2004; Fleming & Strong, 1999; Prigatano, 1996; Prigatano et al., 1990).

As described in Section 1.6, there have been mixed findings in studies examining the neurocognitive correlates of awareness. Severity of injury has shown some relationships with self-awareness deficits (Prigatano, 1999b), but many other studies have not found any associations (Allen & Ruff, 1990; Anderson & Tranel, 1989; Prigatano & Altman, 1990). Other studies found associations in the opposite direction to that predicted (Sawchyn et al., 2005; Sherer et al., 1998), or mixed results depending on which measure of severity was used (GCS or PTA) (Prigatano et al., 1998). Poor performance on intelligence tests has been associated with self-awareness in TBI, with lower post-injury verbal intelligence associated with greater impaired awareness (Anderson & Tranel, 1989). However, this relationship was not found in other studies (Ownsworth et al., 2002; Trudel et al., 1998), and individuals with intact intellectual functioning have also shown deficits in impaired awareness (McGlynn & Schacter, 1989). Executive dysfunction has been associated with ISA in many studies (Allen & Ruff, 1990; Hart et al., 2005; Noe et al., 2005; Ownsworth et al., 2002), but this association was not found, or only partially replicated in others (Bogod et al., 2003; Trudel et al., 1998). In studies that have found this association, the types of executive functioning that have been associated with aspects of awareness include ability to shift-set or form abstract concepts (Noe et al., 2005), planning abilities (Coben et al., 1995), mental flexibility (Coben et al., 1995; Trudel et al., 1998), idea generation or fluency (Coben et al., 1995; Ownsworth & Fleming, 2005), self-regulation and social interaction skills (Bogod et al., 2003; Ownsworth et al., 2002), sustained attention (McAvinue et al., 2005), and reasoning abilities (Malec et al., 1997; Ownsworth et al., 2002). Memory function including WMS-R General Memory Index (Trudel et al., 1998) and delayed recall (Noe et al., 2005; Trudel et

al., 1998) have also been identified in some studies as a significant predictor variable for self-awareness.

As mentioned in Chapter 1, functional or clinically-based models have been developed in an attempt to characterise this complex phenomenon and to guide rehabilitation. At a functional level, Crosson et al (1989) divided awareness into *intellectual awareness*, which represents a patient's ability to recognise his/her deficits or impaired functioning, *emergent awareness*, which represents a patient's ability to describe their difficulties as they are happening, and *anticipatory awareness*, which represents a patient's ability to predict when difficulties will arise because of their deficits. The hierarchical structure of this model has been criticised (Abreu et al., 2001; Toglia & Kirk, 2000), and more interactional models have differentiated between *metacognitive knowledge* or declarative knowledge about one's abilities, which would incorporate elements of intellectual awareness and *online monitoring* of performance during tasks, which relates to emergent awareness and anticipatory awareness (Toglia & Kirk, 2000).

There is little consistency in the literature regarding measures of awareness, with a wide variety of methods adopted and some controversy regarding the best approach (Fleming et al., 1996; Hart & Sherer, 2005; Wise et al., 2005). Methods include comparing self-ratings to significant other or clinician ratings, such as the PCRS (Fleming et al., 1996; Prigatano et al., 1986; Sherer et al., 1998), interviews (Fleming et al., 1996; Ownsworth et al., 2000), observation of actual task performance (Hart et al., 1998), comparison of patients' reports to neuropsychological performance (Allen & Ruff, 1990; Anderson & Tranel, 1989), comparison between patients' predictions of performance and actual performance (Gauggel, Hoop, & Werner, 2002;Fischer,

Trexler et al., 2004) and more recently online-monitoring of errors (McAvinue et al., 2005; O'Keeffe, Dockree, & Robertson, 2004). A number of researchers have highlighted the importance of using a number of different measures to assess the multi-faceted concept of self-awareness of deficits (Fischer, Trexler et al., 2004; Fleming et al., 1996). Ownsworth et al (In Press) point out that the range of different indicators of awareness may not capture the complexity of the construct and Markova and Berrios (2001) suggest that each assessment approach examines a different "object of insight" and that unawareness therefore varies according to assessment type. The numerous inconsistencies in the literature regarding neuroanotomical, neurocognitive, and clinical correlates of awareness may be explained by the fact that conclusions are frequently made based on limited perspectives on unawareness derived from assessment of a single specific "object of insight" (Ownsworth et al., In Press). A richer, multidimensional approach to assessment of awareness has been called for recently, that can inform theoretical models, develop clinical methods of assessment, develop tasks suitable for neuroimaging studies of self-reflection and awareness, and guide rehabilitation of this complex phenomenon (Fischer, Trexler et al., 2004; Ownsworth et al., In Press; Prigatano, 1996; Sherer et al., 2005; Toglia & Kirk, 2000).

The goals of the present study were to incorporate Toglia and Kirk's (2000) model into into a comprehensive, multidimensional approach to assessing awareness in Traumatic Brain Injury. Therefore, awareness was assessed in terms of metacognitive knowledge (intellectual awareness) and online awareness (emergent and anticipatory awareness). Furthermore, it was hoped to clarify which neuropsychological abilities are associated with awareness based on a detailed neuropsychological examination of each participant.

3.2 Method

Participants

Thirty-one adults with traumatic brain injury (TBI) took part in this study. Exclusion criteria for all participants included a pre-trauma history of epilepsy or other neurological condition, a history of major psychiatric disorder, or a history of drug or alcohol problems. Thirty-one neurologically healthy control participants were also recruited. Controls had the additional exclusion criterion that they had never suffered a loss of consciousness from a head injury. The local research ethics committee and hospital ethics committee approved the study and all participants gave informed consent prior to participation.

TBI participants were recruited from the National Rehabilitation Hospital, Dun Laoghaire, Ireland. The mean age of the TBI group was 28.74 (SD 8.52). This group included 27 men and 4 women. The mean length of time from injury to the time of testing was 36.25 months (SD 22.37). Injury severity was measured by post-traumatic amnesia (PTA) duration and Glasgow Coma Scale (GCS), where available. Clinical data for each TBI participant, where available, are displayed on Table 3.1. The comparison control group consisted of 24 men and 7 women, selected to match the TBI group on sex, age and education-level variables.

Table 3.1: Clinical Data for the TBI Patients

TBI Patient	TBI cause	TBI severity (PTA)	TBI severity (GCS)	Months since TBI	Location of damage
BE	Assault	Very Severe	3	11	Left temporal region, cerebellar haemorrhage (CT)
OW	RTA-Motorbike	N/A	5	23	Frontal bilaterally, Basal Ganglia, temporal right (CT)
AMB	Fall	Extremely Severe	9	31	Right subdural haemotoma, right frontal (CT)
DD	RTA- Car	Very Severe	11	34	Normal (CT)
FL	RTA- Car	Extremely Severe	5	38	SAH, left frontal, left superior temporal, left occipital (CT)
CMCG	RTA-Motorbike	Extremely Severe	3	38	Left frontoparietal (CT)
ER	Fall	Very Severe	7	21	Right frontal, SAH (CT)
JC	RTA-Motorbike	Very Severe	8	25	Left fronto-temporal haemotoma (CT)
MOM	Assault	Very Severe	13	19	N/A
RB	RTA- Car	Extremely Severe	7	9	Right frontal, left occipital, DAI (CT)
GC	Fall	Very Severe	3	44	Right frontal ICH(CT)
JC	RTA- Car	Extremely Severe	12	34	Normal DAI (CT)
LM	Assault	Severe	5	76	Right fronto-parietal, temporal (CT)
RC	RTA-Pedestrian	Extremely Severe	8	29	SAH, left occipital(CT)
BF	Fall	Extremely	3	13	SAH, right temporal, left parietal (CT)

		Severe			
FMQ	RTA-Motorbike	Extremely Severe	7	18	Left frontal, right posterior parietal, temporal, occipital (CT)
ER	RTA-Bicycle	Very Severe	14	28	Right temporal contusions (CT)
GM	RTA- Car	Very Severe	N/A	28	Right fronto-parietal, DAI (CT)
AC	RTA- Car	Extremely Severe	3	32	Frontal cyst, otherwise normal CT (CT)
GL	Fall	Severe	N/A	39	Right frontal (MRI)
DF	RTA-Car	Extremely Severe	N/A	N/A	N/A
PJD	Assault	Very Severe	3	53	Left frontal, left subdural haemorrhage (CT)
JG	N/A	Severe	N/A	N/A	N/A
CL	RTA- Car	Extremely Severe	8	67	SAH, right occipital, right hemisphere (CT)
COD	RTA- Car	Extremely Severe	N/A	97	Bilateral frontal, Left occipital(CT)
GH	RTA- Car	Very Severe	5	28	Frontal Lobe, SAH, parietal, occipital (CT)
JK	Work Accident	Mild	N/A	N/A	N/A
ER	RTA- Car	Very Severe	4	58	Bilateral frontal, SAH, DAI (CT)
DP	RTA-Motorbike	Very Severe	3	9	Right frontal lobe, Normal CT(CT)
GB	RTA- Car	Mild	N/A	84	DAI (MRI)
JG	RTA-Motorbike	Extremely Severe	6	29	SAH, bilateral temporal (CT)

PTA: Post Traumatic Amnesia (Mild: <1 Hour; Moderate: 1-24 hours; Severe: 1-7 days; Very Severe: 7-28 days; Extremely Severe: >28 days); RTA: Road Traffic Accident;DAI: Diffuse Axonal Injury;SAH: Subarachnoid Haemorrhage; ICH: Intracerebral Haemorrhage; N/A: Not Available; CT: Computerised Tomography; MRI: Magnetic Resonance Imaging

Demographic details of TBI and controls are displayed on Table 3.2.

Table 3.2: Demographic Details of TBI and Control Participants

	TBI	**Controls**
N	31	31
Sex (m, fm)	27, 4	24, 7
Age (Years)	28.74 (SD 8.52)	30.23 (SD 14.08)
Education Level[1]	5, 10, 15	5, 13, 13

[1]Education Level: <12 yrs: School-Leaving Qualification: >16 yrs/Further Education

Materials and Procedure

Screening Tests

A number of short screening tests for cognitive functioning were administered throughout both sessions. These included:

National Adult Reading Test (NART) (Nelson, 1982; Nelson & Willison, 1991)

In this measure, participants read a graded series of 50 irregularly spelled words to give an estimate of pre-morbid intelligence.

Hospital Anxiety and Depression Scale (HADS) (Zigmond & Snaith, 1983)

The Hospital Anxiety and Depression Scale is a scale comprised of 14 items. Participants chose one of four responses which reflect how they have been feeling in the past week. Seven statements reflect anxiety levels and seven relate to depression levels.

Frenchay Aphasia Screening Test (FAST) (Enderby, Wood, & Wade, 1987; Enderby, Wood, Wade, & Hewer, 1987)

This screening test is designed to cover the four major aspects of language that may be disturbed in a patient with aphasia: comprehension, expression, reading and writing.

Frontal Assessment Battery (FAB) (Dubois, Slachevsky, Litvan, & Pillon, 2000)

This test reflects executive dysfunction, independent of global cognitive deficit. It involves 6 subtests of frontal function, including conceptualisation, mental flexibility, motor programming, sensitivity to interference, inhibitory control, and environmental autonomy.

Neuropsychological Measures

Each participant underwent a comprehensive neuropsychological examination that included the following tests.

Memory

Subtests from the Wechsler Memory Scale (WMS 111) (Wechsler, Wycherly, Benjamin, Crawford, & Mockler, 1998) included: Orientation,

Immediate and Delayed Story Recall (Logical Memory 1 and 11), Digit Span (Forward and Backward), Spatial Span (Forward and Backward).

Prospective Memory Task (Walker, 2003)

Prospective memory function was measured using an event-based computerized task. Participants carried out a sentence verification task in which they indicated whether a sentence was true ('bishops drink tea') or false ('dogs have wheels'). Embedded within 6 sentences was a target word ('hand') that served as a prospective cue for subjects to press the nominated key. Number of 'hits' was taken as a measure of prospective memory performance.

Attention

Subtests from Test of Everyday Attention (Robertson, Ward, Ridgeway, & Nimmo-Smith, 1996)

Selective attention and attention switching were examined using Telephone Search and the Telephone Search While Counting.

Sustained Attention to Response Task (Robertson et al., 1997)

Participants undertook three tasks of sustained attention: the Fixed SART, Random SART and the DART, a dual task variant of the Fixed SART. Each condition was programmed using DMDX software[1]. Each digit was displayed for 580ms followed by an inter-stimulus-interval (ISI) of 920ms. The period from digit onset to digit onset was 1500ms. Digits were presented in white, 0.25° above a central white

[1] DMDX display software, The University of Arizona.

fixation cross on a black background. Five randomly allocated digit sizes were presented to increase the demands for processing the numerical value and to minimize the possibility that subjects would set a search template for an unspecified perceptual feature of the no-go trial ('3'). Digit sizes were 8mm, 11mm, 14mm, 17mm and 20mm respectively. The screen (320mm x 240mm: Dell PC) was positioned approximately 40cm from the participant.

Fixed SART (Manly et al., 2003)

In the Fixed SART digits were presented sequentially from 1 through 9. For each block, 225 digits were presented (repetitions of the 1-9 sequence) over a 6.3 minute period. The participant was instructed to respond with a left mouse press following each digit (go-trials) with the exception of the 25 occasions when the digit 3 (no-go target) appeared.

Random SART (Robertson et al., 1997)

In the Random SART digits were presented in a pseudorandom order. For each block, 225 digits were presented over a 6.3 minute period. The participant was instructed to respond with a left mouse press following each digit (go-trials) with the exception of the 25 occasions when the digit 3 (no-go target) appeared.

DART (Dockree et al., In Press)

In the DART, a secondary continuous performance task was embedded within the basic design of the Fixed SART. In addition to pressing the left mouse button to go-trials and withholding a response to no-go targets ('3s'), participants were also required to make a right button press upon detection of grey-coloured digits. In order to challenge available processing resources during the task but not to challenge

performance in the period before and immediately after the presentation of a target (during the presentation of 1 through 4), the presentation of grey-coloured digits was restricted to numbers 5 through 9. Grey digits were created in Microsoft Paint with a luminance of 120 and a hue of 160. 225 digits were presented in total, including 179 go-trials, 25 targets and 21 greys. All other aspects of the test, namely the trial duration, ITI, stimulus size and software specifications were identical to the Fixed SART.

Executive Functions

Verbal Fluency- FAS test (Spreen & Benton, 1969; Spreen & Strauss, 1998)

Participants were given each letter, F, A, S, and asked to produce as many words as they could in one minute for each letter.

Revised Strategy Application Task (R-SAT) (Levine, Dawson, Boutet, Schwartz, & Stuss, 2000)

The revised Strategy Application Test (R-SAT) was administered to measure planning/strategy performance. The task presents an unstructured environment in a laboratory setting whereby the most efficient strategy can be broken by salient external cues and internal habits. The optimal strategy involves completing the briefest items in three separate activities: figure tracing, sentence copying and object numbering. The primary score reflecting strategy application is the proportion of brief items from all three tasks that are completed.

Self-Report Measures: General Competency, Frontal Symptoms, Everyday Absentmindedness and Empathic Tendency.

Patient Competency Rating Scale (PCRS) (Prigatano & Altman, 1990)

The PCRS is a 30-item self-report instrument which asks the participant to use a 5-point Likert scale to rate his or her degree of difficulty on a variety of tasks and functions, where a rating of 5 represents that the participant rates the item as "can do with ease" and a rating of 1 represents that the participant rates the item as "can't do". The PCRS items are divided into a number of different domains. These include activities of daily living and physical function, behavioural and emotional function, interpersonal skills and cognitive abilities, based on subscale divisions used in Leathem et al (1998).

Frontal Systems Behavioural Scale (FrSBe) (Grace & Malloy, 2002)

The FrSBe is a 46-item rating scale that is designed to measure behaviours associated with damage to the fronto-subcortical systems brain networks. Items relating to apathy, disinhibition and executive dysfunction are rated on a 5-point Likert scale (1=almost never, 5=almost always) for pre-morbid behaviour and current behaviour.

Cognitive Failures Questionnaire (CFQ) (Broadbent et al., 1982)

The Cognitive Failures Questionnaire (CFQ) measures failures in perception, memory and motor-function that have occurred in the last 6 months. The frequency of these mistakes occurring is rated on a five-point scale ranging from "never" to "very often".

Measure of Empathic Tendency (MET) (Mehrabian & Epstein, 1972)

The Measure of Empathic Tendency is a measure of emotional empathy. It consists of 33 statements. Participants rate their agreement on a scale ranging from +4 (very strong agreement) to -4 (very strong disagreement).

Awareness Measures

Metacognitive Knowledge

Awareness Interview- Adapted (Anderson & Tranel, 1989)

The interview schedule for this study is found in the appendices. The standard interview was administered prior to each participant's neuropsychological evaluation. For this study, the standardised procedure comprised 8 sections, consisting of questions regarding diagnosis/reason for hospitalisation, if any, thinking, orientation, memory, attention, speech and language, activities of daily living (ADLs), and overall impression as to how they felt they had performed on the tests and ability to continue with/return to work, education or hobbies. Scoring is carried out in 3 stages, culminating in an "Awareness Index" as described in Anderson & Tranel (1989). In the first stage, participants' verbal responses are scored on a 3-point scale, structured such that a score of 3 indicated that the participant did not report any problem in that area, a score of 2 indicated that the participant reported a mild to moderate impairment and a score of 1 indicated that the participant reported major impairments or significant problems in that area. The second stage involved an independent rater, blind to the participant's responses on their Awareness Interview, coding each participant's neuropsychological performance on a comparable 3-point scale. The primary criterion for each score was

based on deviation from control means from the neuropsychological battery of tests. The third stage involved comparing each subject's self-report on the Awareness Interview with the ratings on their neuropsychological tests. This led to the deviation scores ranging from 0-2 for each item (Reason for Hospitalisation (if any), Thinking, Orientation, Memory, Attention, Language, Activities of Daily Living, Overall Performance and Ability to Return to Work). The eight deviation scores were then added together to get a total deviation score, the "Awareness Index", ranging from 0 (indicating no discrepancy between participant's self-report and neuropsychological performance) to 16 (indicating maximum discrepancy between participant's self-report and neuropsychological performance).

Discrepancy Scores on Questionnaires Self and Other

Three questionnaires were rated for identical items by participants and a significant other, caregiver or friend. These were the Patient Competency Rating Scale (PCRS) (Prigatano et al., 1986), the Frontal Systems Behavioural Scale (FrSBe) (Grace & Malloy, 2002), and the Cognitive Failures Questionnaire (CFQ) (Broadbent et al., 1982). Discrepancy between self-ratings and significant other ratings are used as indicators of inaccurate perception of functional competencies, cognitive, behavioural and social abilities.

Online Emergent Awareness

Error-monitoring

Error-Monitoring on Fixed SART, Random SART and DART (McAvinue et al, 2005; O'Keeffe et al., 2004)

Similar to the method used in Chapter 2, as participants performed the three conditions of the SART, error awareness was measured by asking participants to verbally indicate their awareness of making an error by saying the word "hit" following an error of commission on the no-go target, the number 3. The proportion of aware errors over the total number of errors made gave the overall error awareness score on the SART.

Online Anticipatory Awareness

Prediction Tasks: Discrepancy between predicted and actual performance in cognitive tasks (Fischer, Trexler et al., 2004; Marcel et al., 2004)

Accuracy of prediction on cognitive tasks was assessed using WMS Digit Span (Forwards) and Verbal Fluency (FAS). Participants were asked prior to performing each of these tasks to predict how many numbers they would immediately recall (Digit Span), or words they could come up with in one minute beginning with a certain letter (Verbal Fluency). Prediction accuracy was assessed in each task by calculating an average score [(Prediction minus Performance)/Prediction X 100].

3.3 Results

Section A: Demographic Data

Demographic data and scores of self-report measures for all TBI patients and control participants are detailed in Table 3.2. Groups did not differ significantly in terms of

age [t= -.502, df= 60, p >.05], sex [χ^2 = .995, df= 1, p>.05] or education level [χ^2 = .625, df= 2, p>.05].

Section B: Do TBI participants differ from Control participants overall on Awareness Measures? TBI v Control Group Comparisons

Composite scores for Metacognitive Knowledge (Awareness Index, PCRS discrepancy score, FrSBe discrepancy score and CFQ discrepancy score), Online Emergent Awareness (Overall Error Awareness on Fixed SART, Random SART, and DART) and Online Anticipatory Awareness (Overall Prediction Tasks Accuracy on Digit Span and Verbal Fluency) were calculated based on z-scores from each of the individual awareness measures.

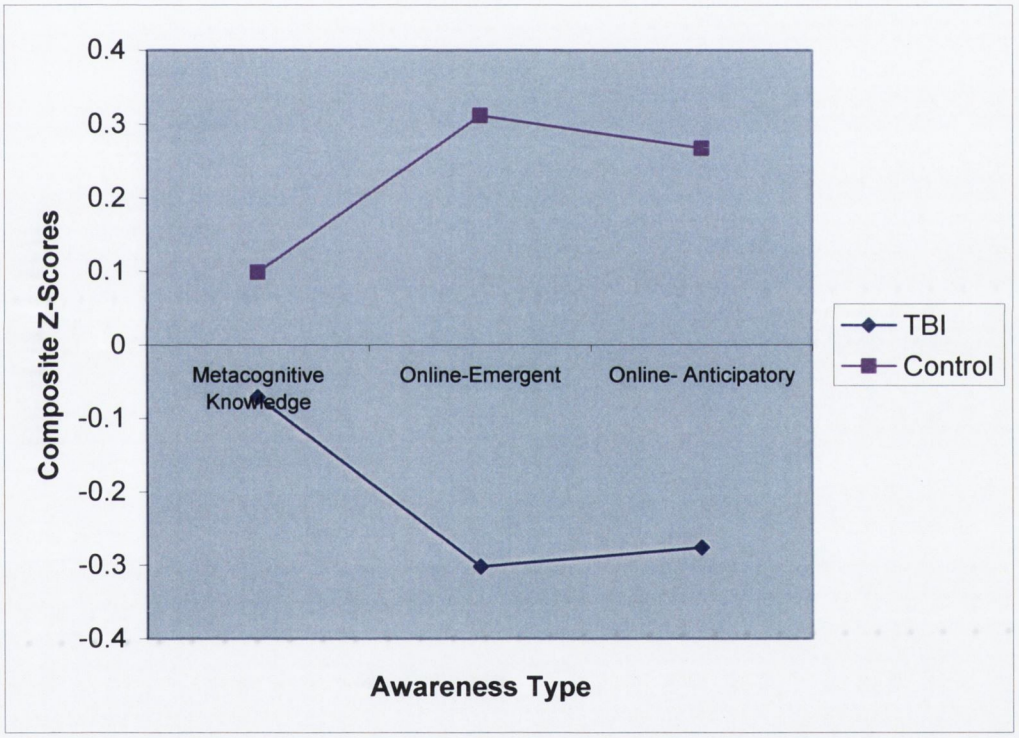
A composite score of Overall Awareness, including z-scores from the metacognitive knowledge tests, online emergent awareness and online anticipatory awareness was calculated [z-scores of all awareness measures: Awareness Interview, discrepancy scores on PCRS, FrSBe, CFQ, online monitoring on SART and Prediction Tasks].

An independent-samples t-test revealed that TBIs scored significantly worse than Controls on Overall Awareness Composite score [t(53.9)= -2.870 p<.01]. The Overall Awareness Composite Scores for TBI and Control Participants are depicted below in Figure 3.1.

In order to investigate performance across the different awareness measures, a mixed ANOVA was run. The between- subjects factor was Group (Two levels: TBI and Control) and the within-subjects factor was Awareness Type (Three levels:

Metacognitive Knowledge, Online Emergent Awareness, Online Anticipatory Awareness). There was a significant main effect of Group [$F(1, 57)= 8.903$, $p<.01$], indicating that TBI participants were significantly impaired across each of the three awareness measures compared to controls. The main effect for Awareness Type [$F(2, 114)= 0.10$, $p>.05$] and the interaction effect between Awareness Type and Group [$F(4, 114) = 1.576$, $p>.05$] did not reach significance. Figure 3.1 below depicts Awareness Type by Group for TBIs and Control participants.

Figure 3.1: Awareness Type by Group for TBI and Control Participants.



Section C: Do TBI Participants with High and Low Awareness groups differ across awareness measures? TBI High Awareness v TBI Low Awareness v Control Comparison by Awareness Type

Following an exploration of raw awareness scores of the TBI data, it became clear that the TBI participant group was not normally distributed across several tests. The

histograms and normality statistics are presented in Figure 3.2. The TBI group was therefore divided into a High Self-Awareness (TBI High SA) Group and a Low Self-Awareness Group (TBI Low SA), based on a median split of the Composite Overall Awareness score. Further analysis of the awareness types was thus performed on 16 TBI High SA, 15 TBI Low SA and 31 Controls. These groups did not differ in terms of age [$F(2, 59) = 0.683$, $p>.05$], sex [$\chi^2 = 1.769$, $df = 2$, $p>.05$], or education level [$\chi^2 = 0.66$, $df = 4$, $p> .05$]. Table 3.3 displays clinical data for TBI High SA and TBI Low SA groups. TBI High SA and TBI Low SA groups did not differ in terms of Time Since Injury [$t(26)=-.488$, $p>.05$] or severity of injury, as measured by PTA [$U=-1.010$, $n_1=15$, $n_2=15$, $p>.05$] or GCS [$U=-1.031$, $n_1=13$, $n_2=11$, $p>.05$].

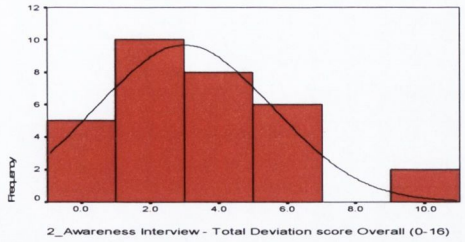
Table 3.3: Clinical Data for TBI High SA and TBI Low SA groups

	TBI High SA	**TBI Low SA**
Time Since Injury (Months)	34.44 (SD 21.3)	38.67 (SD 24.5)
Severity of Injury (PTA):	4.00 (SD 1.1)	4.27 ((SD 1.1)
Severity of Injury (GCS):	6.23 (SD 4.2)	6.73 (SD 2.3)
Neuroradiological Data[1]:		
Right Frontal	6	4
Left Frontal	3	3
Bifrontal	3	4
Diffuse Axonal Injury/Unspecified	4	4

Note 1: Statistical analysis (chi-square) could not be performed on Neuroradiological Data due to cells not reaching minimum expected frequency.

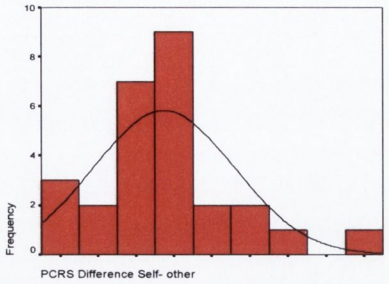
Figure 3.2: Histograms for each Awareness measure for TBI participants and normality tests

Fig 3.2a Awareness Index Scores TBI participants



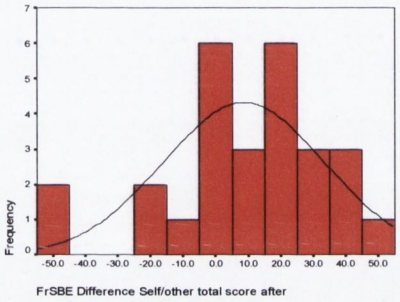
Kolmogorov-Smirnov = .172, p<.05, not normal

Fig 3.2b PCRS Discrepancy Scores TBI participants



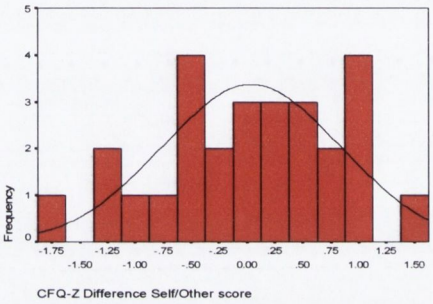
Kolmogorov-Smirnov = .147, p=.1

Fig 3.2c FrSBe Discrepancy Scores TBI participants



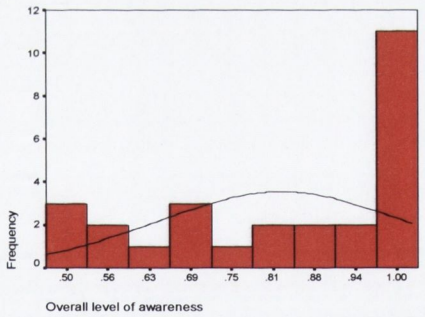
Kolmogorov-Smirnov = .121, p=.2

Fig 3.2d CFQ Discrepancy Scores TBI participants



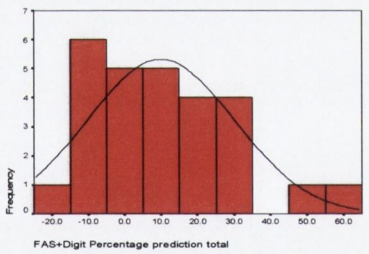
Kolmogorov-Smirnov = .096, p=.2

Fig 3.2e Error-Awareness Scores TBI participants



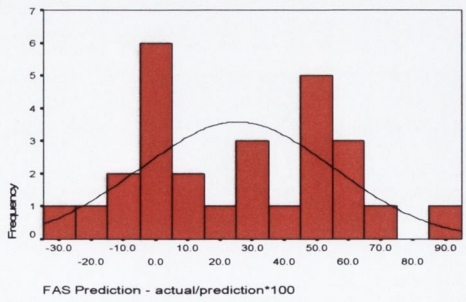
Kolmogorov-Smirnov = .230, p<.01, not normal

Fig 3.2f Prediction Scores TBI participants



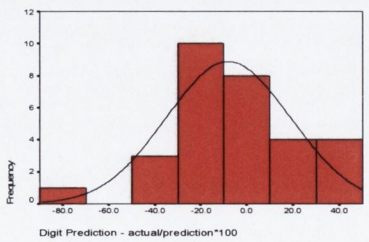
Kolmogorov-Smirnov = .090, p=.2

Fig 3.2g FAS Prediction Scores TBI participants



Kolmogorov-Smirnov = .145, p=.1

Fig 3.2h Digit Prediction Scores TBI participants



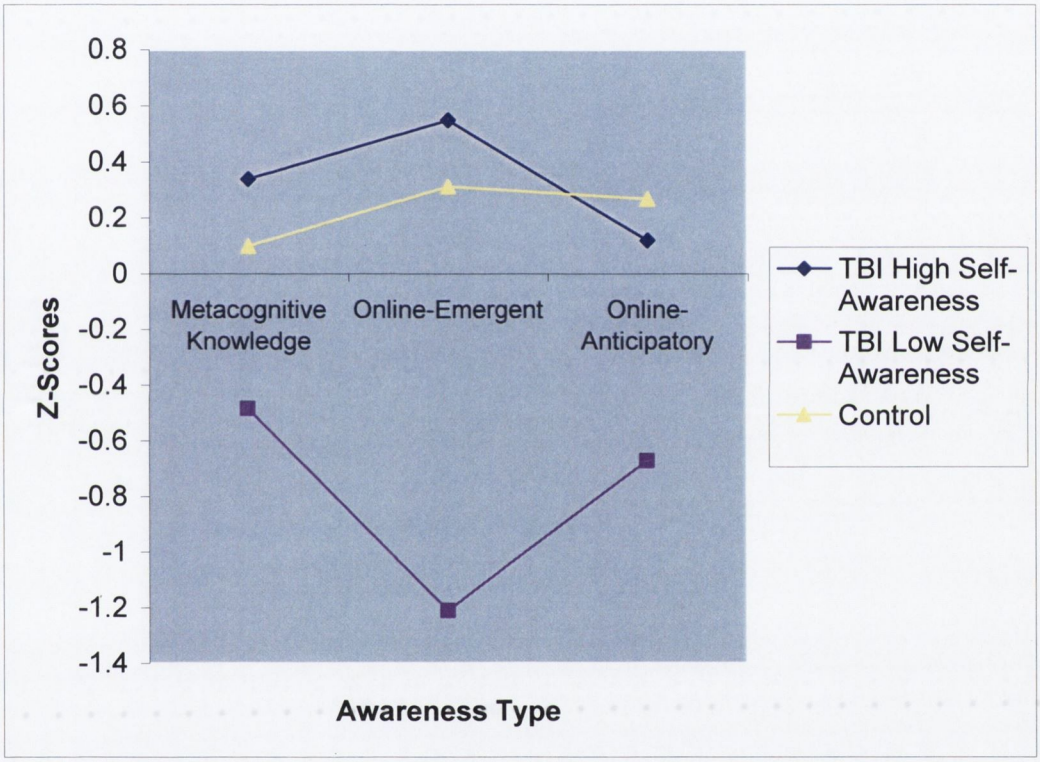
Kolmogorov-Smirnov = .139, p=.1

In order to investigate performance across the different awareness measures, a mixed ANOVA was run. The between- subjects factor was Group (Three levels: TBI High SA, TBI Low SA, and Control) and the within-subjects factor was Awareness Type (Three levels: Metacognitive Knowledge, Online Emergent Awareness, Online Anticipatory Awareness). There was a significant main effect of Group [$F(1, 56)= 29.515$, $p<.0001$], and a significant interaction between Awareness Type and Group [$F(4, 112) = 2.866$]. The main effect for Awareness Type did not reach significance [$F(2, 112)= 0.443$].

Post hoc t tests (independent and paired-samples) indicated that TBI Low SA had significantly lower Metacognitive Knowledge composite score compared to both TBI High SA [$t(28)=-2.868$, $p<.01$] and Controls[$t(18.698)=-2.577$, $p<.05$]. TBI Low SA had significantly lower Online-emergent composite scores than both TBI High SA [$t(17.827)=-6.597$, $p<.001$] and Controls [$t(43)=-5.935$, $p<.001$], and significantly lower Online-Anticipatory composite scores than both TBI High SA [$t(28)=-2.771$, $p<.01$] and Controls [$t(44)=-3.022$, $p<.01$]. TBI High SA and Controls did not differ significantly on Metacognitive Knowledge [$t(19.357)=1.129$, $p>.05$], Online-Emergent [$t(43.983)=1.467$, $p>.05$] or Online- Anticipatory [$t(44)=-.484$, $p>.05$]. These post hoc tests also indicated that TBI Low SA performed significantly worse on Online-Emergent than Online-Anticipatory [$t(14)=2.271$, $p<.05$]. There was no significant differences in their composite scores for Metacognitive Knowledge and Online-Anticipatory [$t(14)=0.542$, $p>.05$]. The difference between Metacognitive Knowledge and Online-Emergent showed a trend towards significance [$t(14)=2.020$, $p=.06$]. There were no significant differences for TBI High SA between scores for Metacognitive Knowledge and Online-Emergent [$t(14)=-1.087$, $p>.05$], or between Metacognitive Knowledge and Online-Anticipatory [$t(13)=1.187$, $p>.05$], or between

Online-Emergent and Online- Anticipatory [t(14)=-1.903, p>.05]. Controls did not differ in their performance between the three awareness types: Metacognitive Knowledge and Online-Emergent [t(29)=-1.155, p>.05], or between Metacognitive Knowledge and Online-Anticipatory [t(30)=-0.821, p>.05], or between Online-Emergent and Online-Anticipatory [t(29)=-0.331, p>.05]. Figure 3.3 below depicts this interaction with the three groups and three awareness types.

Figure 3.3: Interaction between TBI High SA, TBI Low SA and Control Group on three composite awareness scores.



Section D: Do TBI High and Low Awareness groups differ across neuropsychological measures?

In order to investigate whether the TBI High SA and TBI Low SA groups differed across performance on neuropsychological tests, a series of one-way ANOVAs were run, with Group (TBI High SA, TBI Low SA and Controls) as between-subjects

factor and each neuropsychological test as the dependent variable. Table 3.4 displays the mean scores and standard deviations for each test. F-values and p-values are also displayed, as are post-hoc bonferroni comparisons to indicate where differences lie.

As can be seen from Table 3.4, TBI High SA and TBI Low SA groups do not differ significantly from each other on any of the neuropsychological measures. Post hoc comparisons do indicate a trend towards significance between TBI High SA and TBI Low SA on DART mean errors of commission (p =.056). Post hoc comparisons indicated that neither TBI group differed significantly from Controls on the Dual Task subtest of Test of Everyday Attention, the Revised-Strategy Applications Task or the Measure of Emotional Empathy. TBI Low SA group differed from Controls on most other measures, with the exception of aphasia screening test. TBI High SA group did not differ from Controls on NART pre-morbid IQ, HADS anxiety, WMS orientation, Test of Everyday Attention Part A or Part B, Fixed SART, DART and Frontal Assessment Battery.

