The Effectiveness of EEG Biofeedback and Cognitive Rehabilitation as Treatments for Moderate to Severe Traumatic Brain Injury

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A research thesis submitted in partial fulfilment of the requirements for the degree, Doctor of Psychology (Clinical Neuropsychology), 2006

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Declaration

I, Joanne Stephens, declare that the Doctorate of Psychology (Clinical Neuropsychology) thesis entitled The Effectiveness of EEG Biofeedback and Cognitive Rehabilitation as Treatments for Moderate to Severe Traumatic Brain Injury is no more than 40,000 words in length, exclusive of tables, figures, appendices, references and footnotes. This thesis contains no material that has been submitted previously, in whole or in part, for the award of any other academic degree or diploma. Except where otherwise indicated, this thesis is my own work.

Signature: Date:

Joanne Stephens

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Abstract

Background: Cognitive Rehabilitation is an umbrella term which encompasses a number of restorative and compensatory techniques commonly and widely applied to assist with the sequelae following traumatic brain injury (TBI). Such techniques have been well established within the literature. More recently, an increasing body of research has emerged suggesting that electroencephalography (EEG) biofeedback is an effective intervention for sequelae following TBI. The purpose of the study was to investigate the effectiveness of cognitive rehabilitation and EEG biofeedback as treatments for moderate to severe TBI. It aimed to determine the effectiveness of each intervention in treating cognitive, emotional, and behavioural sequelae following TBI. *Methods:* A multiple single case study cross-over (ABBA) design was used with six adult participants, no less than one year post TBI. Three of the participants received the two treatments in the opposite order to the remaining participants, each serving as their own controls. Over ten weeks, each participant received 20 hours of Treatment *A.* Then, following a ten week break they received 20 hours of Treatment *B,* with a final ten week follow-up. A number of cognitive, emotional, and behavioural measures were administered pre-post treatments. Quantitative electroencephalographs (qEEG) were also administered pre-post treatments to evaluate any change in the electrophysiological dynamics of the brain. *Results*: EEG biofeedback appeared to be more effective than cognitive rehabilitation in improving information processing impairments, namely, complex attentional control, response inhibition, and speed of language and comprehension. Cognitive rehabilitation appeared to be more effective than EEG biofeedback in improving visual memory. Both treatments were effective in reducing depression, anxiety, anger, and neurobehavioural symptomatology. Although both treatments were effective in reducing depression, greater reductions were evident following EEG biofeedback. A number of self-reported functional changes were also noted by each participant. EEG biofeedback was more effective than cognitive rehabilitation in the normalisation of dysregulated EEG (as measured by qEEG). *Conclusions:* Overall, EEG biofeedback appeared to be more effective in improving information processing skills, while cognitive rehabilitation was more effective in improving visual memory. Both treatments were effective in the treatment of emotional and behavioural sequelae following TBI. EEG biofeedback was more effective in normalising the participants' EEG. However, the clinical meaningfulness of the qEEG finding is questioned. Speculations are made about the possible functional brain changes which may occur following rehabilitation.

Chapter 1.

INTRODUCTION

PART ONE

1.1 GENERAL INTRODUCTION

Traumatic brain injury (TBI) is a major cause of disability in adults and children, and a significant public health problem. Commonly resulting from motor vehicle accidents (57%), followed by falls (29%), assaults (9%), and drug abuse and gunshots (1%), the incidence of TBI across Australia ranges between 100 to 392 per 100,000 in New South Wales (Lyle, Quine, Bauman & Pierce, 1990; & Tate, McDonald, & Lulham, 1998) and 322 per 100,000 in South Australia (Hillier, Hiller, & Metzer, 1997). Despite the prevalence and impact of TBI on the public health system, the efficacy of rehabilitation approaches for the treatment of TBI sequelae has rarely been examined using sound methodologies. Ultimately, this compromises clinicians' knowledge of which rehabilitation approach best treats the cognitive, emotional, and behavioural sequelae following TBI.

The first chapter of this thesis will define TBI and present a brief overview of the neuropathophysiology, classification, and sequelae following moderate to severe traumatic brain injury (TBI). Detailed attention will be then given (in the second chapter) to rehabilitation approaches applied in TBI, specifically, cognitive rehabilitation and EEG biofeedback. The literature will be reviewed in light of the efficacy of each rehabilitation approach. The rationale for the present study's investigation will be provided, and the efficacy of these rehabilitation approaches will be examined.

1.1.1 Defining Traumatic Brain Injury

Traumatic Brain Injury (TBI) has been defined by the National Head Injury Foundation (NHIF) in the United States of America (USA) 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). More recently, Bruns and Hauser (2003) defined TBI as "…an alteration in brain function manifest as confusion, altered level of consciousness, seizure, coma, or focal sensory or motor neurological deficit resulting from blunt or penetrating force to the head" (p.2). As indicated, TBI can result from either a blunt or penetrating force, often referred to as Closed Head Injuries (CHI) or Open Head Injuries (OHI), respectively. The skull remains intact with no brain exposure following a CHI, however an OHI results from the penetration of the skull and dura by an external object (Lezak, Howieson, & Loring, 2004). Importantly, Lezak et al, (2004) highlight that the pattern of neurological deficit differs between CHI and OHI. The damage occurring in CHI is commonly diffuse, rather than focal. However, although diffuse injury can be observed following OHI, the focal effects are often more pronounced. Given the variable neurological presentation of OHI, the present research will focus on TBI in the context of CHI.

1.1.2 Neuropathophysiology of Traumatic Brain Injury

The mechanisms by which the brain can be injured following trauma are diverse and complex. However, understanding the basic mechanisms of injury, and the brain structures commonly damaged, complements the understanding of the cognitive, emotional/behavioural, and physiological sequelae following TBI. This then assists with the development of an appropriate rehabilitation program.

Brain injury is considered to be the result of 'primary' trauma (the immediate damage to the brain resulting from mechanical forces or pathophysiological mechanism) and/or 'secondary' trauma (developing as a consequence of metabolic disturbances or from the original neuronal damage), (Lezak et al, 2004). Gennarelli and Graham (2005) identify cerebral contusions and diffuse axonal injury (DAI), (more recently termed traumatic axonal injury - TAI), as mechanisms of primary trauma (refer to Figure 1). Intracranial haematoma, brain swelling/oedema, infection, raised intracranial pressure, hypotension, and ischaemic brain damage are identified as possible mechanisms of secondary trauma.

The two principal mechanisms of primary brain injury are contact (contusions) and DAI (Gennarelli & Graham, 2005). Firstly, brain trauma due to contact/contusions results from either an object directly striking the head or from contact occurring directly between the brain and the skull. Such direct contact of the brain can result in a rebounding motion, where contusions occur on opposite sides of the brain known as coup, and contre-coup injuries (Lezak et al, 2004). Secondly, diffuse axonal injury occurs when the brain is subject to severe rotational and acceleration/deceleration forces. This leads to the widespread damage of axons and blood vessels (Gennarelli & Graham, 2005), and results in microscopic damage, shearing, and stretching of axons throughout the brain, brainstem, and corpus callosum (Joseph, 1996).