Table 3.4: Neuropsychological Test Scores for Each Participant Group

Cognitive Functions	*Cognitive Tests*	**TBI Low SA (n = 15)**	**TBI High SA (n= 16)**	**Controls (n= 31)**	**F Value**	***P* Value**
Screening Tests Mean (SD)	**NART Estimated IQ**	99.54 (10.1)[a]	107.20 (11.7)	113.58 (8.4)[a]	9.969	<.001
	HADS- Anxiety	11.0 (4.6)[a]	8.31 (4.6)	6.29 (3.5)[a]	6.934	<.01
	HADS- Depression	8.13 (4.9)[a]	7.75 (4.4)[b]	2.35 (1.8)[ab]	19.398	<.001
	FAST- Aphasia	28.14 (1.3)	28.00 (2.8)[b]	29.29 (0.8)[b]	4.389	<.05
	WMS Orientation	12.29 (1.2)[a]	12.80 (0.9)	13.29 (0.7)[a]	6.30	<.01
	MET	18.27 (34.5)	18.56 (34.5)	37.87 (18.4)	3.774	<.05
Memory Mean (SD)	**WMS Logical Memory I**	36.[illegible][a]	40.93 (10.1)[b]	53.03 (10.5)[ab]	13.694	<.001
	WMS Logical Memory II	21.[illegible] 212	23.21 (10.1)[b]	32.06 (8.2)[ab]	8.947	<.001
	WMS Digit Span Forward	10.[illegible]	11.4 (2.3)[b]	13.19 (1.6)[ab]	14.05	<.001
	Prospective Memory Test	2.73 (2.7)[a]	2.64 (2.7)[b]	5.23 (1.4)[ab]	9.734	<.001
Attention Mean (SD)	**TEA- A**	4.41 (1.6)[a]	3.52 (0.9)	3.03 (0.9)[a]	7.451	<.001
	TEA- B	6.55 (2.9)[a]	6.17 (0.8)	4.27(2.0)[a]	4.780	<.05
	TEA- Dual Task Decrement	2.16 (2.6)	2.55 (3.4)	1.26 (1.5)	1.745	n.s.
	$SART_{fixed}$ Mean Errors of Commision	2.43 (2.8)[a]	1.8 (0.9)	0.94 (0.9)[a]	4.719	<.05
	$SART_{random}$ Mean Errors of Commission	8.61 (3.6)[a]	6.14 (4.8)[b]	2.21 (2.4)[ab]	16.351	<.001
	DART Mean Errors of Commission	6.5 (4.3)[a]	3.96 (2.4)	2.27 (1.6)[a]	12.066	<.001
Executive Functions Mean (SD)	**R-SAT Proportion**	0.736 (0.1)	0.738 (0.1)	0.844 (0.2)	3.713	<.05
	Verbal Fluency FAS (Total)	31.2 (9.1)[a]	36.0 (10.2)[b]	50.74 (13.8)[ab]	16.26	<.001
	FAB	15.13 (1.9)[a]	16.2 (1.9)	17.23 (1.1)[a]	9.084	<.001

NART= National Adult Reading Test Estimated Pre-Morbid Performance IQ; HADS= Hospital Anxiety and Depression Scale; FAST= Frenchay Aphasia Screening Test; WMS= Wechsler Memory Scale; MET= Measure of Empathic Tendency; TEA= Test of Everyday Attention; SART= Sustained Attention to Response Task; DART= Dual-Task Attention To Response Task; R-SAT= Revised Strategy Application Task; FAB= Frontal Assessment Battery

Post-hoc significant comparisons are indicated by the following symbols:

a: Post hoc bonferroni comparison (P<.05): TBI Low SA vs Controls

b: Post hoc bonferroni comparison (P<.05): TBI High SA vs Controls

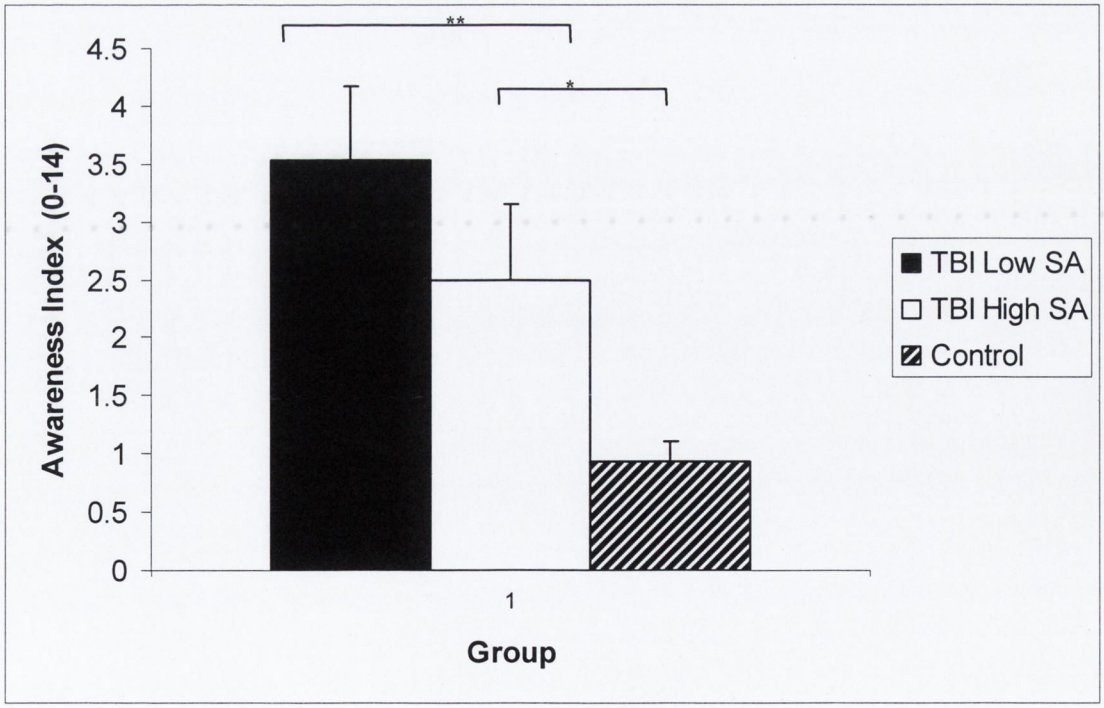
Section E: How do TBI Low SA, TBI High SA and Controls differ on specific awareness measures?

Metacognitive Knowledge

1) Awareness Interview- Awareness Index

A one-way between-groups ANOVA was performed to compare the overall Awareness Index scores for TBI High SA, TBI Low SA and Controls. There was a statistically significant effect of group on the Awareness Index [$F(2, 58)=9.871$, $p<.0001$]. Post hoc bonferroni comparisons indicated that the Awareness Index Mean Score for both TBI Low SA (3.53, SD 2.4) and TBI High SA (2.5, SD 2.6) groups differed significantly from Control participants (0.93, SD 0.9). Figure 3.4 below depicts the overall Awareness Index for each group.

Figure 3.4: Awareness Interview Awareness Index (0-16) for each participant



* Significant at $p<.05$ ** Significant at $p<.01$

The mean deviation scores of the eight categories in the Awareness Interview that contributed to the Overall Awareness Index were then compared between the groups. One-way between subjects ANOVAs were performed. Only the categories of Deviation on Activities of Daily Living and Deviation on overall performance reached statistical significance. [Diagnosis: $F(1, 29) = 3.174$, $p>.05$; Thinking: $F(2, 58)= 2.806$, $p>.05$; Orientation: $F(2, 56) = 2.858$, $p>.05$; Memory: $F(2, 56) = 0.79$, $p>.05$; Attention: $F(2, 55) = 2.149$, $p>.05$; Language: $F(2, 58) = 2.754$, $p>.05$; Activities of Daily Living: $F(2, 54)= 3.855$, $p<.05$; Overall Performance: $F(2, 56) = 9.478$, $p<.001$].

Post hoc bonferroni comparisons indicated that group differences did not reach statistical significance for Deviation on Activities of Daily Living. In the Deviation on Overall Performance category, (Overall Performance on Testing sessions and ability to return to /continue with normal activities), mean deviation scores for TBI Low SA (.533, SD 0.6) and TBI High SA (.333, SD .5) both differed significantly from Controls (0.0).

2) Patient Competency Rating Scale (PCRS)

Mean scores and standard deviations for self-rated and other- rated PCRS are presented in Table 3.5 below for TBI Low SA, TBI High SA and Controls for Total Competency, Activities of Daily Living, Cognitive, Interpersonal and Emotional Subscales. One-way between-subjects' ANOVAs were conducted in order to establish group differences in mean scores on PCRS self-rated scale (Total and subscale) and PCRS other-rated scale (Total and subscales). F values and post hoc bonferroni comparisons are also displayed in Table 3.5. As can be seen from Table 3.5, TBI Low SA are rated significantly lower on competency total and subscales by Significant Others (SOs) compared to TBI High SA on Total Competency, Activites of Daily Living and Interpersonal Relationships Scales. TBI Low SA also rate themselves significantly lower than do controls for competency on all self-rated scales, with the exception of ADL. TBI High SA rate

themselves significantly lower on Total competency, ADL and cognitive subscales compared to Controls' self-ratings.

Table 3.5: Patient Competency Rating Scale (Self- and Other-Rated) Mean Scores and Standard Deviations for Each Group

PCRS Mean (SD)	**TBI Low SA**	**TBI High SA**	**Controls**	**F Value**
PCRS Self Rated				
Total	109.53 (19.6)[a]	111.87 (21.7)[b]	126.19 (10.0)[ab]	7.142**
ADL Self-rated	33.06 (5.2)	32.53 (5.9)[b]	36.16 (2.8)[b]	4.512*
Cognitive Self-rated	28.8 (5.7)[a]	28.2 (5.9)[b]	33.38 (4.0)[ab]	7.326**
Interpersonal Self-rated	24.33 (6.3)[a]	26.73 (5.4)	29.45 (3.3)[a]	6.185**
Emotional Self-rated	23.33 (4.9)[a]	24.13 (6.8)	27.19 (2.7)[a]	4.361*
PCRS Other Rated				
Total	106.36 (26.4)[ac]	123.93 (19.0)[c]	133.38 (9.7)[a]	11.325**
ADL Other-rated	31.0 (6.7)[ac]	35.73 (4.5)[c]	37.33 (2.7)[a]	7.505**
Cognitive Other-rated	28.08 (8.2)[a]	32.06 (6.3)	36.24 (2.8)[a]	8.081**
Interpersonal Other-rated	23.58 (5.1)[ac]	29.46 (4.5)[c]	30.90 (3.6)[a]	11.565**
Emotional Other-rated	23.16 (6.0)[a]	26.67 (5.6)	28.14 (3.3)[a]	3.972*

* Significant at $p<.05$ level
** Significant at $p<.01$ level
[a]: Post hoc bonferroni comparison ($P<.05$): TBI Low SA vs Controls
[b]: Post hoc bonferroni comparison ($P<.05$): TBI High SA vs Controls
[c]: Post hoc bonferroni comparison ($P<.05$): TBI Low SA vs TBI High SA

Discrepancy Ratings on PCRS were calculated by subtracting self-ratings from other-ratings for the total and subscales. The mean and standard deviations for discrepancies between self- and other- rated PCRS for each group are displayed in Table 3.6. One-way between-subjects ANOVAs were conducted to compare discrepancy ratings for TBI Low SA, TBI High SA and Control participants. Group effect was significant for Total Competency, ADL and Cognitive subscales. Interpersonal and Emotional subscales did not reach significance for group effect, although a trend towards significance for Interpersonal subscale was evident

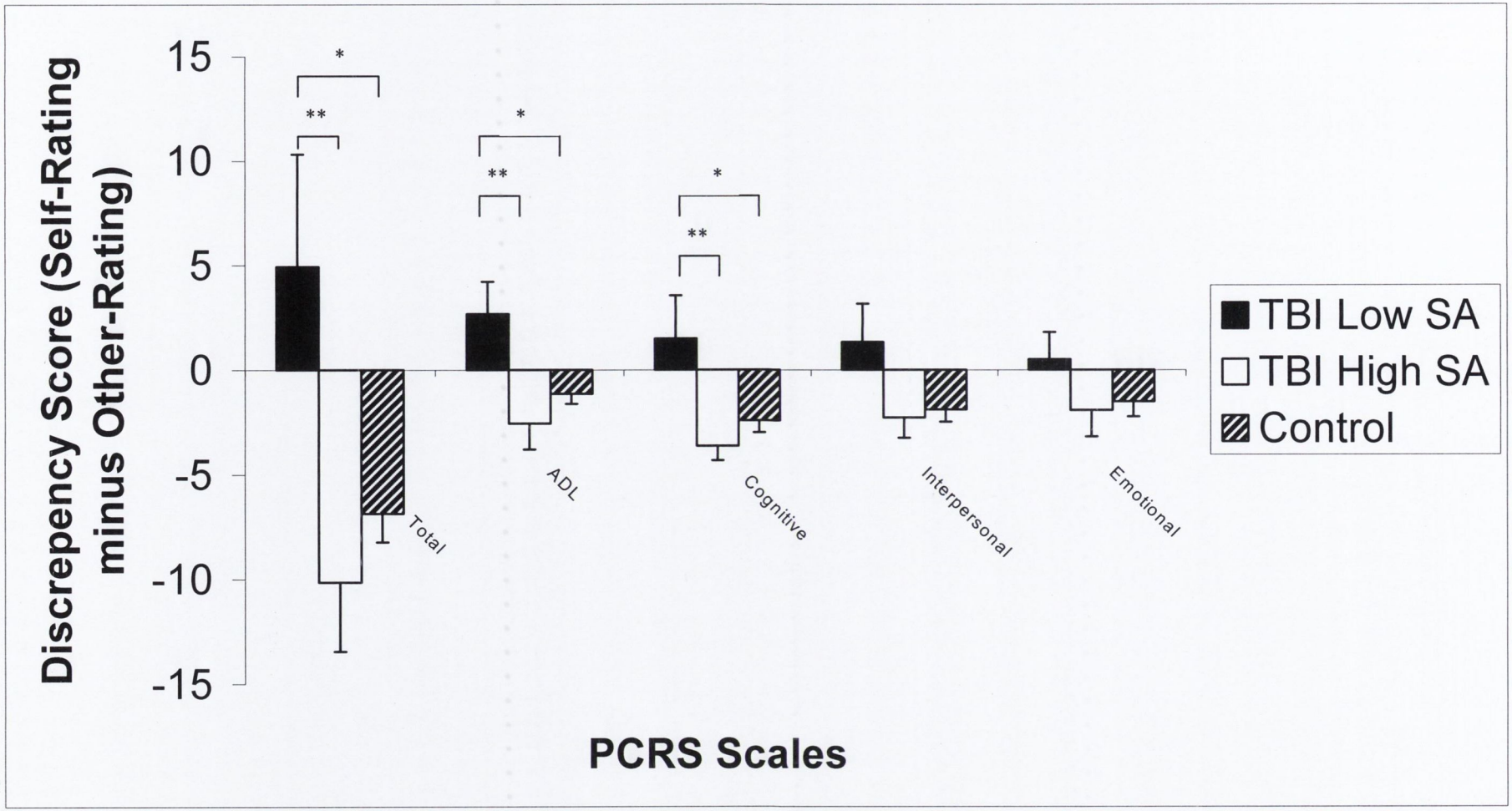
(p=.058). Post hoc bonferroni comparisons on discrepancy scores for TBI Low SA indicated that they significantly overestimated their competencies on Total competency score, ADL and Cognitive subscales, compared to TBI High SA (Total: p<.01; ADL: p<.01; Cognitive: p<.01) and Controls (Total: p<.05; ADL: p<.05; Cognitive: p<.05). The discrepancy scores of the High SA group indicate that they underestimated their competencies. Figure 3.5 displays discrepancy scores on PCRS for total and subscales.

Table 3.6: Patient Competency Rating Scale Discrepancy Mean scores, Standard Deviations, F Values, Significance Level and Post Hoc Comparisons for Each Group

PCRS Discrepancy[1] Mean (SD)	**TBI Low SA**	**TBI High SA**	**Controls**	**F Value**
Total Discrepancy	4.93 (20.2)[ac]	-10.14 (12.4)[c]	-6.86 (7.4)[a]	5.667**
ADL Discrepancy	2.67 (5.4)[ac]	-2.57 (4.6)[c]	-1.14 (2.3)[a]	5.923**
Cognitive Discrepancy	1.50 (7.1)[ac]	-3.64 (2.6)[c]	-2.43 (2.6)[a]	5.923*
Interpersonal Discrepancy	1.33 (6.3)	-2.29 (3.6)	-1.91 (2.7)	3.036
Emotional Discrepancy	0.50 (4.5)	-1.93 (4.7)	-1.52 (3.3)	1.326

* Significant at p<.05 level
** Significant at p<.01 level
[a]: Post hoc bonferroni comparison (P<.05): TBI Low SA vs Controls
[b]: Post hoc bonferroni comparison (P<.05): TBI High SA vs Controls
[c]: Post hoc bonferroni comparison (P<.05): TBI Low SA vs TBI High SA
Note 1: Discrepancy Scores were calculated only for questionnaires that were available for both self and other (n=47), and therefore there is a slight discprepancy between mean scores presented in Table 3.5 above and discrepancy scores here.

Figure 3.5: PCRS Discrepancy Scores for TBI Low SA, TBI High SA and Controls



*Significant at $p<.05$; ** Significant at $p<.01$

3) Frontal Systems Behavioral Scale (FrSBe)

a) Behaviour Ratings of Frontal Behaviour

Mean and standard deviations for Frontal Systems Behavioral Scale (At Present/Since Injury) for apathy, disinhibition and executive dysfunction subscales and total score are presented in Table 3.7 for both self- and other- ratings. One-way between-subjects ANOVAs and post hoc bonferroni comparisons were conducted to investigate group differences between TBI Low SA, TBI High SA and Controls for each of these ratings. F values and post hoc comparisons are also displayed on Table 3.7.

Table 3.7: Mean and Standard Deviation Scores for Self- and Other-Rated Frontal Systems Behavioral Scale (Total and Subscales)

FrSBe At Present Mean (SD)	**TBI Low SA**	**TBI High SA**	**Controls**	**F Value**
FrSBe Self Rated Total	122.87 (28.7)[a]	117.0 (26.6)[b]	87.71 (19.5)[ab]	14.241**
Apathy	38.47 (9.3)[a]	36.47 (9.4)[b]	26.45 (6.55)[ab]	14.548**
Disinhibition	37.6 (9.1)[a]	34.87 (10.7)[b]	27.84 (6.2)[ab]	8.330**
Executive Dysfunction	46.8 (12.7)[a]	45.67 (10.3)[b]	33.42 (9.4)[ab]	11.435**
FrSBe Other Rated Total	121.64 (40.9)[a]	98.33 (24.7)	79.93(19.8)[a]	11.068**
Apathy	34.14 (13.4)[a]	30.07 (7.1)	24.31 (7.6)[a]	5.814**
Disinhibition	37.79 (14.2)[ac]	28.0 (8.1)[c]	25.41 (6.4)[a]	8.625**
Executive Dysfunction	49.0 (16.4)[a]	40.27 (12.4)[b]	30.97 (8.2)[ab]	11.684**

** Significant at $p<.01$ level
[a]: Post hoc bonferroni comparison ($P<.05$): TBI Low SA vs Controls
[b]: Post hoc bonferroni comparison ($P<.05$): TBI High SA vs Controls
[c]: Post hoc bonferroni comparison ($P<.05$): TBI Low SA vs TBI High SA

As can be seen from Table 3.7, both TBI Low SA and TBI High SA rate themselves as demonstrating significantly more symptoms of frontal systems dysfunction compared to Controls. SO ratings for frontal systems dysfunction for TBI Low SA are also significantly higher than Controls. Furthermore, SO ratings for TBI Low SA

are significantly higher than TBI High SA for Disinhibition subscale. TBI High SA significant-other ratings are significantly higher than Controls for executive dysfunction subscale only.

One-way between-subjects ANOVAs were performed to examine the discrepancy between self- and other-rated FrSBe Scales (Self-rated minus SO-rated). The discrepancies between self- and other-rated scores for frontal systems behaviour did not differ significantly between groups, although there was a trend towards significance for Total FrSBe ($F(2, 55)= 2.980$, $p=.059$; Apathy: $F(2, 55)= 1.964$, $p=.15$; Disinhibition: $F(2, 55)= 2.945$, $p=.06$; Executive Dysfunction: $F(2, 55)= 2.808$, $p=.06$].

Table 3.8: Discrepancy Scores (Total and Subscale) on Frontal Systems Behavioral Scale for Each Group

FrSBe Discprepancy[1] Mean (SD)	**TBI Low SA**	**TBI High SA**	**Controls**	**F Value**
Total Discrepancy	0.50 (27.2)	18.67 (20.9)	6.48(16.7)	2.980
Apathy Discrepancy	4.50 (9.8)	6.40 (6.2)	1.79 (6.9)	1.964
Disinhibition Discrepancy	-0.57 (10.9)	6.87 (10.1)	2.03 (5.9)	2.945
Executive Dysfunction Discrepancy	-2.71 (11.8)	5.40 (8.3)	1.89 (8.2)	2.808

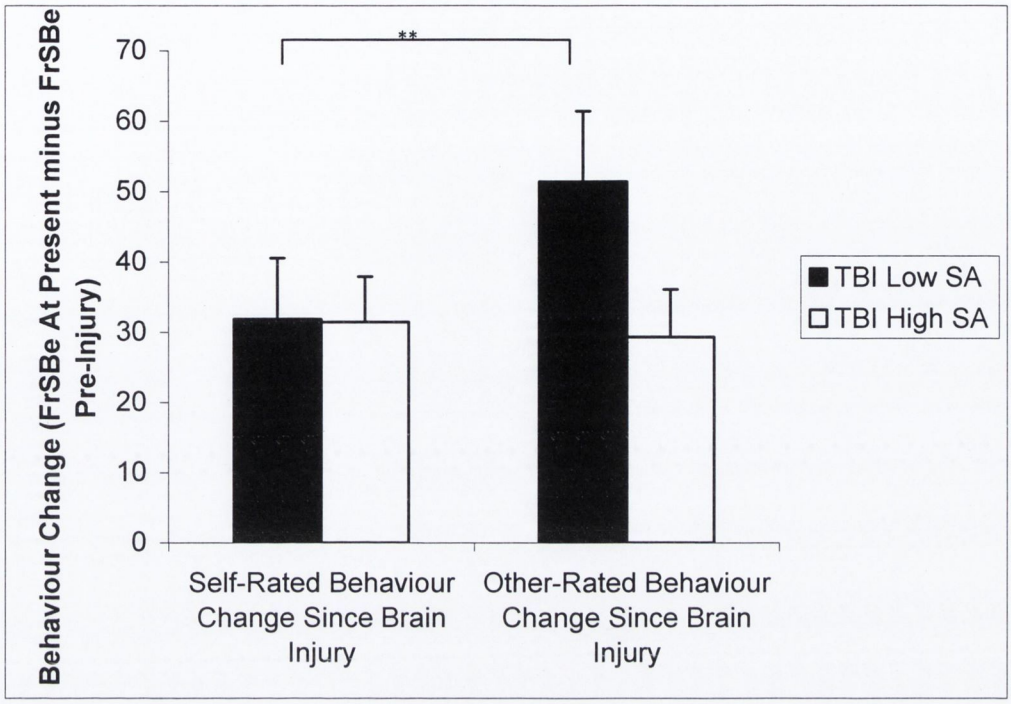
Note 1: Discrepancy Scores were calculated only for questionnaires that were available for both self and other (n=58), and therefore there is a slight discrepancy between mean scores and discrepancy scores here.

b) Behaviour-Change Ratings since injury for TBI participants

The FrSBe consists of a scale for rating pre-injury behaviour and a scale for rating current behaviour. TBI participants and their significant others rated both the pre-injury and current behaviour scales, which gave an indication of perceived behaviour-change since injury for both the TBI participants themselves and their

SOs. A Behaviour Change score was calculated by subtracting FrSBe pre-injury Total Score from FrSBe At Present Total score for both self and significant other ratings, in order to examine TBI participants' and their significant-others' perceptions of how their behaviour may or may not have changed since their brain injury. This led to a Self-Rated Behaviour Change score and an Other-Rated Behaviour Change score. To examine if perceptions differed between TBI Low SA and TBI High SA of Behaviour Change, a mixed between-subjects ANOVA was run. Between-subjects variable was Group (Two levels: TBI Low SA and TBI High SA) and the within-subjects variable was Behaviour Change (Two levels: Self-Rated Behaviour Change and Other-Rated Behaviour Change). There was a significant effect for Group [$F(1, 27) = 5.622$, $p<.05$] and a significant interaction between group and Behaviour Change [$F(1, 27) = 8.752$, $p<.01$]. Main effect for Behaviour Change did not reach significance [$F(1, 27)= 1.067$, $p>.05$]. Post hoc (independent and paired samples) t tests showed that for the TBI Low SA group, other-rated Behaviour Change [M = 51.43, SD= 37.59] was significantly larger in magnitude than self-rated Behaviour Change [M= 31.87, SD= 33.66] ($t = -2.988$, $df=13$, $p<.01$). For the TBI High SA group, Behaviour Change self-ratings [M=31.467, SD=24.9] and other-ratings [29.333, SD=26.2] did not differ ($t=0.597$, $df= 14$, $p>.05$). The difference between Other-rated Behaviour Change for TBI Low SA and TBI High SA was approaching significance ($t = -1.853$, $df= 27$, $p=.07$), whereas there was no difference between self-rated Behaviour Change for TBI groups ($t= -0.037$, $df=28$, $p>.05$). These differences in perceived behaviour change since injury are displayed in Figure 3.6.

Figure 3.6: Self-and Other-Rated Behaviour Change (FrSBe At Present minus FrSBe Pre-Injury) Since Brain Injury for TBI Participants



** Significant at $p<.01$

Cognitive Failures Questionnaire Self- and Other-Ratings

Mean actual scores and z-scores for self-rated CFQ and other-rated CFQ are detailed in Table 3.9. Discrepancy scores between self- and other-rated scales are also detailed. One-way between-subjects ANOVAs indicated that only self-rated CFQ differed significantly between groups. Post hoc bonferroni comparisons indicated that both TBI groups rated themselves significantly higher on CFQ than Controls ($p<.05$).

Table 3.9: Mean and Standard Deviations (z-scores and Actual) Self- and Other-rated CFQ Scores and Discrepancy Scores

	TBI Low SA	**TBI High SA**	**Controls**	**F Value**
CFQ self (Actual, *z-scores*)	50.47, *0.428*	50.63, *0.435*	32.71, *-.432*	6.885**
CFQ other (Actual, *z-scores*)	17.64, *0.508*	12.53, *-0.031*	10.66, *-.229*	2.734
CFQ Discrepancy (*z-scores*)	*-0.1496*	*0.446*	*-.159*	2.099

** Significant at $p<.01$

Mean error awareness scores and standard deviations on Sustained Attention to Response Task (Fixed SART, Random SART and DART) are displayed as proportion of aware errors in Table 3.10.

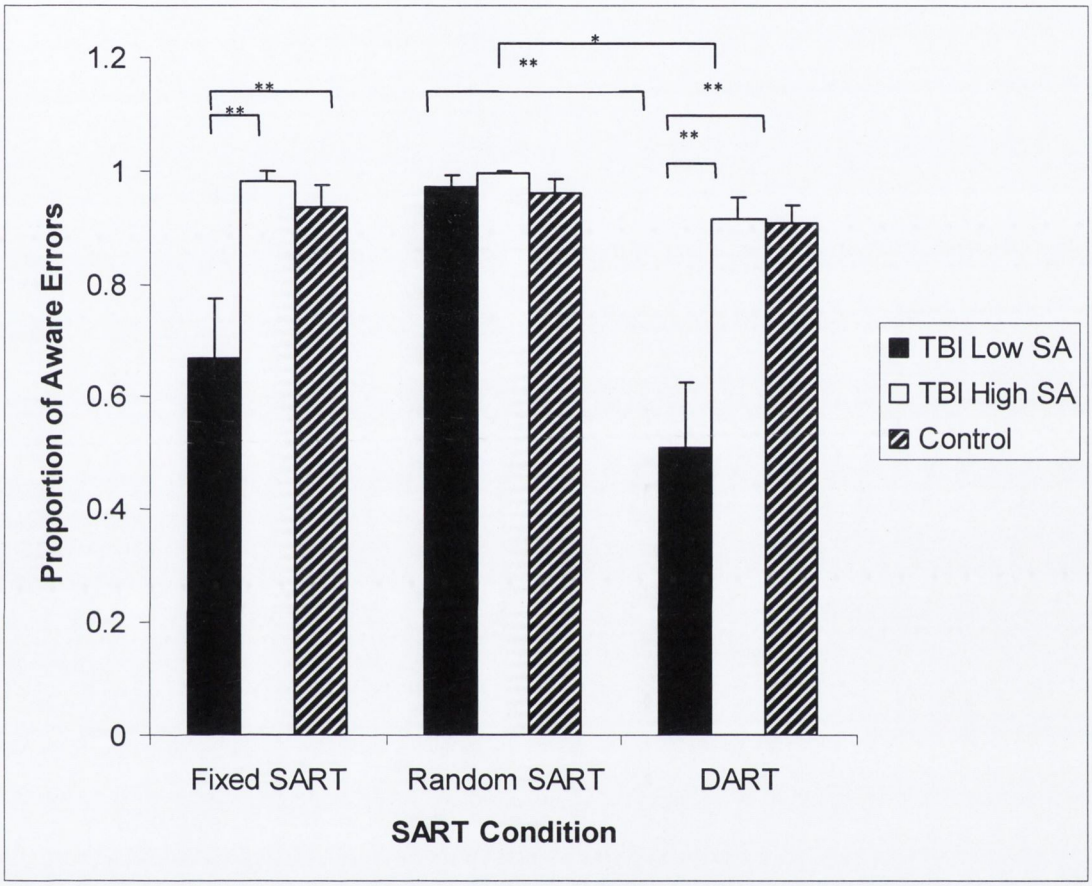
Table 3.10: Proportion of Aware Errors on SART Conditions for Each Group

Proportion Error Awareness Mean (SD)	**TBI Low SA**	**TBI High SA**	**Controls**
Error Awareness Fixed SART	.6699 (0.4)	.9833 (0.1)	.9361 (0.2)
Error Awareness Random SART	.9719 (0.1)	.9949 (0.01)	.960 (0.1)
Error Awareness DART	.5168 (0.4)	.9140 (0.1)	.9066 (0.2)

A mixed between-subjects ANOVA was run in order to examine error awareness proportion between the groups. The within-subjects variable was SART condition (Three levels: Fixed SART, Random SART and DART) and the between-subjects variable was Group (Three levels: TBI Low SA, TBI High SA and Controls). There was a significant interaction between Group and SART condition [$F(3.11, 70)=3.087$, $p<.05$]. Main effects were also significant for Group [$F(2, 45)= 14.974$, $p<.001$] and SART condition [$F(1.56, 70)= 7.997$, $p<.01$]. Post hoc bonferroni comparisons indicated that TBI Low SA had a significantly lower error-awareness proportion score on Fixed SART and DART conditions, compared to both TBI High SA (Fixed: $p<.01$, DART: $p<.01$) and Controls (Fixed: $p<.01$, DART: $p<.001$). The three groups did not differ on Error Awareness on Random SART. Paired-samples t-tests indicated that both TBI Low SA group and TBI High SA group had a significantly lower proportion of aware errors on the DART condition than on

Random SART. For the Control group, there were no significant differences in proportion of error awareness across the three SART conditions. Figure 3.7 displays error awareness proportion scores for each participant group.

Figure 3.7: Error Awareness Proportion Scores on Each SART Task for Each Group



* Significant at $p < .05$
** Significant at $p < .01$

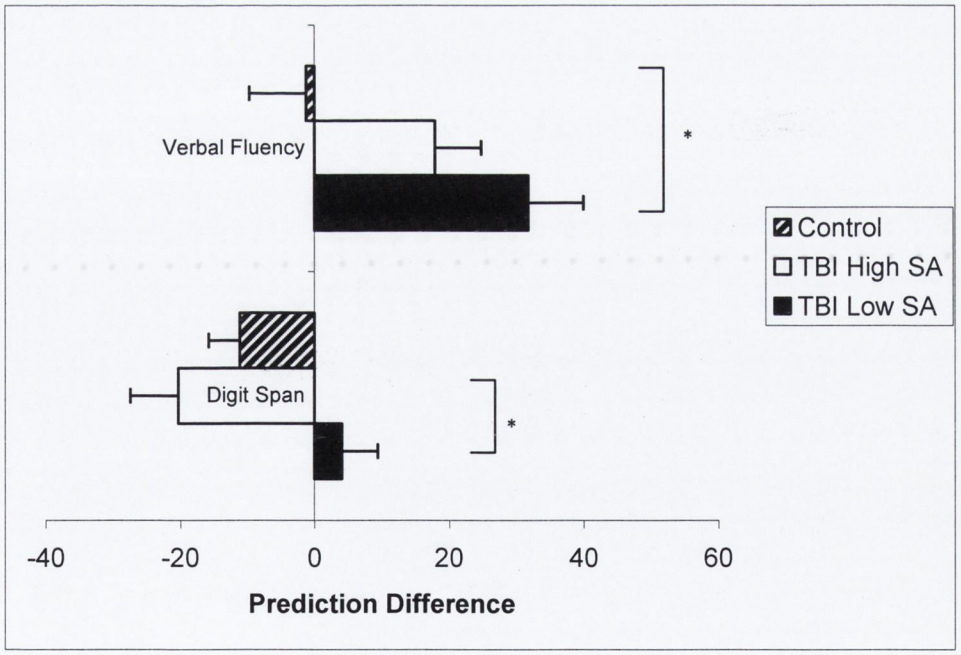
Online Monitoring of Errors: Anticipatory Awareness

Prediction Tasks

In the prediction tasks (Digit Span Forwards and Verbal Fluency), the average difference between predictions and performances was converted into a percentage

score [(prediction minus performance)/prediction X 100]. In order to investigate differences in prediction accuracy between groups, a one-way between-subjects ANOVA was run for prediction on Digit Span. Group was a significant factor [F(2, 58)= $p<.05$]. Post hoc bonferroni comparisons indicated that TBI High SA [M=-20.31, SD= 27.5] significantly underestimated their predictions to performance compared to TBI Low SA [M= 4.1, SD= 20.6] who showed a trend towards overestimation predictions to performance ($p<.05$). A one-way ANOVA examining prediction accuracy on FAS, showed that group was a significant factor [F(2, 59)=3.841, $p<.05$]. Post hoc comparisons showed that TBI Low SA [M=31.75, SD= 31.6] overpredicted their performance on FAS compared to Controls [M=-1.26, SD= 46.7] ($p<.05$). Figure 3.8 depicts each group's prediction accuracy on both Digit Span and FAS tasks.

Figure 3.8: Percentage Differences Between Prediction and Performance on Digit Span and Verbal Fluency Task for Each Group



* Significant at $p<.05$ level

Section F: What is the relationship of Awareness Composite Scores to other measures for TBI participants?

Table 3.11 shows selected correlations between each category of composite awareness and other measures of cognitive function.

Higher Metacognitive Knowledge in TBI participants was associated with lower total frontal behaviour as rated by significant others on the FrSBe, specifically with lowered disinhibition and executive dysfunction symptoms. Higher Metacognitive Knowledge also appeared to be associated with higher total competency as rated by significant others, and specifically on activities of daily living and interpersonal subscales. Though it did not reach significance level, there was a trend towards higher Metacognitive Knowledge associated with higher HADS depression (p=.059).

Higher levels of Online-Emergent Awareness were associated with lower levels of HADS Anxiety. Higher levels of Online-Emergent Awareness were also associated with lower ratings of frontal behaviour, on total FrSBe and each subscale. Higher Online-Emergent Awareness was associated with higher ratings of competency as rated by significant others on PCRS Total and specifically on the interpersonal subscale. Fewer errors of commission on the Fixed SART was also associated with higher levels of online-emergent awareness.

Higher levels of Online-Anticipatory Awareness were associated with lower errors of commission on the DART. There were no other significant correlations with the awareness measures with any other demographic or neuropsychological variable for TBI participants.

Table 3.11: Pearson Product-Moment Correlations between Awareness Category and Selected Measures for TBI participants

Variable	Metacognitive Knowledge	Online-Emergent Awareness	Online-Anticipatory Awareness
HADS Anxiety	.009	-.501**	-.239
HADS Depression	.349	-.242	.036
FrSBe Total (Other-rated)	-.379*	-.429*	.025
FrSBe Apathy	-.115	-.412*	-.153
FrSBe Disinhibition	-.460*	-.392*	.059
FrSBe Executive Dysfunction	-.421*	-.370*	.145
PCRS (Other-rated)	.443*	.394*	-.121
PCRS ADL	.490**	.378	-.271
PCRS Interpersonal	.419*	.491**	-.148
Fixed SART Errors	.219	-.474*	-.046
DART Errors of Commission	-.113	-.242	-.422*

* Significant at $p < .05$
** Significant at $p < .01$

Section G: What is the relationship of the three awareness measures to each other?

In order to examine the relationship between the three different types of awareness measures, correlational analyses were conducted. Metacognitive Knowledge was not associated with either Online-Emergent [r=.184, p>.05] or Online-Anticipatory awareness [r=.009, p>.05]. A strong association between Online-Emergent and Online-Anticipatory awareness was evident [r=.717, p<.0001].

3.4 Discussion

This study used a multidimensional approach to comprehensively assess awareness in Traumatic Brain Injury participants and matched controls. The TBI group, as a whole, demonstrated impaired awareness on Metacognitive Knowledge, Online-Emergent Awareness and Online-Anticipatory Awareness, compared to Controls. TBI participants with High Self-Awareness, performed equally as well as Controls in all of the awareness assessments, with the exception of total overall Awareness Index from the Awareness Interview. Directly in contrast to this, the TBI Low Self-Awareness group were significantly impaired on every measure of awareness, with the exception of Cognitive Failures Questionnaire discrepancy scores and overall Frontal Systems Behavioural Scale discrepancy scores. The TBI High SA group and the TBI Low SA group did not differ from each other in terms of severity, time since injury or any neuropsychological test. However, the TBI Low SA group were rated as demonstrating poorer interpersonal skills, more symptoms of disinhibition, less competency in activities of daily living and less overall competency than the TBI High SA group, as rated by significant others on the FrSBe and PCRS. Furthermore, significant relationships were found between executive dysfunction symptoms and interpersonal difficulties and overall Metacognitive Knowledge and Online Emergent Awareness. Sustained attention abilities were associated with Online Emergent Awareness and Online Anticipatory Awareness. Measures of Metacognitive Knowledge did not correlate with the other two types of awareness. Both types of Online Awareness, Emergent and Anticipatory, showed a strong relationship with each other.

Looking at the Awareness Index scores from the Awareness Interview, both TBI High SA and Low SA groups were significantly impaired at assessing their overall abilities across each category. In keeping with previous studies, this suggests that TBI participants, as a group, have inaccurate perceptions of their strengths and weaknesses overall (Anderson & Tranel, 1989; Sherer, Boake et al., 1998). However, the groups did not differ significantly from Controls across any individual category, with the exception of ability to assess how they performed overall on the tests and based on their current performance, their ability to predict whether or not they could return or continue with work or education. Also of note, is that this was the only measure of awareness in which the High SA group differed significantly from controls. The awareness interview compares individual responses to neuropsychological measures. Despite showing high levels of awareness overall, the TBI High SA group were significantly impaired across a number of neuropsychological measures, indicating impaired immediate and delayed recall, impaired short-term memory, impaired sustained attention on the Random SART and impaired verbal fluency. It is possible that despite being aware of having difficulties in each cognitive area, TBI High SA group are nevertheless inaccurate at assessing what the functional implications of these deficits are for returning to employment, hobbies and activities of daily living, or the ability to predict ones future accurately. This is consistent with Fleming and Strong's three level model of awareness that separates objective knowledge of deficits from subjective appreciation of these deficits or the ability to predict ones future accurately (Fleming & Strong, 1995; Fleming et al., 1996).

Consistent with numerous studies investigating awareness deficits in TBI populations (Fleming & Strong, 1999; Prigatano & Altman, 1990), the TBI Low SA group

significantly overestimated their overall competency compared to Controls and the TBI High SA group on the PCRS, indicating that the TBI Low SA group have an inaccurately elevated perception of their everyday abilities. Examining the subscale discrepancy scores indicates that the TBI Low SA group significantly overestimated their competencies on the ADL subscale and Cognition subscale, whereas their discrepancy ratings for Emotional and Interpersonal subscales did not differ significantly from TBI High SA group or Controls. It is not unusual for TBI participants to overestimate their competencies on the cognitive subscale, as it is considered that cognitive items are more abstract than physical or more concrete items and therefore more difficult to accurately assess for TBI participants (Fleming & Strong, 1999). However, the finding that TBI Low SA group significantly overestimated their activities of daily living competencies compared to both TBI High SA group and Controls is contrary to numerous studies that have found that TBI participants are most accurate at assessing their abilities on physical functioning, such as activities of daily living (Fischer, Trexler et al., 2004; Fleming & Strong, 1999; Prigatano et al., 1990). Included in the subscale for ADL are items that require rating for "Driving a car if had to", "Taking care of my finances" and "Meeting daily responsibilities". Two previous studies found that TBIs overestimated their level of competencies on the first two of these tasks, despite rating their other ADL items similarly to significant others. They suggested that there may be non-neurological reasons for this, such as denial or desire to make a good impression, as these items are extremely important for independent living and may have legal consequences. Neurological reasons may also be involved, such that the TBI patients may focus on the physical elements required to drive whereas the SO may base their ratings more on the cognitive abilities required in these tasks (Fleming & Strong, 1999; Prigatano et al., 1990). As the TBI Low SA group also significantly overestimated their

competencies on the Cognitive subscale, the reason applying to inaccurate perception of the cognitive components of some of the ADL items may be more likely for overestimation on this subscale. An alternative explanation to this may come from a more socio-cultural perspective than a neurocognitive one. In a series of cross-cultural studies on awareness of deficits, it was shown that using the PCRS, Japanese TBI participants did not overestimate their social and emotional functioning competencies as their American TBI counterparts did but significantly overestimated their ADL and self-care abilities. This was interpreted as suggesting the greater social cost in Japanese culture of disclosing dependence required for ADLs and self-care activities (Prigatano et al., 1997). However, it is unlikely that an Irish socio-cultural context would reflect Japanese culture more than American. It is most likely that ADLs and self-care activities are important for independent-living, especially for a TBI patient group that are, on average, three-years post-injury and have all undergone various lengths of rehabilitation as both inpatients and outpatients in the National Rehabilitation Hospital. Hillier and Metzer (1997) report similar findings where TBI patients report a lower rate of physical impairments and self-care difficulties than SOs and clinicians, possibly reflecting a longer time since injury also (on average 5 years post injury).

The trend for the High SA TBI group to underestimate their competencies compared to SO ratings was interesting and has also been found in some previous studies of awareness (Prigatano & Altman, 1990). This trend for some TBI participants to underestimate their competencies, or exaggerate their deficits has been described as these patients being "hyperaware" (Lanham et al., 2000) or being highly deficit focussed (Sawchyn et al., 2005), with negative associations of greater psychological and emotional distress (Fleming et al., 1998; Prigatano & Altman, 1990; Ranseen et

al., 1990). The High and Low SA group in the present study did not differ significantly from each other in terms of HADS Anxiety or Depression, and in fact, though not statistically different, the Low SA group showed slightly higher ratings on both scales. However, relatively few previous studies in awareness have used a neurologically-healthy control group (Hart et al., 2005). In this study, the controls also underestimated their competencies compared to SO ratings, consistent with Leathem et al (1998) and Prigatano et al (1996; Prigatano et al., 1998). Roche et al (Roche, Fleming, & Shum, 2002) demonstrated that control participants reported more frequent prospective memory failures compared to their SO reports on a questionnaire of prospective memory. It is possible that instead of the negative connotations associated with underestimating one's competencies, that this is in fact the "normal" trend on this type of questionnaire. Hoofien et al (2004) also demonstrated that those TBI patients who underestimated in their study performed just as well and sometimes better than those who showed good awareness in terms of outcome. It may be that healthy control participants and TBI participants with High SA are aware of and acknowledge difficulties for a number of items on the PCRS but are capable of controlling these difficulties in everyday life so that these difficulties are not evident to their SOs (Leathem et al., 1998).

Considering the Frontal Systems Behavioral Scale (FrSBe), it appears that TBI groups with High and Low SA are relatively accurate at assessing their frontal symptoms, apathy, disinhibtion and executive dysfunction. The TBI Low SA group rate themselves as demonstrating more of these frontal behaviours than controls on each scale, in a similar pattern as to how their SO rates them. The SOs of this group rate the TBI Low SA as significantly more disinhibited than the TBI High SA group also. The TBI High SA group rate themselves as demonstrating more of these

behaviours than the control group, but their SOs rate them similarly as the Controls, with the exception of executive dysfunction subscale. However, the discrepancy scores do not reach significance level for either group, which may be a result of the large variability on this scale (For example SO Total Rating for TBI Low SA group has a mean of 121. 64 and a standard deviation of 40.9). The non-significant trend of discrepancy scores is a similar pattern to the PCRS discrepancy scores with the TBI Low SA generally underestimating their frontal behaviours compared to their SO raters and the TBI High SA overestimating their frontal behaviours compared to their SO raters, similar to Controls but to a greater extent.

In terms of perceived behaviour change, the TBI High SA group and their SO raters were similar in their assessment of perceived change for this group. However, the TBI Low SA group rated their behaviour change as similar to the TBI High SA group which was significantly less than how their SOs rated their behaviour change. This indicates that although the TBI Low SA group do recognise and report some changes in their behaviour, it is to a much smaller degree than how their SOs see significant changes in the TBI Low SA participants' behaviour since before their injury. This finding could have significant implications for the emotional impact on family members of this group of TBI patients, considering it has been shown that family members' distress level and quality of life ratings was significantly correlated with their perception of the patients' impaired awareness (Koskinen, 1998; Prigatano et al., 2005) and in the absence of social support, impaired awareness of behaviour change on the part of the patient leads to increased caregiver burden and life dissatisfaction for the family members and caregivers (Ergh, Hanks, Rapport, & Coleman, 2003).

Both TBI High and Low SA rated themselves as demonstrating more everyday cognitive failures than the Controls. The discrepancy scores between self- and other-ratings were not significantly different in this study. This is contrary to a study by Hart et al (2005) that demonstrated that discrepancy scores on the CFQ between self and SO were significantly different for TBI participants and healthy controls. However, as noted by Fleming et al (1999), TBI participants may have a greater awareness of memory problems than of more higher cognitive, social and emotional difficulties. Similar results were found by Mckinlay and Brooks (1984), who showed that there was moderate agreement about memory impairment (65% agreement) between persons with TBI and relatives.

Considering all the three questionnaire discrepancy scores together, it is somewhat surprising that the TBI Low SA group do not follow a similar pattern to other studies, in the areas that they appear to be inaccurate on, such as ADLs and general behaviour change since injury. The TBI Low SA group appear to be reasonably accurate at assessing their emotional, interpersonal and dysexecutive symptoms, which is contrary to numerous studies that have indicated that it is these exact areas that pose most problems for accurate awareness for TBI patients (Fischer, Trexler et al., 2004; Fleming & Strong, 1999; Prigatano, 1996; Prigatano et al., 1990). One explanation for this may be that the TBI participants recruited for the present study were all on average three years post-injury (mean 36 months since injury), longer chronicity than some mentioned above, such as 16 months in Prigatano et al (Prigatano et al., 1990), between 3 and 12 months post-injury in the study by Fleming et al (1999), and a mean of 141 days since injury in Fischer et al (Fischer, Trexler et al., 2004). In another study that recruited patients with a slightly longer chronicity (mean 687 days for mild and 745 days for severe TBI patients), no

significant self-other discrepancies were found on the PCRS for the entire sample of TBI participants, and when divided into mild, moderate and severe, the moderate and severe patients showed the expected discrepancies in a trend towards overestimating competencies, but without reaching significant differences. Only the mildly injured group in this study significantly underestimated their abilities compared to SO ratings (Sawchyn et al., 2005). It has been shown that there can be some improvement in awareness of deficits over time in TBI patients (Fleming & Strong, 1999) and some studies have also shown that through specific interventions awareness of deficits can also improve through group educational and psychotherapy programs (Ownsworth, McFarland, & Young, 2000), or through educational games (Chittum, Johnson, Chittum, Guercio, & McMorrow, 1996; Zhou et al., 1996). All the TBI participants from this study were recruited from the National Rehabilitation Hospital and would have undergone significant rehabilitation and psychoeducation as both inpatients and outpatients. It may be that throughout the course of their rehabilitation that they have been made aware of the greater likelihood of having emotional, interpersonal and social problems following their injury and are now able to accurately rate their difficulties in these areas. The TBI Low SA group in particular are nevertheless still significantly impaired across these and each of the other measures of awareness. This has both clinical and theoretical relevance with regard to both assessment and rehabilitation of these participants. Indeed, as also pointed out by Ownsworth et al (In Press), it demonstrates the importance of using a multidimensional approach to assessment, rather than limiting assessment of ISA to one discrepancy score, as in several previous studies outlined in Chapter 1.

The TBI Low SA group were significantly impaired on Online-Emergent Awareness tasks, monitoring their errors as they performed three different conditions of a

sustained attention task. The TBI High SA group were significantly better than the TBI Low SA group on this task, and similar to, if not better than, Controls in their ability to monitor their errors on sustained attention tasks. The overall error monitoring impairment for the TBI Low SA group is in keeping with previous studies using this error-monitoring methodology that showed poor error awareness on these tasks of sustained attention in TBI participants (McAvinue et al., 2005; O'Keeffe et al., 2004- Chapter 2).

However, there are a number of particularly interesting findings in this study in relation to online monitoring of errors. Firstly, this is the first of these series of studies that has used three different conditions of the Sustained Attention to Response Task (SART) - Fixed, Random and DART. The TBI Low SA group were significantly impaired at monitoring their errors on both the Fixed SART and DART versions of this task compared to both TBI High SA and Control participants but perfectly able to monitor at a similar level to Controls and TBI High SA on the Random SART. This impairment of online-monitoring of errors cannot be related to the actual number of errors made, as the most errors in all participant groups were made on the Random SART condition, the exact condition that shows the highest level of error-awareness for both TBI groups. The Fixed SART has been described as a "purer" measure of sustained attention, by placing fewer demands on the response inhibition system by using a predictable fixed series of numbers from one to nine compared to the Random SART, where the digits are presented randomly, in an unpredictable fashion. The unpredictable nature of the Random SART may exert an exogenous alerting effect, which would require the maintenance of endogenous alertness to a lesser extent than the more monotonous, predictable Fixed SART. The Fixed SART showed greater increased activation in the right dorsolateral prefrontal

and right parietal regions than the more challenging Random SART (Manly et al., 2003). Dockree et al (2004) found that TBI patients failed to show alpha desynchronization during the Fixed SART, suggesting that brain injury may disrupt the cortical-cortical or thalamo-cortical networks required to increase alert responding intensity during the Fixed SART. The DART, the dual-task conditions of the Fixed SART, was developed to further enhance the sensitivity to sustained attention errors in a brain-injured and neurologically-healthy population, by adding greater challenge to the sustained attention system that is already compromised following brain injury. TBI participants did make more errors of sustained attention on the dual-task paradigm than the Fixed SART, as did neurologically-healthy controls. It was argued that TBI patients are more susceptible to transient drifts of attention, which leads to a loss of controlled processing for the maintenance of endogenous attention (Dockree et al., In Press; Manly et al., 2003; Robertson et al., 1997). McAvinue et al (2005) found that the magnitude of TBI participants' deficits in sustained attention and error awareness was significantly correlated, which supported the idea that the ability to maintain vigilant attention in routine and monotonous tasks may contribute to the maintenance of error awareness.

The findings from the present study are consistent with the notion that sustained attention abilities may be one of the executive functions required in monitoring of errors, as a significant association was found between better Online-Emergent Awareness and fewer errors of sustained attention on the Fixed SART. The fact that error-awareness on the Random SART was almost perfect for both TBI groups suggests that when an error is more impulsive, it may be more easily monitored. There is a significant reduction in awareness when the error is characterised by attentional drift off-task, as is more likely in both the Fixed SART and DART, as

described above, due to the greater demands on the maintenance of endogenous alertness. The TBI High SA group perform just as well as controls overall on this type of awareness, however, in the most demanding dual-task condition, this high-performance brain-injured group still monitor significantly fewer errors than they do on the Random SART, as do the more impaired Low SA group. The TBI High SA group are not significantly impaired from Controls on either the Fixed SART or the DART. It may be that the added challenge of monitoring errors on these tasks adds demands to an already-challenged sustained attention system, in particular in the most challenging condition of the DART, which is evident in a reduced ability to monitor errors even for this high-performance group of TBI participants. The control group did not differ in their ability to monitor errors across the three conditions of the SART, indicating an effective maintenance of endogenous alertness and an ability to simultaneously monitor their errors successfully, even with the increased dual task demands of the DART condition.

The findings from the tests of Online-Anticipatory Awareness indicated that TBI Low SA are particularly impaired in the ability to accurately predict their performance on two separate cognitive tasks, showing a trend to overestimate their ability on both the Digit Span and verbal fluency task. This is in contrast to both the TBI High SA and Controls who do not differ significantly from each other in their accuracy, and who show a trend to underestimate overall predictions. Fischer et al (2004) also showed that brain-injured participants overestimated their predictions on a different cognitive task (Selective Reminding Test). In another study, patients with anterior lesions also showed a tendency to overestimate their performance on a digit symbol subtest of the WAIS-R compared to patients with posterior lesions (Vilkki & Holst, 1991). The TBI High SA participants in the present study significantly

underestimated their accuracy on the Digit Span task compared to the TBI Low SA group, showing a similar trend to Controls who also appeared to show a trend to underpredict their performance on this task. On the verbal fluency task, Controls are almost perfectly accurate at predicting their performance, with a slight trend again to underestimate, whereas the TBI High SA group show a trend, though nonsignificant, to overestimate on this task. It is possible that although the TBI High SA group show a trend to underestimate overall on the prediction tasks, the verbal fluency task is more difficult to accurately estimate performance, as everyday analogues of Digit Span, such as attempting to remember telephone numbers, are more easily accessible from memory. The TBI High SA group may be aware that they are impaired on short-term working memory due to more frequent exposure to this type of task.

The TBI High SA and TBI Low SA patient groups did not differ significantly from each other in severity of injury, chronicity or on any test on the extensive neuropsychological test battery, which may be somewhat surprising considering the distinctively different performances of the two patient groups across each of the three categories of awareness and the many specific tests. The TBI Low group showed a significantly poorer neuropsychological profile compared to Controls than the TBI High SA group did compared to Controls, in that the TBI Low SA group were significantly impaired across almost all of the neuropsychological battery of tests compared to Controls, with the exception of the Revised- Strategy Application Task, the dual task subtest of the Test of Everyday Attention, the FAST apashia screening test and the Measure of Empathic Tendency. This is in contrast to the TBI High SA group, who despite showing significant impairment on the memory tests, performed quite well on the test battery overall, and did not differ significantly from Controls on the attention tests, with the exception of the Random SART or the executive function

tests. Interestingly, the TBI High SA group performed significantly worse on the FAST Aphasia screening test compared to Controls and although they did not differ from Controls on the HADS Anxiety scale, they had a significantly higher HADS Depression scale compared to Controls. However, considering the rating scales, the TBI Low SA group were rated as being more impaired on total competence ratings than the TBI High SA group, as rated by their SO on the PCRS scale. The TBI Low SA group were also rated as significantly less competent on activities of daily living and interpersonal abilities on the PCRS and more disinhibited on the FrSBe.

These findings are only partially consistent with previous studies who divided their TBI participants by awareness group. Noe et al (2005) showed no significant differences in terms of chronicity, GCS or length of coma between TBI High and Low SA groups. They did show that the TBI Low SA group had a significantly longer PTA than the High SA group, a finding that was not replicated in the present study. Prigatano and Altman (1990) also did not find any significant differences in severity (as measured by GCS scores) and chronicity between their three groups of TBI (Overestimators, Accurate and Underestimators). In terms of neuropsychological performance differences, the findings from this study are partially consistent with Prigatano and Altman (1990) and Prigatano et al (1997), who did not find any relationship between awareness group membership and neuropsychological measurements. Speed of finger tapping for the left hand was found to be the only discriminatory measure between groups. Hoofien et al (2004) also showed no significant difference across memory and attention domains but TBI patients who overestimated were significantly worse on comprehension and showed generally worse behavioural outcome as measured by the Behaviour Evaluation Checklist, examining the frequency of behavioural disturbances, including impulsivity and aggressiveness. The TBI patients who overestimated their abilities

also showed a worse psychiatric outcome than those with accurate estimates of their functioning and underestimators.

Hoofien et al's (2004) findings are particularly relevant in relation to this study, as the family members of the overestimators attributed significantly more behavioural disturbances to those in this awareness group than the family members of those patients who were accurate or underestimated. This is somewhat consistent with the findings in this study that show that TBI Low SA group were rated as showing significantly more disinhibition and interpersonal problems than those with High SA. It has been said that standard neuropsychological measures may fail to capture the more complex neuropsychological impairments when TBI patients are divided in terms of disturbances in self-awareness (Prigatano, 1991), a suggestion that the present study would support. Noe et al (2005) did show numerous differences between the High SA group and Low SA group in their study however, in terms of both neuropsychiatric and neuropsycholgocial profile. Despite numerous neuropsychological differences in their study however, only one task of executive function (WCST number of categories) and delayed recall (Tavec Free delayed recall) were independent predictors of self-awareness in the groups.

Other studies have found similar results to Noe et al (2005) showing that certain measures of executive function and memory predict awareness (e.g. McAvinue et al., 2005; Ownsworth et al., 2002; Trudel et al., 1998). The present study only partially replicated some of these findings by showing an association between sustained attention and Online-Emergent Awareness and Online-Anticipatory Awareness. The ability to maintain alertness in the context of routine tasks appears to predict the ability to monitor ongoing performance while performing a task, but also in relation

to predicting future performances. Greater Metacognitive Knowledge was predicted by greater total competence and interpersonal abilities, and competence on ADLs. Similarly, the more symptoms of executive dysfunction, disinhibition and frontal symptoms in general displayed, the more likely a TBI patient was impaired on Metacognitive Knowledge. The total ratings of general frontal symptoms and each of the subscales of apathy, disinhibition and executive dysfunction, as well as the total competency ratings and the interpersonal subscale predicted levels of Online Emergent Awareness also. These findings show that executive deficits and difficulties in the control of behaviour are intrinsically related to awareness deficits. No relationships were found to associate memory and awareness, despite some previous findings showing this association (Noe et al., 2005; Trudel et al., 1998).

The findings that Metacognitive Knowledge did not correlate with the two types of online measures (Emergent and Anticipatory), while both Online Emergent and Online-Anticipatory Awareness measures strongly correlate with one another (r=.717), support Toglia and Kirk's categorisation of awareness into Metacognitive Knowledge versus Online Awareness (Toglia & Kirk, 2000). The data from this study also provide further evidence for the neurocognitive factors involved in the DICE and CAM theories of awareness differentiating global awareness and online monitoring (Ownsworth et al., In Press; Schacter, 1990). The associations found in this study between the awareness measures and symptoms of frontal behaviour implicate the frontal lobes in each type of awareness, both Metacogntive Knowledge and Online Monitoring, as proposed by the aforementioned models and the Hierarchy of Brain Function Model (Stuss et al., 2001). As is evident from the neuroradiological data of the TBI patients in the present study, discrete lesion location is unusual in the context of Traumatic Brain Injury. Sherer et al (2005)

indicated that it may be more likely that broadly distributed networks may be disrupted to cause impairments in self-awareness, than any specific lesion location. As DAI in TBI is associated with frontal, prefrontal and cingulate hypoperfusion and damage to the subcortical areas (Bonne et al., 2003; Fontaine et al., 1999), it would seem likely that frontal networks, including their subcortical projections are involved in self-awareness. The recent fMRI study by Schmitz et al (in press) provides support for this, indicating that the medial pre-frontal and retrosplenial cortical regions play a primary role in self-referential evaluative processes and that activation of the right anterior dorsolateral prefrontal cortex was associated with reduced accuracy on the PCRS.

The emotional impact of either preserved or impaired self-awareness has been investigated in a number of previous studies. Greater awareness has frequently, though not exclusively, been linked with poorer emotional adjustment, or greater distress (Sawchyn et al., 2005; Sohlberg et al., 1998). However, in this study, there was no significant difference between depression and anxiety scores for the High and Low SA groups, with the High SA group showing slightly lower, though non-sigificant, ratings. Both patient groups showed significantly greater depression levels than Controls and only the Low SA group differed significantly from Controls for anxiety levels. Indeed, there was a moderate association between anxiety levels and Online-Emergent Awareness, in the opposite direction to that found in previous studies. TBI participants who were better able to monitor their errors scored lower on anxiety levels. Noe et al (2005) also showed no difference between High and Low SA groups on the Beck Depression Scale and the Zung Anxiety Scale. However, as mentioned above, the Low SA group in the Noe study nevertheless showed greater neuropsychiatric symptoms as measured by the Neuropscyhiatric Inventory. Wallace

and Bogner (2000) also found that while many TBI patients reported mild or greater depression and anxiety, it was not associated with awareness ratings. Ownsworth and Fleming (2005) found that intellectual awareness (Metacognitive Knowledge) was not associated with better or worse emotional adjustment, but that higher levels of hope for the future was related to better online-awareness. These authors also point out that the association between greater emotional distress and higher levels of awareness found in some previous studies may be related to earlier stages of recovery. Consistent with this, the TBI participants in the present study were, on average, three years post injury.

In conclusion, this study has shown that Awareness of Deficits almost certainly cannot be either described or measured using a single, unidimensional approach to assessment. It has confirmed the multidimensional, complex nature of awareness and is one of the first studies that comprehensively examines awareness across the three levels of awareness, as outlined by clinical models suggested by Crosson et al (1989) and Toglia and Kirk (2000). In a recent review by Prigatano (2005), certain "Facts" about impaired awareness after TBI were described. For example, findings that relate severity of injury and emotional distress to awareness, and the assumption that physical deficits are more readily acknowledged by TBI patients than emotional/interpersonal (Fischer, Trexler et al., 2004). These facts need to be considered carefully with regards to the "object of insight" that was used to assess impaired awareness (Markova & Berrios, 2001), as a huge number of studies have only used one single measure to assess awareness in TBI clinical groups, typically a discrepancy score on one questionnaire. It is clear from the present findings that behaviour suggesting executive dysfunction and complex social and interpersonal difficulties are related to each type of awareness. However, standard

neuropsychological assessments may not reflect these underlying associations, as this data demonstrated. The association between sustained attention, Online-Emergent Awareness and Online-Anticipatory Awareness may suggest a possible route for rehabilitation, as it has been shown in a study using similar methodology to the present one, that the provision of auditory feedback on error led to an improvement in sustained attention performance on the SART for TBI participants. Although it was unclear whether feedback on error enhanced error awareness or general arousal, the possibility that feedback on error may improve online error-monitoring certainly merits further investigation (McAvinue et al., 2005). Despite the small patient sample size (n=31) in the present study, the distinct lack of association between the composite scores of Metacognitive Knowledge and the two Online-Awareness types, strongly suggests that previous studies may not have examined the whole, multifaceted picture of awareness of deficits following TBI. This study has taken us a step closer to understanding the complex phenomenon that is awareness of deficits.

Chapter 4

Study 3

Evidence of Impaired Metacognitive Knowledge and Online Awareness in Corticobasal Degeneration and Progressive Supranuclear Palsy Patients

Abstract

Loss of insight is one of the core features of fronto-termporal dementia (FTD). FTD shares many clinical and pathological feaures with corticobasal degeneration (CBD) and progressive supranuclear palsy (PSP). The aim of this study was to investigate awareness of cognitive deficits in FTD, CBD and PSP using a multidimensional approach to assessment, that examines metacognitive knowledge of the disorders, online monitoring of errors (emergent awareness) and ability to accurately predict performance on future tasks (anticipatory awareness). 35 patients (14 FTD, 11 CBD, 10 PSP) and 20 controls were recruited. Results indicated that loss of insight was a feature of each of the three patient groups. FTD patients were most impaired on online monitoring of errors compared to the other two patient groups. Executive functions and memory abilities were associated with awareness.

4.1 Introduction

Early loss of insight, or the inability to accurately perceive changes in behaviour and personality, is one of the core clinical features of frontal-variant Frontotemporal Dementia (fvFTD) (Neary et al., 1998). Systematic studies of the degree and nature of loss of insight in FTD are rare however, and clinically, loss of insight is most often only assessed using clinical interviews rather than using validated quantitative measures (Rankin et al., 2005; Diehl & Kurz, 2002). In two studies that have explicitly investigated loss of insight in FTD, both found that FTD patients were significantly more impaired than patients with Alzheimer's Disease (AD) at identifying personality changes since their illness (Rankin et al., 2005) and self-monitoring their memory performances (Souchay et al., 2003).

A proportion of patients with AD also show loss of insight or anosognosia. The clinical relevance for investigating loss of insight becomes apparent when one considers that in AD, higher levels of awareness are associated with better treatment outcome (Morris & Hannesdottir, 2004). Lack of insight has been demonstrated to cause increased stress and caregiver burden (Seltzer, Vasterling, Yoder, & Thompson, 1997) and can result in poor patient-caregiver interaction (Hutchinson, Leger-Krall, & Wilson, 1997). Poor compliance with medication and performing dangerous or difficult activities have also been associated with loss of insight (Cotrell & Wild, 1999; McGlynn & Schacter, 1989).

FTD shares many clinical, radiological, and pathological features with the atypical sporadic parkinsonian movement disorders known as Corticobasal Degeneration

(CBD) and Progressive Supranuclear Palsy (PSP) (Cordato, Duggins, Halliday, Morris, & Pantelis, 2005; Kitagaki, Hirono, Ishii, & Mori, 2000). All three disorders may exhibit atrophy of the frontal and/or temporal lobes. Furthermore, at the molecular level, many cases of FTD (especially familial cases) and all cases of both CBD and PSP are related to abnormal function/levels of the microtubule associated protein tau. For CBD, most diagnostic criteria define a movement disorder, including rigidity, apraxia, cortical sensory loss or alien limb (Lang, Riley, & Bergeron, 1994; Litvan et al., 2003). However, it has been noted that many patients with CBD develop or present with a frontal dementia or features of a progressive nonfluent aphasia, indicating CBD is a cognitive disorder as well as a motor disorder, making this condition difficult to distinguish from other neurodegenerative disorders such as FTD, PSP, and AD (Bak, Crawford, Hearn, Mathuranath, & Hodges, 2005; Frattali, Grafman, Patronas, Makhlouf, & Litvan, 2000; Graham, Bak, Patterson, & Hodges, 2003; Kertesz, Martinez-Lage, Davidson, & Munoz, 2000; Lang, 2000; Litvan et al., 1997; Mathuranath, Xuereb, Bak, & Hodges, 2000). PSP is another commonly misdiagnosed neurodegenerative akinetic rigid disorder which can present with axial rigidity, vertical gaze palsy and recurrent falls. Neurocognitive deficits can also be a feature of PSP, and executive function deficits, including set-shifting, planning and categorisation are a frequent feature of PSP (Bak et al., 2005; Boeve, Lang, & Litvan, 2003; Grafman., Litvan, & Stark, 1995; Litvan et al., 1996; Litvan et al., 2003; Soliveri et al., 1999). Misdiagnosis of both CBD and PSP with Parkinson Disease (PD) are common (Litvan et al., 2003). Recent evidence indicates that patients with PD can demonstrate impaired self-awareness in multiple domains, based on discrepancy between patients and their caregivers (Leritz et al., 2004; Seltzer et al., 2001). No studies, that that this researcher is aware of at least, have examined loss of insight specifically in CBD and PSP.

The Dissociable Interactions and Conscious Experience (DICE) model (McGlynn & Schacter, 1989; Schacter, 1990) and its later developments to the Conscious Awareness Model (CAM) (Agnew & Morris, 1998; Morris & Hannesdottir, 2004) propose neurocognitive models of awareness. The CAM was developed to explain awareness deficits in AD, and so may be useful in order to help explain loss of insight in other neurodegenerative disorders, such as FTD, CBD and PSP. This model includes a comparator system within the central executive to monitor mismatches between a personal data base and experiencing cognitive failures and successes. When a mismatch is detected, a signal is sent to the metacognitive awareness system which leads to conscious experience of failure. If the executive system is not functioning correctly, the comparator mechanism may not pick up mismatches, and so failures in cognitive performance may not reach metacognitive output or conscious awareness. This is referred to as "executive anosognosia" in the CAM model.

Consistent with this type of model, studies examining lack of insight and deficits in self-awareness in AD have shown associations with impaired insight and dysexecutive symptoms (Dalla Barba, Parlato, Iavarone, & Boller, 1995; Souchay et al., 2003; E. Weinstein, Friedland, & Wagner, 1992), as described in Chapter 1 Section 1.4. A relationship between impaired self-monitoring on a memory task and executive function deficits was also found in one study with patients with FTD (Souchay, Isingrini et al. 2003). Other studies have shown that tasks of frontal function predicted anosognosia in AD (Lopez et al., 1994). Further investigations with patients with AD showed that anosognosia correlated with hypoperfusion of the

right dorsolateral frontal lobe (Reed et al., 1993; Starkstein et al., 1995). McDaniel et al (McDaniel, Edland, & Heyman, 1995) suggested that spread of pathology to the frontal cortex resulted in more impaired insight.

A number of recent neuroimaging studies have implicated various aspects of self-awareness with the frontal lobes (Fossati et al., 2003; Miller et al., 2001; Phan et al., 2004; Vogeley et al., 2001) and have also implicated parietal structures (Vogeley, Bussfeld et al. 2001). Abu-Akel (2003) differentiated mentalising about oneself into three modules, relating to three different neuroanatomical areas. He argues that parietal structures are where information about oneself is initially represented, followed by evaluation in the limbic-paralimbic module for personal relevance and meaning, and finally the information about oneself undergoes executive processes in the prefrontal module, including error monitoring, planning, inhibition of responses, similar to Morris and Hannessdottir's "executive anosognosia" proposal (Morris & Hannesdottir, 2004). Rankin et al (2005) propose that according to the Abu-Akel model, FTD patients have more damage to prefrontal regions of this third module which is involved in the processing of information about oneself.

Researchers have called for more *"quanititative operationalisation of this core criterion"* in FTD patients to improve characterisation of this symptom and the disease as a whole (Rankin et al., 2005). More complete and systematic studies of impaired awareness in atypical and subcortical dementias have also been called for (Aalten et al., 2005; Leritz et al., 2004). In AD, where assessment of awareness has been studied widely, four main approaches are used to assess level of awareness, similar to ISA assessment in TBI (Clare et al., 2002): Clinician Global Ratings (Reed et al., 1993), discrepancy scores between patient's own perception of their difficulties and a caregiver on a rating scale or questionnaire (Vasterling, Seltzer, &

Watrous, 1997), discrepancy between patients self-ratings and scores on objective tests (Anderson & Tranel, 1989) or some combination of these methods (Green et al., 1993). In a study by Rankin et al (2005) that explicitly examined awareness for changes in personality in FTD patients, discrepancy scores between patient and SO on a personality questionnaire was used. Predictions on a memory task was the method adopted by Souchay et al (2003) in their study that examined self-monitoring of memory processes in FTD patients. A multidimensional approach to the assessment of insight has been called for, as conclusion based on a single assessment can provide only a limited perspective on the multifaceted phenomenon (Ownsworth et al., In Press).

The present study is the first systematic and quantitative examination of insight deficits in FTD, CBD and PSP in relation to neuropsychological performance. The goal of this study was to examine awareness of deficit impairment across patient groups with FTD, CBD and PSP and controls with a multidimensional approach using the models of awareness proposed by Toglia and Kirk (2000) and Crosson and colleagues (1989) as a guideline. Insight was assessed on three levels: Metacognitive Knowledge, Online Emergent Awareness and Online Anticipatory Awareness, similar to the method adopted in Chapter 3.

4.2 Method

Eligible participants who agreed to participate gave informed consent according to the Declaration of Helsinki, the Mater Misericordiae Hospital Ethics Committee and TCD Ethics Committee.

Participants

55 participants took part in this study. This included 35 patients and 20 neurologically healthy controls.

Patients (N= 35)

The patients were recruited from academic hospitals in the Republic of Ireland from June 2003 to March 2005. The referred patients were then screened for core criteria for the disorders (FTD, PSP, CBD) by the neurologist at a Dementia and Movement Disorders Clinic. Exclusion criteria included previous head trauma, a history of major psychiatric disorder, evidence of previous or concurrent neurological condition such as stroke or epilepsy, use of neuroleptic agents or history of drug or alcohol abuse. The clinical diagnosis of FTD was assigned using the current consensus criteria as first proposed by the Lund and Manchester Groups (1994), Neary et al (1998) and reviewed in 2001 (McKhann et al., 2001). Clinical diagnosis of PSP was assigned according to the consensus clinical criteria as first published by Litvan et al (1996), the modified version of this original set of criteria as suggested by a Scientific Issues Committee (SIC) taskforce in 1993 that distinguishes clinically possible, clinically probable and clinically definite disease (Litvan et al., 2003). Clinical diagnosis of CBD was assigned according to suggested clinical research

criteria from the SIC taskforce (Litvan et al., 2003). Using these criteria 14 FTD, 11 CBD, and 10 PSP were defined.[2]

Controls (N= 20)

The control groups consisted of caregivers of the patient participants. The caregiver group consisted of 20 neurologically healthy adults, 16 of whom were spouses/partners of the recruited patients and 4 of whom were caregiver children of patients.

Table 4.1 shows demographic and screening data for all groups participating in this study.

Materials and Procedure

The clinical neurological examination (2 hours) was conducted by a clinical neurologist and neuropsychological assessment was examined by the research psychologist (2.5 hours) and included all awareness measures. All participants were given rest sessions at regular intervals and upon request during both sessions. Testing took place in a clinical research room in the Mater Misericordiae University Hospital or in the home of the patient.

---

[2] Of the 14 cases assigned the clinical diagnosis of FTD, 11 were frontal (behavioural) variant FTD from clinical onset, 1 was the Primary Progressive Aphasia subtype at onset (pure word finding deficits in first 2 years) with subsequent involvement of other frontal dysexecutive features, 1 was Semantic Dementia with prominent new disinhibition at time of testing) and 1 case was Motor Neurone Disease with FTD (impaired lexical fluency with attentional deficit).

Screening Tests

A number of short screening tests for cognitive functioning were administered throughout both sessions. These included:

National Adult Reading Test (NART) (Nelson, 1982; Nelson & Willison, 1991) as an estimate of pre-morbid intellect. *Mini-Mental State Examination (MMSE)* (Folstein, Folstein, & McHugh, 1975) as a measure of general cognitive function. *Hospital Anxiety and Depression Scale (HADS),* an indication of anxiety and depression levels (Zigmond & Snaith, 1983).

Neuropsychological Measures

Each participant underwent a comprehensive neuropsychological examination that included the following tests:

Memory

Subtests from the Wechsler Memory Scale (WMS 111) (Wechsler et al., 1998) included: Immediate and Delayed Story Recall (Logical Memory 1 and 11) , Immediate and Delayed Face Recognition (Faces 1 and 11), Digit Span (Forward and Backward), Spatial Span (Forward and Backward).