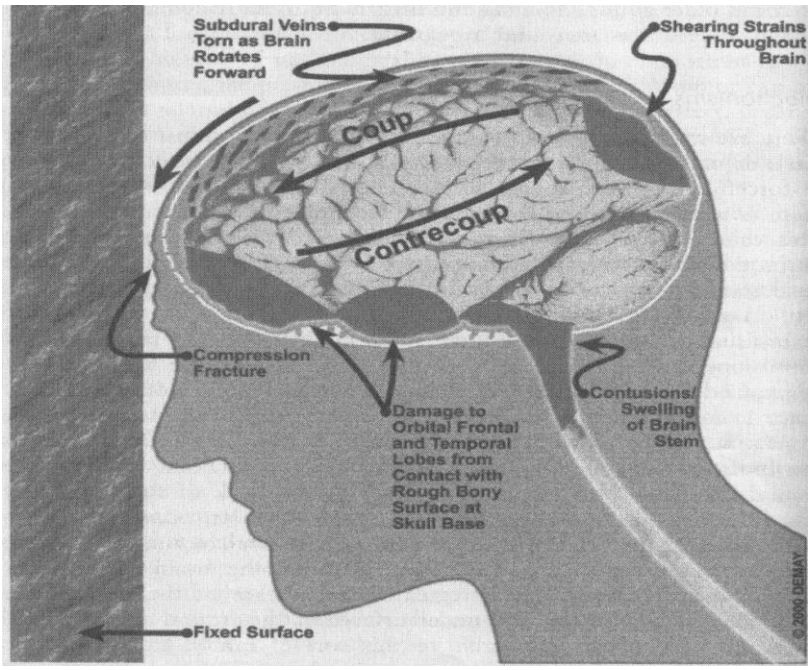

Figure 1. *Mechanisms of Brain Damage caused by closed brain injury (Sohlberg & Mateer, 2001, p.28).*

Structural brain damage has been identified in a number of cortical regions following both contusions and DIA. Brain injury often occurs in brain regions which are particularly vulnerable and susceptible to damage. Following cortical contusion, the orbital and lateral under-surfaces of the frontal and temporal lobes forcefully move over the bony surface on the base of the skull (e.g., the sphenoid and crista galli). Consequently, they are particularly vulnerable to injury, and are the most common sites of damage (Sohlberg & Mateer, 2001). More specifically, Gennarelli and Graham (2005) reported a characteristic distribution of damage following contusion affecting the poles of the frontal lobes; the inferior aspects of the frontal lobes, including the gyri recti; the cortex above and below the operculum of the Sylvian fissures; the temporal poles; and the lateral inferior aspects of the temporal lobes. Shearing lesions from DAI, although diffuse, are most commonly evident in the white and grey matter, particularly in the frontal and temporal regions, the corpus callosum (Bigler, 2005), and superior cerebellar peduncles (Sohlberg & Mateer, 2001).

Functional imaging studies using single photon emission computed tomography (SPECT), have demonstrated functional impairments in the frontal, temporal and parietal cortical regions (Goethals, Audenaert, Jacobs, Lannoo, Van De Wiele, et al, 2004). Even in the absence of structural lesions, functional imaging using positron emission tomography (PET) has identified impaired cerebral functioning (Fontaine, Azouvi, Remy, Bussel, & Samson, 1999). In particular, using functional magnetic resonance imaging (fMRI), impaired functioning has been frequently identified following TBI in the *anterior cingulate cortex* (Easdon, Levine, O'Conner, Tisserand, & Hevenor, 2004; & Soeda, Nakashima, Okumura, Kuwata, Shinoda, et al, 2005) and *prefrontal cortex* (Scheibel, Pearson, Faria, Kotrla, Aylward, et al, 2003; & Easdon et al 2004).

1.1.3 Definition and Classification of Moderate and Severe Traumatic Brain Injury

Studies have frequently identified different outcomes following moderate TBI as compared to severe TBI (Rimel, Giordani, Barth, & Jane, 1982; & Hellawell, Taylor, & Pentland, 1999). As demonstrated by Hellawell et al (1999), at six months post injury, 49% of their moderate TBI sample had made a good recovery, increasing to 53% by 24 months. Comparatively, only 23% of the severe TBI sample had made a good recovery at six months, and 23% at 24 months. Therefore, it is important to accurately classify injury severity following TBI to assist in the prognosis, and subsequently, the rehabilitation process.

As highlighted by Lezak et al (2004), there is a lack of consistent definitions of moderate TBI within the literature, and depending on which definition is used, there is also the presence of considerable overlap with 'mild-to-moderate' (Levin et al, 2001;

Wallesch, Curio, Kutz, Jost, Bartels, et al, 2001; & Brown, McCauley, Levin, Contant, & Boake, 2004) and also 'moderate-to-severe' classifications (Lannoo, Colardyns, Jannes, & De Soete, 2001; Colantonio, Ratcliff, Chase, Kelsey, Escobar, et al, 2004; & Hanlon, Demery, Kuczen, & Kelly, 2005).

The duration of loss of consciousness (Symonds, 1928) and Post Traumatic Amnesia (Russell, 1932) were characteristic features of brain injury originally proposed as measures of injury severity. The Glasgow Coma Scale (GCS) was devised by Teasdale and Jennett (1974) based on their definition of coma as the absence of eye opening, a failure to obey commands, and a failure to give any comprehensive verbal response. Table 1, displays the GCS classification of brain injury severity.

Table 1: *Severity Classification Criteria for the Glasgow Coma Scale (GCS)*

Classification	***GCS***		***Coma Duration***
Mild	≥ 13	or	≥ 20 minutes
Moderate	9-12	or	No longer than within 6 hours of admission
Severe	≤ 8	or	> 6 hours after admission

Measuring the duration of Post Traumatic Amnesia (PTA) is an additional method of classifying brain injury severity. This concept is characterised by the inability to lay down new memories reliably from one day to the next (Shores, Marosszeky, Sandanam & Batchelor, 1986). Russell and Smith (1961) originally proposed a classification of TBI according to the duration of PTA, which was expanded by Jennett and Teasdale (1981) as can be observed in Table 2.