Attention

Trail Making Test (Reitan, 1958) Psychomotor Processing Speed (Part A) and Mental Flexibility Component (Part B)

Sustained Attention to Response Task (SART) (Robertson et al., 1997)

The Fixed SART consisted of digits from 1 to 9 being presented in a fixed order from 1 to 9 for 25 presentations of this sequence. Participants are required to respond by a mouse-click to every go-target and withhold response for the rare no-go target

(the number '3'). The no-go target was presented 25 times. In this study, a modified version of the Fixed SART was administered to allow for slower processing of information by the patients. Presentation of the targets and masks were slowed by 50% compared to previous studies. Three blocks of the Fixed SART were run.

Executive Functions

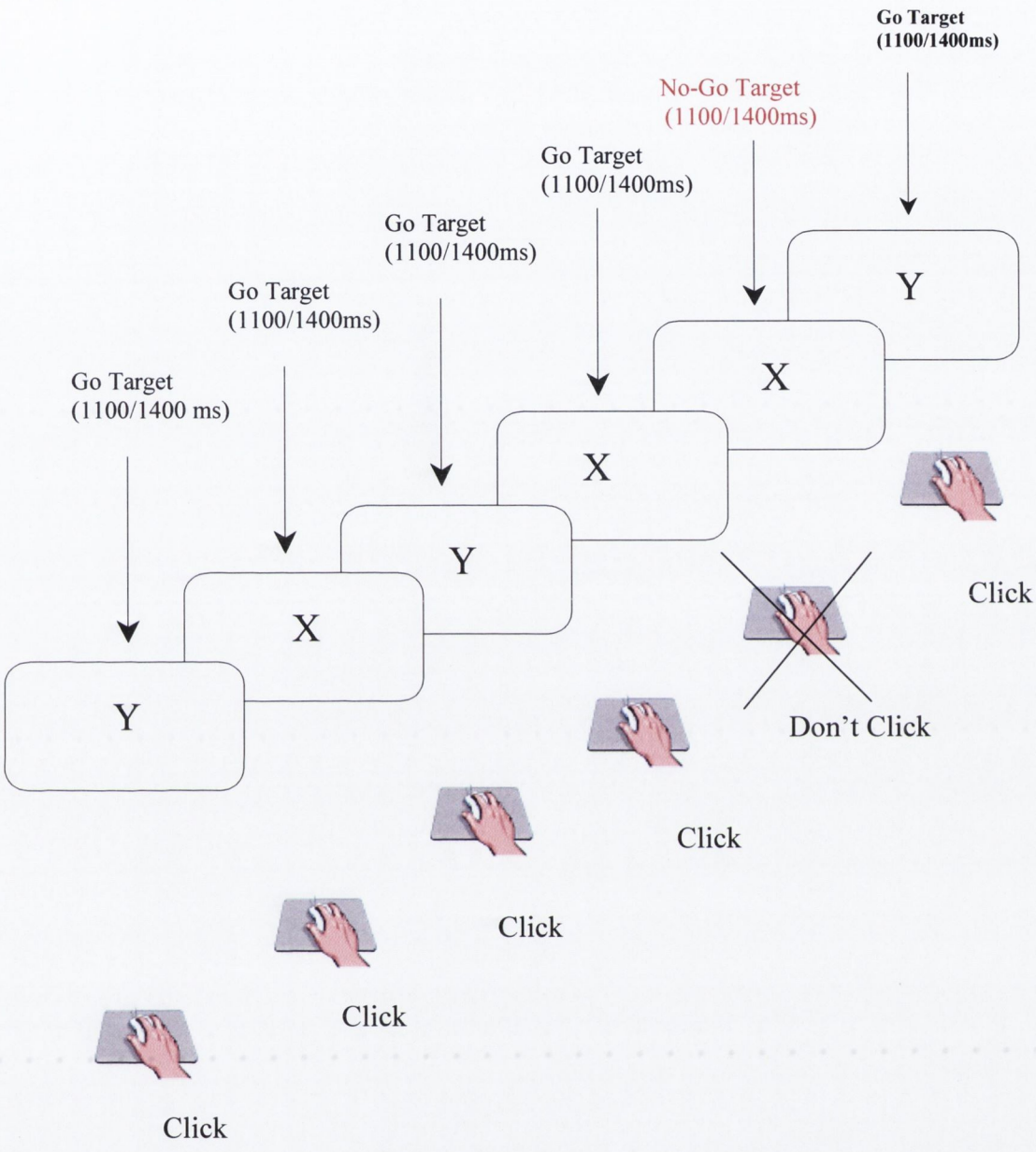
Verbal Fluency- FAS test (Benton & Hamsher, 1976; Spreen & Strauss, 1998)

Frontal Assessment Battery (Dubois et al., 2000).

XY response inhibition task (Garavan, Ross, Murphy, Roche, & Stein, 2002; Garavan, Ross, & Stein, 1999)

A modified version of the XY response inhibition task described in Garavan et al (2002) was run. In this task, participants were required to respond with a mouse click to the letters X and Y. The letters X and Y are presented in a series of an alternating pattern (X, Y, X, Y, X, Y....). Participants were required to intermittently inhibit their responses when the alternating sequence was interrupted, that is when two X's or two Y's were presented in succession. (X, Y, X, Y, X, X....). There were 40 lures (no-go target) and 590 go-targets. Figure 4.1 below depicts the XY response inhibition task.

Figure 4.1: An Example of the XY Task



Visuospatial Orientation

Subtest from Visual Object and Space Perception Battery (VOSP) (Warrington, 1991)

Position Discrimination.

Real-life/Other

Naturalistic Actions

NAT Naturalistic Actions Test (Schwartz, Buxbaum, Ferraro, Veramonti, & Segal, 2002)

The NAT is a performance-based test to measure how well participants perform naturalistic action, learned sequential, object-oriented behaviour in the pursuit of everyday goals. Task 3 subtest was used in this study (Packing a lunchbox and a schoolbag).

Facial Emotion Processing (Best, Williams, & Coccaro, 2002; Ekman & Friesen, 1976)

Participants were presented with 60 black-and-white photographs of men and women expressing happiness, surprise, fear, disgust, anger and sadness. Photographs were presented for 5 seconds each and participants were asked to choose the label that best described the expression, as in Best et al (2002).

Self and Significant Other Questionnaire Ratings for competency on everyday Activities, frontal systems behaviours, everyday cognitive failures, empathic tendency.

Each patient completed the following scales during testing sessions, as did a significant other (SO) regarding each patient's behaviour. For controls, a reliable informant also filled in a significant other rating regarding the controls' competency (usually a neurologically-healthy sibling, friend or child). Therefore, each participant (both patients and controls) had a self-rated and significant other-rated questionnaire from the three questionnaires, the Patient Competency Rating Scale (PCRS) (Prigatano & Altman, 1990), the Frontal Systems Behavioural Scale (FrSBe) (Grace & Malloy, 2002) Cognitive Failures Questionnaire (CFQ) (Broadbent et al., 1982). The Measure of Empathic Tendency (MET) (Mehrabian & Epstein, 1972) was also administered to each participant but was not rated by a SO.

Awareness Measures

Metacognitive Knowledge

1) Awareness Interview- Adapted (Anderson & Tranel, 1989)

The interview schedule and scoring was identical to that described in Chapter 3, with the exception that it included an additional two questions regarding vision and motor function (see Appendix). This meant that the Awareness Index ranged from 0-20 in this study.

2) Discrepancy Scores on Questionnaires Self and Other (Grace & Malloy, 2002; Grace et al., 1999; Prigatano & Altman, 1990)

Of the questionnaires that were administered, three were given to both the participants and a significant other to rate. These were the Patient Competency Rating Scale (PCRS) (Prigatano et al., 1986), the Frontal Systems Behavioural Scale

(FrSBe) (Grace & Malloy, 2002) and the Cognitive Failures Questionnaire (CFQ) (Broadbent et al., 1982). These three questionnaires were rated for identical items by participants and a SO. The discrepancy score was derived by subtracting the significant-other rated score from the self-rated score.

Online Emergent Awareness

3) Error-Monitoring on the SART (McAvinue et al., In Press; O'Keeffe et al., 2004)

As participants performed the three blocks of the SART, error awareness was measured by asking participants to verbally indicate their awareness of making an error by saying the word "hit" following an error of commission on the no-go target (the number '3'). The proportion of aware errors over the total number of errors made gave the overall error awareness score on the SART, similar to the method used in Chapter 2 and Chapter 3.

4) Error-Monitoring on XY

Participants were asked to verbally indicate their awareness of their errors by saying "hit" following an error of commission-failing to inhibit their response on the no-go target. The proportion of aware errors over the total number of errors made gave the overall error awareness score on the XY task.

5) Prediction Tasks- Discrepancy between predicted and actual performance in cognitive tasks (Fischer, Trexler et al., 2004; Marcel et al., 2004).

Accuracy of prediction on cognitive tasks was assessed using WMS Digit Span (Forwards and Backwards); WMS Spatial Span (Forwards and Backwards) and Verbal Fluency (FAS). Participants were asked prior to performing each of these tasks to predict how many numbers they would remember (Digit Span), or patterns they would remember (Spatial Span) or words they could come up with in one minute beginning with a certain letter (Verbal Fluency). Prediction accuracy was assessed in each task by calculating an average score [(Prediction minus Performance)/Prediction X 100].

4.3 Results

Section A: Demographic and Screening Data

Groups were well matched in terms of sex [χ^2= 5.495, df=3, p>.05], education level [F(3, 49)=0.625, p>.05], estimated pre-morbid IQ [F(3, 44)=1.713, p>.05], HADS Anxiety [F(3, 48)=1.831, p>.05], HADS Depression [F(3, 48)=0.353, p>.05]. The three patient groups did not differ significantly in terms of disease duration [F(2, 31)=.519, p>.05]. There was a significant effect for participant age [F(3, 50)=4.118, p<.05]. Post hoc bonferroni comparisons indicated that PSP participants were significantly older than control participants (p<.05). No other groups differed significantly in terms of age. There was a significant effect for MMSE scores [F(3, 50)=0.519, p<.05]. Post hoc bonferroni comparisons indicated that FTD patients had significantly lower MMSE scores than both PSP patients (p<.05) and controls (p<.01). Demographic data, screening scales and scores of self-report measures for all participants are detailed in Table 4.1.

Table 4.1: Demographic Data for Participants (N= 55)

	FTD	CBD	PSP	Control
N	14	11	10	20
Sex (m, fm)	8, 6	4, 7	7, 3	6, 14
Age (Years)	58.23 (SD 10.10)	64.82 (SD 7.48)	67.6 (SD 5.89)	57.1 (SD 10.13)
Education Level (Yrs)	12.92 (SD 3.09)	11.36 (SD 3.56)	13.11 (SD 4.05)	12.73 (SD 3.28)
NART Estimated Performance IQ	107.33 (SD 13.6)	98.11 (SD 14.21)	109.10 (SD 14.47)	107.20 (SD 7.32)
HADS-Anxiety	6.46 (SD 4.81)	4.91 (SD 4.46)	3.22 (SD 3.53)	7.11 (SD 4.45)
HADS-Depression	5.77 (SD 4.43)	5.63 (SD 4.43)	4.67 (SD 3.61)	4.63 (SD 2.71)
MMSE	22.5 (SD 7.81)	24.91 (SD 4.72)	28.3 (SD 2.39)	29.3 (SD .92)
Disease Duration (Months)	40.08 (SD 25.99)	32.18 (SD 15.24)	47.40 (SD 53.63)	N/A.

Section B: Patient and Control performance on Neuropsychological Tests

One-way between-groups Analysis of Variance revealed a number of significant between-group differences. Table 4.2 displays the mean scores and standard deviations for each group on each neuropsychological test. ANOVA F- values and significance level are also displayed, as are post hoc bonferroni comparisons, indicating where the specific group differences lie. Only Trail Making Test, Sustained Attention to Response Task and XY Response Inhibition Task did not show significant effect of group, although a trend towards significance was evident for group on the Sustained Attention to Response Task (p=.06). These post hoc bonferroni comparisons indicated that the patient groups did not show significant differences from each other in the neuropsychological tests. Patients differed significantly from controls across a number of measures. The FTD patient group performed significantly worse than controls on immediate and delayed verbal recall (WMS Logical Memory I and WMS Logical Memory II), non-verbal immediate and delayed recall (WMS Faces I and Faces II), WMS Digit Span, Verbal Fluency (FAS test), Frontal Assessment Battery, Percentage Accuracy on Recognition of Facial Emotions and Naturalistic Actions Task. The CBD patient group performed significantly worse than the control participants on immediate and delayed verbal recall (WMS Logical Memory I, WMS Logical Memory II), delayed non-verbal recall (WMS Faces II), WMS Spatial Span, Verbal Fluency (FAS test), Frontal Assessment Battery, VOSP Dot Discrimination, Percentage Accuracy on Recognition of Facial Emotions and Naturalistic Action Task. The PSP patient group performed significantly worse than control participants on non-verbal immediate and delayed recall (WMS Faces I and Faces II) Verbal Fluency (FAS test), Frontal Assessment Battery, VOSP Dot Discrimination, Percentage Accuracy on Recognition of Facial Emotions and the NAT.

Table 4.2: Neuropsychological Test Scores for Each Participant Group

Cognitive Functions	*Cognitive Tests*	*FTD*	*CBD*	*PSP*	*Caregivers*	F *Value*	P *Value*
Memory Mean (SD)	WMS Logical Memory I	19.0 (12.9)[a]	23.09 (8.8)[b]	29.2 (6.5)	34.45 (9.6)[ab]	7.446	<.001
	WMS Logical Memory II	7.38 (8.1)[a]	12.27 (8.7)[b]	15.8 (5.4)	20.15 (7.6)[ab]	7.818	<.001
	WMS Faces I	28.57 (5.7)[a]	29.91 (6.4)	28.8 (3.9)[c]	35.35 (5.5)[ac]	5.642	<.01
	WMS Faces II	28.86 (4.5)[a]	29.64 (4.7)[b]	29.1 (4.7)[c]	35.6 (4.7)[abc]	8.027	<.001
	WMS Digit Span	13.67 (6.0)[a]	15.55 (5.2)	15.5 (3.4)	18.5 (3.3)[a]	3.230	<.05
	WMS Spatial Span	9.31 (3.1)	8.55 (5.2)[b]	10.6 (5.1)	13.35 (4.1)[b]	3.883	<.05
Attention Mean (SD)	TMT-Difference B-A (secs)	119.35 (77.3)	149.52 (82.8)	144.94 (98.6)	79.07 (76.7)	1.956	>.05
	SART (Mean Errors of Commission)	6.185 (4.4)	4.542 (3.0)	2.815 (2.4)	3.316 (2.2)	2.585	>.05
Executive Functions Mean (SD)	XY Task (Errors of Commission)	20.56 (10.0)	15.63 (8.9)	18.44 (8.9)	14.3 (9.3)	1.099	>.05
	Verbal Fluency FAS (Total)	25.43 (19.9)[a]	20.09 (9.3)[b]	27.11 (15.8)[c]	47.75 (14.8)[abc]	9.976	<.001
	Frontal Assessment Battery	11.43 (5.3)[a]	9.09 (5.0)[b]	12.22 (3.5)[c]	16.60 (1.6)[abc]	10.132	<.001
Visuospatial Mean (SD)	VOSP Dot Discrimination	19.0 (1.2)	17.55 (3.2)[b]	17.2 (2.9)[c]	19.8 (0.5)[bc]	5.105	<.01
Other/Real-Life Functions Mean (SD)	Recognition of Facial Emotions (% Acc.)	61.53 (23.8)[a]	57.88 (18.9)[b]	59.67 (17.9)[c]	78.99 (9.4)[abc]	5.252	<.01
	Naturalistic Actions Task	1.69 (2.3)[a]	2.11 (1.9)[b]	2.22 (1.9)[c]	4.35 (1.2)[abc]	6.491	<.01

Post hoc comparisons between each group are indicated by the following symbols:
[a] Post hoc comparisons ($p<.05$): Controls vs patients with FTD
[b] Post hoc comparisons ($p<.05$): Controls vs patients with CBD
[c] Post hoc comparisons ($p<.05$): Controls vs patients with PSP

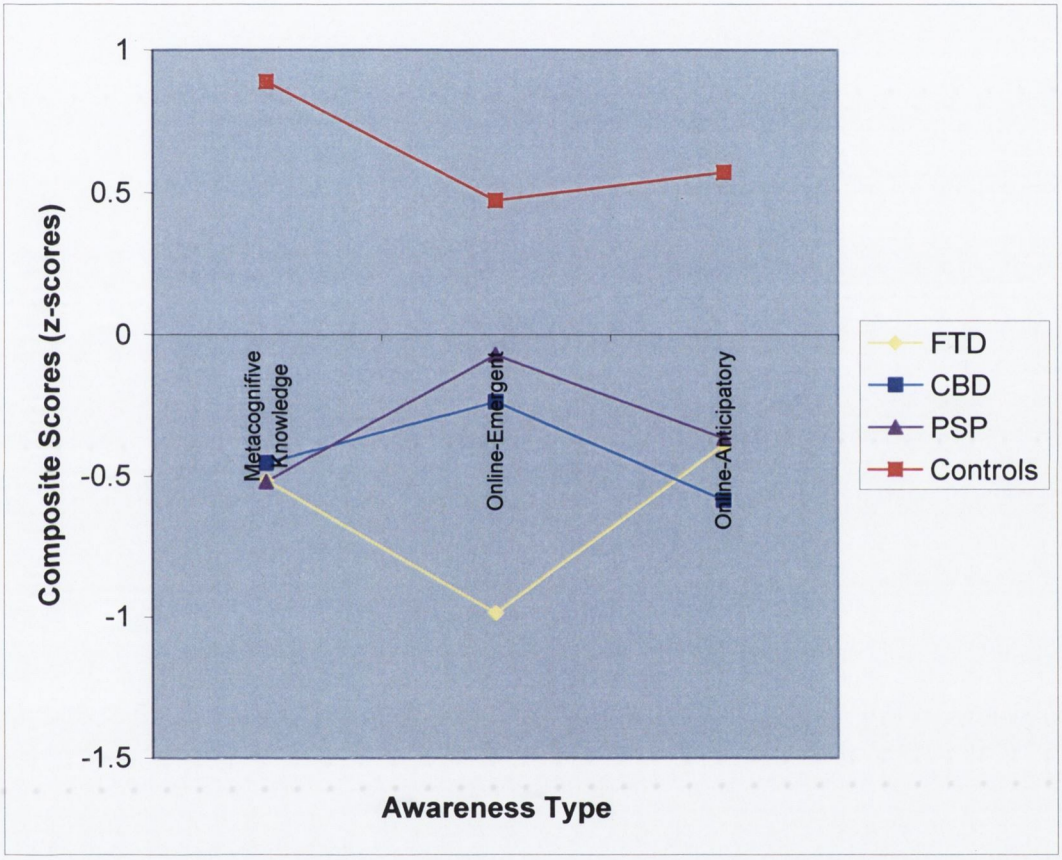
Section C: How do Groups differ on Overall Composite Measures of Insight?

Composite scores for Metacognitive Knowledge (Awareness Index, PCRS discrepancy score, FrSBe discrepancy score and CFQ discrepancy score), Online Emergent Awareness (Overall Error Awareness on Fixed SART and XY task) and Online Anticipatory Awareness (Overall Prediction Tasks Accuracy, Digit Span, Spatial Span, Verbal Fluency) were calculated based on z-scores from each of the individual awareness measures. In order to investigate performance across the different awareness measures, a mixed ANOVA was run. The between- subjects factor was Group (Four levels: FTD, CBD, PSP and Controls) and the within-subjects factor was Awareness Type (Three levels: Metacognitive Knowledge, Online Emergent Awareness, Online Anticipatory Awareness). The interaction effect between Group and Awareness Type was significant [$F(6, 78) = 2.603$, $p<.05$] There was a significant main effect of Group [$F(1, 39)= 13.094$, $p<.001$]. The main effect for Awareness Type [$F(2, 78)= 1.988$, $p>.05$] did not reach significance.

Post hoc t tests (independent and paired-samples) indicated that each patient group had poorer awareness composite scores for each of the three types of awareness composite scores compared to controls [Metacognitive Knowledge: Controls V FTD: $t(15.253)=4.768$, $p<.001$; Controls V CBD: $t(12.579)=5.429$, $p<.001$; Controls V PSP: $t(28)=8.425$, $p<.001$; Online-Emergent: Controls V FTD: $t(26)=5.281$, $p<.001$; Controls V CBD: $t(27)=2.531$, $p<.05$; Controls V PSP: $t(27)=2.081$, $p<.05$; Online-Anticipatory: Controls V FTD: $t(28)=2.738$, $p<.05$; Controls V CBD: $t(27)=3.113$, $p<.01$; Controls V PSP: $t(28)=2.887$, $p<.01$]. Furthermore, FTD patients were significantly worse on Online-Emergent Composite scores than both CBD patients [$t(15)=-2.200$, $p<.05$] and PSP patients [$t(15)=-3.097$, $p<.01$]. Post hoc t tests also

revealed that FTD patients were significantly worse on Online-Emergent than Metacogntive Knowledge [t(7)=5.391, p<.001] and than Online- Anticipatory [t(6)=-4.290, p<.01]. Controls scored significantly lower on Online-Emergent compared to Metacognitive Knowledge also [t(19)=3.242, p<.01]. No other significant differences were evident for within-subject factors.

Figure 4.2: Awareness Type by Group for Each Participant Group



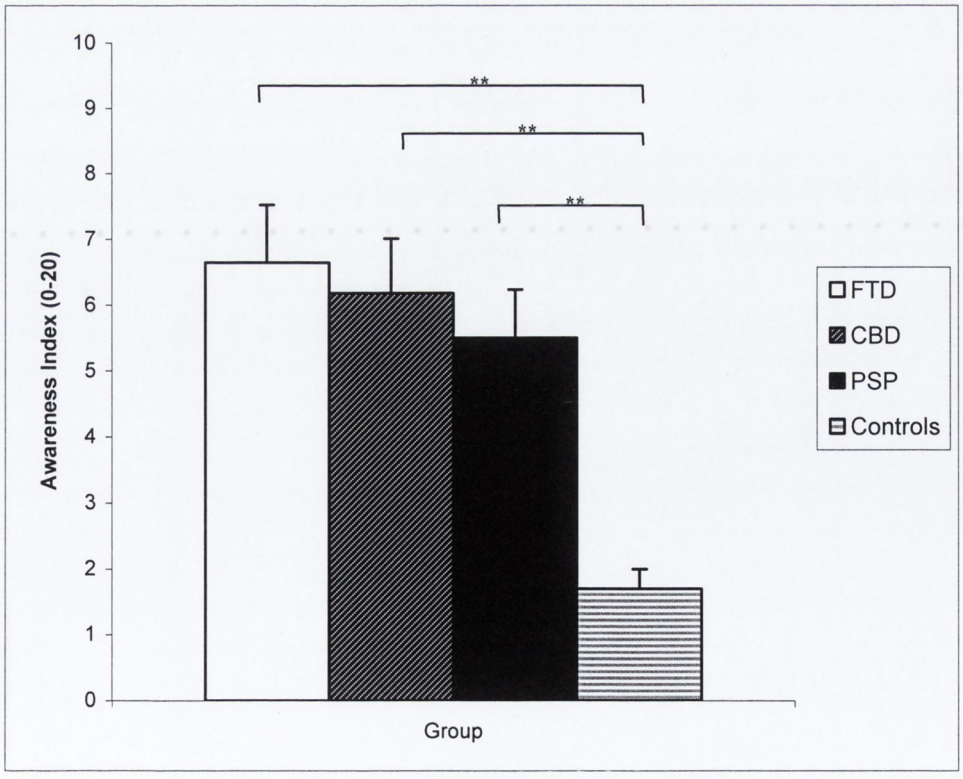
Section D: How do groups differ on Specific Awareness Measures?

Metacognitive Knowledge

1) Awareness Interview- Awareness Index

A one-way between-groups ANOVA was performed on the overall Awareness Index scores of all participants, patient and controls. There was a statistically significant effect of group on the Awareness Index [$F(3, 51)=14.874$, $p<.0001$]. Post hoc bonferroni comparisons indicated that the Awareness Index mean score for each of the FTD (6.64, SD 3.3), CBD (6.18, SD 2.8) and PSP (5.5, SD 2.3) patient groups differed significantly from controls (1.7, SD 1.3). Figure 4.3 below depicts the overall Awareness Index for each participant and where the group differences lie.

Figure 4.3: Awareness Interview Awareness Index (0-20) for each participant



** Significant at $p<.01$

The mean deviation scores of the ten categories in the Awareness Interview that contributed to the overall Awareness Index were then compared between the patient groups. One-way between subjects ANOVAs were performed. The mean deviation scores, the standard deviations and the F-values for each of the categories are displayed in Table 4.3.

Post hoc bonferroni comparisons indicated that in the Motor Category, PSP patients' mean deviation scores were significantly larger than those of both the FTD and CBD patients. In the "Overall Performance and Ability to Return to Work" category, FTD patients' mean deviation scores were significantly larger than those of CBD and PSP patients. In the Diagnosis category, there was a trend towards significance for larger mean deviation scores for FTD patients compared to PSP patients ($p = .06$).

Table 4.3: Awareness Interview Category Deviation Means, Standard Deviations and F-Values for Each Patient Group

Category Deviation Scores	**FTD**	**CBD**	**PSP**	**F Value**
Diagnosis	1.15 (SD 3.3)	0.64 (SD .5)	0.5 (SD .7)	3.43*
Motor	0.07 (SD .3)	0.55 (SD .5)	1.2 (SD .6)	16.38**
Thinking	0.77 (SD .9)	1.27 (SD .8)	0.78 (SD .7)	1.37
Orientation	0.79 (SD .8)	0.55 (SD .8)	0.2 (SD .4)	1.92
Memory	0.83 (SD .7)	0.55 (SD .7)	0.7 (SD .8)	.43
Attention	1.0 (SD .7)	1.13 (SD .6)	1.1 (SD .9)	.07
Language	0.5 (SD .8)	1.09 (SD .8)	0.4 (SD .5)	2.93
Visual Perception	0.07 (SD .3)	0.09 (SD .3)	0.2 (SD .4)	.49
Activities of Daily Living	1.14 (SD .9)	0.44 (SD .7)	0.44 (SD .7)	3.07
Overall Performance	0.93 (SD .7)	0.27 (SD .6)	0.2 (SD .4)	5.08*

* Significant at $p < .05$
** Significant at $p < .01$

2) Patient Competency Rating Scale (PCRS)

Mean scores and standard deviations for self-rated and other- rated PCRS are presented in Table 4.4 below for FTD, CBD, PSP and Controls for PCRS Total Competency, Activities of Daily Living, Cognitive, Interpersonal and Emotional Subscales. One-way between-subjects' ANOVA were conducted in order to establish group differences in mean scores on PCRS self-rated scale (total and subscale) and PCRS other-rated scale (total and subscales). F values and post hoc bonferroni comparisons are also displayed on Table 4.4.

As is evident from Table 4.4, post hoc bonferroni comparisons indicated that for self-rated PCRS, CBD patients rated themselves as significantly lower total competency than FTD patients ($p<.01$) and controls ($p<.05$). On the ADL subscale, CBD patients rated themselves as significantly lower competency than FTD patients ($p<.001$), PSP patients ($p<.05$) and Control participants ($p<.001$). PCRS Total Competency was rated by significant-others as significantly higher for Control participants compared to FTD patients ($p<.001$), CBD patients ($p<.001$) and PSP patients($p<.001$). The SO raters also rated each patient group significantly lower than Control participants on ADL (FTD: $p<.001$; CBD: $p<.001$; PSP: $p<.001$). ADL competency (other-rated) showed a trend towards statistically lower ADL ratings for CBD patients than FTD patients ($p=.07$). Each participants' SO rated cognitive competency as significantly lower for each patient than controls (FTD: $p<.01$; CBD: $p<.01$; PSP: $p<.001$). Interpersonal competency was rated as significantly lower for FTD patients ($p<.01$) and PSP patients ($p<.01$) compared to ratings for Controls. CBD were rated as significantly lower competency on the Emotional subscale than Controls by their respective significant others ($p<.01$).

Table 4.4: Patient Competency Rating Scale (Self- and Other-Rated) Mean Scores and Standard Deviations for Each group

PCRS Mean (SD)	**FTD**	**CBD**	**PSP**	**Controls**	**F Value**
PCRS Self Rated Total	126.62 (19.2)[d]	104.36 (12.5)[bd]	117.1(21.9)	123.95(11.5)[b]	4.719**
ADL Self-rated	34.39 (6.4)[d]	22.82 (5.3)[bdf]	29.9 (8.2)[cf]	37.25 (2.9)[bc]	17.136**
Cognitive Self-rated	33.46 (4.7)	30.64 (5.2)	33.3 (5.3)	33.7 (4.4)	1.081
Interpersonal Self-rated	30.0 (5.2)	25.91 (5.2)	26.7 (6.8)	27.2 (4.3)	1.429
Emotional Self-rated	28.77 (5.8)	25.0 (5.4)	26.7 (4.6)	25.8 (3.3)	1.564
PCRS Other Rated Total	100.85 (23.4)[a]	3.6 (6.0)[b]	90.78 (24.5)[c]	131.88 (9.7)[abc]	14.045**
ADL Other-rated	27.62 (7.7)[a]	20.6 (6.5)[b]	21.67 (9.3)[c]	38.12 (2.4)[abc]	21.082**
Cognitive Other-rated	27.31 (6.8)[a]	27.2 (6.3)[b]	24.56 (8.5)[c]	36.41 (3.9)[abc]	9.729**
Interpersonal Other-rated	21.62 (7.0)[a]	23.7 (5.4)	20.33 (5.3)[c]	28.94 (4.6)[ac]	6.381**
Emotional Other-rated	24.85 (4.9)	22.1 (3.5)[b]	24.22 (5.1)	28.41 (3.8)[b]	5.430**

* Significant at $p<.05$ level
** Significant at $p<.01$ level
[a]: Post hoc bonferroni comparison ($p<.05$): FTD vs Controls
[b]: Post hoc bonferroni comparison ($p<.05$): CBD vs Controls
[c]: Post hoc bonferroni comparison ($p<.05$): PSP vs Controls
[d]: Post hoc bonferroni comparison ($p<.05$): FTD vs CBD
[e]: Post hoc bonferroni comparison ($p<.05$): FTD vs PSP
[f]: Post hoc bonferroni comparison ($p<.05$): CBD vs PSP

Discrepancy Ratings on PCRS were calculated by subtracting self-ratings from other-ratings for the total competency score and each subscale. The mean and standard deviations for discrepancies between self- and other- rated PCRS for each group are displayed in Table 4.5. One-way between-subjects ANOVAs were conducted to compare discrepancy ratings for FTD, CBD, PSP and Control participants. Group effect was significant for total competency and each subscale.

Post hoc bonferroni comparisons indicated that FTD and PSP patients significantly overestimated their competencies on Total competency score compared to controls, who showed a tendency to underestimate their competencies. Examining the subscale discrepancy scores, FTD and PSP patients overestimated their competencies on ADL and cognitive competencies. On interpersonal competencies, FTD patients significantly overestimated compared to control discrepancy scores, while PSP patients overestimation compared to controls was approaching significance level (p=.07). Discrepancy scores for FTD and CBD patients were significantly higher than Controls on Emotional competencies subscale. Figure 4.4 displays discrepancy scores on PCRS for Total and Subscales for all participants.

Table 4.5: Patient Competency Rating Scale Discrepancy Mean scores, Standard Deviations, F Values, Significance Level and Post Hoc Comparisons for Each Group

PCRS Discrepancy[1] Mean (SD)	**FTD**	**CBD**	**PSP**	**Controls**	**F Value**
Total Discrepancy	22.58 (28.9)[a]	11.3 (17.5)	23.56 (19.1)[c]	-8.41 (11.9)[ac]	7.961**
ADL Discrepancy	6.0 (9.1)[a]	2.5 (5.8)	7.22 (7.5)[c]	-0.58 (1.8)[ac]	4.194*
Cognitive Discrepancy	5.0 (8.1)[a]	3.2 (3.7)	8.33 (8.5)[c]	-3.17 (4.9)[ac]	7.618**
Interpersonal Discrepancy	7.67 (9.5)[a]	2.2 (5.9)	5.67 (6.1)	-1.76 (5.5)[a]	4.993**
Emotional Discrepancy	3.58 (7.4)[a]	3.4 (6.8)[b]	1.78 (4.4)	-2.88 (3.03)[ab]	4.346**

* Significant at $p<.05$ level ** Significant at $p<.01$ level

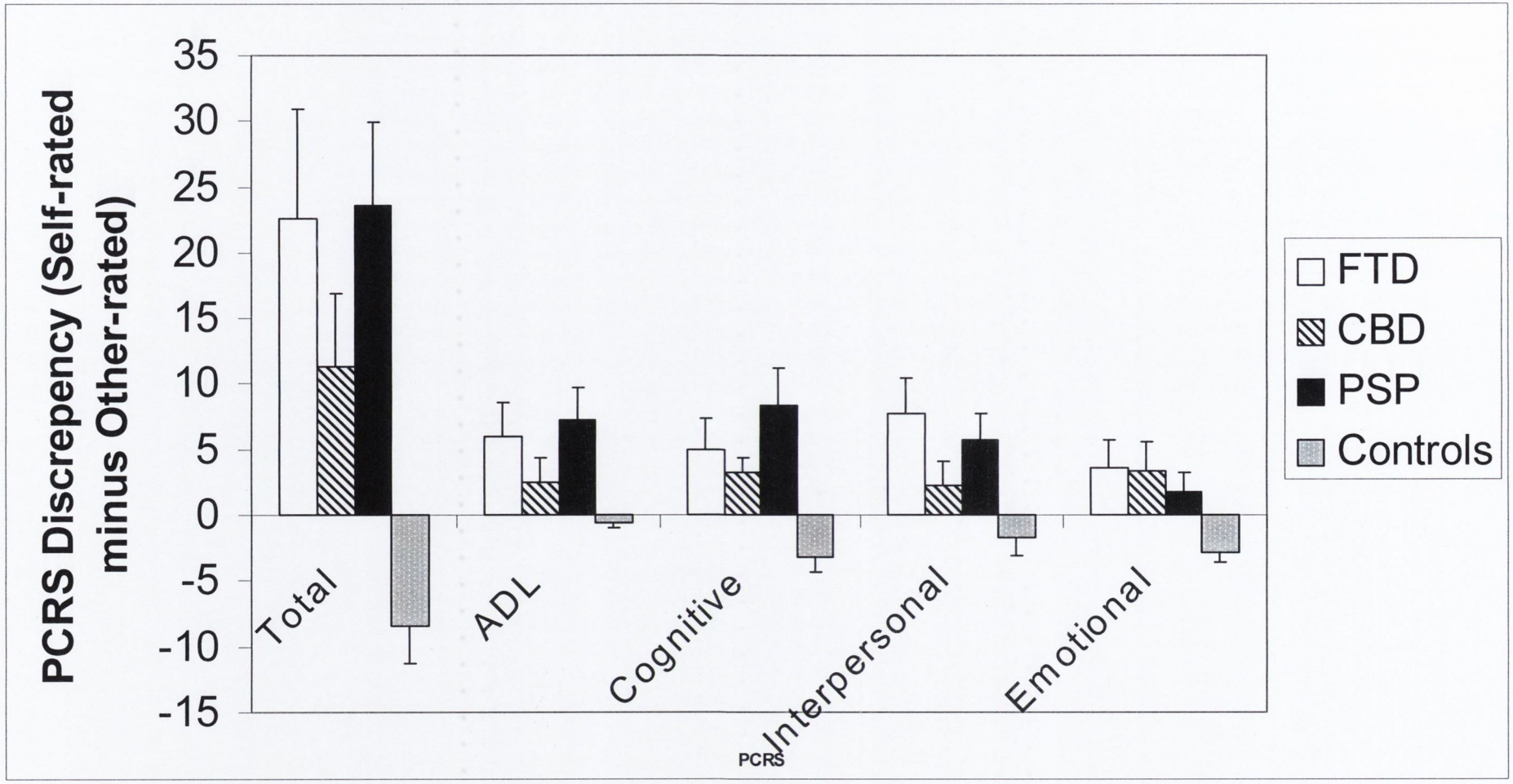
[a]: Post hoc bonferroni comparison ($P<.05$): FTD vs Controls

[b]: Post hoc bonferroni comparison ($P<.05$): CBD vs Controls

[c]: Post hoc bonferroni comparison ($P<.05$): PSP vs Controls

Note 1: Discrepancy Scores were calculated only for questionnaires that were available for both self and other (n=48)

Figure 4.4 Patient Competency Rating Scale Discrepancy Scores for Each Participant Group



3) Frontal Systems Behavioral Scale (FrSBe)

a) "At Present" FrSBe Ratings

Mean and standard deviations for Frontal Systems Behavioral Scale (At Present/Since Injury) for apathy, disinhibition and executive dysfunction subscales and total score are presented in Table 4.6 for both self and significant-other ratings. One-way between subjects ANOVAs and post hoc bonferroni comparisons were conducted to investigate group differences between each participant group for each of these ratings. F values and post hoc comparisons are also displayed on Table 4.5.

As is displayed in Table 4.6, there was no significant effect of Group for self-rated FrSBe total or subscale scores- Self-ratings on the FrSBe for FTD, CBD, PSP and Controls did not differ significantly from each other. For FrSBe other-rated total and subscales, the significant-others of FTD, CBD and PSP patients all rated them significantly higher on symptoms of frontal behaviour than the significant others of control participants. The one exception to this pattern was on the FrSBe disinhibition subscale, where the difference between ratings for FTD and Control participants did not reach significance level, although it appears to be approaching significance (p=.06).

Table 4.6: Mean and Standard Deviation Scores for Self and Other- Rated Frontal Systems Behavioral Scale (Total and Subscales).

FrSBe At Present Mean (SD)	FTD	CBD	PSP	Control	F Value
FrSBe Self Rated					
Total	104.27(28.)	100.4(20.5)	117.2 (26.8)	97.1 (24.7)	1.450
Apathy	36.09 (9.8)	35.9 (8.5)	39.2 (9.7)	31.2 (8.8)	1.909
Disinhibition	26.45 (12.7)	26.0 (6.6)	33.5 (12.3)	29.05 (10.4)	1.042
Executive Dysfunction	41.72 (10.1)	38.5 (8.7)	44.3 (10.1)	36.85 (8.9)	1.639
FrSBe Other Rated					
Total	121.23(34.3)[a]	129.3 (42.7)[b]	143.13(34.0)[c]	75.58(17.4)[abc]	11.671**
Apathy	40.62 (13.4)[a]	42.11 (14.8)[b]	49.38 (11.1)[c]	23.88 (1.8)[abc]	11.412**
Disinhibition	30.23 (9.9)	33.11 (12.9)[b]	36.13 (9.0)[c]	21.41 (5.1)[bc]	6.304**
Executive Dysfunction	50.38 (15.1)[a]	54.11 (16.3)[b]	56.88 (16.9)[c]	30.35 (7.32)[abc]	10.957**

** Significant at $p<.01$ level

[a]: Post hoc bonferroni comparison ($P<.05$): FTD vs Controls

[b]: Post hoc bonferroni comparison ($P<.05$): CBD vs Controls

[c]: Post hoc bonferroni comparison ($P<.05$): PSP vs Controls

One-way between-subjects ANOVAs were conducted to examine the discrepancy between self and other rated FrSBe Scales (Self-rated FrSBe minus Significant-Other rated FrSBe). Table 4.7 displays mean and standard deviations and each significant F value for total and subscale discrepancy scores.

As can be seen from Table 4.7, post hoc bonferroni comparisons indicated that the discrepancy scores for each patient group were significantly different from the discrepancy scores of the Control participants, for Total FrSBe, Apathy and Executive Dysfunction subscale. For discrepancy on the disinhibition subscale, only discrepancy scores of CBD patients differed significantly from Controls, although differences between those of FTD patients and Controls was approaching significance (p=.06). As can be seen from Figure 4.5, the direction of discrepancy scores for FTD, CBD and PSP patients indicate that the SOs of each of these patient groups rated them as demonstrating significantly more symptoms of executive dysfunction than the patients rated themselves. For Control participants, SOs rated them as having fewer frontal syndrome behaviours than they rated themselves.

Table 4.7: Discrepancy Scores (Total and Subscale) on Frontal Systems Behavioral Scale for each group

FrSBe Discprepancy[1] Mean (SD)	**FTD**	**CBD**	**PSP**	**Controls**	**F Value**
Total Discrepancy	-16.27 (40.8)[a]	-27.78 (36.3)[b]	-22.63(29.2)[c]	23.94 (23.5)[abc]	2.980**
Apathy Discrepancy	-5.0 (15.6)[a]	-5.89 (15.3)[b]	-9.38 (10.8)[c]	8.4 (7.6)[abc]	1.964**
Disinhibition Discrepancy	-3.27 (14.8)	-6.78 (10.9)[b]	-2.13 (11.4)	8.76(9.6)[b]	2.945**
Executive Dysfunction Discrepancy	-8.0 (14.4)[a]	-15.11 (12.8)[b]	-10.63 (10.8)[c]	6.71(10.1)[abc]	2.808**

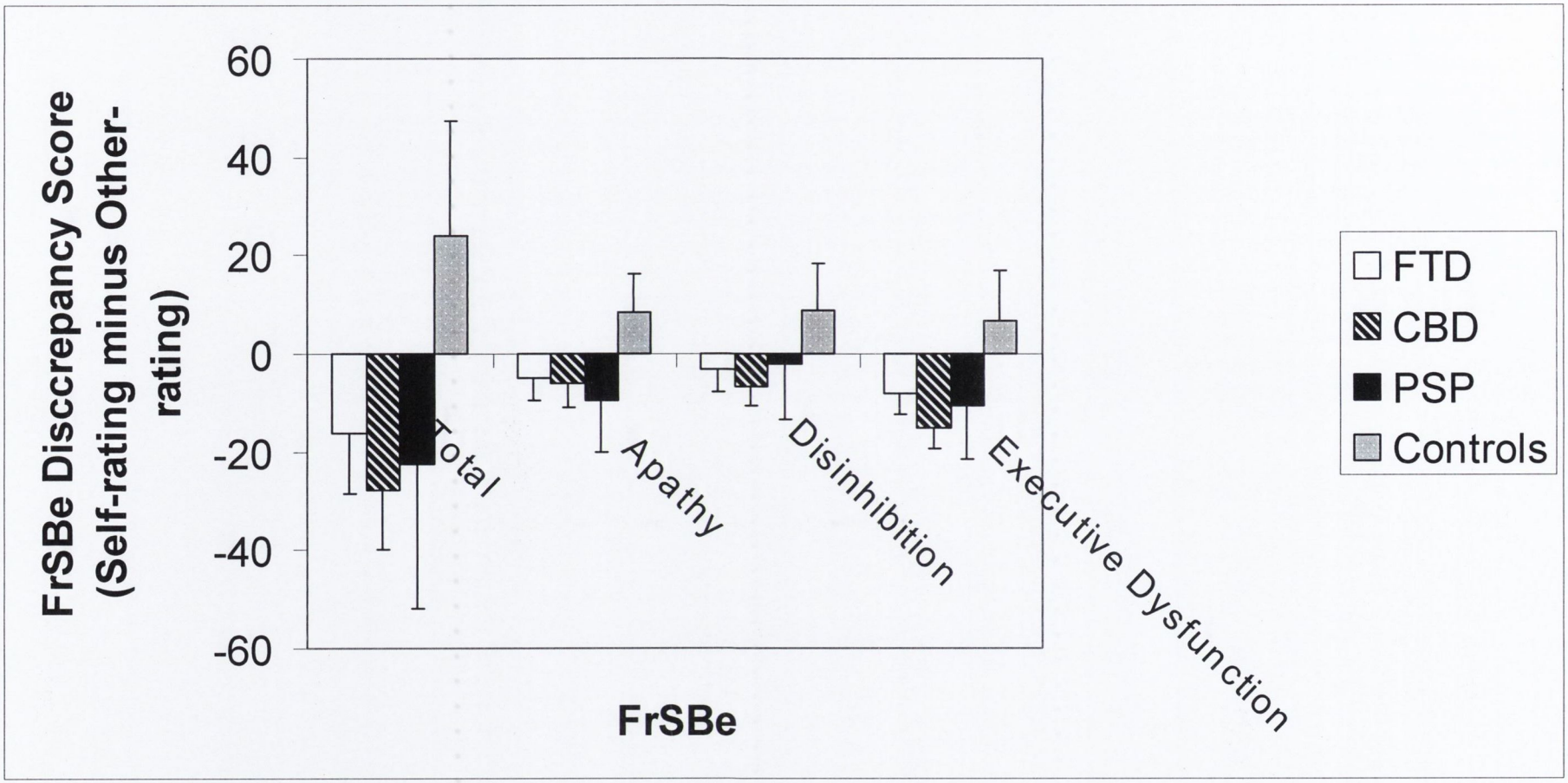
Note 1: Discrepancy Scores were calculated only for questionnaires that were available for both self and other (n=45), and therefore there is a slight difference between FrSBe mean scores and FrSBe discrepancy scores.

[a]: Post hoc bonferroni comparison ($P<.05$): FTD vs Controls

[b]: Post hoc bonferroni comparison ($P<.05$): CBD vs Controls

[c]: Post hoc bonferroni comparison ($P<.05$): PSP vs Controls

Figure 4.5: FrSBe Total and Subscale Discrepancy Scores (Self-Rated minus Other-Rated) for Each Participant Group



b) Ratings of Behaviour Changes for TBI participants

FTD, CBD and PSP participants and their SOs rated the FrSBe scale for all items on the pre-morbid scale and on the "At Present" scale also. In order to examine patient participants' and their SO's perceptions of how their behaviour may or may not have changed since their brain injury, a Behaviour-Change score was calculated by subtracting FrSBe before-illness Total score from FrSBe At Present Total score for both self and significant other ratings. This led to a Self-Rated Behaviour Change score and an Other-Rated Behaviour Change score, presented in Table 4.8 below.

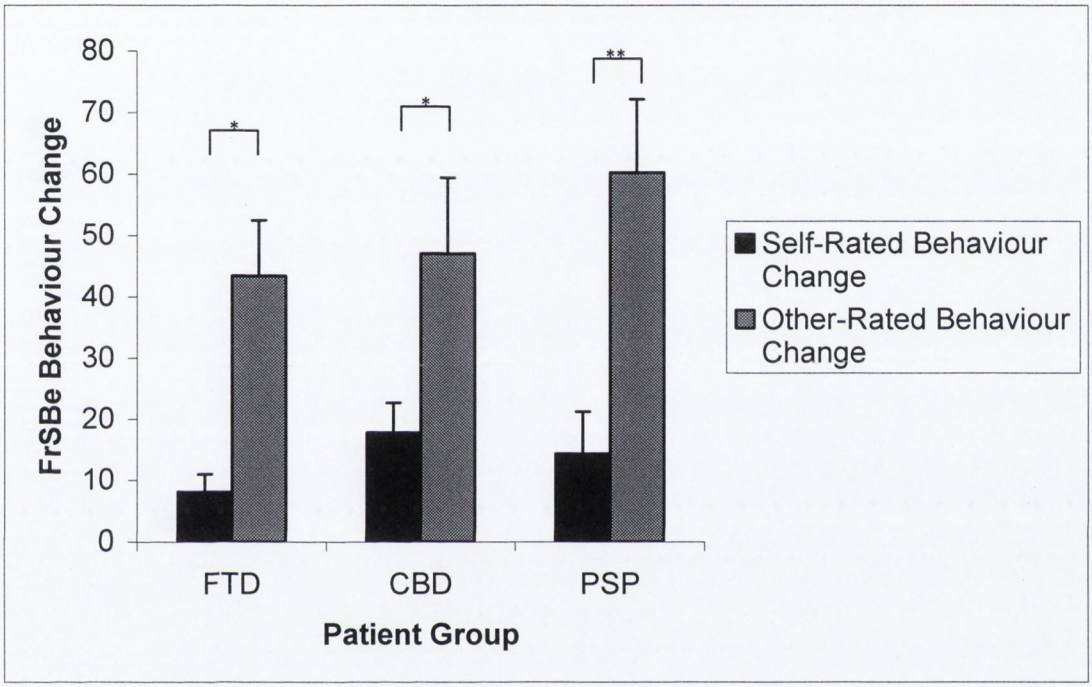
Table 4.8: Magnitude of Behaviour Change (FrSBe At Present minus FrsBe Pre-illness) Self-rated and Other-Rated for all Patient Groups

FrSBe Behaviour Change	**FTD**	**CBD**	**PSP**
FrSBe Self Rated Behaviour Change Mean (SD)	8.1 (9.4)	17.8 (15.7)	14.4 (21.9)
FrSBe Other Rated Behaviour Change Mean (SD)	43.5(9.1)	47.1 (12.3)	60.3(33.6)

To examine if perceptions differed between patient groups of Behaviour Change, a mixed between-subjects ANOVA was run. Between-subjects variable was Group (three levels: FTD, CBD and PSP) and the within-subjects variable was Behaviour Change (two levels: Self-rated Behaviour Change and Other-rated Behaviour Change). Main effect for Behaviour Change (Self rated and Other Rated) was significant [$F(1, 24)=29.377$, $p<.0001$]. The effect for Group [$F(2, 24) = .647$, $p>.05$] and interaction between Group and Behaviour Change [$F(2, 24) = .591$, $p>.05$] were not significant. Figure 4.6 shows that SO rated Behaviour Change is significantly

higher than self-rated Behaviour Change for all three patient groups, indicating that SOs rate each patient's behaviour change as much greater than the patients rate their own behaviour change.

Figure 4.6: Self- and Other-Rated Behaviour Change (FrSBe At Present minus FrSBe Pre-Illness) Since Diagnosis for FTD, CBD and PSP Patients



1) Self-rated and Other-rated Cognitive Failures Questionnaire (CFQ)

Mean CFQ actual and z-scores are displayed below in Table 4.9 for self-rated CFQ, other-rated CFQ and discrepancy scores between self and other rated CFQ. One-way ANOVAs were conducted to examine if groups differed on self-rated, other-rated and discrepancy scores on CFQ. Groups did not differ significantly on self-rated CFQ. Post hoc comparisons indicated that for other-rated CFQ, each patient group was rated as having more incidents of everyday cognitive failures than control

participants' ratings (FTD: $p<.01$; CBD: $p<.001$; PSP: $p<.001$). Discrepancy scores for CBD and PSP patient group were significantly different from Control discrepancy scores (CBD: $p<.001$; PSP: $p<.001$). The difference between discrepancy scores for FTD and Controls showed a trend towards significance ($p=.08$).

Table 4.9: Self- and Other-Rated Cognitive Failures Questionnaire Ratings and Discrepancy Scores Between Self- and Other-Ratings for all Participant Groups.

	FTD	**CBD**	**PSP**	**Controls**	**F Value**
CFQ self (Actual, *z-scores*)	35.6, *0.05*	29.6, *-0.30*	24.5, *-0.60*	39.6, *0.29*	1.850
CFQ other (Actual, *z-scores*)	16.7, *0.39*[a]	19.7, *0.76*[b]	19.8, *0.77*[c]	7.7, *-0.72*[abc]	10.133**
CFQ Discrepancy (*z-scores*)	*-0.27*	*-0.93*[b]	*-1.3*[c]	*1.08*[bc]	8.758**

** Significant at $p<.01$

[a]: Post hoc bonferroni comparison ($P<.05$): FTD vs Controls

[b]: Post hoc bonferroni comparison ($P<.05$): CBD vs Controls

[c]: Post hoc bonferroni comparison ($P<.05$): PSP vs Controls

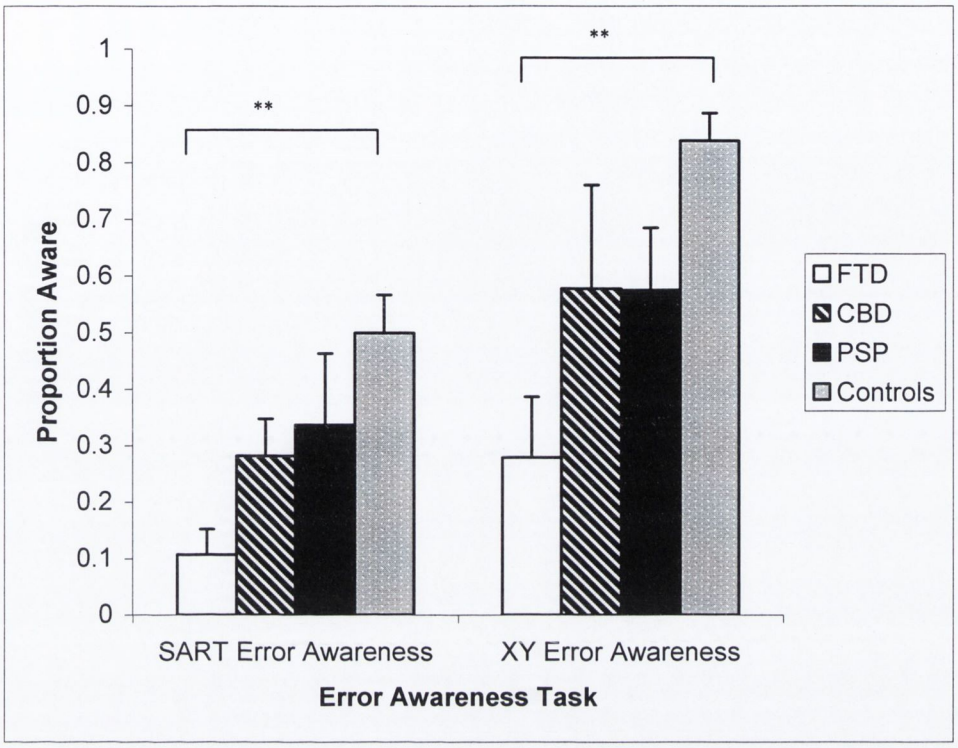
Mean error awareness scores and standard deviations on Sustained Attention to Response Task (Fixed SART) and XY Response Inhibition task are displayed as proportion of aware errors in Table 4.10.

A mixed between-subjects ANOVA was conducted in order to compare error-monitoring on SART and XY tasks. Group was the between-subjects factor (Four levels: FTD, CBD, PSP and Controls) and Awareness Task was the within-subjects factor (Two levels: SART and XY). Main effect for group was significant [$F(3, 31) = 12.652$, $p<.001$]. The main effect for Awareness Task was also significant [$F(1, 31)= 15.844$, $p<.001$], indicating that across all groups, participants monitored more errors on XY error-monitoring task than SART error-monitoring task. No significant interaction between Group and Awareness Task was evident [$F(3, 31)=0.775$, $p=0.775$]. In order to discern which groups differed significantly from each other, post hoc independent samples t-tests were run. These tests indicated that the FTD group monitored significantly fewer errors than Controls on both SART and XY tasks [SART: $t(20)=-3.424$, $p<.01$; XY: $t(26)=-5.465$, $p<.001$]. The difference between FTD and CBD monitoring on the SART was approaching significance [$t(12)=-2.047$, $p=.06$]. The other patient groups did not differ significantly from Controls, although the differences for CBD and Controls on SART was approaching significance ($t(22)= -2.045$, $p=.053$) and for PSP and Controls on XY were also approaching significance level ($t(20)= -2.186$, $p=.051$). Figure 4.7 depicts proportion of aware errors on SART and XY error monitoring tasks for each participant group and where the significant differences lie.

Table 4.10: Mean Proportions of Error Awareness on SART and XY tasks for all groups.

Awareness Tasks	**FTD**	**CBD**	**PSP**	**Controls**
Mean proportion SART Error Awareness Mean (SD)	0.1069 (SD. 11)	0.2818(SD .18)	.3369 (SD .36)	0.4990 (SD .27)
Mean proportion XY Error Awareness Mean (SD)	0.2790 (SD .30)	0.5778 (SD .45)	0.5750 (SD .33)	0.8379 (SD .22)

Figure 4.7: Error Monitoring on the SART and XY for Each Participant Group



** Significant at $p<.01$

Online Monitoring of Errors: Anticipatory Awareness

Prediction Tasks

In the prediction tasks (Digit Span Forwards and Backwards, Spatial Span Forwards and Backwards and Verbal Fluency), the average difference between predictions and performances on each task was converted into a percentage score [(prediction-performance)/prediction X 100]. In order to investigate differences in prediction accuracy between groups, a series of one-way between-subjects ANOVA was run for prediction accuracy on each prediction task. Table 4.11 below displays mean

prediction accuracy and SD for each of the prediction tasks, as well as the overall total of prediction accuracy.

Post hoc bonferroni comparisons indicated that only for prediction accuracy on Spatial Backward tasks, CBD patient groups significantly overestimated their prediction compared to Controls who underestimated their predictions ($p<.05$). On total overall prediction accuracy, CBD significantly overestimated their predictions compared to Controls who again underestimated their predictions ($p<.05$), as did PSP patients ($p<.05$). FTD patients showed a trend towards overestimating their predictions compared to Controls ($p=.06$).

Table 4.11: Prediction-Performance Tasks for Each Participant Group. Mean Percentage Accuracy for Each Task and F-Values with Significance Levels.

Prediction Task Accuracy Mean Percentage (SD)	**FTD**	**CBD**	**PSP**	**Controls**	**F Value**
Digit Forward Percentage Accuracy	-15.01 (51.4)	-28.02 (25.5)	-7.51 (28.6)	-51.32 (48.8)	3.010*
Digit Backward Percentage Accuracy	17.86 (41.3)	16.78 (47.2)	7.83 (33.5)	-22.39 (58.0)	2.284
Spatial Forward Percentage Accuracy	3.66 (33.8)	29.54 (45.3)	12.38 (44.4)	-10.57 (32.2)	2.539
Spatial Backward Percentage Accuracy	27.59 (41.2)	41.2 (41.1)	8.89 (43.8)	-9.89 (45.9)	3.492*
Verbal Fluency Percentage Accuracy	-27.45 (53.9)	4.11 (49.3)	10.06 (51.8)	-42.48 (57.5)	2.494
Overall Percentage Accuracy	3.65 (33.2)	14.05 (35.0)	6.42 (24.9)	-27.27 (30.2)	5.343**

* Significant at $p<.05$ level
** Significant at $p<.01$ level

Section E: What is the relationship of Awareness Composite Scores to other measures for patient participants?

Table 4.12 shows selected correlations between each category of composite awareness and other measures of cognitive function.

Table 4.12: Pearson Product-Moment Correlations Between Awareness Category and Selected Measures for Patients

Variable	**Metacognitive Knowledge**	**Online-Emergent Awareness**	**Online-Anticipatory Awareness**
NART IQ	.155	.421*	**.252**
HADS Anxiety	.286	.106	**-.027**
HADS Depression	.115	-.030	**-.457***
FAB	.370*	.267	**.706****
Total Digit Span	.376*	.420*	**.480****
CFQ (Other-rated)	-.651**	.317	**-.295**
Total FAS words	.594**	.144	**.567****
Emotion Recognition Accuracy	.590**	.164	**.791****
MET	.383*	-.217	**.227**
NAT	.556**	.386	**.251**
FrSBe Total (Other-rated)	-.765**	.148	**-.548****
PCRS (Other-rated)	.663**	-.156	**.471***
Fixed SART Mean Errors	-.141	**-.439***	**-.095**
XY Response Inhibition Errors	**-.423***	**-.280**	**-.574****

* Significant at $p < .05$; ** Significant at $p < .01$

NART: National Adult Reading Test; HADS: Hospital Anxiety and Depression Scale; FAB: Frontal Assessment Battery; CFQ; Cognitive Failures Questionnaire; FAS: F, A, S Verbal Fluency Test; FrSBe: Frontal Systems Behavioural Scale; MET: Measure of Empathic Tendency; NAT: Naturalistic Actions Task; PCRS: Patient Competency Rating Scale; SART: Sustained Attention to Response Task

As can be seen from Table 4.12, higher Metacognitive Knowledge in patients was associated with a number of measures sensitive to frontal damage, including fewer frontal symptoms as indicated by higher scores on the Frontal Assessment Battery (FAB), a screening tool for frontal symptoms, higher ability on Verbal Fluency (FAS test), fewer frontal symptoms as measured by lower total scores on Frontal Systems Behavioral Scale (FrSBe), better recognition of facial emotions, fewer naturalistic

action errors (NAT) and fewer errors on XY response inhibition task. Metacognitive Knowledge was also associated with generally higher patient competency, as measured by total other-rated PCRS. Higher digit span scores and lower cognitive failures as measured by CFQ other-rated, were also correlated with better Metacognitive Knowledge, as was higher empathic scores on the Measure of Empathic Tendency.

Higher levels of Online-Emergent Awareness were associated with higher pre-morbid IQ scores, and higher digit span scores. It was also associated with better sustained attention abilities, as measured by the SART.

Higher levels of Online-Anticipatory Awareness was associated with a number of measures sensitive to frontal lobe damage, indicating that fewer symptoms of frontal dysexecutive syndrome were associated with better Online-Anticipatory Awareness, as measured by FAB, FAS Verbal Fluency, FrSBe total Score, XY response inhibition task, and facial emotion recognition. Higher other-rated patient competency was also correlated with better Online-Anticipatory Awareness. Better scores on Digit Span were also associated with this type of awareness. Higher scores on Online-Anticipatory Awareness were also associated with lower depression levels, as measured by self-rated HADS- Depression scale.

Section F: What is the relationship of the three awareness measures to each other?

In order to examine the relationship between the three different types of awareness measures, correlational analysis were conducted. There was a weak association between Metacognitive Knowledge and Online-Anticipatory Awareness [r=.443,

$p<.05$]. Online-Emergent Awareness did not show an association between either Metacognitive Knowledge [$r=.148$, $p>.05$] or Online-Anticipatory Awareness[$r=.309$, $p>.05$].

Section G: Were Caregivers representative as controls?

How representative were caregivers as control participants in this study was of slight concern, as initially some appeared to be making a slightly greater number of errors on some of the neuropsychological tests than other control groups in comparable studies. For this reason, a small number of age and education matched uninvolved controls were recruited from the Trinity College Department of Psychology Participant Database, to examine the possibility that the caregivers were performing differently than uninvolved controls on some measures of cognitive functioning. These "uninvolved controls" ($n=11$) were well matched to caregivers in terms of age [$t(29)= -1.255$, $p>.05$], sex [$\chi^2= .18$, df=1, $p>.05$]and education level [$t(29)=.412$, $p>.05$]. Each of the uninvolved controls underwent identical neuropsychological testing and were examined using identical awareness and insight measures as outlined above.

In terms of neuropsychological assessment, screening tools and questionnaire ratings, caregiver controls differed from unrelated controls on some select measures. Caregivers rated themselves as significantly higher on the HADS-Depression Scale than uninvolved controls [Caregivers (M=4.63, SD=2.7); Unrelated (M=2.18, SD=1.2); $t(26.48)=3.426$, $p<.01$]. Caregivers made significantly more errors of commission on the SART than unrelated controls [Caregivers (M=3.315, SD=2.2); Unrelated (M=1.33, SD=0.8); $t(24.84)=3.536$, $p<.01$] and on the XY response

inhibition task [Caregivers (M=14.3, SD=9.3); Unrelated (M=8.4, SD=5.2); t(27.528)=2.233, $p<.05$]. Caregivers controls also took longer in seconds on the Trail-Making Task Part A than unrelated controls [Caregivers (M=49.92s, SD=28.3); Unrelated (M=34.25, SD=10.5); t(26.6)=2.211, $p<.05$]. In terms of self-rated apathy, as measured on the FrSBe subscale, caregiver controls rated themselves as significantly higher apathy scores than uninvolved controls [Caregivers (M=31.2, SD=8.8); Unrelated (M=22.72, SD=4.3); t(28.713)=3.59, $p<.001$]. No other neuropsychological test, screening tool or self or other-rated questionnaire differed significantly between the caregiver control group and the uninvolved control group.

As there were some significant differences on some tests of attention and executive control (SART, TMT-Part A, XY) and screening tools related to emotional functioning (HADS-Depression, FrSBe-Apathy), it was deemed necessary to compare performance between the control groups across each of the awareness and error-monitoring tasks. There were no significant differences between caregiver control group and unrelated controls across any awareness or online monitoring measures [Awareness Interview: t(29)=1.301, $p>.05$; FrSBe Difference Score: t(25)=1.316, $p>.05$; PCRS Difference Score: t(25)=0.290, $p>.05$; CFQ Difference Score: t(25)=1.117, $p>.05$; SART Error-Awareness: t(25)=-1.184, $p>.05$; XY Error Awareness: t(28)=-0.596, $p>.05$; Total prediction Accuracy: t(21.762)=-0.457, $p>.05$], indicating that for the purposes of this study into indices of awareness, the caregivers were representative as controls.

4.4 Discussion

The findings from this study offer quantifiable evidence that patients with FTD, CBD and PSP all showed significant loss of insight across each level of awareness: Metacognitive Knowledge, Online-Emergent Awareness and Online-Anticipatory Awareness. Overall, FTD patients were significantly worse than CBD and PSP patients on Online-Emergent Awareness. For measures of Metacognitive Knowledge, each patient group overestimated their abilities or competencies compared to neuropsychological performance and compared to their significant other (SO) ratings of behaviour. For Online-Emergent Awareness measures, FTD patients were significantly impaired at monitoring their errors on two separate tasks of executive function. For Online-Anticipatory Awareness, CBD and PSP patients significantly overestimated their predicted performance on cognitive tasks and FTD patients showed a similar trend. There were significant relationships between impaired executive functions, memory, and general competencies and ISA for the patient participants. The results therefore indicate that, as hypothesized, impaired insight is not only a feature of FTD, but also of CBD and PSP on each level of insight.

Examining the results of the Awareness Interview in more detail indicates that overall, each patient group were inaccurate in their perceptions of difficulties across each of the cognitive abilities. This is consistent with findings by Anderson and Tranel (1989) who showed that patients with dementia showed unawareness of deficits using the Awareness Interview. The findings in the present study also showed that PSP patients were less accurate at reporting their deficits in the questions regarding motor functioning compared to both FTD and CBD patients.

This suggests that contrary to the findings in the brain injury literature which report that patients are more accurate at describing physical and motor difficulties than more abstract cognitive ones (Fleming & Strong, 1999), PSP patients may be less aware of the extent of their physical difficulties. FTD patients were less accurate at perceived performance on the tests overall and on their ability to return to normal activities when compared to CBD and PSP patients. This suggests that the FTD patients may be specifically impaired overall in terms of global or intellectual awareness of their difficulties and the ability to accurately predict the consequences of their difficulties on future activities compared to the other two patient groups.

Patients with FTD and PSP overestimated their abilities to perform everyday tasks, as measured by the Patient Competency Rating Scale (PCRS), compared to how their SO's rate these abilities. FTD patients overestimated significantly on each of the subscales, whereas PSP overestimated only for the ADL and Cognitive subscales. The overestimation on the ADL subscale is consistent with the findings for PSP patients from the Awareness Interview, that they inaccurately reported their motor problems. CBD patients only significantly overestimated on the emotional subscale of the PCRS. Each patient group significantly underestimated their frontal symptoms (FrSBe). This finding is consistent with the brain injury literature that suggests higher order cognitive functions and social and emotional changes are most difficult to accurately assess (Fleming & Strong, 1999). Rankin et al (2005) also showed that FTD patients were significantly inaccurate at describing their personality changes since disease onset. Interestingly in the present study, each patient group did report *some* change in their behaviour since before their illness, however, the magnitude of these reported changes was significantly smaller than the magnitude of behaviour change reported by their SO's on the same scale. For example, the magnitude of

behaviour change reported by SO's was five times greater than that reported by the FTD patients themselves, two times greater than CBD patients' own reports and four times greater for PSP patients' self-rated changes. For the CFQ discrepancy scores, CBD and PSP patients underestimated their everyday absentmindedness (CFQ discrepancy).

In terms of error-monitoring on the Sustained Attention to Response Task and XY Response Inhibition Task, although CBD and PSP patients appeared to monitor fewer of their errors compared to controls, FTD patients were the only patient group that significantly differed on each of the tasks from controls. Error-monitoring is thought to be largely reliant on the effective functioning frontal lobes (Stuss, 1991) and thus it is therefore intuitive that FTD patients, the patient group with potentially most frontal and prefrontal atrophy, would be most significantly impaired on this task. Indeed, as the composite scores for Online-Emergent Awareness indicated, FTD patients were more significantly impaired on this type of awareness than the controls and both the other two patient groups. This suggests that these error-monitoring tasks can sensitively discriminate FTD patients from CBD and PSP patients, which may have clinical relevance for diagnosis.

The composite scores from the prediction tasks (Online-Anticipatory Awareness) indicated that each of the patient groups overpredicted how they thought they could perform on cognitive tasks, compared to controls, who tended to underestimate compared to actual performance. This finding is consistent with that of Souchay et al (2003) who found that FTD patients significantly overestimated their predictions on a memory task. On the individual prediction-performance tasks, the Spatial Span Backward proved most difficult for CBD patients to accurately predict, as they

significantly overestimated their performance on this task compared to controls. This may be due to the finding that the other two patient groups, FTD and PSP, did not differ significantly from Control participants on the spatial span task.

On the neuropsychological test battery, the patient groups did not differ significantly from each other. Only differences between the patient groups and control group were observed. Compared to Control participants, patients with FTD performed significantly worse on each test of memory except for WMS Spatial Span, made significantly more errors on the Naturalistic Action Task, were significantly poorer in terms of accuracy at recognising facial emotions, scored lower on the Frontal Assessment Battery and had impaired verbal fluency. These results indicate that overall patients with FTD have quite an impaired neurocognitive profile compared to neurologically-healthy controls. On the visuospatial task they performed as well as controls, indicating that visuospatial capacities are spared, at least in our FTD group. These results are comparable to previous findings indicating that patients with FTD were significantly impaired on cognitive tasks compared to controls but not impaired on visuoconstruction tasks compared to controls (Diehl and Kurz, 2002). Patients with CBD were impaired on immediate and delayed logical memory recall, delayed non-verbal recall and spatial span. They were also impaired on tasks sensitive to frontal system functioning, including verbal fluency, Frontal Assessment Battery, accuracy at recognising facial emotion. They were also impaired on visuospatial task and Naturalistic Actions Task compared to controls. Patients with PSP performed significantly worse than controls only on the non-verbal sub-tests of the memory tests (Faces 1 and Faces 2, WMS 111). They were impaired on three of the tasks sensitive to frontal function (Recognition of Facial Emotions, Frontal Assessment Battery and Verbal Fluency) and were also impaired on the visuospatial tasks and

Naturalistic Actions Task compared controls. Soliveri et al (1999) showed that patients with PSP and CBD differed significantly only on a task of ideomotor apraxia (CBD more impaired) and on a task of executive dysfunction (PSP more impaired). In the present study, both patient groups performed worse than controls on similar tasks of executive dysfunction, visuospatial ability and naturalistic actions, but patients with CBD showed impairment across more of the memory subtests than patients with PSP. Patients with FTD were significantly impaired compared to Controls across a greater number of neuropsychological tests than either CBD or PSP patient groups. However, despite the trend towards patients with FTD performing worst, followed by patients with CBD, and patients with PSP performing slightly better on the cognitive tasks, there were no statistically significant differences across the neuropsychological tests between the three patient groups. This lack of significant differences between the patient groups may be as a result of the small number of participants in each patient group in the present study.

A number of significant associations were found in terms of neuropsychological performance and lack of insight. Better Metacognitive Knowledge, or global awareness of deficits, was quite strongly associated with preservation of a number of tests sensitive to frontal function (FAB, emotion recognition, FrSBe) and executive function (response inhibition, verbal fluency). Preserved memory abilities (short term working memory and everyday cognitive failures) were also associated with preserved Metacognitive Knowledge. Better general everyday competencies, as measured by the PCRS and the NAT also predicted better Metacognitive Knowledge. Naturalistic Actions are sequential, learned, object-orientated behaviour in the pursuit of accomplishing everyday goals and errors of these actions are thought to be as a result of depletion in cognitive resources related to self-monitoring, sustaining

action plans, selection of appropriate actions and in particular, the capacity to manipulate and monitor information in working memory (Schwartz, Buxbaum et al., 2002; Schwartz et al., 1999; Schwartz, Segal, Veramonti, Ferraro, & Buxbaum, 2002). Better Metacognitive Knowledge was also associated with higher levels of empathic tendency. Online-Anticipatory Awareness was also found to be strongly associated with tasks sensitive to frontal function, executive function, memory and general competency. Better Online-Anticipatory Awareness, the ability to accurately predict performance in future tasks was associated with lower levels of depression on the HADS. Emotional disturbance and loss of insight has been investigated in the AD literature, though findings are inconclusive with some researchers reporting lower depression levels associated with less awareness (Starkstein et al., 1997) and others failing to demonstrate this (Vasterling, Seltzer, Foss, & Vanderbrook, 1995). The finding from the present study suggests that the relationship between mood disturbances, such as depression and anxiety and loss of insight, should be investigated further, as it suggests that preserved insight may be linked with lower depression. Deficits in Online-Emergent Awareness, the ability to accurately monitor errors during performance was associated with poorer short term working memory and poorer premorbid intellectual capacities. Errors on a sustained attention task also predicted poorer ability to monitor these errors on two separate tasks. This association was also found in brain injury patients by McAvinue et al (2005) and in Chapter 3 of this thesis. The findings from this study show further support for the notion that the ability to maintain vigilant attention in routine and monotonous tasks may contribute to the maintenance of error awareness. The findings that executive functions and memory are related to both Metacognitive Knowledge and Online Awareness are consistent with investigations into loss of insight and AD (Feher, Mahurin, Inbody, Crook, & Pirozzolo, 1991; Lopez et al., 1994). An association

between impaired ability to predict performance on a memory task and executive dysfunction in FTD patients was also found in Souchay et al's (2003) metamemory study. It appears that preserved insight is also associated with better everyday functioning in terms of general competencies, better empathic tendencies, and less behavioural disturbances. Findings in the AD literature suggest that lack of insight leads to greater stress and caregiver burden (Seltzer et al., 1997). Although not directly investigated in this study, this association may be related to the behavioural disturbance and everyday functional impairments associated with more impaired insight, as found with the patients in the present study.

The control group in the present study were the caregivers, consisting mainly of spouses/partners and some children, of the recruited patients. Although perfectly representative on the awareness tasks which were the primary interest in this study, it is nevertheless interesting to note that compared with a completely uninvolved control group, the caregivers performed worse on attention tasks and also had significantly higher levels of depression and apathy than the uninvolved controls. This provides indirect evidence of burden and possibly stress related cognitive and emotional consequences of caring for a person with cognitive and physical impairment, such as the patients in the present study. The area of caregiver burden and stress certainly deserves further investigation in relation to loss of insight in patients with neurodegenerative disorders, such as FTD, CBD, and PSP and also PD and HD.