Table 2: *Estimates of Severity of Brain Injury Based on Posttraumatic Amnesia (PTA) Duration.*

PTA Duration	*Severity*
<5 minutes	Very mild
5-60 minutes	Mild
1-24 hours	Moderate
1-7 days	Severe
1-4 weeks	Very Severe
More than 4 weeks	Extremely Severe

These measures, using commonly accepted criteria for the classification of TBI severity, define moderate TBI as: GCS of 9 to 12 (coma duration no longer than within 6 hours of admission), and PTA of one to 24 hours (refer to Table 1 & Table 2). This classification defines severe TBI as a GCS of 3 to 8, six hours following hospital admission (refer to Table 1), with a PTA subdivided into three categories; 1) 1-7 hours of PTA which equates to a 'Severe' TBI; 2) 1-4 weeks of PTA which is a 'Very Severe' TBI; and 3) more than 4 weeks which is an 'Extremely Severe' TBI (refer to Table 2).

1.1.3.1 Sequelae following Moderate and Severe Traumatic Brain Injury

1.1.3.1.1 Cognitive Sequelae

As previously highlighted, it is difficult to find research which accurately reflects the sequelae of moderate TBI, given much overlap exists between other classifications. Despite this, global cognitive impairments have been reported following both moderate (Rimel et al, 1982) and severe (Lezak et al, 2004) TBI. However, although global cognitive impairments follow both moderate and severe brain injuries, TBI victims who sustain a severe brain injury, tend to demonstrate poorer cognitive performances than that of the moderate TBI category.

Global deficits following moderate and severe TBI have been well demonstrated in

the literature with impairments reported in various aspects of *attentional functioning* (Bate, Mathias, & Crawford, 2001; Kersel, Marsh, Havill, & Sleigh, 2001a; & Rios, Perianez, & Munoz-Cesepdes, 2004), *speed of information processing* (Colantonio et al, 2004) *working memory,* (Hanten, Stallings-Roberson, Song, Bradshaw, & Levin, 2003), *learning and/or memory* (Shum, Harris, & O'Gorman, 2000; & Vanderploeg, Crowell, & Curtiss, 2001), *prospective memory* (Roche, Fleming, & Shum, 2002), *language,* (Hellawell et al, 1999), and *executive functioning* (Spikman, Deelman, & Van Zomeren, 2000; & Greve, Love, Sherwin, Mathias, Ramzinski, et al, 2002).

Importantly, cognitive impairments may have a pervasive and persistent impact following TBI. Research has demonstrated that information processing deficits (such as attention, speed of information processing, and working memory) appear to be more often severely impaired in TBI individuals than other cognitive functions (Hoofien, Gilboa, Vakil, & Donovick, 2001). Furthermore, processing deficits, in particular slow processing speed, have been associated with poorer performances on neuropsychological measures of other cognitive domains (Madigan, DeLuca, Diamond, Tramontano, & Averill, 2000). This suggests that such processing difficulties may have a pervasive impact on a number of cognitive functions, compromising an individual's global cognitive performance. Additionally, following the acute stages of moderate and severe TBI, cognitive impairments in a number of domains may continue to persist over a long period of time, if not for life. Marked verbal learning deficits have been identified at an average of 14.1 (Hoofien et al, 2001) and 14.2 years post injury (Colantonio et al, 2004). Likewise, research findings have also demonstrated the persistence of marked language deficits (Hellawell et al, 1999), impaired verbal abstract reasoning and verbal fluency, (Kersel et al, 2001a) for greater than 12 months following TBI. These findings clearly demonstrated the

persistence of a number of cognitive deficits into the long-term.

1.1.3.1.2 Emotional and Behavioural Sequelae

Individuals who sustain a moderate or severe TBI may experience a number of emotional and/or behavioural problems. Interestingly, minimal difference has been reported between moderate and severe TBI in the prevalence of mood disorders (Hellawell et al, 1999). Research findings have reported difficulties following TBI with *depression* (Kersel Marsh, Havill, & Sleigh, 2001b; & Milders, Fuchs, & Crawford, 2003), *anxiety*, (Kersel et al, 2001b; & Sohlberg & Mateer, 2001), *post-traumatic stress disorder* (Bryant, Marosszeky, Crooks, Baguley & Gurka, 2000; & Williams, Evans, Wilson, & Needham, 2002), and *obsessional behaviours* (Childers, Holland, Ryan, & Rupright, 1998). Additionally, behaviours such as hostility (Hoofien et al, 2001), impatience, irritability, argumentativeness, anger, difficulty becoming interested, lack of initiative, irresponsibility, aggression, and lack of control over social behaviour, have been reported following TBI (Kersel et al, 2001b). Unusual and/or inappropriate behaviours, apathy, withdrawal, and decreased communicative ability have also been noted (Milders et al, 2003).

Emotional difficulties may become evident within the early stages of recovery, however they may also emerge several years post injury. Importantly, just like the cognitive sequelae, emotional/behavioural sequelae can be persistent and have pervasive effects on a person's functional outcomes affecting employment, independent living, relationships, social, and leisure activities (Hoofien et al, 2001; & Kersel et al, 2001b). A number of these studies have reported ongoing emotional and behavioural difficulties, whereby symptoms continued to persist between 3 and 14 years following TBI (Douglas & Spellacy, 2000; Hoofien et al, 2001; & Milders et al,

2003). These studies support the view that emotional and behavioural changes following TBI can last for a very long time and in many cases may be permanent

Importantly, as highlighted by Sohlberg and Mateer (2001), premorbid personality traits or disorders may interact with the brain injury, affecting the emotional and behavioural outcome significantly.

Chapter 2.

INTRODUCTION

PART TWO

2.1 REHABILITATION OF TRAUMATIC BRAIN INJURY

As identified in the previous chapter, the cognitive, emotional, and behavioural sequelae of moderate and severe TBI may persist indefinitely over time, and can have pervasive effects on a person's functional outcomes affecting employment, independent living, relationships, social, and leisure activities. Therefore, it is important to implement rehabilitation to reduce the impact of the previously described sequelae on an individual's everyday functioning in life.

2.1.1 Rehabilitation Approaches for Traumatic Brain Injury

Rehabilitation has been defined in two different ways by McLellan (1997):

*"**1**. A process of active change by which a person who has become disabled acquires the knowledge and skills needed for optimal physical, psychological, and social function. **2**. The application of all measures aimed at reducing the impact of disabling and handicapping conditions and enabling disabled and handicapped people to achieve social integration."(p.1)*

The definition provided by McLellan encapsulates the purpose of rehabilitation, whereby an injured individual can work towards achieving their goal lifestyle. Similarly, Wilson (2002) indicated that the main purpose of rehabilitation is to achieve the maximum physical, psychological, social, and vocational wellbeing, and to enable people to return to their own pre-injury environment. Importantly, rehabilitation should implement realistic aims to reduce the impact of disabling and

handicapping conditions (Wilson, 1997).

A number of approaches are used in the rehabilitation of TBI to reduce the impact of cognitive, emotional, and behavioural sequelae. Some common approaches include: psychotherapy (in particular cognitive behavioural rehabilitation), behaviour modification, and cognitive rehabilitation. Another relatively new approach to the rehabilitation of TBI is electroencephalogram (EEG) biofeedback. In practice, it is difficult to separate each approach described as they are often implemented collaboratively, and tailored to the specific needs of the individual. Wilson, Evans, and Keobane (2002) described a holistic approach to cognitive rehabilitation using a collection of approaches (psychotherapy, behavioural, and cognitive techniques). Cognitive rehabilitation was suggested by Wilson and colleagues to consist of four main approaches: 1) cognitive retraining; 2) strategies derived from cognitive neuropsychology; 3) a combination of different methods and techniques (particularly from neuropsychology, cognitive psychology, and behavioural psychology); and 4) holist approaches that address the cognitive, social, and emotional sequelae of brain injury. In the present research, a holistic approach to cognitive rehabilitation has been taken. However, for the purpose of the literature review, each approach will be discussed and reviewed as an individual therapeutic method.

2.1.1.1 Psychotherapy

Psychotherapy is a broad concept encompassing a variety of counselling techniques and therapies (Miller, 1993) such as psychoanalytic therapy, gestalt therapy, reality therapy, narrative therapy, and cognitive behavioural therapy (Corey, 2001). It is defined by Prigatano (1999) as a method of teaching patients to learn to behave in their own best self-interest. He indicates that the overall goal of psychotherapy is to

establish or re-establish a sense of purpose or meaning in a patient's life. Following TBI, injured persons, like non-injured individuals, are likely to struggle with unresolved interpersonal conflicts; may operate on irrational assumptions about themselves and their world; may demonstrate anxiety, depression, phobias, or obsessions; and they may feel alienated (Pollack, 2005). Brain injured persons may also struggle with a profound state of personal loss and experience failures, but may not understand why (Prigatano, 1999). Such struggles may respond to psychotherapeutic interventions.

A specific form of psychotherapy is cognitive behavioural therapy (CBT). Cognitive behavioural therapy is fundamentally concerned with information processing, that is: peoples' perceptions and interpretations of their experiences alter and shape their behaviour (Alderman, 2003). Such therapies assist the patient to identify distortions in their thinking, and help them generate more rational interpretations of the events (Alderman, 2003). A number of studies have evaluated the effectiveness of CBT in treating the emotional and behavioural sequelae of TBI. Recent research (Bedard, Felteau, Mazmanian, Fedyk, Klein, et al, 2003) delivered 12 weekly group sessions providing insight mediation, breathing exercises, guided visualisation, and group discussion. They aimed to encourage a new way of perceiving life and its difficulties, in order to bring a sense of acceptance and allow patients to move beyond limiting beliefs. Following therapy, patients reported significant improvements in their quality of life, as compared to drop-out controls.

Anecdotal evidence and case studies demonstrate that CBT reduces anxiety symptomatology in TBI patients who have obsessive compulsive disorder (Williams, Evans, & Fleminger, 2003) and post traumatic stress disorder (Williams, Evans, &

Wilson, 2003). Khan-Bourne and Brown (2003) present CBT as a potentially suitable treatment for depression following TBI. However, little research has evaluated the effectiveness of CBT as a treatment for depression following TBI. A study investigating the effectiveness of a 16 week group programme combining components of CBT, cognitive rehabilitation, and social skills training, demonstrated significant improvements in the self-awareness and psychosocial functioning of 21 chronic TBI participants (Ownsworth, Farland & Young, 2000).

Aggressive behaviour has also been reported to be effectively managed through CBT techniques. Medd and Tate (2000) found a significant reduction in the outward expression of anger following the implementation of CBT in 8 TBI subjects, compared to 8 waiting-list controls. Improvements in the management of anger were maintained at a two month follow-up assessment. Interestingly, research findings suggest that CBT not only assists in changing maladaptive thought processes, but also may change impaired cerebral functioning. Although not using TBI populations, very recent studies have reported functional brain changes (in particular, the anterior cingulate cortex) following CBT (Straube, Glauer, Dilger, Mentzel, & Miltner, 2006).

Alderman (2004) indicates that self awareness, attention, monitoring skills, memory, the ability to think abstractly, and the ability to generate alternatives, are all crucial abilities to possess in the CBT process. Therefore, CBT may not be suitable for all TBI patients. This limitation has been reported to compromise the effective use of all psychotherapeutic techniques for the TBI population. Similarly, a list of requisite abilities a person possesses in order to gain benefit from psychotherapy include: the capacity for abstract thinking; a degree of self-awareness and the ability to self-monitor; the ability to tolerate frustration and anxiety; memory that is intact enough to

recall significant information both within and across therapy sessions; and the ability to transfer what is learned in the treatment environment to other life situations (Pollack, 2005). Alderman (2004) highlights that given these requisite abilities, psychotherapeutic methods are not suitable for all people with TBI. Psychotherapy has limited effectiveness in individuals following severe TBI with poor awareness, who lack insight, are inaccurate in their self-report, and who have motivational problems. Consequently, in cases where emotional and behavioural difficulties are not amiable to psychotherapy, behaviour modification may be a useful rehabilitation approach.

2.1.1.2 Behavioural Modification

Many behavioural problems observed following TBI do not ameliorate easily and may require strategic behavioural interventions (Corrigan & Bach, 2005). Behavioural therapy is best defined by Erwin (1978) as a "nonbiological form of therapy that developed largely out of learning theory research and that is normally applied directly, incrementally, and experimentally in the treatment of specific maladaptive behaviour patterns" (p.44). Ponsford (1999) described behaviour modification as a procedure that is designed to either increase the probability of desirable behaviours, or decrease the probability of undesirable behaviours. This is accomplished by altering the response, consequence, or antecedent of the behaviour (Alderman, 2003). A number of behavioural techniques may be applied including, positive or negative reinforcement, differential reinforcement (Hegel & Ferguson, 2000; Watson, Rutterford, Shortland, Williamson, & Alderman, 2001; & Knight, Rutterford, Alderman, & Swan, 2002), non-contingent reinforcement (Persel, Persel, Ashley, & Krych, 1997), token economy and response cost (Bellus, Kost, Vergo, & Dinezza, 1998), feedback (Schlund & Pace, 1999), covert sensitization, time out,

overcorrection (restitution and positive practice), and desensitization (Thorpe & Olson, 1997).

Research has yielded evidence for the efficacy of behavioural modification in the rehabilitation of maladaptive behaviour following TBI. A meta-analysis conducted by Ager and O'May (2001) reported that empirical evidence supports the effectiveness of behavioural interventions at changing behaviour, in particular: in addressing socially disruptive and internally maladaptive behaviour. However, a significant proportion of the experimental literature is individual case studies, compromising the ability to generalise the findings to the general TBI population.