Overall, the findings from the present study fit with theoretical and functional models of self-awareness that differentiate between Metacognitive Knowledge and Online Awareness (Emergent and Anticipatory) (Toglia & Kirk, 2000). It appears that each

patient group are impaired across all three types of awareness, though, FTD patients are most dramatically impaired on Online-Emergent Awareness compared to CBD and PSP patients. This can in some way relate to Abu-Akel's (2003) neuroanatomical model of mentalising about the self. CBD and PSP patients may have more posterior and subcortical atrophy initially, linking in with the first two levels of Abu-Akel's model, relating to perceiving information about oneself and evaluating it for personal relevance and emotional meaning. However, the more prefrontal regions may be more preserved for the more detailed self-monitoring. FTD patients appear to have impairment at each level of awareness, and most notably at the self-monitoring level of executive functions. These correlate with Rankin et al's (2005) interpretation that FTD patients may have more severe damage to these structures required for the third module of self-related processing. The findings also relate to CAM, the neurocognitive model of Morris and Hannesdottir (2004), with FTD patients again most impaired on emergent-awareness or "executive anosognosia", perhaps due to most significant damage to their central executive system, and therefore the comparator mechanism of the CAM. Metacognitive Knowledge may represent "primary anosognosia" shown in the present study to be impaired in each patient group.

The clinical and theoretical implications of this study are manifold. From a clinical perspective, this is the first study to our knowledge to systematically examine loss of insight in FTD patients in terms of Metacognitive Knowledge and Online Awareness. As a core criterion, this study takes the process of quantifying this symptom further. Furthermore for CBD and PSP patients, it is clinically relevant for diagnosis, disease progression and effective assessment that both of these patient groups showed significant loss of insight, for both Metacognitive Knowledge and

Online Awareness. Loss of insight can lead to a number of negative outcomes such as increased stress and caregiver burden (Seltzer et al., 1997), poor patient-caregiver interaction (Hutchinson et al., 1997) and poor medication compliance (McGlynn & Schacter, 1989). With regard to the assessment of loss of insight, it is clear from these findings that it must be investigated quantitatively using a multidimensional approach to elucidate the nature and correlates of this multifaceted phenomenon that is awareness. These findings also support theories which fractionate different elements of self-awareness functionally and neuroanatomically. It is vital that further research is conducted to further examine the neural, cognitive and emotional correlates of loss of insight in these patient groups, and the effect that unawareness has on caregivers.

Chapter 5

General Discussion

Impaired self-awareness of deficits following brain injury or neurodegeneration has been identified as a significant barrier to motivation for rehabilitation, community re-integration and successful vocational outcome. It has also been strongly associated with maladaptive and psychopathological symptoms (Fleming et al., 1998; Hoofien et al., 2004; Noe et al., 2005; Sherer, Bergloff, Levin et al., 1998; Trudel et al., 1998). Therefore, it is clear that the mechanisms underlying and the factors associated with impaired self awareness must be better understood. Clinical models have identified at least three different levels or areas of awareness: Metacognitive Knowledge or intellectual awareness of deficits or illness; Online-Awareness consisting of Emergent Awareness, that is the ability to monitor errors in one's performance during a task; and Anticipatory Awareness, the ability to predict when deficits or difficulties are going to impact on future task performances (Crosson et al., 1989; Toglia & Kirk, 2000). However, previous research has primarily relied on a single, unimodal approach to assessment of intellectual awareness which has led to lack of consensus about predictors and correlates and a narrow understanding of the phenomenon of impaired self-awareness as a whole. There is certainly a paucity of research into impaired online awareness, its neuropsychological and behavioural correlates and its association with Metacognitive Knowledge or intellectual awareness. The central aim of this thesis was to explore impaired self-awareness by adopting a multimodal approach to assessment based on Toglia and Kirk's (2000)

clinical model of Metacognitive Knowledge and online awareness in order to further elucidate the neuropsychological correlates of the impairment as a whole, initially with individuals with Traumatic Brain Injury in Chapters 2 and 3, and later with individuals with neurodegenerative illnesses of FTD, CBD and PSP in Chapter 4.

5.1 Summary of Main Findings

The first study found a significant impairment of online awareness of errors in a group of 16 TBI patients compared to 16 matched neurologically-healthy controls. This replicated earlier work by McAvinue et al (2005) and is also consistent with a study by Hart et al (1998) that found impairments in error detection in TBI participants. The earlier work was extended to include a psychophysiological measure of this error-monitoring task. When control participants made an error on the simple laboratory task, they showed a large autonomic response, reflected in a significant increase of phasic EDA. The TBI participants did not show this specific error-related EDA increase even when they correctly monitored their errors, suggesting an attenuated emotional response to error-processing. A general arousal deficit in TBI participants was ruled out, as there was no significant differences between the baseline tonic measure of EDA or the phasic EDA to correct withholds on the task between TBI participants and controls. It was proposed that this may be an extension of Damasio's "Somatic Marker Hypothesis". He proposed that damage to the frontal lobe, particularly to the ventromedial cortex and its limbic connections may preserve cognitive evaluations but lack emotional content (Damasio, 1994). Even if patients detect errors, their emotional responses to the significance of committing errors may be reduced and this diminished emotional response may affect error correction as well as error recognition mechanisms. In terms of

contributing to overall knowledge of impaired self-awareness, this simple laboratory task provided a short, easy-to-administer, and non-intrusive way to assess online emergent awareness in a group of TBI participants.

Study 2 utilised this task as part of a multidimensional approach to assess three aspects of impaired self-awareness in 31 TBI participants and matched controls; Metacognitive Knowledge or intellectual awareness, Online-Emergent Awareness and Online-Anticipatory Awareness. In general, the TBI participants who demonstrated high self-awareness performed as well as, if not better than, the neurologically-healthy controls on most awareness tasks. In contrast, the low self-awareness group performed significantly worse across each aspect of awareness measured, and were particularly impaired on the emergent online awareness measures. The High and Low TBI self-awareness groups did not differ across the standard neuropsychological tests, but this study indicated that the low self-awareness group were rated as significantly more impaired in terms of poorer complex behaviour and interpersonal relationships. This is consistent with a number of previous studies that have failed to find an association between impaired awareness and standard neuropsychological assessments (Lanham et al., 2000; Newman et al., 2000; Prigatano & Altman, 1990). The findings that ISA was significantly associated with a number of complex behaviours indicative of executive dysfunction, as measured by the FrSBe including apathy, disinhibition and executive dysfunction is consistent with numerous findings that ISA appears to be associated with complex executive functioning (Hart et al., 2005; Ownsworth & Fleming, 2005; Trudel et al., 1998). This study further provided unequivocal evidence that the phenomenon of ISA following brain injury cannot be described adequately using a single, unidimensional approach and represented one of the first studies to

comprehensively examine ISA at a multidimensional level. Support for Toglia and Kirk's (2000) differentiation between Metacognitive Knowledge and Online-Awareness was demonstrated in that Online-Emergent composite score and Online-Anticipatory composite score were strongly associated with each other, whereas there was no such association with the Metacognitive Knowledge composite score, suggesting they are different constructs of the awareness phenomenon.

Study 3 extended this research to examine ISA in neurodegenerative patients with several overlapping clinical and pathological features. There has been a dearth of research in relation to ISA in atypical dementias, both cortical and subcortical. This study clearly demonstrated that ISA is not only a feature of FTD, but that CBD and PSP patients also show significant impairment across numerous measures of ISA. This is the first investigation, at least that this researcher is aware of, to demonstrate these deficits in ISA in CBD and PSP patients. FTD patients were significantly impaired on Online-Emergent Awareness, which may be a useful clinical marker of this disorder. Several neuropsychological correlates of ISA were found with these patient groups, suggesting that executive function and memory may underlie ISA with these neurodegenerative patients. These findings are consistent with the small number of studies that have examined aspects of ISA in FTD patients. Souchay et al (2003) found that metamemory impairments in FTD patients was associated with measures of executive function. Rankin et al (2005) demonstrated that FTD patients showed significant lack of awareness for their personality changes. In the current body of work, FTD patients indicated a small but very limited knowledge of behaviour change by rating their behaviour on the FrSBe as slightly different from pre-morbid levels. However SO's rated their behaviour change as over 5 times

greater than their self-rated behaviour changes, consistent with Rankin et al's findings that FTD patients lack awareness of personality changes.

The findings of an association between Metacognitive Knowledge and Online-Anticipatory Awareness are inconsistent with the findings from Study 2. However, the association in this study, Study 3, was a moderately weak one (.44), compared to the stronger association found between the two online awareness composite scores in Study 2 (.71). Furthermore, in Study 3, CBD and PSP patients did not differ significantly from controls on Online-Emergent Awareness tasks, whereas FTD patients did, which may have led to a lack of association found with online-emergent and Online-Anticipatory Awareness, when the three patient groups were analysed together.

5.2 Integration with Theoretical and Clinical Frameworks

In an effort to integrate the findings from each of Studies 1, 2, and 3, it is useful to examine the findings in the context of clinical and theoretical frameworks of ISA. Each of the studies from the current body of work strongly supports Toglia and Kirk's (2000) and Crosson et al's (1989) model that differentiates between Metacognitive Knowledge and Online Awareness. Toglia and Kirk's (2000) model also views awareness as a dynamic process which varies with task and situational context. They propose that individuals can show awareness in some areas but not in others as they view awareness as a whole to be the result of a complex interaction between Metacognitive Knowledge, online-awareness, beliefs, affective state, cognitive deficits, motivation and task demands. This would explain for example that while the TBI High self-awareness group in Study 2 perform exceptionally well

across the majority of awareness tasks, they still fail to recognise deficits that are revealed on neuropsychological testing. It would also explain that although both CBD and PSP patients in Study 3 are impaired on Metacognitive Knowledge and Online-Anticipatory Awareness, their Online-Emergent Awareness does not differ significantly from controls.

In terms of theoretical frameworks, the findings from each of these studies also support the neuropsychological and neurological theories that emphasise the role of the frontal network and its subcortical connections in self-awareness rather than discrete focal lesions, at least for patients where diffuse damage is a highly likely possibility, such as TBI and atypical dementias and parkinsonian syndromes and where ISA can be clearly seen for a wide variety of neuropsychological and behavioural symptoms. Prigatano (1991) proposed that the heteromodal cortex (including large areas of the prefrontal and frontal cortex, the inferior parietal lobe, suprmarginal and angular gyri and the anterior temporal lobes) is responsible for the integration of information about the external world with information about the internal state of the individual, necessary requirements for effective self-awareness. The distributed nature of the neurological networks proposed by Prigatano correlates well with the neural regions that are most vulnerable following TBI. As aspects of executive dysfunction have been related to the deficits in ISA in both Study 2 and Study 3, this would also support Stuss et al's Hierarchy of Brain Function model (Stuss & Benson, 1986; Stuss et al., 2001). The reformulation of the DICE model into the CAM model (Agnew & Morris, 1998; Morris & Hannesdottir, 2004) to explain different manifestations of ISA in AD is also useful here, in that it differentiates between executive anosognosia and primary anosognosia. In Study 2, the TBI low self-awareness group were most significantly impaired on Online-

Emergent Awareness, perhaps a manifestation of executive anosognosia. The interaction between the three measures indicated that although this group were impaired on both Metacognitive Knowledge and anticipatory awareness, suggesting a primary anosogonosia also, it appears that executive anosognosia is most starkly impaired in this group. This may also be reflected in their relatives' ratings of significant impairment on scales of interpersonal behaviour, disinhibition and basic living skills as measured by the FrSBe and PCRS compared to TBI high awareness group, as each of these behaviours and abilities may require an intact ability to monitor errors and maintain executive control of one's behaviour. In Study 3, FTD patients were the only patient group that significantly differed from controls on both tasks of online-emergent behaviour, although the three patient groups differed significantly in terms of more primary anosognosia or general Metacognitive Knowledge of deficits. Again, this may indicate that FTD patients showed most significant executive anosognosia, suggesting a failed comparator mechanism from an impaired executive system. While the three patient groups did not differ significantly from each other across any of the neuropsychological measures or behavioural scales, the correlation matrix did show a significant association between Online-Emergent Awareness and sustained attention and working memory ability, both purported to involve executive control of behaviour.

5.3 Limitations of the Current Studies

The small sample sizes and heterogeneity of TBI patients may have limited the generalisability of the findings and precluded further statistical analysis such as regression or structural equation modelling. However, the trend that approximately 50% of individuals with TBI demonstrate severe ISA and almost all demonstrate

some element of ISA suggests that the findings are quite consistent with the prevalence of ISA in the literature. Furthermore, the findings on ISA and the conclusions drawn are not based on a single score, as found in many previous studies. Considering the comprehensive nature of the assessments, these findings may provide a richer, more in-depth analysis of the types of deficits in awareness that exist in TBI patients. In relation to Study 3, the numbers of FTD patients assessed (n= 14) is equivalent to the 12 FTD patients assessed in Rankin et al's (2005) study and more than double the number of those assessed in Souchay et al's (2003) metamemory study with only 6 FTD patients assessed. Both CBD and PSP are relatively rare neurodegenerative disorders, with estimates of an incidence of 1 PSP patient per 100,000 of the general population. Considering the rarity and the related difficulty in recruitment of suitable patients who could partake in such an in-depth neuropsychological and neurological assessment, it is felt that 11 CBD and 10 PSP patients is quite representative of the Irish population of such patients. Every patient that was referred from across Ireland who consented to partake in the research and who met the inclusion criteria within the 18 month referral period was assessed. With regards to the small sample sizes in Study 3, there were a number of trends towards significance in terms of neuropsychological performance between the patient groups. It is felt that with even slightly larger patient groups, these trends would have reached significance level and differentiated between the groups. Nevertheless, the significant findings with regard to impaired awareness of deficits across each level of awareness measured are perhaps even more remarkable considering the small sample sizes. Future research could continue the present work with larger sample sizes of TBI participants and FTD, CBD and PSP patients, examining specifically intellectual awareness, Online Emergent Awareness and Online Anticipatory Awareness.

As the TBI patients from Study 1 were recruited from Headway Ireland, the National Head Injuries Association, the researcher did not have access to medical files or scan data from these patients. However, these limitations were rectified in Study 2, as the TBI patients in this study were recruited from the National Rehabilitation Hospital in Co. Dublin, allowing access to medical files and scan details for the majority of recruited patients.

This study focussed on the neuropsychological aspects of impaired awareness. It is clear that psychological and socio-cultural factors also influence the manifestation of impaired awareness in clinical populations (Clare, 2004b; Ownsworth et al., In Press). Future investigations could certainly investigate the interactions of psychological and socio-cultural factors with the neuropsychological factors found in the present studies.

5.4 Future Research

Following the findings from the current body of work, a number of interesting possible future research possibilities arises. For example, the findings of a specific error-monitoring deficit in TBI patients could be further explored with imaging studies, employing ERP analysis to examine error-related negativity as they perform error-monitoring tasks. fMRI studies, such as that in Hester et al's (2005) recent fMRI study examining error-awareness in healthy individuals could inform the area of neural correlates of online awareness in clinical populations enormously, as could many of the other recent developments in imaging studies and self-reflection in neurologically-healthy individuals (Johnson et al., 2002; Vogeley et al., 2004), as suggested by the very recent fMRI study with TBI participants involving self-

reflection of personality traits (Schmitz et al, In Press). As was shown in particular in Study 3, further research is urgently required regarding the impact on carers of patients with neurodegenerative disorders with behavioural disturbances and in particular ISA on stress, cognitive performance and emotional distress in the carers. With regard to variability in the normal population, a large-scale study of online-awareness abilities in neurologically-healthy individuals may inform the clinical research in the area.

The next step in relation to research with TBI and neurodegenerative patients is to examine possible rehabilitative steps that may aid the development of ISA. One potential route was identified in a recent study by McAvinue et al (2005) who showed that feedback on error led to a reduction of errors of commission in a simple laboratory task of sustained attention. As errors of commission were significantly correlated with error awareness in that study and in two studies in the current thesis, it is possible that feedback on error may also improve error awareness. Future research could examine this possibility.

Many of the studies in the literature in relation to rehabilitation and vocational or behavioural outcome have based their findings on one single discrepancy score or rating, representative of intellectual awareness only. It is essential for a more comprehensive understanding of the relationship between ISA and outcome that future investigations adopt a multidimensional approach, such as that adopted here, which will also show the correlation of impaired online awareness with outcome.

5.5 Conclusion

The current body of work has shown that without a doubt, awareness is a multifaceted phenomenon and must be assessed as such. This thesis has shown that the oft-neglected aspects of impaired awareness in clinical populations, online error awareness, can, and should be assessed in conjunction with intellectual awareness of deficits or Metacognitive Knowledge. It has also been shown that TBI patients with impaired self-awareness of deficits display more behavioural disturbances, and have more difficulties with interpersonal relationships than TBI patients with more preserved awareness. This thesis has also been the first to systematically and comprehensively examine impaired self-awareness of deficits in terms of intellectual, emergent and anticipatory awareness in FTD patients. The findings are also the first to show that impaired self-awareness may also be a feature of CBD and PSP patients as well as FTD patients.

References

Aalten, P., van Valen, E., Clare, L., Kenny, G., & Verhey, F. (2005). Awareness in dementia: a review of clinical correlates. *Aging Ment Health, 9*, 414-422.

Abell, F., Krams, M., & Ashburner, J. (1999). The neuroanatomy of autism: a volex-based structural analysis of whole brain scans. *Cognitive Neuroscience, 10*, 1647-1651.

Abreu, B. C., Seale, G., Scheibel, R. S., Huddleston, N., Zhang, L., & Ottenbacher, K. J. (2001). Levels of self-awareness after acute brain injury: how patients' and rehabilitation specialists' perceptions compare. *Arch Phys Med Rehabil, 82*, 49-56.

Abu-Akel, A. (2003). A neurobiological mapping of theory of mind. *Brain Res Brain Res Rev, 43*, 29-40.

Adams, J. (1984). Head Injury. In J. Adams, A. N. Corsellis & C. W. Ducjen (Eds.), *Greenfield's neuropathology* (pp. 85-124). London: Edward Arnold.

Adams, J. H., Doyle, D., Graham, D. I., Lawrence, A. E., McLellan, D. R., Gennarelli, T. A., et al. (1985). The contusion index: a reappraisal in human and experimental non-missile head injury. *Neuropathol Appl Neurobiol, 11*, 299-308.

Agnew, S. K., & Morris, R. G. (1998). The heterogeneity of anosognosia for memory impairment in alzheimer's disease: a review of the literature and a proposed model. *Aging and Mental Health, 2*, 7-19.

Alexander, M. P., Benson, D. F., & Stuss, D. T. (1989). Frontal lobes and language. *Brain Lang, 37*, 656-691.

Allen, C. C., & Ruff, R. M. (1990). Self-rating versus neuropsychological performance of moderate versus severe head-injured patients. *Brain Inj, 4*, 7-17.

Amador, X. F., Flaum, M., Andreasen, N. C., Strauss, D. H., Yale, S. A., Clark, S. C., et al. (1994). Awareness of illness in schizophrenia and schizoaffective and mood disorders. *Arch Gen Psychiatry, 51*, 826-836.

Amador, X. F., Strauss, D. H., Yale, S. A., Flaum, M. M., Endicott, J., & Gorman, J. M. (1993). Assessment of insight in psychosis. *Am J Psychiatry, 150*, 873-879.

Anderson, S. W., & Tranel, D. (1989). Awareness of Disease States following cerebral infarction, dementia, and head trauma: standardized assessment. *The Clinical Neuropsychologist, 3*, 327-339.

Anton, G. (1899). Ueber die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit. *Archiv fuer Psychiatrie und Nervenkrankheiten, 32*, 86-127.

Babinski, J. (1914). Contribution a l'etude des troubles mantaux dans l'hemiplegie organique cerebale (anosognosie). *Revue Neurologique, 27*, 845-848.

Baddeley, A., Harris, J., Sunderland, A., Watts, K., & Wilson, B. A. (1987). Closed head injury and memory. In H. S. Levin, J. Grafman, & & H. M. Eisenberg (Eds.), *Neurobehavioural recovery from head injury*. New York: Oxford University Press.

Baddley, A. D. (1986). *Working Memory*. Oxford: Oxford University Press.

Bak, T., Crawford, L. M., Hearn, V. C., Mathuranath, P. S., & Hodges, J. R. (2005). Subcortical dementia revisited: Similarities and differences in cognitive function between progressive supranuclear palsy (PSP), corticobasal degeneration (CBD) and multiple system atrophy (MSA). *Neurocase, 11*, 268-273.

Barco, P. P., Crosson, B., Bolesta, M. M., Werts, D., & Stout, R. (1991). Training awareness and compensation in postacute head injury rehabilitation. In J. Kreutzer & P. Wehman (Eds.), *Cognitive Rehabilitation for Persons with Traumatic Brain Injury* (pp. 129-146). Baltimore: Paul H. Brookes.

Baron-Cohen, S., Ring, H. A., Wheelwright, S., Bullmore, E. T., Brammer, M. J., Simmons, A., et al. (1999). Social intelligence in the normal and autistic brain: an fMRI study. *Eur J Neurosci, 11*, 1891-1898.

Baron-Cohen, S., Tager-Flusberg, H., & Cohen, D. (2000). *Understanding other minds. Perspectives from developmental cognitive neuroscience*. Oxford: Oxford University Press.

Ben-Yishay, Y., Rattok, J., & Piasetsky, E. B. (1985). Neuropsychological rehabilitation: Quest for a holitstic approach. *Seminars in Neuroscience, 5*, 252-259.

Ben-Yishay, Y., Silver, B., Piasetsky, E. B., & Rattok, J. (1987). Relationship between employability and vocational outcome after intensive holistic cognitive rehabilitation. *Journal of Head Trauma Rehabilitation, 2*, 35-48.

Benton, A. L., & Hamsher, K. (1976). *Multilingual aphasia examination* (2nd ed.). Iowa City: AJA Associates Inc.

Bergman, H., & Deuschl, G. (2002). Pathophysiology of Parkinson's disease: From clinical neurology to basic neuroscience and back. *Movement Disorders, 17*, S28-S40.

Bernstein, P. S., Scheffers, M. K., & Coles, M. G. (1995). "Where did I go wrong?" A psychophysiological analysis of error detection. *J Exp Psychol Hum Percept Perform, 21*, 1312-1322.

Best, M., Williams, J. M., & Coccaro, E. F. (2002). Evidence for a dysfunctional prefrontal circuit in patients with an impulsive aggressive disorder. *Proc Natl Acad Sci U S A, 99*, 8448-8453.

Bigler, E. D. (1990). Neuropathology of traumatic brain injury. In E. D. Bigler (Ed.), *Traumatic Brain Injury*. Austin: Pro-Ed.

Bigler, E. D. (1999). Neuroimaging in mild TBI. In N. R. V. R. J. Roberts (Ed.), *The Evaluation and Treatment of Mild Traumatic Brain Injury*. Mahwah: Erlbaum.

Bigler, E. D., & Snyder, J. L. (1995). Neuropsychological outcome and quantitative neuroimaging in mild head injury. *Arch Clin Neuropsychol, 10*, 159-174.

Birnboim, S. (1995). A metacognitive approach to cognitive rehabilitation. *British Journal of Occupational Therapy, 58*, 61-64.

Bisiach, E., & Geminiani, G. (1991). Anosognosia related to hemiplegia and hemianopia. In G. P. Prigatano & D. L. Schacter (Eds.), *Awareness of Deficit after Brain Injury. Clinical and Theoretical Issues*. New York: Oxford University Press.

Bisiach, E., Vallar, G., Perani, D., Papagno, C., & Berti, A. (1986). Unawareness of disease following lesions of the right hemisphere: anosognosia for hemiplegia and anosognosia for hemianopia. *Neuropsychologia, 24*, 471-482.

Bisiach, E. M., S. & Berti, A. (1985). *Mechanisms of production-control and belief-fixation in human visuospatial processing. Clinical evidence from hemispatial neglect.* Paper presented at the Eighth Symposium on Quantitative Analysis of Behaviour, Harvard University.

Blakemore, S. J., Wolpert, D., & Frith, C. (2000). Why can't you tickle yourself? *Neuroreport, 11*, R11-16.

Boeve, B. F., Lang, A. E., & Litvan, I. (2003). Corticobasal degeneration and its relationship to progressive supranuclear palsy and frontotemporal dementia. *Ann Neurol, 54 Suppl 5*, S15-19.

Bogod, N. M., Mateer, C. A., & MacDonald, S. W. (2003). Self-awareness after traumatic brain injury: a comparison of measures and their relationship to executive functions. *J Int Neuropsychol Soc, 9*, 450-458.

Bohac, D. L., Malec, J. F., & Moessner, A. M. (1997). Factor analysis of the Mayo-Portland Adaptability Inventory: structure and validity. *Brain Inj, 11*, 469-482.

Bonne, O., Gilboa, A., Louzoun, Y., Kempf-Sherf, O., Katz, M., Fishman, Y., et al. (2003). Cerebral blood flow in chronic symptomatic mild traumatic brain injury. *Psychiatry Res, 124*, 141-152.

Borgaro, S. R., & Prigatano, G. P. (2002). Early cognitive and affective sequelae of traumatic brain injury: a study using the BNI Screen for Higher Cerebral Functions. *J Head Trauma Rehabil, 17*, 526-534.

Boucsein, W. (1992). *Electrodermal Activity*. New York: Plenum.

Broadbent, D. E., Cooper, P. F., FitzGerald, P., & Parkes, K. R. (1982). The Cognitive Failures Questionnaire (CFQ) and its correlates. *Br J Clin Psychol, 21 (Pt 1)*, 1-16.

Brooks, D. N. (1984). Cognitive deficits after head injury. In N.Brooks (Ed.), *Closed Head Injury: Psychological, social and family consequences*. Toronto: Oxford University Press.

Brooks, D. N., Campsie, L., symington, C., Beattie, A., & McKinlay, W. W. (1986). The effects of severe head injury on patient and relative within seven years of injury. *Journal of Head Trauma Rehabilitation, 2*, 1-13.

Brooks, D. N., & McKinlay, W. (1983). Personality and behavioural change after severe blunt head injury--a relative's view. *J Neurol Neurosurg Psychiatry, 46*, 336-344.

Brooks, N. (1984). *Closed Head Injury: Psychological, Social and Family Consequences.* Oxford: Oxford University Press.

Burgess, P. W., Alderman, N., Evans, J., Emslie, H., & Wilson, B. A. (1998). The ecological validity of tests of executive function. *J Int Neuropsychol Soc, 4*, 547-558.

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends Cogn Sci, 4*, 215-222.

Bychowski, Z. (1920). Ueber das Fehlen der Wahrnehmung der eigenen Blindheit bei zwei Kriegsverletzten. *Neurologisches Centralblatt, 39*, 354-357.

Campodonico, J. F. (1992). *Impaired awareness for complex deficits following traumatic brain injury.* Unpublished PhD, California School of Professional Psychology, Fresno.

Cavallo, M. M., Kay, T., & Ezrachi, O. (1992). Problems and changes after traumatic brain injury: differing perceptions within and between families. *Brain Inj, 6*, 327-335.

Chan, R. C. (2001). A further study on the sustained attention response to task (SART): the effect of age, gender and education. *Brain Inury, 15*, 819-829.

Chittum, W. R., Johnson, K., Chittum, J. M., Guercio, J. M., & McMorrow, M. J. (1996). Road to awareness: an individualized training package for increasing knowledge and comprehension of personal deficits in persons with acquired brain injury. *Brain Inj, 10*, 763-776.

Cicerone, K. D., & Tanenbaum, L. N. (1997). Disturbance of social cognition after traumatic orbitofrontal brain injury. *Arch Clin Neuropsychol, 12*, 173-188.

Clare, L. (2004a). Awareness in early-stage Alzheimer's disease: a review of methods and evidence. *Br J Clin Psychol, 43*, 177-196.

Clare, L. (2004b). The construction of awareness in early-stage Alzheimer's disease: a review of concepts and models. *Br J Clin Psychol, 43*, 155-175.

Clare, L., Markova, I., Verhey, F., & Kenny, G. (2005). Awareness in dementia: A review of assessment methods and measures. *Aging and Mental Health, 9*, 394-413.

Clare, L., Wilson, B. A., Carter, G., Roth, I., & Hodges, J. R. (2002). Assessing awareness in early-stage Alzheimer's disease: Development and piloting of the Memory Awareness Rating Scale. *Neuropsychological Rehabilitation, 12*, 341-362.

Coben, R. A., Boksenbaum, S. I., & Kulberg, A. M. (1995). Cognitive determinants of unawareness of deficits: The importance of specific frontal-mediated executive functions. *Archives of Clinical Neuropsychology, 10*, 309-310.

Cocchini, G., Beschin, N., & Sala, S. D. (2002). Chronic anosognosia: a case report and theoretical account. *Neuropsychologia, 40*, 2030-2038.

Cooke, D. L., & Kausler, D. H. (1995). Content memory and temporal memory for actions in survivors of traumatic brain injury. *J Clin Exp Neuropsychol, 17*, 90-99.

Cordato, N. J., Duggins, A. J., Halliday, G. M., Morris, J. G., & Pantelis, C. (2005). Clinical deficits correlate with regional cerebral atrophy in progressive supranuclear palsy. *Brain, 128*, 1259-1266.

Cotrell, V., & Wild, K. (1999). Longitudinal study of self-imposed driving restrictions and deficit awareness in patients with Alzheimer disease. *Alzheimer Dis Assoc Disord, 13*, 151-156.

Crisp, R. (1992). Awareness of deficit after traumatic brain injury: a literature review. *The Australian Occupational Therapy Journal, 39*, 15-21.

Critchley, H. D. (2002). Electrodermal responses: what happens in the brain. *Neuroscientist, 8*, 132-142.

Critchley, M. (1953a). *The Parietal Lobes*. London: Edward Arnold.

Critchley, M. (1953b). The problem of awareness or non-awareness of hemianopic field defects. *Transactions of the Ophthalmological Society of U.K., 69*, 95-109.

Crosson, C., Barco, P. P., Velozo, C., Bolesta, M. M., Cooper, P. V., Werts, D., et al. (1989). Awareness and Compensation in postacute head injury rehabilitation. *Journal of Head Trauma Rehabilitation, 4*, 46-54.

Cuesta, M. J., & Peralta, V. (1994). Lack of insight in schizophrenia. *Schizophr Bull, 20*, 359-366.

Cummings, J. L. (1990). *Subcortical dementia*. New York: Oxford University Press.

Cummings, J. L., & Benson, D. F. (1984). Subcortical dementia. Review of an emerging concept. *Arch Neurol, 41*, 874-879.

Cummings, J. L., & Benson, D. F. (1992). *Dementia: A Clinical Approach* (2nd ed.). Boston: Butterworth-Heinemann.

Dalla Barba, G., Parlato, V., Iavarone, A., & Boller, F. (1995). Anosognosia, intrusions and 'frontal' functions in Alzheimer's disease and depression. *Neuropsychologia, 33*, 247-259.

Damasio, A. R. (1994). *Descartes' Error: Emotion, Reason, and the Human Brain.* New York: GP Putnam.

Damasio, A. R., Tranel, D., & Damasio, H. (1990). Individuals with sociopathic behavior caused by frontal damage fail to respond autonomically to social stimuli. *Behav Brain Res, 41*, 81-94.

Danielczyk, W. (1983). Various mental behavioral disorders in Parkinson's disease, primary degenrative senile dementia, and multiple infarction dementia. *Journal of Neural Transmission, 56*, 161-176.

David, A., Buchanan, A., Reed, A., & Almeida, O. (1992). The assessment of insight in psychosis. *Br J Psychiatry, 161*, 599-602.

David, A., van Os, J., Jones, P., Harvey, I., Foerster, A., & Fahy, T. (1995). Insight and psychotic illness. Cross-sectional and longitudinal associations. *Br J Psychiatry, 167*, 621-628.

Dawson, M. E., Schell, A. M., & Filion, D. L. (2000). The Electrodermal System. In J. T. Cacioppo, L. G. Tassinary & G. G. Berntson (Eds.), *Handbook of Psychophysiology* (2nd ed.). Cambridge: Cambridge University Press.

Deaton, A. V. (1986). Denial in the aftermath of traumatic head injury: Its manifestations, measurement, and treatment. *Rehabilitation Psychology, 31*, 231-240.

DeBettignies, B. H., Mahurin, R. K., & Pirozzolo, F. J. (1990). Insight for impairment in independent living skills in Alzheimer's disease and multi-infarct dementia. *J Clin Exp Neuropsychol, 12*, 355-363.

Deckel, A. W., & Morrison, D. (1996). Evidence of a neurologically based 'Denial of illness' in patients with Huntington's Disease. *Archives of Clinical Neuropsychology, 11*, 295-302.

Dehaene, S., Posner, M., & Tucker, D. (1994). Localisation of a neural system for error detection and compensation. *Psychological Science, 5*, 303-305.

Derogatis, L. (1977). *The SCL-90 Manual: Scoring, administration and procedure for the SCL-90.* Baltimore: John Hopkins University School of Medicine.

Derouesne, C., Thibault, S., Lagha-Pierucci, S., Baudouin-Madec, V., Ancri, D., & Lacomblez, L. (1999). Decreased awareness of cognitive deficits in patients with mild dementia of the Alzheimer type. *Int J Geriatr Psychiatry, 14*, 1019-1030.

Diehl, J., & Kurz, A. (2002). Frontotemporal dementia: patient characteristics, cognition, and behaviour. *Int J Geriatr Psychiatry, 17*, 914-918.

Dikmen, S. S., Machamer, J. E., Winn, H. R., & Temkin, N. R. (1995). Neuropsychological Outcome at 1-Year Post Head Injury. *Neuropsychology, 9*, 80-90.

Dockree, P. M., Bellgrove, M. A., O'Keeffe, F., Moloney, P., Aimola, L., Carton, S., Robertson, IH. (2005). Sustained Attention in traumatic brain injury (tbi) and healthy controls: enhanced sensitivity with dual-task load. *Experimental Brain Research,* 1-12.

Dockree, P. M., Kelly, S. P., Roche, R. A., Hogan, M. J., Reilly, R. B., & Robertson, I. H. (2004). Behavioural and physiological impairments of sustained attention after traumatic brain injury. *Brain Res Cogn Brain Res, 20*, 403-414.

Douglas, J. M., & Spellacy, F. J. (1996). Indicators of long-term family functioning following severe traumatic brain injury in adults. *Brain Inj, 10*, 819-839.

Dubois, B., Slachevsky, A., Litvan, I., & Pillon, B. (2000). The FAB: a Frontal Assessment Battery at bedside. *Neurology, 55*, 1621-1626.

Ekman, P., & Friesen, W. V. (1976). *Pictures of Facial Affect*. Palo Alto: Consulting Psychologists Press.

Enderby, P., Wood, V. A., & Wade, D. T. (1987). *Frenchay Aphasia Screening Test-Test Manual*: NFER-Nelson.

Enderby, P. M., Wood, V. A., Wade, D. T., & Hewer, R. L. (1987). The Frenchay Aphasia Screening Test: a short, simple test for aphasia appropriate for non-specialists. *Int Rehabil Med, 8*, 166-170.

Ergh, T. C., Hanks, R. A., Rapport, L. J., & Coleman, R. D. (2003). Social support moderates caregiver life satisfaction following traumatic brain injury. *J Clin Exp Neuropsychol, 25*, 1090-1101.

Ergh, T. C., Rapport, L. J., Coleman, R. D., & Hanks, R. A. (2002). Predictors of caregiver and family functioning following traumatic brain injury: social support moderates caregiver distress. *J Head Trauma Rehabil, 17*, 155-174.

Ezrachi, O., Ben-Yishay, Y., Kay, T., & al, e. (1991). Predicting employment in traumatic brain injury following neuropsychological rehabilitation. *Journal of Head Trauma Rehabilitation, 6*, 71-84.

Falkenstein, M., Hoorman, J., Christ, S., & Hohnsbein, J. (2000). ERP components on reaction errors and their functional significance: a tutorial. *Biological Psychology, 51*, 87-107.

Feher, E. P., Mahurin, R. K., Inbody, S., Crook, T. H., 3rd, & Pirozzolo, F. J. (1991). Anosognosia in Alzheimer's disease. *Neuropsychiatry Neuropsychol Behav Neurol, 4*, 136-146.

Fischer, S., Gauggel, S., & Trexler, L. E. (2004). Awareness of activity limitations, goal setting and rehabilitation outcome in patients with brain injuries. *Brain Inj, 18*, 547-562.

Fischer, S., Trexler, L. E., & Gauggel, S. (2004). Awareness of activity limitations and prediction of performance in patients with brain injuries and orthopedic disorders. *J Int Neuropsychol Soc, 10*, 190-199.

Flashman, L. A. (2002). Disorders of awareness in neuropsychiatric syndromes: an update. *Curr Psychiatry Rep, 4*, 346-353.

Flashman, L. A., Amador, X., & McAllister, T. W. (1998). Lack of Awareness of Deficits in Traumatic Brain Injury. *Semin Clin Neuropsychiatry, 3*, 201-210.

Flashman, L. A., & McAllister, T. W. (2002). Lack of awareness and its impact in traumatic brain injury. *NeuroRehabilitation, 17*, 285-296.

Flashman, L. A., McAllister, T. W., Andreasen, N. C., & Saykin, A. J. (2000). Smaller brain size associated with unawareness of illness in patients with schizophrenia. *Am J Psychiatry, 157*, 1167-1169.

Flashman, L. A., McAllister, T. W., Johnson, S. C., Rick, J. H., Green, R. L., & Saykin, A. J. (2001). Specific frontal lobe subregions correlated with unawareness of illness in schizophrenia: a preliminary study. *J Neuropsychiatry Clin Neurosci, 13*, 255-257.