Although behavioural modifications are often employed when patients have been excluded from psychotherapy due to lack of awareness, poor motivation, or severe cognitive impairment (Alderman, 2004), behavioural modifications bear limitations similar to that of psychotherapeutic techniques. It can be difficult to identify reinforcers, or even negative consequences, which have any impact on behaviour in individuals who are lacking drive and motivation (Ponsford, 1999). Ponsford (1999) highlights a number of further limitations on the effective implementation of behaviour techniques. Firstly, it is reported that behaviour problems which are an entrenched part of a person's behavioural repertoire prior to the brain injury are likely to be resistant to change. Secondly, the efficacy of the treatment is reliant on the consistency of application, support, and co-operation of particular staff and family members involved with TBI individual. If any of these are compromised the success of the treatment is limited. Furthermore, the ability to generalise the effects of behavioural treatments outside the setting in which they have been implemented has been questioned (Mottram, 2004).

2.1.1.3 Cognitive Rehabilitation

Cognitive Rehabilitation is a widely used umbrella term encompassing a variety of intervention strategies such as: Cognitive Retraining, Cognitive Remediation, and Compensatory Strategy Training (Ricker, 1998). Carney, Chesnut, Maynard, Mann, Patterson and Helfand, (1999) indicate that cognitive rehabilitation is often divided into two major approaches, namely Restorative and Compensatory.

The restorative approach focuses on intervening at the level of areas of impaired cognitive functioning, thereby aiming to improve specific cognitive functions. It attempts to directly retrain the damaged cognitive function (Park & Ingles, 2001). An example of a restorative approach is attention processing training (APT). This involves patients engaging in a series of hierarchically organised and repetitive drills that exercise and place increasing demands on complex attentional control and working memory systems (Sohlberg & Mateer, 2001). Sohlberg and Mateer (2001) describe a number of restorative methods for improving memory function including: memory practice drills; meta-memory training; and prospective memory training. They also describe mnemonic strategy training which commonly involves the use of visual imagery, verbal organisation techniques (e.g. making paired associations with target words), or semantic elaboration (linking target words or ideas in a story). The underlying rationale for the restorative approach is that practice on carefully selected exercises promotes recovery of the damaged neural circuits, which in turn regulates the cognitive function of interest (Park & Ingles, 2001). In support of this, quantitative electroencephalogram (qEEG) research has demonstrated that following cognitive retraining, changes can be identified in the electrophysiological dynamics of a TBI subject's brain (Stathopoulou & Lubar, 2004).

The compensatory approach attempts to teach strategies and skills to reduce the impact of the cognitive impairments as they manifest in everyday settings (Sloan & Ponsford, 1999). It focuses on functional goals and interventions which are designed to teach the individual how to reach goals using residual abilities and relative strengths, hence adapting to the cognitive deficit. Compensatory techniques include internal, external, and environmental adaptation methods (Evans, Wilson, Needham, & Brentnall, 2003). Internal compensatory strategies may include such techniques as verbalisations (orally repeating information to oneself), chunking (mental grouping of information in segments), and pacing (performing a task with an intermittent pause), (Fasotti, Kovacs, Eling, & Brouwer, 2000). External compensatory strategies may include: written planning systems (e.g. planners, notebooks, & calendars); electronic planners and computerised systems (e.g. mobile phones, alarms, paging systems, and watches); and auditory or visual systems (electronic voice organisers, voice recorders, and pictorial systems), (Sohlberg & Mateer, 2001). Environmental adaptation may include techniques of labelling objects within the environment, such as the cupboards (Evans et al, 2003).

Although the restorative approach, as opposed to the compensatory approach, has been suggested to promote recovery of the damaged neural circuits (Park & Ingles, 2001), research has also demonstrated changes in SPECT (Laatsch, Pavel, Jobe, Lin, & Quintana, 1999) and functional MRI (Laatsch, Little, & Thulborn, 2004; & Laatsch, Thulborn, Krisky, Shobat, & Sweeney, 2004) following the implementation of compensatory strategies in TBI participants. While the range of treatment sessions and period of time to implement them varied, for each of these studies compensatory strategies were administered over 6 to 36 sessions, 27 sessions over eight months, and over a four to 13 month period, respectively. Irrespective of the relative frequency

and duration of these interventions, the findings provide some evidence for the neurobiological mechanisms of recovery following the use of compensatory strategies in TBI.

Some have questioned whether rehabilitation should aim to restore a damaged cognitive function, or to develop compensatory or alternative ways of performing a task (Park & Ingles, 2001). However, studies have demonstrated significant improvements in cognition of a comparable degree following the application of both cognitive rehabilitation approaches (Dirette, Hinojosa, & Carnevale, 1999). Furthermore, the distinction between teaching compensatory strategies and restoring a cognitive process is somewhat blurred, since both methods require learning and depend upon repetitive activation of associated cognitive processes (Sohlberg and Mateer, 2001). Importantly, as highlighted by Sohlberg and Mateer (2001) the selection of the different approaches is highly individualised and most often techniques from each method are collectively implemented to achieve the best outcome.

2.1.1.3.1 The Effectiveness of Cognitive Rehabilitation in TBI

Comprehensive and well controlled studies have investigated the effectiveness of cognitive rehabilitation, evaluating its efficacy in the treatment of a broad range of sequelae including: cognitive, emotional/behavioural, and functional abilities. A meta-analysis conducted by Carney et al (1999) reviewed 32 studies on the efficacy of cognitive rehabilitation. They concluded that there is some evidence that certain cognitive rehabilitation methods improve cognitive performance, reduce anxiety, and improve self-concept and interpersonal relationships for persons with TBI. Although Carney and colleagues reported improvements from pre- to post-treatment, they

indicated that the well designed randomised controlled trials evaluated produced no significant treatment effects in the statistical analysis. Consequently, this questions the efficacy of cognitive rehabilitation.

Additionally, recent randomised controlled studies have failed to find support for the effectiveness of cognitive rehabilitation (Salazar, Warden, Schwab, Spector, Braverman, et al, 2000; & Warden, Salazar, Martin, Schwab, Coyle, et al, 2000). Salazar, Warden and colleagues reported no statistically significant difference between an inpatient cognitive rehabilitation (modelled after Prigatano's milieu-oriented approach) and a home rehabilitation program (consisting mainly of a weekly telephone call from a psychiatric nurse) on cognitive and functional measures in moderate to severe TBI patients. However, it is worth noting that the authors describe that the home rehabilitation patients were given strategies for enhancing cognitive and organisational skills (Salazar et al, 2000), and that during the phone calls the nurse and patients developed problem-solving techniques (Warden et al, 2000). Therefore, it would appear that the home rehabilitation patients were in fact receiving some form of cognitive rehabilitation, making the conclusions somewhat questionable.

Compensatory and remediation approaches have been used in isolation and collectively to assist in the rehabilitation of a number of cognitive functions. A number of other studies evaluating the effectiveness of specific cognitive rehabilitation methods within specific cognitive domains including information processing, memory, and executive function, will be reviewed.

2.1.1.3.1.1 Information Processing (Attention, visual processing, and speed of processing)

Park & Ingles, (2001) published a meta-analysis evaluating 30 studies on the effectiveness of cognitive rehabilitation approaches in retraining attention following TBI. They compared studies seeking to improve cognitive function versus those attempting to teach specific functional skills. Of the 30 studies included in the analysis (mean of 31.2 hours treatment), it was concluded that specific-skills training significantly improved performance on the tasks requiring attention, but the cognitive retraining methods did not significantly affect outcome. Similarly, research conducted by Cicerone (2002) has provided evidence for the strategy training model. Cicerone implemented a prospective case comparison design, comparing four mild TBI treatment participants with a matched untreated sample. The treatment facilitated the participants' conscious use of strategies (verbal mediation, rehearsal, anticipation of task demands, self-pacing, and n-back task) to effectively allocate attentional resources and manage the rate of information during the task performance. Following strategy training, the treatment group demonstrated clinically meaningful change on 58.3% of the measures, whereas the non-treatment comparison group showed clinically meaningful change in only 12.5% of the measures. Cicerone concluded that the benefits of treatment were due to the subjects' improved ability to compensate for, and adopt strategies for the more effective allocation of their residual attentional resources.

Recent small-scale and single case studies have demonstrated the effectiveness of cognitive retraining for attentional difficulties in the TBI population. Palmese and Raskin (2000) investigated the effectiveness of cognitive retraining using a 10 week Attention Process Training-II programme (APT-II), on attention deficits in three mild TBI subjects. Pre-tests and post-tests were administered, and individually tailored cognitive retraining programs were implemented. Results suggested improvements in

the attentional functioning of two subjects and improvements in performance speed in all three participants. Similarly, Sohlberg, McLaughlin, Pavese, Heidrich, and Posner (2000) evaluated the effectiveness of the APT program on attentional networks in 14 TBI patients one year post injury. Subjects served as their own controls in a crossover design, and were randomly assigned into two groups of seven. The study demonstrated improvements over a wide range of tasks that involved executive functions and attentional control, namely the Paced Auditory Serial Test (PASAT), Stroop, Trail Making Test, and memory for locations. Importantly, through structured interviewing they ascertained that the participants were able to generalise their improvements in attentional skills to day-to-day life.

With advancing technology, computerised methods of cognitive retraining have been explored. Nevertheless, there has been much controversy and scepticism with this method of implementing cognitive retraining. Early research within the field has demonstrated no significant differences between TBI subjects receiving computer-assisted and non-computer assisted cognitive retraining methods (Batchelor, Shores, Marosszeky, Sandanam, & Lovarini, 1988). Despite these findings, a recent historical review of the literature conducted by Lynch (2002) concluded that computer-assisted cognitive retraining can be an effective adjunct to a comprehensive program of cognitive rehabilitation. A well-controlled study conducted by Niemann, Ruff, and Baser (1990) investigated the effectiveness of a computer-assisted attention retraining program in 29 TBI subjects randomly assigned into either the treatment or matched control group. Their findings indicated that the computer-assisted attention retraining was effective in significantly improving performance on measures of attention, compared to the control group who received compensatory memory strategies. Unfortunately, subjects failed to generalise their cognitive improvements to real-life

situations.

Similarly, a more recent multiple single case study conducted by Stathopoulou and Lubar (2004) evaluated the effectiveness of a cognitive retraining program (Captain's Log) in five TBI participants, using psychometric assessment and qEEG. Findings indicated that following 22 sessions of computerised cognitive remediation, subjects showed improvements in sustained, alternating, and selective attention. Furthermore, EEG changes were observed on qEEG, namely, a decrease in alpha. It was concluded that the reduction in alpha was consistent with an improvement in attentional functioning. It follows that a link between cognitive retraining and EEG changes can occur.

The effectiveness of cognitive rehabilitation in the management of information and speed of processing deficits following TBI has been evaluated in well controlled studies. Dirette and Hinojosa (1999) implemented three compensatory strategies (verbalisation, chunking, and pacing) in 15 TBI subjects to assist with visual processing, compared to a matched control group who received remedial computer intervention. Findings revealed that both the experimental and control group significantly improved to a comparable degree following intervention. However, it was noted that 80% of the subjects used compensatory strategies with or without instruction. Furthermore, it was identified that subjects who used internal compensatory strategies performed better on performance speed. Fasotti et al (2000) used a Time Pressure Management (TPM) compensatory approach (teaching both prevention and management strategies) to rehabilitate processing speed in 12 TBI participants, compared with a matched control group who received concentration training utilising generic verbal suggestions. The results indicated that the TPM

group, while they used the management strategies more frequently than controls, did not improve in the application of prevention strategies. Importantly, both approaches were effective in improving processing speed, and this was maintained at a six month follow-up.

2.1.1.3.1.2 Memory

A large proportion of the literature evaluating cognitive rehabilitation techniques has been conducted in the memory domain. Compensatory strategies appear to be more commonly evaluated than the restorative/retraining approach in the rehabilitation of memory functioning. Such strategies also have been demonstrated to be the preferred choice by TBI individuals and their carers (Evans et al, 2003). Evans and colleagues interviewed 101 TBI subjects and their care givers to identify the most commonly used aids/strategies to compensate for memory impairment. They found that external aids such as calendars, wall charts, and notebooks were the most commonly used memory aids, but electronic organisers were not used by many participants. Similarly, Gillette and DePompei (2004) surveyed 53 cognitively impaired individuals on their use of technological devises. It was established that there was limited use of computers and/or electronic organisers (11% of the sample) for the compensation of memory.

Despite findings reporting the limited usage of technological devises in the TBI population, research has provided support for the efficacy of various technological methods in the compensation of prospective memory difficulties. Such technological memory aids include cognitive prosthetics, tele-rehab, NeuroPage, Smart house, environmental control systems, information technology, mobile phones, navigational hardware, and palmtops (Gartland, 2004). In a case study (Kim, Burke, Dowds, &

George, 1999) the effectiveness of a microcomputer (palmtop computer) as a memory aid for a TBI patient was evaluated. With the implementation of the microcomputer in an in-patient rehabilitation setting, the patient demonstrated an improvement in therapy attendance and initiated the request for his medication on schedule. Similarly, Kim, Burke, Dowds, Robinson Boone, and Park (2000) also demonstrated some level of effectiveness for the palmtop in an outpatient TBI population. Their findings indicated that nine (75%) of the 12 participants who had been trained in the use of the palmtop microcomputer, found the strategy useful during supervised trials. Seven (58.3%) of the 12 subjects continued to use the device on a daily basis for several years following the withdrawal of the supervised trial.