Fleming, J., & Strong, J. (1995). Self awareness of deficits following acquired brain injury. *British Journal of Occupational Therapy, 58*, 55-60.

Fleming, J., & Strong, J. (1999). A longitudinal study of self-awareness: Functional deficits underestimated by persons with brain injury. *The Occupational Therapy Journal of Research, 19*, 3-17.

Fleming, J. M., Strong, J., & Ashton, R. (1996). Self-awareness of deficits in adults with traumatic brain injury: how best to measure? *Brain Inj, 10*, 1-15.

Fleming, J. M., Strong, J., & Ashton, R. (1998). Cluster analysis of self-awareness levels in adults with traumatic brain injury and relationship to outcome. *J Head Trauma Rehabil, 13*, 39-51.

Fletcher, P. C., Happe, F., Frith, U., Baker, S. C., Dolan, R. J., Frackowiak, R. S., et al. (1995). Other minds in the brain: a functional imaging study of "theory of mind" in story comprehension. *Cognition, 57*, 109-128.

Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res, 12*, 189-198.

Fontaine, A., Azouvi, P., Remy, P., Bussel, B., & Samson, Y. (1999). Functional anatomy of neuropsychological deficits after severe traumatic brain injury. *Neurology, 53*, 1963-1968.

Fordyce, D. J., & Roueche, J. R. (1986). Changes in perspectives of disability among patients, staff, and relatives during rehabilitation of brain injury. *Rehabilitation Psychology, 31*, 217-229.

Fordyce, D. J., Roueche, J. R., & Prigatano, G. P. (1983). Enhanced emotional reactions in chronic head trauma patients. *J Neurol Neurosurg Psychiatry, 46*, 620-624.

Fossati, P., Hevenor, S. J., Graham, S. J., Grady, C., Keightley, M. L., Craik, F., et al. (2003). In search of the emotional self: an fMRI study using positive and negative emotional words. *Am J Psychiatry, 160*, 1938-1945.

Frackowiak, R. S., Pozzilli, C., Legg, N. J., Du Boulay, G. H., Marshall, J., Lenzi, G. L., et al. (1981). Regional cerebral oxygen supply and utilization in dementia. A clinical and physiological study with oxygen-15 and positron tomography. *Brain, 104*, 753-778.

Frattali, C. M., Grafman, J., Patronas, N., Makhlouf, F., & Litvan, I. (2000). Language disturbances in corticobasal degeneration. *Neurology, 54*, 990-992.

Freeland, J. C. (1996). Awareness of deficits: A complex interplay of neurological, personality, social and rehabilitation factors. *Magazine, 4*, 32-34.

Freeman, W., & Watts, J.W. (1950). *Psychosurgery in the Treatment of Mental Disorders and Intractable Pain* (2nd ed.). Springfield: Charles Thomas.

Frith, C., & Dolan, R. (1996). The role of the prefrontal cortex in higher cognitive functions. *Cognitive Brain Research, 5*, 175-181.

Frith, C. D., & Frith, U. (1999). Interacting Minds--A Biological Basis. *Science, 286*, 1692-1695.

Gainotti, G. (1972). Emotional behavior and hemispheric side of the lesion. *Cortex, 8*, 41-55.

Gainotti, G. (1993). Emotional and psychosocial problems after brain injury. *Neuropsychological Rehabilitation, 3*, 259-277.

Garavan, H., Ross, T. J., Kaufman, J., & Stein, E. A. (2003). A midline dissociation between error-processing and response-conflict monitoring. *Neuroimage, 20*, 1132-1139.

Garavan, H., Ross, T. J., Murphy, K., Roche, R. A., & Stein, E. A. (2002a). Dissociable executive functions in the dynamic control of behavior: inhibition, error detection, and correction. *Neuroimage, 17*, 1820-1829.

Garavan, H., Ross, T. J., & Stein, E. A. (1999). Right hemispheric dominance of inhibitory control: an event-related functional MRI study. *Proc Natl Acad Sci USA, 96*, 8301-8306.

Gasquoine, P. G. (1992). Affective State and Awareness of Sensory and Cognitive Effects After Closed Head Injury. *Neuropsychology, 6*, 187-196.

Gauggel, S., Hoop, M., & Werner, K. (2002). Assigned versus self-set goals and their impact on the performance of brain-damaged patients. *J Clin Exp Neuropsychol, 24*, 1070-1080.

Gauggel, S., Peleska, B., & Bode, R. K. (2000). Relationship between cognitive impairments and rated activity restrictions in stroke patients. *J Head Trauma Rehabil, 15*, 710-723.

Gehring, W. J., Gross, B., Coles, M., Meyer, D., & Donchin, E. (1993). A neural system for error detection and compensation. *Psychological Science, 4*, 385-390.

Gehring, W. J., & Knight, R. T. (2000). Prefrontal-cingulate interactions in action monitoring. *Nat Neurosci, 3*, 516-520.

George, M. S., Costa, D. C., Kouris, K., Ring, H. A., & Ell, P. J. (1992). Cerebral blood flow abnormalities in adults with infantile autism. *J Nerv Ment Dis, 180*, 413-417.

Gerstmann, J. (1942). Problem of imperception of disease and of impaired body territories with organic lesions; relation to body scheme and its disorders. *Archives of Neurology and Psychiatry, 48*, 890-913.

Giacino, J. T., & Cicerone, K. D. (1998). Varieties of deficit unawareness after brain injury. *J Head Trauma Rehabil, 13*, 1-15.

Gillen, R., Tennen, H., Affleck, G., & Steinpreis, R. (1998). Distress, depressive symptoms, and depressive disorder among caregivers of patients with brain injury. *J Head Trauma Rehabil, 13*, 31-43.

Godfrey, H. P., Knight, R. G., & Partridge, F. M. (1996). Emotional adjustment following traumatic brain injury: A stress-appraisal-coping formulation. *Journal of Head Trauma Rehabilitation, 11*, 29-40.

Godfrey, H. P., Partridge, F. M., Knight, R. G., & Bishara, S. (1993). Course of insight disorder and emotional dysfunction following closed head injury: a controlled cross-sectional follow-up study. *J Clin Exp Neuropsychol, 15*, 503-515.

Goel, V., Grafman, J., Sadato, N., & Hallett, M. (1995). Modeling other minds. *Neuroreport, 6*, 1741-1746.

Goldberg, E., & Barr, W. B. (1991). Three possible mechanisms of unawareness of deficit. In G. P. Prigatano & D. L. Schacter (Eds.), *Awareness of Deficit After Brain Injury*. New York: Oxford University Press.

Grace, J., & Malloy, P. F. (2002). *Frontal Systems Behavior Scale. Professional Manual.*

Grace, J., Stout, J. C., & Malloy, P. F. (1999). Assessing frontal lobe behavioral syndromes with the Frontal Lobe Personality Scale. *Assessment, 6*, 269-284.

Grady, M. S., & McIntosh, T. C. (2002). Head Injury. In A. K. Asbury (Ed.), *Diseases of the Nervous System* (3rd ed.). Cambridge: Cambridge University Press.

Grafman J., Litvan I., & Stark M. (1995). Neuropsychological Features of Progressive Supranuclear Palsy. *Brain and Cognition, 28*, 311-320.

Graham, D. I. (1996). Neuropathology of head trauma. In R. K. Narayan (Ed.), *Neurotrauma*. New York: McGraw-Hill.

Graham, D. P., Kunik, M. E., Doody, R., & Snow, A. L. (2005). Self-reported awareness of performance in dementia. *Cognitive Brain Research, 25*, 144-152.

Graham, N. L., Bak, T., Patterson, K., & Hodges, J. R. (2003). Language function and dysfunction in corticobasal degeneration. *Neurology, 61*, 493-499.

Granacher, R. P. (2004). *Traumatic Brain Injury. Methods for Clinical and Forensic Neuropsychiatric Assessment*. New York: CRC Press.

Green, J., Goldstein, F. C., Sirockman, B. E., & Green, R. C. (1993). Variable awareness of deficits in Alzheimer's Disease. *Neuropsychiatry Neuropsychol Behav Neurol, 6*, 159-165.

Gregory, C., & Hodges, J. R. (1996). Frontotemporal dementia: Use of consensus criteria and prevalence of psychiatric features. *Neuropsychiatry Neuropsychol Behav Neurol, 9*, 145-153.

Groswasser, Z., Reider-Groswasser, I., Soroker, N., & Machtey, Y. (1987). Magnetic resonance imaging in head injured patients with normal late computed tomography scans. *Surg Neurol, 27*, 331-337.

Gusnard, D. A., Akbudak, E., Shulman, G. L., & Raichle, M. E. (2001). Medial prefrontal cortex and self-referential mental activity: relation to a default mode of brain function. *Proc Natl Acad Sci U S A, 98*, 4259-4264.

Gusnard, D. A., & Raichle, M. E. (2001). Searching for a baseline: functional imaging and the resting human brain. *Nat Rev Neurosci, 2*, 685-694.

Hacker, D. J. (1998). Definitions and empirical foundations. In J. D. Hacker, & AC Graesser (Ed.), *Metacognition in Educational Theory and Practice*. Mahweh: Lawrence Erlbaum.

Halliday, A. L. (1999). Pathophysiology. In D. W. Marion (Ed.), *Traumatic Brain Injury*. New York: Thieme.

Hart, T., Giovannetti, T., Montgomery, M. W., & Schwartz, M. F. (1998). Awareness of errors in naturalistic action after traumatic brain injury. *J Head Trauma Rehabil, 13*, 16-28.

Hart, T., & Sherer, M. (2005). Preface to Special Edition. *Journal of Head Trauma Rehabilitation, 20*, 285-286.

Hart, T., Sherer, M., Whyte, J., Polansky, M., & Novack, T. A. (2004). Awareness of behavioral, cognitive, and physical deficits in acute traumatic brain injury. *Arch Phys Med Rehabil, 85*, 1450-1456.

Hart, T., Whyte, J., Kim, J., & Vaccaro, M. (2005). Executive Function and Self-awareness of "Real-world" Behavior and Attention Deficits Following Traumatic Brain Injury. *J Head Trauma Rehabil, 20*, 333-347.

Hart, T., Whyte, J., Polansky, M., Millis, S., Hammond, F. M., Sherer, M., et al. (2003). Concordance of patient and family report of neurobehavioral symptoms at 1 year after traumatic brain injury. *Arch Phys Med Rehabil, 84*, 204-213.

Harwood, D. G., & Sultzer, D. L. (2002). "Life is not worth living": hopelessness in Alzheimer's disease. *J Geriatr Psychiatry Neurol, 15*, 38-43.

Harwood, D. G., Sultzer, D. L., & Wheatley, M. V. (2000). Impaired insight in Alzheimer disease: association with cognitive deficits, psychiatric symptoms,

and behavioral disturbances. *Neuropsychiatry Neuropsychol Behav Neurol, 13*, 83-88.

Heilbronner, R. L., Roueche, J. R., Everson, S. A., & Epler, L. (1989). Comparing patient perspectives of disability and treatment effects with quality of participation in a post-acute brain injury rehabilitation programme. *Brain Inj, 3*, 387-395.

Heilman, K. M. (1991). Anosognosia: Possible neuropsychological mechanisms. In G. P. Prigatano, D. (Ed.), *Awareness of Deficit After Brain Injury: Clinical and Theoretical Issues*. New York: Oxford University Press.

Heilman, K. M., Barrett, A. M., & Adair, J. C. (1998). Possible mechanisms of anosognosia: a defect in self-awareness. *Philos Trans R Soc Lond B Biol Sci, 353*, 1903-1909.

Heilman, K. M., Watson, R. T., & Valenstein, E. (1985). Neglect and related disorders. In K. M. Heilman & E. Valenstein (Eds.), *Clinical Neuropsychology* (pp. 243-294). New York: Oxford University Press.

Henry, G., Gross, H., Herndon, C., & Furst, C. (2000). Non-impact brain injury: Neuropsychological and behavioral correlates with consideration of physical findings. *Applied Neuropsychology, 7*, 65-75.

Hester, R., Foxe, J. J., Molholm, S., Shpaner, M., & Garavan, H. (2005). Neural mechanisms involved in error processing: A comparison of errors made with and without awareness. *NeuroImage, 27*, 602-608.

Hickey, A. M., O'Boyle, C. A., McGee, H. M., & McDonald, N. J. (1997). The relationship between post-trauma problem reporting and carer quality of life after severe head injury. *Psychology and Health, 12*, 822-838.

Hillier, S. L., & Metzer, J. (1997). Awareness and perceptions of outcomes after traumatic brain injury. *Brain Inj, 11*, 525-536.

Hoofien, D., Gilboa, A., Vakil, E., & Barak, O. (2004). Unawareness of cognitive deficits and daily functioning among persons with traumatic brain injuries. *J Clin Exp Neuropsychol, 26*, 278-290.

Hsiang, J., & Marshall, L. F. (1998). Head Injury. In M. Swash (Ed.), *Outcome in neurological and neurosurgical disorders*. Cambridge: Cambridge University Press.

Hugdahl, K. (1995). *Psychophysiology: The mind-body perspective*. Cambridge, Mass.: Harvard University Press.

Hutchinson, S. A., Leger-Krall, S., & Wilson, H. S. (1997). Early probable Alzheimer's disease and awareness context theory. *Soc Sci Med, 45*, 1399-1409.

James, W. (1890). *The Principles of Psychology*. Chicago: Encyclopaedia Britannica.

Janowsky, J. S., Shimamura, A. P., & Squire, L. R. (1989). Memory and metamemory: Comparisons between patients with frontal lobe lesions and amnesic patients. *Psychobiology, 17*, 3-11.

Jennett, B., Teasdale, G., & Knill-Jones, R. (1975). Prognosis after severe head injury. *Ciba Found Symp*, 309-324.

Johnson, S. C., Baxter, L. C., Wilder, L. S., Pipe, J. G., Heiserman, J. E., & Prigatano, G. P. (2002). Neural correlates of self-reflection. *Brain, 125*, 1808-1814.

Kanner, L. (1973). Autistic disturbances of affective contact. In L. Kanner (Ed.), *Childhood Psychosis: Initial studies and New Insights* (pp. 217-250). Washington DC: Winston.

Kaszniak, A. W., & Zak, M. G. (1996). On the neuropsychology of metamemory: Contributions from the study of amnesia and dementia. *Learning and Individual Differences, 8*, 355-381.

Katz, D. I., & Alexander, M. P. (1994). Traumatic brain injury. Predicting course of recovery and outcome for patients admitted to rehabilitation. *Arch Neurol, 51*, 661-670.

Katz, N., & Hartman-Maeir, A. (2005). Higher-level cognitive functions. Awareness and executive functions enabling engagement in occupation. In N. Katz (Ed.), *Cognition and Occupation across the life span. Models for Intervention in Occupational Therapy* (2nd ed.): The American Occupational Therapy Association, Inc.

Kelley, W. M., Macrae, C. N., Wyland, C. L., Caglar, S., Inati, S., & Heatherton, T. F. (2002). Finding the self? An event-related fMRI study. *J Cogn Neurosci, 14*, 785-794.

Kennedy, M. R. T., & Yorkston, K. M. (2004). The effects of frontal injury on "on-line" self-monitoring during verbal learning by adults with diffuse brain injury. *Neuropsychological Rehabilitation, 14*, 449-465.

Kertesz, A., Martinez-Lage, P., Davidson, W., & Munoz, D. G. (2000). The corticobasal degeneration syndrome overlaps progressive aphasia and frontotemporal dementia. *Neurology, 55*, 1368-1375.

Kiehl, K. A., Liddle, P. F., & Hopfinger, J. B. (2000). Error processing and the rostral anterior cingulate: an event-related fMRI study. *Psychophysiology, 37*, 216-223.

Kitagaki, H., Hirono, N., Ishii, K., & Mori, E. (2000). Corticobasal degeneration: evaluation of cortical atrophy by means of hemispheric surface display generated with MR images. *Radiology, 216*, 31-38.

Kjaer, T. W., Nowak, M., & Lou, H. C. (2002). Reflective self-awareness and conscious states: PET evidence for a common midline parietofrontal core. *Neuroimage, 17*, 1080-1086.

Kliegel, M., Eschen, A., & Thone-Otto, A. I. (2004). Planning and realization of complex intentions in traumatic brain injury and normal aging. *Brain Cogn, 56*, 43-54.

Koehler, P. J., Endtz, L. J., Te Velde, J., & Hekster, R. E. M. (1986). Aware or non-aware. On the significance of awareness for the localization of the lesion responsible for homonymous hemianopia. *Journal of the Neurological Sciences, 75*, 255-262.

Kortte, K. B., Wegener, S. T., & Chwalisz, K. (2003). Anosognosia and denial: Their relationship to coping and depression in acquired brain injury. *Rehabilitation Psychology, 48*, 131-136.

Koskinen, S. (1998). Quality of life 10 years after a very severe traumatic brain injury (TBI): the perspective of the injured and the closest relative. *Brain Inj, 12*, 631-648.

Kraus, J. (1996). Epidemiology of brain injury. In R. Narayan (Ed.), *Neurotrauma*. New York: McGraw-Hill.

Kraus, J. F., & McArthur, D. L. (1996). Epidemiologic aspects of brain injury. *Neurol Clin, 14*, 435-450.

Kreutzer, J. S., Marwitz, J. H., & Kepler, K. (1992). Traumatic brain injury: family response and outcome. *Arch Phys Med Rehabil, 73*, 771-778.

Lam, C. S., McMahon, B. T., Priddy, D. A., & Gehred-Schultz, A. (1988). Deficit awareness and treatment performance among traumatic head injury adults. *Brain Inj, 2*, 235-242.

Lang, A. E. (2000). Corticobasal ganglionic degeneration and/or frontotemporal dementia? *J Neurol Neurosurg Psychiatry, 69*, 705.

Lang, A. E., Riley, D., & Bergeron, C. (1994). Cortico-basal ganglionic degeneration. In C. DB (Ed.), *Neurodegenerative Diseases* (pp. 877-894). Philadelphia: WB Saunders.

Langer, K., & Padrone, F. J. (1992). Psychotherapeutic treatment of awareness in acute rehabilitation of traumatic brain injury. *Neuropsychological Rehabilitation, 2*, 59-70.

Lanham, R. A., Jr., Weissenburger, J. E., Schwab, K. A., & Rosner, M. M. (2000). A longitudinal investigation of the concordance between individuals with traumatic brain injury and family or friend ratings on the Katz adjustment scale. *J Head Trauma Rehabil, 15*, 1123-1138.

Leathem, J. M., Murphy, L. J., & Flett, R. A. (1998). Self- and informant-ratings on the patient competency rating scale in patients with traumatic brain injury. *J Clin Exp Neuropsychol, 20*, 694-705.

Lebrun, Y. (1987). Anosognosia in aphasics. *Cortex, 23*, 251-263.

Lehrer, P. M., Groveman, A., Randolph, C., Miller, M. H., & Pollack, I. (1989). Physiological response patterns to cognitive testing in adults with closed head injuries. *Psychophysiology, 26*, 668-675.

Leritz, E., Loftis, C., Crucian, G., Friedman, W., & Bowers, D. (2004). Self-awareness of deficits in Parkinson Disease. *The Clinical Neuropsychologist, 18*, 352-361.

Levin, H., Williams, D., Eisenberg, D., High, W. J., & Guinto, F. J. (1992). Serial MRI and neurobehavioral findings after mild to moderate closed head injury. *Journal of Neurology, Neurosurgery and Psychiatry, 55*, 255-262.

Levine, B., Dawson, D., Boutet, I., Schwartz, M. L., & Stuss, D. T. (2000). Assessment of strategic self-regulation in traumatic brain injury: its relationship to injury severity and psychosocial outcome. *Neuropsychology, 14*, 491-500.

Lezak, M. D. (1979). Recovery of memory and learning functions following traumatic brain injury. *Cortex, 15*, 63-72.

Lezak, M. D. (1995). *Neuropsychological Assessment* (3rd ed.). New York: Oxford University Press.

Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological Assessment* (4th ed.). New York: Oxford University Press.

Litvan, I., Agid, Y., Calne, D., Campbell, G., Dubois, B., Duvoisin, R. C., et al. (1996). Clinical research criteria for the diagnosis of progressive supranuclear palsy (Steele-Richardson-Olszewski syndrome): report of the NINDS-SPSP international workshop. *Neurology, 47*, 1-9.

Litvan, I., Agid, Y., Goetz, C., Jankovic, J., Wenning, G. K., Brandel, J. P., et al. (1997). Accuracy of the clinical diagnosis of corticobasal degeneration: a clinicopathologic study. *Neurology, 48*, 119-125.

Litvan, I., Bhatia, K. P., Burn, D. J., Goetz, C. G., Lang, A. E., McKeith, I., et al. (2003). Movement Disorders Society Scientific Issues Committee report: SIC Task Force appraisal of clinical diagnostic criteria for Parkinsonian disorders. *Mov Disord, 18*, 467-486.

Livingston, M. G., Brooks, D. N., & Bond, M. R. (1985). Patient outcome in the year following severe head injury and relatives' psychiatric and social functioning. *J Neurol Neurosurg Psychiatry, 48*, 876-881.

Loeb, P. A. (1986). *The Independent Living Scales Manual*. San Antonio: The Psychological Corporation.

Lopez, O. L., Becker, J. T., Somsak, D., Dew, M. A., & DeKosky, S. T. (1994). Awareness of cognitive deficits and anosognosia in probable Alzheimer's disease. *Eur Neurol, 34*, 277-282.

Lough, S., Kipps, C. M., Treise, C., Watson, P., Blair, J. R., & Hodges, J. R. (In Press). Social reasoning, emotion and empathy in frontotemporal dementia. *Neuropsychologia, In Press.*

Luria, A. R. (1980). *Higher Cortical Functions in Man.* New York: Basic Books.

Lysaker, P., & Bell, M. (1994). Insight and cognitive impairment in schizophrenia. Performance on repeated administrations of the Wisconsin Card Sorting Test. *J Nerv Ment Dis, 182*, 656-660.

MacKenzie, J. D., Siddiqi, F., Babb, J. S., Bagley, L. J., Mannon, L. J., Sinson, G. P., et al. (2002). Brain atrophy in mild or moderate traumatic brain injury: a longitudinal quantitative analysis. *AJNR Am J Neuroradiol, 23*, 1509-1515.

Macrae, C. N., Moran, J. M., Heatherton, T. F., Banfield, J. F., & Kelley, W. M. (2004). Medial prefrontal activity predicts memory for self. *Cereb Cortex, 14*, 647-654.

Malec, J. F., Buffington, A. L., Moessner, A. M., & Degiorgio, L. (2000). A medical/vocational case coordination system for persons with brain injury: an evaluation of employment outcomes. *Arch Phys Med Rehabil, 81*, 1007-1015.

Malec, J. F., Machulda, M. M., & Moessner, A. M. (1997). Differing problem perceptions of staff, survivors, and significant others after brain injury. *Journal of Head Trauma Rehabilitation, 12*, 1-13.

Malec, J. F., & Moessner, A. M. (2000). Self-Awareness, Distress, and postacute rehabilitation outcome. *Rehabilitation Psychology, 45*, 227-241.

Malec, J. F., Smigielski, J. S., & DePompolo, R. W. (1991). Goal attainment scaling and outcome measurement in postacute brain injury rehabilitation. *Arch Phys Med Rehabil, 72*, 138-143.

Malia, K. (1997). Insight after brain injury: What does it mean? *The Journal of Cognitive Rehabilitation, 15*, 10-16.

Malia, K., Torode, S., & Powell, G. (1993). Insight and progress in rehabilitation after brain injury. *Clinical Rehabilitation, 7*, 23-29.

Mangone, C. A., Hier, D. B., Gorelick, P. B., Ganellen, R. J., Langenberg, P., Boarman, R., et al. (1991). Impaired insight in Alzheimer's disease. *J Geriatr Psychiatry Neurol, 4*, 189-193.

Manly, T., Owen, A. M., McAvinue, L., Datta, A., Lewis, G. H., Scott, S. K., et al. (2003). Enhancing the sensitivity of a sustained attention task to frontal damage: convergent clinical and functional imaging evidence. *Neurocase, 9*, 340-349.

Manly, T., Robertson, I. H., Galloway, M., & Hawkins, K. (1999). The absent mind: further investigations of sustained attention to response. *Neuropsychologia, 37*, 661-670.

Marcel, A. (1983). Conscious and unconcscious perception: experiments on visual masking and word recognition. *Cognitive Psychology, 15*, 197-237.

Marcel, A. J., Tegner, R., & Nimmo-Smith, I. (2004). Anosognosia for plegia: specificity, extension, partiality and disunity of bodily unawareness. *Cortex, 40*, 19-40.

Marcia, L. (1991). *The relationship of awareness of deficit following traumatic brain injury to the psychosocial well being, marital adjustment and community integration of the individual; and the psychological well being and marital adjustment of the spouse.* Unpublished PhD, State University of New York at Buffalo.

Markova, I. S., & Berrios, G. E. (1992). The assessment of insight in clinical psychiatry: a new scale. *Acta Psychiatr Scand, 86*, 159-164.

Markova, I. S., & Berrios, G. E. (2001). The 'object' of insight assessment: relationship to insight 'structure'. *Psychopathology, 34*, 245-252.

Markova, I. S., Clare, L., Wang, M., Romero, B., & Kenny, G. (2005). Awareness in dementia: conceptual issues. *Aging Ment Health, 9*, 386-393.

Maslow, A. (1954). *Motivation and Personality*. New York: Harper & Row.

Mathuranath, P. S., Xuereb, J. H., Bak, T., & Hodges, J. R. (2000). Corticobasal ganglionic degeneration and/or frontotemporal dementia? A report of two overlap cases and review of literature. *J Neurol Neurosurg Psychiatry, 68*, 304-312.

Matthews, G., Jones, D. M., & Chamberlain, A. G. (1990). Refining the measurement of mood: The UWIST Mood adjective Checklist. *British Journal of Psychology, 81*, 17-42.

Mattson, A. J., & Levin, H. S. (1990). Frontal lobe dysfunction following closed head injury. A review of the literature. *J Nerv Ment Dis, 178*, 282-291.

McAllister, T. W., Saykin, A. J., Flashman, L. A., Sparling, M. B., Johnson, S. C., Guerin, S. J., et al. (1999). Brain activation during working memory 1 month after mild traumatic brain injury: a functional MRI study. *Neurology, 53*, 1300-1308.

McAvinue, L., O'Keeffe, F., McMackin, D., & Robertson, I. H. (2005). Impaired sustained attention and error awareness in traumatic brain injury: Implications for insight. *Neuropsychological Rehabilitation, 15*.

McAvinue, L., O'Keeffe, F., McMackin, D., & Robertson, I. H. (2005). Impaired sustained attention and error awareness in traumatic brain injury: Implications for insight. *Neuropsychological Rehabilitation*, 15(5), 569-587.

McDaniel, K., Edland, S., & Heyman, A. (1995). Relationship between level of insight and severity of dementia in Alzheimer's disease. *Alzheimer's Disease and Associated Disorders, 9*, 101-104.

McDowell, S., Whyte, J., & D'Esposito, M. (1997). Working memory impairments in traumatic brain injury: evidence from a dual-task paradigm. *Neuropsychologia, 35*, 1341-1353.

McEvoy, J. P., Freter, S., Everett, G., Geller, J. L., Appelbaum, P., Apperson, L. J., et al. (1989). Insight and the clinical outcome of schizophrenic patients. *J Nerv Ment Dis, 177*, 48-51.

McGlynn, S. M., & Kaszniak, A. W. (1991). Unawareness of deficits in dementia and schizophrenia. In G. P. S. Prigatano, D. (Ed.), *Awareness of Deficits following Brain Injury. Clinical and Theoretical Issues*. New York: Oxford University Press.

McGlynn, S. M., & Kaszniak, A. W. (1991). When metacognition fails: Impaired awareness of deficit in Alzheimer's Disease. *Journal of Cognitive Neuroscience, 3*, 183-189.

McGlynn, S. M., & Schacter, D. L. (1989). Unawareness of deficits in neuropsychological syndromes. *J Clin Exp Neuropsychol, 11*, 143-205.

McKhann, G. M., Albert, M. S., Grossman, M., Miller, B., Dickson, D., & Trojanowski, J. Q. (2001). Clinical and pathological diagnosis of frontotemporal dementia: report of the Work Group on Frontotemporal Dementia and Pick's Disease. *Arch Neurol, 58*, 1803-1809.

McKinlay, W. W., & Brooks, D. N. (1984). Methodological problems in assessing psychoscial recovery following severe head injury. *Journal of Clinical Neuropsychology, 6*, 87-99.

McKinlay, W. W., Brooks, D. N., Bond, M. R., Martinage, D. P., & Marshall, M. M. (1981). The short-term outcome of severe blunt head injury as reported by relatives of the injured persons. *J Neurol Neurosurg Psychiatry, 44*, 527-533.

Mehrabian, A., & Epstein, N. (1972). A measure of emotional empathy. *Journal of Personality, 40*, 525-543.

Mesulam, M. M. (1985). *Principles of Behavioral Neurology*. Philadelphia: FA Davis.

Mesulam, M. M. (2000). *Principles of Behavioral and Cognitive Neurology*. New York: Oxford University Press.

Michon, A., Deweer, B., Pillon, B., Agid, Y., & Dubois, B. (1994). Relation of anosognosia to frontal lobe dysfunction in Alzheimer's disease. *J Neurol Neurosurg Psychiatry, 57*, 805-809.

Migliorelli, R., Teson, A., Sabe, L., Petracchi, M., Leiguarda, R., & Starkstein, S. E. (1995). Prevalence and correlates of dysthymia and major depression among patients with Alzheimer's disease. *Am J Psychiatry, 152*, 37-44.

Miller, B. L., Seeley, W. W., Mychack, P., Rosen, H. J., Mena, I., & Boone, K. (2001). Neuroanatomy of the self: evidence from patients with frontotemporal dementia. *Neurology, 57*, 817-821.

Moore, A. D., Stambrook, M., & Peters, L. C. (1989). Coping strategies and adjustment after closed-head injury: a cluster analytical approach. *Brain Inj, 3*, 171-175.

Morris, R. G., & Hannesdottir, K. (2004). Loss of "Awareness" in Alzheimer's Disease. In R. G. Morris & J. T. Becker (Eds.), *The Cognitive Neuropsychology of Alzheimer's Disease*. Oxford: Oxford University Press.

Murrey, G. J., Hale, F. M., & Williams, J. D. (2005). Assessment of anosognosia in persons with frontal lobe damage: clinical utility of the Mayo-Portland Adaptability Inventory (MPAI). *Brain Inj, 19*, 599-603.

Nair, J. (2004). Knowing me, knowing you. Self-awareness in Asperger's and Autism. In B. D. Beitman & J. Nair (Eds.), *Self-Awareness Deficits in*

Psychiatric Patients. Neurobiology, Assessment, and Treatment. London: W.W. Norton & Co.

Neary, D., Snowden, J. S., Gustafson, L., Passant, U., Stuss, D., Black, S., et al. (1998). Frontotemporal lobar degeneration: a consensus on clinical diagnostic criteria. *Neurology, 51*, 1546-1554.

Nedd, K., Sfakianakis, G., Ganz, W., Uricchio, B., Vernberg, D., Villanueva, P., et al. (1993). 99mTc-HMPAO SPECT of the brain in mild to moderate traumatic brain injury patients: compared with CT--a prospective study. *Brain Inj, 7*, 469-479.

Nelson, H. E. (1982). *The National Adult Reading Test (NART): Test Manual* (First ed.). Windsor: NFER- Neslon.

Nelson, H. E., & Willison, J. (1991). *The National Adult Reading Test Manual* (2nd ed.). Windsor: NFER- Nelson.

Newman, A. C., Garmoe, W., Beatty, P., & Ziccardi, M. (2000). Self-awareness of traumatically brain injured patients in the acute inpatient rehabilitation setting. *Brain Inj, 14*, 333-344.

NHIF. (1989). *Task Force on Special Education*. Southborough: NHIF (National Head Injuries Foundation).

Nockelby, D. M., & Deaton, A. V. (1987). Denial versus distress. Coping patterns in post-head trauma patients. *International Journal of Clinical Neuropsychology, 4*.

Noe, E., Ferri, J., Caballero, M. C., Villodre, R., Sanchez, A., & Chirivella, J. (2005). Self-awareness after acquired brain injury Predictors and rehabilitation. *J Neurol, 252*, 168-175.

O'Connell, R. G., Bellgrove, M. A., Dockree, P. M., & I.H., Robertson. (2005). Effects of Self Alert Training (SA) on Sustained Attention Performance in Adult ADHD. *A supplement of the Journal of Cognitive Neuroscience., Cognitive Neuroscience Society Annual Meeting 2005. Abstract Supplement.*

O'Keeffe, F. M., Dockree, P. M., & Robertson, I. H. (2004). Poor insight in traumatic brain injury mediated by impaired error processing? Evidence from electrodermal activity. *Brain Res Cogn Brain Res, 22*, 101-112.

Oddy, M., Coughlan, T., Tyerman, A., & Jenkins, D. (1985). Social adjustment after closed head injury: a further follow-up seven years after injury. *J Neurol Neurosurg Psychiatry, 48*, 564-568.

Ommaya, A. K., & Gennarelli, T. A. (1974). Cerebral concussion and traumatic unconsciousness. Correlation of experimental and clinical observations of blunt head injuries. *Brain, 97*, 633-654.

Ownsworth, T., Clare, L., & Morris, R. (In Press). An integrated biopsychosocial approach to understanding awareness deficits in Alzheimer's disease and brain injury. *Neuropsychological Rehabilitation.*

Ownsworth, T., & Fleming, J. (2005). The Relative Importance of Metacognitive Skills, Emotional Status, and Executive Function in Psychosocial Adjustment Following Acquired Brain Injury. *J Head Trauma Rehabil, 20*, 315-332.

Ownsworth, T., McFarland, K., & Young, M. R. (2000). Self-awareness and psychosocial functioning following acquired brain injury: An evaluation of a group support programme. *Neuropsychological Rehabilitation, 10*, 465-484.

Ownsworth, T. L., McFarland, K., & Young, R. M. (2002). The investigation of factors underlying deficits in self-awareness and self-regulation. *Brain Inj, 16*, 291-309.

Ownsworth, T. L., McFarland, K. M., & Young, R. M. (2000). Development and standardization of the Self-regulation Skills Interview (SRSI): a new clinical assessment tool for acquired brain injury. *Clin Neuropsychol, 14*, 76-92.

Pang, D. (1989). Physics and pathology of closed head injury. In M. D. Lezak (Ed.), *Assessment of the Behavioral Consequences of Head Trauma* (Vol. 7). New York: Alan R. Liss.

Phan, K. L., Taylor, S. F., Welsh, R. C., Ho, S. H., Britton, J. C., & Liberzon, I. (2004). Neural correlates of individual ratings of emotional salience: a trial-related fMRI study. *Neuroimage, 21*, 768-780.

Platek, S. M., Keenan, J. P., Gallup, G. G., Jr., & Mohamed, F. B. (2004). Where am I? The neurological correlates of self and other. *Brain Res Cogn Brain Res, 19*, 114-122.

Ponsford, J. (1988). Neuropsychological assessment: the need for a more pragmatic approach. *Australian Psychologist, 23*, 349-360.

Ponsford, J. (1995). Mechanisms, recovery, and sequelae of traumatic brain injury: A foundation for the R.E.A.L. approach. In J. Ponsford, Sloan, S. & Snow, P. (Ed.), *Traumatic Brain Injury: Rehabilitation for Everyday Adaptive Living.* Hove: Lawrence Erlbaum Associates.

Ponsford, J., & Kinsella, G. (1991). The use of a rating scale of attentional behaviour. *Neuropsychological Rehabilitation, 1*, 241-257.

Ponsford, J., & Kinsella, G. (1992). Attentional deficits following closed-head injury. *J Clin Exp Neuropsychol, 14*, 822-838.

Ponsford, J. L., Olver, J. H., & Curran, C. (1995). A profile of outcome: 2 years after traumatic brain injury. *Brain Inj, 9*, 1-10.

Port, A., Willmott, C., & Charlton, J. (2002). Self-awareness following traumatic brain injury and implications for rehabilitation. *Brain Inj, 16*, 277-289.

Posner, M. (1978). *Chronometric Explorations of the Mind*. Baltimore: John Hopkins University Press.

Powell, J. M., Machamer, J. E., Temkin, N. R., & Dikmen, S. S. (2001). Self-report of extent of recovery and barriers to recovery after traumatic brain injury: a longitudinal study. *Arch Phys Med Rehabil, 82*, 1025-1030.

Premack, D., & Woodruff, G. (1978). Does the chimpanzee have a theory of mind? *Behavioural Science, 4*, 515-526.

Prigatano, G. P. (1988). Emotion and motivation in recovery and adaptation after brain damage. In S. Finger, T. LeVere, C. Almi & D. Stein (Eds.), *Brain Injury and Recovery: Theoretical and Controversial Issues*. NY: Plenum Press.

Prigatano, G. P. (1991). Disturbances of self-awareness of deficit after traumatic brain injury. In G. P. Prigatano & D. L. Schacter (Eds.), *Awareness of Deficit after Brain Injury: Clinical and Theoretical Issues*. New York: Oxford University Press.

Prigatano, G. P. (1992). Personality disturbances associated with traumatic brain injury. *Journal of Clinical and Counselling Psychology, 60*, 360-368.

Prigatano, G. P. (1996). Behavioral limitations TBI patients tend to underestimate: A replication and extension to patients with lateralized cerebral dysfunction. *Clinical Neuropsychologist, 10*, 191-201.