Other technological devices such as paging systems have been reported as useful external memory aids, particularly as reminders (Kapur, Glisky, & Wilson, 2004). Research has identified that of 40 clients trained in the use of a paging system (NeuroPage), 31 (77.5%) clients reported the pager to be successful in the management of memory (Wilson, Scott, Evans, & Emslie, 2003). Wilson, Emslie, Quirk, and Evans (2001) evaluated the effectiveness of a NeuroPage in a randomised control crossover design study with 143 brain injured patients. Their results indicated that 80% of those who completed the 16 week trial were significantly more successful in carrying out everyday activities including: self-care; taking medication; and keeping appointments. These improvements were reported to be maintained in a majority of patients for seven weeks after returning the pager. It was concluded that the paging system significantly reduced everyday failures of memory and planning in people with brain injury. Similarly, a recent case study (Kirsch, Shenton, & Rowan, 2004) demonstrated the effectiveness of an alphanumeric paging system in a TBI patient. Kirsch and colleagues found that the paging system increased the reliability

of the patient's memory. It was reported that the paging system provided structure, time-dependent and activity relevant cueing, which facilitated the patient's task performance at specific times during the day. Despite the improvement identified, the patient's memory performance returned to baseline when the paging system was withdrawn. This questions the ability to generalise this technique to every-day life.

Non-technological compensatory strategies, particularly external memory aids, have been reported to be effective in the rehabilitation of prospective memory. A literature review conducted by Kapur et al (2004) evaluated the effectiveness of memory aids for memory impairments. They reported that external memory aids were effective in improving everyday memory functioning, particularly evident in the rehabilitation of prospective memory. Schmitter-Edgecombe, Fahy, and Long (1995) investigated that effectiveness of notebook training in four TBI subjects, compared to a matched control group receiving supportive counselling. Following treatment, the notebook training group reported significantly fewer everyday memory failures on a daily checklist measure than the control group. However, at a six month follow-up assessment there was no significant difference between the two groups, suggesting that the notebook group did not maintain the strategies taught during intervention once it was withdrawn.

External memory aids in the form of diaries have been appraised to assist with prospective memory difficulties in TBI. A recent multiple single case study demonstrated successful diary use following the implementation of prospective memory rehabilitation in three TBI participants (Fleming, Shum, Strong, & Lightbody, 2005). Fleming and colleagues improved prospective memory functions following eight weeks of intervention which was designed to enhance prospective

memory function by: 1) identifying potential barriers; 2) establishing self-awareness of memory deficits; 3) customising a compensatory tool (diary); 4) and implementing a cueing system and organisational strategies. This approach to diary training supports an older study conducted by Ownsworth and McFarland (1999). They investigated two methods of diary training, the Diary Only approach (utilising task specific methods) and the Self-Instructional Training approach (which taught compensation using the higher cognitive skills of self-awareness and self-regulation). In a sample of 20 TBI subjects randomly assigned to the two groups, compared with 31 non-TBI controls, Ownsworth and McFarland demonstrated that the Self-Instructional group more consistently made diary entries, reported less memory problems, and made more positive ratings associated with treatment efficacy.

Internal compensatory strategies for memory impairment following TBI have also been investigated. Research has evaluated the effectiveness of mnemonic compensatory strategies in the rehabilitation of memory for people's names and faces (Milders, Deelman, & Berg, 1998; & Hux, Manasse, Wright & Snell, 2000). Milders et al (1998) taught internal compensatory strategies to improve learning of new names and retrieval of familiar people's names to 13 TBI participants, compared to 13 healthy controls (not taught strategies). Brain injury subjects were directed to increase the meaningfulness of people's names, to improve learning of new names and to assist with the retrieval of known names. Participants' performance on learning and memory retrieval tasks substantially improved immediately following the strategy training. At six months, the improvement in learning new names was maintained; however, improvement was not maintained in the retrieval of familiar people's names. More recently, Hux, et al (2000) implemented a multiple baseline study which demonstrated that mnemonics and visual imagery strategies were

effective in increasing four out of the seven TBI participants' ability to recall face-name associations. No follow-up assessment was conducted.

2.1.1.3.1.3 Executive Functions

Various cognitive rehabilitation approaches have been implemented in the treatment of a number of executive functions including: planning and organisation, problem-solving, and abstract thinking. Often, strategies used in the rehabilitation of memory are concurrently used in the rehabilitation of planning and organisational problems following TBI. In particular, both technological/electronic and non-technological external memory aids have demonstrated effectiveness in the treatment of planning and organisational difficulties. In studies previously described, external strategies such as palmtop (Kim et al, 1999; & Kim et al, 2000), paging systems (Wilson et al, 2001; Wilson et al, 2003; & Kirsch et al, 2004), and diaries (Ownsworth & McFarland, 1999; & Fleming et al, 2005), have demonstrated effectiveness in managing executive difficulties in addition to memory problems following TBI.

Research has illustrated the effectiveness of meta-cognitive strategies in the rehabilitation of problem-solving following TBI (Marshall, Karow, Morelli, Iden, Dixon, et al, 2004). Marshall and colleagues taught 20 TBI participants to use meta-cognitive strategies (semantic category labelling) to solve verbal problems. Their findings suggested that TBI participants improved in problems-solving post rehabilitation and three meta-cognitive strategies (1. better planning, 2. reduced impulsivity, and 3. strategy shifting) appeared to account for much of the improvement. The improvements were maintained at one month follow-up.

Similarly, a randomised controlled study applied a comprehensive rehabilitation

program in 46 TBI subjects to compensate for problem-solving deficits (Rath, Simon, Langenbahn, Sherr, & Diller, 2003). Twenty-seven of the subjects participated in 24 sessions of the innovative group treatment programme. This provided compensatory strategies to address and rehabilitate the underlying processes of problem-solving, that is, emotional self-regulation and logical thinking/reasoning deficits. Innovative subjects were required to report and analyse precursors to everyday problematic situations, evaluate the situation, and then develop and revise a plan of action via a structured reframing process. The comparison group of 19 participants completed 24 sessions of conventional group rehabilitation, consisting of a broad range of cognitive remediation techniques and a psychosocial component. Rath and colleagues demonstrated that the innovative group showed significant improvements on tests of executive functioning, in problem-solving, problem-solving self-appraisal, self-appraised clear thinking, and emotional self-regulation. In contrast, the conventional group improved on only one reasoning measure. The improvements made in the innovative group were maintained at six month follow-up.

Cognitive remediation of abstract thinking and flexibility of thinking has also been evaluated (Kaplan, 2001). Kaplan applied an abstraction task to remediate problems of abstract thinking and flexibility of thinking in a TBI single case study. The subject was taught strategies to decipher the similarities and dissimilarities within multiple abstract concepts. Following rehabilitation, the subject's performance on neuropsychological measures showed significant improvements in: verbal abstraction skills (91st vs. 75th percentiles); cause and effect reasoning (63rd vs. 16th percentiles); visual reasoning; and in attention to detail. No follow-up assessment was conducted.