Prigatano, G. P. (1999a). Diller lecture: Impaired awareness, finger tapping, and rehabilitation outcome after brain injury. *Rehabilitation Psychology, 44*.

Prigatano, G. P. (1999b). Disorders of self-awareness after brain injury. In G. P. Prigatano (Ed.), *Principles of Neuropsychological Rehabilitation* (pp. 265-293). New York: Oxford University Press.

Prigatano, G. P. (2005). Disturbances of self-awareness and rehabilitation of patients with traumatic brain injury: a 20-year perspective. *J Head Trauma Rehabil, 20*, 19-29.

Prigatano, G. P., & Altman, I. M. (1990). Impaired awareness of behavioral limitations after traumatic brain injury. *Arch Phys Med Rehabil, 71*, 1058-1064.

Prigatano, G. P., Altman, I. M., & O'Brien, K. P. (1990). Behavioral limitations that traumatic-brain-injured patients tend to underestimate. *Clinical Neuropsychologist, 4*, 163-176.

Prigatano, G. P., Borgaro, S., Baker, J., & Wethe, J. (2005). Awareness and Distress After Traumatic Brain Injury: A Relative's Perspective. *J Head Trauma Rehabil, 20*, 359-367.

Prigatano, G. P., Bruna, O., Mataro, M., Munoz, J. M., Fernandez, S., & Junque, C. (1998). Initial disturbances of consciousness and resultant impaired awareness in Spanish patients with traumatic brain injury. *Journal of Head Trauma Rehabilitation, 13*, 29-38.

Prigatano, G. P., & Fordyce, D. J. (1986). Cognitive dysfunction and psychosocial adjustment after brain injury. In G. P. Prigatano (Ed.), *Neuropsychological Rehabilitation after Brain Injury*. Baltimore: John Hopkins University Press.

Prigatano, G. P., Fordyce, D. J., & Zeiner, H. K. (1986). *Neuropsychological Rehabilitation After Brain Injury*. Baltimore: John Hopkins University Press.

Prigatano, G. P., & Johnson, S. C. (2003). The three vectors of consciousness and their disturbances after brain injury. *Neuropsychological Rehabilitation, 13*, 13-29.

Prigatano, G. P., & Klonoff, P. S. (1988). Psychotherapy and Neuropsychological assessment after brain injury. *Journal of Head Trauma Rehabilitation, 3*, 45-46.

Prigatano, G. P., & Klonoff, P. S. (1998). A clinician's rating scale for evaluating impaired self-awareness and denial of disability after brain injury. *Clinical Neuropsychologist, 12*, 56-67.

Prigatano, G. P., & Leathem, J. M. (1993). Awareness of behavioral limitations after traumatic brain injury: a cross-cultural study of New Zealand Maoris and non-Maoris. *Clinical Neuropsychologist, 7*, 123-135.

Prigatano, G. P., Ogano, M., & Amakusa, B. (1997). A cross-cultural study on impaired self-awareness in Japanese patients with brain dysfunction. *Neuropsychiatry Neuropsychol Behav Neurol, 10*, 135-143.

Prigatano, G. P., & Schacter, D. L. (1991). *Awareness of Deficit after Brain Injury: Clinical and Theoretical Issues*. New York: Oxford University Press.

Prigatano, G. P., & Weinstein, E. A. (1996). Edwin A. Weinstein's contribution to neuropsychological rehabilitation. *Neuropsychological Rehabilitation, 6*, 305-326.

Ptak, R., & Schnider, A. (1999). Spontaneous confabulations after orbitofrontal damage: The role of temporal context confusion and self-monitoring. *Neurocase, 5*, 243-250.

Raichle, M. E., MacLeod, A. M., Snyder, A. Z., Powers, W. J., Gusnard, D. A., & Shulman, G. L. (2001). A default mode of brain function. *Proc Natl Acad Sci U S A, 98*, 676-682.

Rankin, K. P., Baldwin, E., Pace-Savitsky, C., Kramer, J. H., & Miller, B. L. (2005). Self awareness and personality change in dementia. *J Neurol Neurosurg Psychiatry, 76*, 632-639.

Ranseen, J., Bohaska, L., & Schmitt, F. (1990). An investigation of anosognosia following traumatic head injury. *International Journal of Clinical Neuropsychology, 12*, 29-36.

Reed, B. R., Jagust, W. J., & Coulter, L. (1993). Anosognosia in Alzheimer's disease: relationships to depression, cognitive function, and cerebral perfusion. *J Clin Exp Neuropsychol, 15*, 231-244.

Reitan, R. M. (1955). *Manual for Administration of Neuropsychological Test Batteries for Adults and Children.* Indiana: Indiana University Medical Center, Indianapolis.

Reitan, R. M. (1958). Validity of the Trail Making Test as an indicator of organic brain damage. *Perceptual & Motor Skills, 8*, 271-276.

Richardson, J. (2000). *Clinical and Neuropsychological Aspects of Closed Head Injury* (2nd ed.). Uxbridge: Psychology Press.

Robertson, I. H., Manly, T., Andrade, J., Baddeley, B. T., & Yiend, J. (1997). 'Oops!' Performance correlates of everyday attentional failures in traumatic brain injured and normal subjects. *Neuropsychologia, 35*, 747-758.

Robertson, I. H., Ward, T., Ridgeway, V., & Nimmo-Smith, I. (1996). The structure of normal human attention: The Test of Everyday Attention. *J Int Neuropsychol Soc, 2*, 525-534.

Roche, N. L., Fleming, J., & Shum, D. H. K. (2002). Self-awareness of prospective memory failure in adults with traumatic brain injury. *Brain Injury, 16*, 931-945.

Romano, M. D. (1974). Family response to traumatic brain injury. *Scandinavian Journal of Rehabilitation Medicine, 6*, 1-4.

Russel, W. R., & Nathan, P. W. (1946). Traumatic Amnesia. *Brain, 69*, 280-300.

Sachs, E. (1927). Symptomatology of a group of frontal lobe lesions. *Brain, 50*, 474-479.

Sander, A., Seel, R. T., Kreutzer, J., Hall, K., High, W. M., Jr., & Rosenthal, M. (1997). Agreement between persons with traumatic brain injury and their relatives regarding psychosocial outcome using the community integration questionnaire. *Arch Phys Med Rehabil, 78*, 353-357.

Sawchyn, J. M. (2001). *Awareness of neurobehavioral deficits and emotional adjustment in acute- and post-acute rehabilitation following traumatic brain injury.* Unpublished PhD, University of Victoria.

Sawchyn, J. M., Mateer, C. A., & Suffield, J. B. (2005). Awareness, emotional adjustment, and injury severity in postacute brain injury. *J Head Trauma Rehabil, 20*, 301-314.

Sbordone, R. J., Seyranian, G. D., & Ruff, R. M. (1998). Are the subjective complaints of traumatically brain injured patients reliable? *Brain Inj, 12*, 505-515.

Schacter, D., Glisky, E. L., & McGlynn, S. M. (1990). Impact of memory disorder on everyday life: Awareness of deficits and return to work. In D. C. Tupper, K. (Ed.), *The Neuropsychology of Everyday life.* Boston: Martinus Nijhoff.

Schacter, D. L. (1990). Toward a cognitive neuropsychology of awareness: implicit knowledge and anosognosia. *J Clin Exp Neuropsychol, 12*, 155-178.

Schacter, D. L., & Prigatano, G. P. (1991). Forms of unawareness. In G. P. Prigatano & D. L. Schacter (Eds.), *Awareness of Deficits following Brain Injury. Clinical and Theoretical Issues*. New York: Oxford University Press.

Schmitz, T.W., Rowley, H.A., Kawahara, T.N., Johnson, S.C. Neural correlates of self evaluative accuracy after traumatic brain injury. Neuropsychologia, In Press.

Schmitter-Edgecombe, M., & Woo, E. (2004). Memory self-awareness and memory self-monitoring following severe closed-head injury. *Brain Injury, 18*, 997-1016.

Schnider, A., & Ptak, R. (1999). Spontaneous confabulators fail to suppress currently irrelevant memory traces. *Nat Neurosci, 2*, 677-681.

Schwartz, M. F., Buxbaum, L. J., Ferraro, M., Veramonti, T., & Segal, M. (2002). *NAT The Naturalistic Action Test Manual*. Bury St. Edmunds.: Thames Valley Test Company.

Schwartz, M. F., Buxbaum, L. J., Montgomery, M. W., Fitzpatrick-DeSalme, E., Hart, T., Ferraro, M., et al. (1999). Naturalistic action production following right hemisphere stroke. *Neuropsychologia, 37*, 51-66.

Schwartz, M. F., Segal, M., Veramonti, T., Ferraro, M., & Buxbaum, L. J. (2002). The Naturalistic Action Test: A standardised assessment for everyday action impairment. *Neuropsychological Rehabilitation, 12*, 311-339.

Seel, R. T., Kreutzer, J. S., & Sander, A. M. (1997). Concordance of patients' and family members' ratings of neurobehavioral functioning after traumatic brain injury. *Arch Phys Med Rehabil, 78*, 1254-1259.

Seiffer, A., Clare, L., & Harvey, I. (In Press). The role of personality and coping in relation to awareness of current functioning in early-stage dementia. *Aging and Mental Health.*

Seltzer, B., Vasterling, J. J., Hale, M. A., & Khurana, R. (1995). Unawareness of memory deficit in Alzheimer's Disease: Relation to mood and other disease variables. *Neuropsychiatry Neuropsychol Behav Neurol, 8*, 176-181.

Seltzer, B., Vasterling, J. J., Mathias, C. W., & Brennan, A. (2001). Clinical and neuropsychological correlates of impaired awareness of deficits in

Alzheimer's disease and Parkinson's disease: a comparative study. *Neuropsychiatry Neuropsychol Behav Neurol, 14*, 122-129.

Seltzer, B., Vasterling, J. J., Yoder, J. A., & Thompson, K. A. (1997). Awareness of deficit in Alzheimer's disease: relation to caregiver burden. *Gerontologist, 37*, 20-24.

Shallice, T. (1999). The origin of confabulations. *Nat Neurosci, 2*, 588-590.

Shallice, T., & Burgess, P. (1988). *From Neuropsychology to Mental Structure*. Cambridge: Cambridge University Press.

Shallice, T., & Burgess, P. (1996). The domain of supervisory processes and temporal organization of behaviour. *Philos Trans R Soc Lond B Biol Sci, 351*, 1405-1411; discussion 1411-1402.

Sherer, M., Bergloff, P., Boake, C., High, W., Jr., & Levin, E. (1998). The Awareness Questionnaire: factor structure and internal consistency. *Brain Inj, 12*, 63-68.

Sherer, M., Bergloff, P., Levin, E., High, W. M., Jr., Oden, K. E., & Nick, T. G. (1998). Impaired awareness and employment outcome after traumatic brain injury. *J Head Trauma Rehabil, 13*, 52-61.

Sherer, M., Boake, C., Levin, E., Silver, B., Ringholz, G., & High, W. M., Jr. (1998). Characteristics of impaired awareness after traumatic brain injury. *Journal of the International Neuropsychological Society, 4*, 380-387.

Sherer, M., Hart, T., & Nick, T. G. (2003). Measurement of impaired self-awareness after traumatic brain injury: a comparison of the patient competency rating scale and the awareness questionnaire. *Brain Inj, 17*, 25-37.

Sherer, M., Hart, T., Nick, T. G., Whyte, J., Thompson, R. N., & Yablon, S. A. (2003). Early impaired self-awareness after traumatic brain injury. *Arch Phys Med Rehabil, 84*, 168-176.

Sherer, M., Hart, T., Whyte, J., Nick, T. G., & Yablon, S. A. (2005). Neuroanatomic Basis of Impaired Self-awareness After Traumatic Brain Injury: Findings From Early Computed Tomography. *J Head Trauma Rehabil, 20*, 287-300.

Sherer, M., Oden, K. E., Bergloff, P., Levin, E., & High, W. M., Jr. (1998). Assessment and treatment of impaired awareness after brain injury: Implications for community re-integration. *NeuroRehabilitation, 10*, 25-37.

Shimamura, A. P., Jernigan, T. L., & Squire, L. R. (1988). Korsakoff's syndrome: radiological (CT) findings and neuropsychological correlates. *J Neurosci, 8*, 4400-4410.

Shimamura, A. P., & Squire, L. R. (1986). Memory and metamemory: a study of the feeling-of-knowing phenomenon in amnesic patients. *J Exp Psychol Learn Mem Cogn, 12*, 452-460.

Silver, J. M., Hales, R. E., & Yudofsky, S. (2002). Neuropsychiatric aspects of traumatic brain injury. In S. C. Y. R. E. Hales (Ed.), *American Psychiatric Press Textbook of Neuropsychiatry* (3rd ed.). Washington DC: American Psychiatric Press.

Simmond, M., & Fleming, J. (2003a). Occupational therapy assessment of self-awareness following traumatic brain injury. *British Journal of Occupational Therapy, 66*, 447-453.

Simmond, M., & Fleming, J. (2003b). Reliability of the self-awareness of deficits interview for adults with traumatic brain injury. *Brain Inj, 17*, 325-337.

Sohlberg, M. M. (2000). Assessing and managing unawareness of self. *Semin Speech Lang, 21*, 135-150; quiz 150-131.

Sohlberg, M. M., Mateer, C. A., Penkman, L., Glang, A., & Todis, B. (1998). Awareness intervention: who needs it? *J Head Trauma Rehabil, 13*, 62-78.

Soliveri, P., Monza, D., Paridi, D., Radice, D., Grisoli, M., Testa, D., et al. (1999). Cognitive and magnetic resonance imaging aspects of corticobasal degeneration and progressive supranuclear palsy. *Neurology, 53*, 502-507.

Souchay, C., Isingrini, M., Pillon, B., & Gil, R. (2003). Metamemory accuracy in Alzheimer's disease and frontotemporal lobe dementia. *Neurocase, 9*, 482-492.

Sperry, R. W. (1974). Lateral specialization in the surgically separated hemispheres. In F. O. Schmitt & F. G. Worden (Eds.), *The Neurosciences*. Cambridge: Massachusetts Institute of Technology.

Spreen, O., & Benton, A. L. (1969). *Neurosensory Center Comprehensive Examination for Aphasia (NCCEA)*. Victoria: University of Victoria Neuropsychology Laboratory.

Spreen, O., & Strauss, E. (1998). *A Compendium of Neuropsychological Tests. Administration, Norms and Commentary* (2nd ed.). New York: Oxford University Press.

Squire, L. R., Amaral, D. G., & Press, G. A. (1990). Magnetic resonance imaging of the hippocampal formation and mammillary nuclei distinguish medial temporal lobe and diencephalic amnesia. *J Neurosci, 10*, 3106-3117.

Starkstein, S. E., Chemerinski, E., Sabe, L., Kuzis, G., Petracca, G., Teson, A., et al. (1997). Prospective longitudinal study of depression and anosognosia in Alzheimer's disease. *British Journal of Psychiatry, 171*, 47-52.

Starkstein, S. E., Fedoroff, J. P., Price, T. R., Leiguarda, R., & Robinson, R. G. (1992). Anosognosia in patients with cerebrovascular lesions. A study of causative factors. *Stroke, 23*, 1446-1453.

Starkstein, S. E., Vazquez, S., Migliorelli, R., Teson, A., Sabe, L., & Leiguarda, R. (1995). A single-photon emission computed tomographic study of anosognosia in Alzheimer's disease. *Arch Neurol, 52*, 415-420.

Stemmer, B., Segalowitz, S. J., Witzke, W., & Schonle, P. W. (2004). Error detection in patients with lesions to the medial prefrontal cortex: an ERP study. *Neuropsychologia, 42*, 118-130.

Stern, R., Ray, W. J., & Quigley, K. S. (2001). *Psychophysiological Recording*. Oxford: Oxford University Press.

Stuss, D. (1987). Contribution of frontal lobe injury to cognitive impairment after closed head injury: Methods of assessment and recent findings. In H. Levin, J. Grafman & D. Eisenberg (Eds.), *Neurobehavioral Recovery after brain injury*. New York: Oxford University Press.

Stuss, D., & Alexander, M. P. (2000). Affectively burnt in: A proposed role of the right frontal lobe. In E. Tulving (Ed.), *Memory, consciousness and the brain: The Tallin Conference*. Philadelphia: Psychology Press.

Stuss, D., & Gow, C. A. (1992). 'Frontal Dysfunction' after traumatic brain injury. *Neuropsychiatry, neuropsychology and behavioral neurology, 5*, 272-282.

Stuss, D. T. (1991). Disturbance of self-awareness after frontal system damage. In G. P. Prigatano & D. L. Schacter (Eds.), *Awareness of deficit after brain injury:Clinical and theoretical issues* (pp. 63-83). New York: Oxford University Press.

Stuss, D. T., & Benson, D. F. (1986). *The Frontal Lobes*. New York: Raven Press.

Stuss, D. T., Gallup, G. G., Jr., & Alexander, M. P. (2001). The frontal lobes are necessary for 'theory of mind'. *Brain, 124*, 279-286.

Stuss, D. T., Picton, T. W., & Alexander, M. P. (2001). Consciousness, self-awareness and the frontal lobes. In S. Salloway, P. Malloy & J. Duffy (Eds.), *The Frontal Lobes and Neuropsychiatric Illness*. Washington: American Psychiatric Press.

Sunderland, A., Harris, J. E., & Gleave, J. (1984). Memory failures in everyday life following severe head injury. *J Clin Neuropsychol, 6*, 127-142.

Teasdale, G. (1995). Head Injury. *Journal of Neuorlogy, Neurosurgery and Psychiatry, 58*, 526-539.

Teasdale, G. M., & Mathew, P. (1996). Mechanism of cerebral concussion, contusion and other effects of head injury. In J. Youmans (Ed.), *Neurological Surgery*. Philadelphia: Saunders.

Tepper, S., Beatty, P., & DeJong, G. (1996). Outcomes in traumatic brain injury: self-report versus report of significant others. *Brain Inj, 10*, 575-581.

Toglia, J. (1998). A dynamic interactional model to cognitive rehabilitation. In N. Katz (Ed.), *Cognition and occupation in rehabilitation* (pp. 5-50). Bethesda: American Occupational Therapy Association.

Toglia, J., & Kirk, U. (2000). Understanding awareness deficits following brain injury. *NeuroRehabilitation, 15*, 57-70.

Tranel, D., & Damasio, H. (1994). Neuroanatomical correlates of electrodermal skin conductance responses. *Psychophysiology, 31*, 427-438.

Tranel, E., & Damasio, A. R. (1985). Knowledge without awareness: An automatic index of facial recognition by prosopagnosics. *Science, 228*, 1453-1454.

Trosset, M. W., & Kaszniak, A. W. (1996). Measures of deficit unawareness for predicted performance experiments. *J Int Neuropsychol Soc, 2*, 315-322.

Trudel, T. M., Tryone, W. W., & Purdum, C. M. (1998). Awareness of Disability and Long-term outcome after traumatic brain injury. *Rehabilitation Psychology, 43*, 267-281.

Tulving, E. (1985). Memory and consciousness. *Canadian Psychology, 25*, 1-12.

Turner, G., & Levine, B. (2004). Disorders of Executive Functioning and Self-Awareness. In J. Ponsford (Ed.), *Cognitive and Behavioral Rehabilitation: From Neurobiology to Clinical Practice* (pp. 224-268). New York: Guilford Press.

Tyerman, A., & Humphrey, M. (1984). Changes in self-concept following severe head injury. *International Journal of Rehabilitation Research, 7*, 11-23.

Ullsperger, M., von Cramon, D. Y., & Muller, N. G. (2002). Interactions of focal cortical lesions with error processing: evidence from event-related brain potentials. *Neuropsychology, 16*, 548-561.

Umilta, C., & Stablum, F. (1998). Control processes explored by the study of closed head injury patients. In G. Mazzoni & T. O. Nelson (Eds.), *Metacognition and Cognitive Neuropsychology*. Mahwah, NJ.: Lawrence Erlbaum Associates.

vanZomeren, A. H., & Brouwer, W. H. (1994). *Clinical Neuropsychology of Attention*. New York: Oxford University Press.

Varney, N. R., & Medefee, L. (1993). Psychosocial and executive deficits following closed head injury: implications for the orbital frontal cortex. *Journal of Head Trauma Rehabilitation, 8*, 32-44.

Vasterling, J. J., Seltzer, B., Foss, J. W., & Vanderbrook, V. (1995). Unawareness of deficit in Alzheimer's disease. Domain-specific differences and disease correlates. *Neuropsychiatry Neuropsychol Behav Neurol, 8*, 26-32.

Vasterling, J. J., Seltzer, B., & Watrous, W. E. (1997). Longitudinal assessment of deficit unawareness in Alzheimer's disease. *Neuropsychiatry Neuropsychol Behav Neurol, 10*, 197-202.

Vilalta-Franch, J., Lozano-Gallego, M., Hernandez-Ferrandiz, M., Llinas-regla, J., Lopez-Pousa, S., & Lopez, O. (1999). The Neuropsychiatric Inventory. Psychometric properties of its adaptation into Spanish. *Spanish Revue Neurologie, 29*, 15-19.

Vilkki, J. (1985). Amnesic syndromes after surgery of anterior communicating artery aneurysms. *Cortex, 21*, 431-444.

Vilkki, J. (1992). Cognitive flexibility and mental programming after closed head injuries and anterior or posterior cerebral excisions. *Neuropsychologia, 30*, 807-814.

Vilkki, J., & Holst, P. (1991). Mental Programming after frontal lobe lesions: Results on digit symbol performance with self-selected goals. *Cortex, 27*, 203-211.

Vogeley, K., Bussfeld, P., Newen, A., Herrmann, S., Happe, F., Falkai, P., et al. (2001). Mind reading: neural mechanisms of theory of mind and self-perspective. *Neuroimage, 14*, 170-181.

Vogeley, K., May, M., Ritzl, A., Falkai, P., Zilles, K., & Fink, G. R. (2004). Neural correlates of first-person perspective as one constituent of human self-consciousness. *J Cogn Neurosci, 16*, 817-827.

von Monakow, C. (1885). Experimentelle und pathologisch-anatomische Untersuchungen ueber die Beziehungen der sogentannen Sehsphaere zu den infracorticalen Opticuscentren und zum N. opticus. *Archiv fuer Psychiatrie, 16*, 151-199.

Walker, D. (2003). *Remembrance of things future: involuntary and strategic processes in prospective memory reminders.* Unpublished PhD, University of Reading, Reading, UK.

Wallace, C. A., & Bogner, J. (2000). Awareness of deficits: emotional implications for persons with brain injury and their significant others. *Brain Inj, 14*, 549-562.

Walsh, K. W. (1991). *Understanding brain damage: A primer of neuropsychological evaluation* (2nd ed.). Edinburgh: Churchill Livingstone.

Warrington, E. K. J., M. (1991). *The Visual Object and Space Perception Battery.* Suffolk: Thames Valley Test Company.

Wechsler, D., Wycherly, R., Benjamin, L., Crawford, J. R., & Mockler, D. (1998). *Wechsler Memory Scale-111* (Third ed.). London: The Psychological Corporation Limited.

Weinstein, E., Friedland, R., & Wagner, E. E. (1992). Denial/unawareness of impairment in Alzheimer's Disease. *Neurology, 42 (Suppl 3)*, 200.

Weinstein, E. A. (1991). Anosognosia and denial of illness. In G. P. Prigatano & D. L. Schacter (Eds.), *Awareness of Deficits after brain injury: Clinical and Theoretical issues* (pp. 240-257). New York: Oxford University Press.

Weinstein, E. A., Friedland, R., & Wagner, E. E. (1994). Denial/unawareness of impairment and symbolic behavior in Alzheimer's disease. *Neuropsychiatry Neuropsychol Behav Neurol, 7*, 176-184.

Weinstein, E. A., & Kahn, R. L. (1955). *Denial of Illness. Symbolic and Physiological Aspects*. Springfield: Charles C. Thomas.

Weinstein, E. A., Lyerly, O. G., & Cole, M. (1966). Meaning in jargon aphasia. *Cortex, 2*, 165-187.

Weiskrantz, L. (1986). *Blindsight*. New York: Oxford University Press.

Whitlock, F. A. (1981). Some observations on the meaning of confabulation. *British Journal of Medical Psychology, 54*, 213-218.

Wilson, B. A., Alderman, N., Burgess, P. W., Emslie, H., & Evans, J. (1996). *Behavioural Assessment of the Dysexecutive Syndrome*. Bury St Edmunds: Thames Valley Test Company.

Wise, K., Ownsworth, T., & Fleming, J. (2005). Convergent validity of self-awareness measures and their association with employment outcome in adults following acquired brain injury. *Brain Injury, 19*, 765-775.

Zahn, T. P., Grafman, J., & Tranel, D. (1999). Frontal lobe lesions and electrodermal activity: effects of significance. *Neuropsychologia, 37*, 1227-1241.

Zahn, T. P., & Mirsky, A. F. (1999). Autonomic activity during task performance in adults with closed head injury. *Int J Psychophysiol, 33*, 113-126.

Zaidel, E. (1987). Hemispheric monitoring. In D. Ottoson (Ed.), *Duality and Unity of the Brain* (pp. 247-281). London: Macmillan.

Zangwill, O. L. (1966). The amnesic syndrome. In C. W. M. Z. Whitty, O.L. (Ed.), *Amnesia* (pp. 104-117). London: Butterworths.

Zeman, A. (2001). Consciousness. *Brain, 124*, 1263-1289.

Zhang, Q., & Sachdev, P. S. (2003). Psychotic disorder and traumatic brain injury. *Curr Psychiatry Rep, 5*, 197-201.

Zhou, J., Chittum, J. M., Johnson, K., Poppen, R., Guercio, J. M., & McMorrow, M. J. (1996). The utilization of a game format to increase knowledge of residuals among people with acquired brain injury. *Journal of Head Trauma Rehabilitation, 11*, 51-61.

Zigmond, A. S., & Snaith, R. P. (1983). The hospital anxiety and depression scale. *Acta Psychiatr Scand, 67*, 361-370.

Zimmer, H. (2000). Frequency and mean amplitude of spontaneous electrodermal fluctuations are not interchangeable indicators of psychological processes. *Z Exp Psychol, 47*, 129-143.

Appendices

Awareness Interview for Study 2

Awareness Interview for Study 3

Cognitive Failures Questionnaire- Self (CFQ-Self)

Cognitive Failures Questionnaire- Other (CFQ-Other)

Hospital Anxiety and Depression Scale (HADS)

National Adult Reading Test (NART)

Measure of Empathic Tendency (MET)

Awareness Interview Schedule for Study 2

1) Awareness of the reason for hospitalisation
What is wrong with you?
Why are you in hospital?

If Patient does not explicitly describe the primary reason for hospitalisation
Did you have an accident or hit your head?

2) Awareness of impairments of intellect or "thinking ability"
How is your thinking?
Are you thinking as clearly as you normally do?

3) Awareness of orientation problems
Are you ever confused about where you are or what month of the year it is?

4) Awareness of memory impairment
Are you having any trouble with your memory?

5) Awareness of attention impairment
How is your concentration? Do you have trouble paying attention?

6) Awareness of speech or language problems
How is your speech? Has it been affected at all? Do you have any difficulty understanding what other people say?

7) Awareness of activities of daily living impairments
In your current state do you have any problems with daily activities?
Eating?
Dressing?
Washing?
Getting about?

8) Awareness of quality of test performance and ability to return to normal activities
How do you think you did on these tests today?
Based on how you are doing now, do you think you will be able to return to your normal activities in the next several weeks? (Specify activities, eg. Employment, hobbies, activities of daily living)

Awareness Interview Schedule for Study 3

1) Awareness of the reason for hospitalisation
What is wrong with you?
Why are you in hospital?

If Patient does not explicitly describe the primary reason for hospitalisation
Did anything happen to you, or are you having difficulties which may have brought you in?

2) Awareness of motor impairments
Question patient regarding movement of his or her arms and legs.
How do your arms work?
Can you move them normally?
Both of them?

3) Awareness of impairments of intellect or "thinking ability"
How is your thinking?
Are you thinking as clearly as you normally do?

4) Awareness of orientation problems
Are you ever confused about where you are or what month of the year it is?

5) Awareness of memory impairment
Are you having any trouble with your memory?

6) Awareness of attention impairment
How is your concentration? Do you have trouble paying attention?

7) Awareness of speech or language problems
How is your speech? Has it been affected at all? Do you have any difficulty understanding what other people say?

8) Awareness of Visual impairments
Are you having trouble with your vision?

9) Awareness of activities of daily living impairments
In your current state do you have any problems with daily activities?
Eating?
Dressing?
Washing?
Getting about?

10) Awareness of quality of test performance and ability to return to normal activities
How do you think you did on these tests today?
Based on how you are doing now, do you think you will be able to return to your normal activities in the next several weeks? (Specify activities, eg. Employment, hobbies, activities of daily living)

CFQ

Name ……. Date……

…….

The following questions are about minor mistakes which everyone makes from time to time, but some of which happen more often than others. We want to know how often these things have happened to you in the last six months. Please circle the appropriate number.

	Very often	**Quite often**	**Occasio-nally**	**Very rarely**	**Never**
Do you read something and find you haven't been thinking about it and must read it again?	4	3	2	1	0
Do you find you forget why you went from one part of the house to the other?	4	3	2	1	0
Do you fail to notice signposts on the road?	4	3	2	1	0
Do you find you confuse right and left when giving directions?	4	3	2	1	0
Do you bump into people?	4	3	2	1	0
Do you find you forget whether you've turned off a light or a fire or locked the door?	4	3	2	1	0
Do you fail to listen to people's names when you are meeting them?	4	3	2	1	0
Do you say something and realise afterwards that it might be taken as insulting?	4	3	2	1	0
Do you fail to hear people speaking to you when you are doing something else?	4	3	2	1	0
Do you lose your temper and regret it?	4	3	2	1	0
Do you leave important letters unanswered for days?	4	3	2	1	0
Do you find you forget which way to turn on a road you know well but rarely use?	4	3	2	1	0
Do you fail to see what you want in a supermarket (although it's there)?	4	3	2	1	0
Do you find yourself suddenly wondering whether you've used a word correctly?	4	3	2	1	0
Do you have trouble making up your mind?	4	3	2	1	0

Do you find you forget appointments?	4	3	2	1	0
Do you forget where you put something like a newspaper or a book?	4	3	2	1	0
Do you find you accidentally throw away the thing you want and keep what you meant to throw away as in the example of throwing away the matchbox and putting the used match in your pocket?	4	3	2	1	0
Do you daydream when you ought to be listening to something?	4	3	2	1	0
Do you find you forget people's names?	4	3	2	1	0
Do you start doing one thing at home and get distracted into doing something else (unintentionally)?	4	3	2	1	0
Do you find you can't quite remember something although it's 'on the tip of your tongue'?	4	3	2	1	0
Do you find you forget what you came to the shops to buy?	4	3	2	1	0
Do you drop things?	4	3	2	1	0
Do you find you can't think of anything to say?	4	3	2	1	0

CFQ (For Others)

The questions given below are about mistakes and difficulties which everybody has from time to time. We would like you to think of a recent two-month period when _____ has shown any of these difficulties. After each question please tick only one of five possible answers. Please make sure you read them carefully because for some questions "very often" appears on the left side of the page and "never" is on the right, but for other questions "never" is on the left and "very often" is on the right.

During the last two months _____ seemed to be:

1. Absentminded, that is making mistakes in what he/she is doing because he/she is thinking of something else?

Very often	Quite often	Occasionally	Very rarely	Never
4	3	2	1	0

2. Finding it difficult to concentrate on anything because his/her attention tends to wander from one thing to another?

Very often	Quite often	Occasionally	Very rarely	Never
4	3	2	1	0

3. Forgetful, such as forgetting where he/she has put things, or about appointments, or about what he/she has done?

Very often	Quite often	Occasionally	Very rarely	Never
4	3	2	1	0

4. Busy thinking about his/her own affairs and so not noticing what is going on around him/her?

Very often	Quite often	Occasionally	Very rarely	Never
4	3	2	1	0

5. Clumsy, for example, dropping things or bumping into people?

Very often	Quite often	Occasionally	Very rarely	Never
4	3	2	1	0

6. Having difficulty in making up his/her mind?

Very often	Quite often	Occasionally	Very rarely	Never
4	3	2	1	0

7. Disorganised, that is, getting into a muddle when doing something because of lack of planning or concentration?

Very often	Quite often	Occasionally	Very rarely	Never
4	3	2	1	0

8. Getting unduly cross about minor matters?

Very often	Quite often	Occasionally	Very rarely	Never
4	3	2	1	0

HAD Scale

Read each item and place a firm tick in the box opposite the reply which comes closest to how you have been feeling in the past week. Don't take too long over your replies: your immediate reaction to each item will probably be more accurate than a long thought-out response.

1. I feel tense or 'wound up'

- Most of the time ☐
- A lot of the time ☐
- Time to time, Occasionally ☐
- Not at all ☐

2. I still enjoy the things I used to enjoy

- Definitely as much ☐
- Not quite so much ☐
- Only a little ☐
- Hardly at all ☐

3. I get a sort of frightened feeling as if something awful is about to happen

- Very definitely and quite badly ☐
- Yes, but not too badly ☐
- A little, but it doesn't worry me ☐
- Not at all ☐

4. I can laugh and see the funny side of things.

- As much as I always could ☐
- Not quite so much now ☐
- Definitely not so much now ☐
- Not at all ☐

5. Worrying thoughts go through my mind

- A great deal of the time ☐
- A lot of the time ☐
- From time to time but not often ☐
- Only occasionally ☐

6. I feel cheerful

- Not at all ☐
- Not often ☐
- Sometimes ☐
- Most of the time ☐

7. I can sit at ease and feel relaxed

- Definitely ☐
- Usually ☐
- Not often ☐
- Not at all ☐

8. I feel as if I am slowed down

- Nearly all the time ☐
- Very often ☐
- Sometimes ☐
- Not at all ☐

9. I get a sort of frightened feeling like 'butterflies' in the stomach.

- Not at all ☐
- Occasionally ☐
- Quite often ☐
- Very often ☐

10. I have lost interest in my appearance

- Definitely ☐
- I don't take so much care as I should ☐
- I may not take quite as much care ☐
- I take just as much care as ever ☐

11. I feel restless as if I have to be on the move.

- Very much indeed ☐
- Quite a lot ☐
- Not very much ☐
- Not at all ☐

12. I look forward with enjoyment to things.

- As much as ever I did ☐
- Rather less than I used to ☐
- Definitely less than I used to ☐
- Hardly at all ☐

13. I get sudden feelings of panic

- Very often indeed ☐
- Quite often ☐
- Not very often ☐
- Not at all ☐

14. I can enjoy a good book or radio or TV programme

- Often ☐
- Sometimes ☐
- Not often ☐
- Very seldom ☐

National Adult Reading Test

Please read aloud the following words:

Ache ____
Debt ____
Psalm ____
Depot ____
Chord ____
Bouquet ____
Deny ____
Capon ____
Heir ____
Aisle ____
Subtle ____
Nausea ____
Equivocal ____
Naïve ____
Thyme ____
Courteous ____
Gaoled ____
Procreate ____
Quadruped ____
Catacomb ____
Superfluous ____
Radix ____
Assignate ____
Gist ____
Hiatus ____

Simile ____
Aeon ____
Cellist ____
Zealot ____
Abstemious ____
Gouge ____
Placebo ____
Façade ____
Aver ____
Leviathan ____
Chagrin ____
Détente ____
Gauche ____
Drachm ____
Idyll ____
Beatify ____
Banal ____
Sidereal ____
Puerperal ____
Topiary ____
Demesne ____
Labile ____
Phlegm ____
Syncope ____
Prelate ____

MET

Date:_____

Name:_____ Age:_____ Sex:_____

Please read each item and circle the response that comes closest to how you feel.

1. It makes me sad to see a lonely stranger in a group.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

2. People make too much of the feelings and sensitivity of animals.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

3. I often find public displays of affection annoying.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

4. I am annoyed by unhappy people who are just sorry for themselves

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

5. I become nervous if others around me seem to be nervous.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

6. I find it silly for people to cry out of happiness.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

7. I tend to get emotionally involved with a friend's problems.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

8. Sometimes the words of a love song can move me deeply.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

9. I tend to lose control when I am bringing bad news to people.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

10. The people around me have a great influence on my moods.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

11. Most foreigners I have met seemed cool and unemotional.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

12. I would rather be a social worker than work in a job in a training centre.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

13. I don't get upset just because a friend is acting upset.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

14. I like to watch people open presents.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

15. Lonely people are probably unfriendly.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

16. Seeing people cry upsets me.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

17. Some songs make me happy.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

18. I really get involved with the feelings of the characters in a novel.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

19. I get very angry when I see someone being ill-treated.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

20. I am able to remain calm even though those around me worry.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

21. When a friend starts to talk about his/her problems, I try to steer the conversation to something else.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

22. Another's laughter is not catching for me.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

23. Sometimes at the cinema I am amused by the amount of crying and sniffling around me.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

24. I am able to make decisions without being influenced by people's feelings.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

25. I cannot continue to feel ok if people around me are depressed.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

26. It is hard for me to see how some things upset people so much.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

27. I am very upset when I see an animal in pain.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

28. Becoming involved in books or films is silly.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

29. It upsets me to see helpless people.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

30. I become more irritated than sympathetic when I see someone's tears.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

31. I become very involved when I watch a film.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

32. I often find that I can remain cool in spite of the excitement around me.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree

33. Little children sometimes cry for no apparent reason.

-4	-3	-2	-1	0	1	2	3	4
Very Strongly Disagree				Neither Agree nor Disagree				Very Strongly Agree