2.1.1.3.2 Limitations of Cognitive Rehabilitation

Similar to that of behavioural approaches, the ability to maintain and generalise the skills targeted in treatment to every day function, is a major and continuing concern (Sohlberg & Mateer, 2001). Ben-Yishay & Diller (1993) indicate that difficulty with concrete thinking following TBI places limitation on the patient's ability to transfer what they have been taught from one context to another. The difficulty in maintaining changes following the withdrawal of active rehabilitation was demonstrated in the implementation of computerised cognitive retraining (Niemann et al, 1990), external compensatory memory aids (Schmitter-Edgecombe et al, 1995; & Kirsch, et al 2004) and internal compensatory strategies (Milders et al, 1998). Such methods appeared to have good effect during the course of rehabilitation, but when active rehabilitation was withdrawn, TBI individuals tended to return to pre-rehabilitation functioning. Despite the need for validating the maintenance of improvements following the withdrawal of rehabilitation, a number of studies reviewed do not include follow-up assessments (Niemann et al, 1990; Dirette & Hinojosa, 1999; Kim et al, 1999; Ownsworth & McFarland, 1999; Hux et al, 2000; Palmese & Raskin, 2000; Kaplan, 2001; Stathopoulou & Lubar, 2001; Cicerone, 2002; & Fleming et al, 2005).

Methodological issues consistently arising within the cognitive rehabilitation literature include the inclusion of TBI subjects during the early natural/spontaneous recovery stages, and poorly controlled study designs. Research has identified that significant improvements in TBI functioning occurs within the first six months (Lannoo, Colardyn, Jannes, & De Soete, 2001) and has further demonstrated that the majority of symptoms reported at six months post TBI maintained the same clinical classification at one year post TBI (Bowen, Chamberlain, Tennant, Neumann & Conner 1999). Some research has failed to fully account for the impact of natural

recovery, including TBI participants less than six months post injury. This occurred within the rehabilitation of attention (Cicerone, 2002); visual processing (Dirette & Hinojosa, 1999); speed of processing (Fasotti et al, 2000); prospective memory and planning (Fleming et al, 2005) and in general cognitive and functional abilities (Salazar *et al*, 2000; & Warden *et al*, 2000). Given most natural recovery occurs during these early months following TBI, it was difficult to determine whether the improvement in rehabilitation was attributable to either natural recovery or to cognitive rehabilitation. Additionally, a number of poorly controlled studies were also evident within the literature, with a number of studies failing to include randomised controlled trials. This was evident in the studies of rehabilitation of attention (Palmese & Raskin, 2000; & Stathopoulou & Lubar, 2001); memory (Hux, et al, 2000); prospective memory and planning (Kim at al, 1999; Kim et al, 2000; Kirsch, et al 2004; & Fleming et al, 2005); and executive functioning (Kaplan, 2001; & Marshall et al, 2004).

Additionally, most of the studies in the literature reviewed used formal cognitive measures, and the ecological validity and generalisability of neuropsychological measures to the functional environment has been questioned (Bowman, 1996). Oddy, Alcott, Francis, Jenkins, & Fowlie (1999) indicated that neuropsychological tests are not suitable for the measurement of outcome. It is common for significant functional improvements to exist, despite the lack of significant changes on formal cognitive measures (Teasdale, Hanson, Gade, & Christiansen, 1997; & Wilson, 2002).

The cognitive rehabilitation literature has frequently failed to control for the effect of perceived self-efficacy on the rehabilitation outcome, and finally, has not used placebo designs, or sham controlled studies. Research has established that higher

levels of perceived self efficacy are accompanied by higher performance attainments (Tam, 1996; & Marks, 2001). Therefore, it is possible that perceived self-efficacy may have influenced the rehabilitation outcomes. Designing placebo controlled studies would account for any change not directly attributable to the treatment, such as: the subjects' expectation of the therapy; their attitude; motivation; co-operation; and the therapeutic relationship or alliance. However, the ethical principles of implementing placebo or sham controlled studies have been questioned (La Vaque & Rossiter, 2001).

2.1.1.4 Electroencephalography Biofeedback

2.1.1.4.1 Basic Principles of Clinical Electrophysiology

In order to understand the application of EEG biofeedback, the basic principles of electrophysiology will be briefly discussed. Arciniegas, Anderson, & Rojas (2005), indicated that neurons of the cortical mantle are organised into columns. The electrical activity of columns of cortical neurons is generated by amino acid and other neurotransmitter afferents, which in turn, are regulated by subcortical structures, in particular the thalamus and reticular activating system (Hughes, 1982). Hughes and John (1999) report that EEG activity in healthy human beings can be expected to be reasonably stable, as a result of the homeostatic regulation of these processes. However, dysrhythmia in thalamocortical oscillations arising due to deficiencies or excesses of neurotransmitters may produce marked departure from the homeostatistically regulated EEG (Hughes & John, 1999).

Abnormal EEG rhythms may occur across four wide frequency bands, which are defined as Delta (1.5 – 3.5 Hz), Theta (3.5 – 7.5 Hz), Alpha (7.5 – 12.5 Hz) and Beta

(12.5 – 20 Hz), (Hughes and John, 1999). See Table 3 for a detailed description of each band. A further frequency band (12-15 Hz) referred to as Sensory Motor Rhythm (so named because the frequency exhibits rhythmic bursts at the sensorimotor cortex), has been identified and holds relevance in the application of EEG biofeedback (Sterman, Wywicka & Roth, 1969). Sensory Motor Rhythm (SMR) is characterised by an active mind and quiet body, an external focus of attention, paying attention, sequencing, information storage and retrieval (Laibow, 1999). Each frequency band is represented in the form of: Absolute Power in each band (total microvolts: μV^2); Relative Power in each band (percentage of total power in each channel); Coherence (a measure of synchronization between activity in two channels); or Symmetry (the ratio of power in each band between a symmetrical pair of electrodes), (Hughes & John, 1999).

Table 3: *Major EEG bands, their respective frequencies, probable neural generators, and most characteristic location in a normal surface EEG recording.*

** Adapted from Arciniegas, Anderson & Rojas (2005), page 137.*

Band	Frequency Range (Hz)	Principle Neural Generators	Characteristic Surface Electrode Location
β (beta)	12.5-20	Corticocortical & thalamocortical networks involved in information-processing	Maximal over frontal and central regions
α (alpha)	7.5-12.5	Thalamic pacemaker neurons	Occipital and perhaps central when eyes are closed
θ (theta)	3.5-7.5	Thalamic pacemaker neurons under the influence of inhibitory input from the reticular nucleus of the thalamus	If present in waking recording at all, amplitude is low and content is small; may be most obvious in central regions; becomes more obvious with drowsiness and sleep
δ (delta)	1.5-3.5	Oscillatory neurons in the deep cortical layers and within the thalamus	Not typically seen in the awake record of healthy adults; diffusely present in deeper sleep stages; may be focally located over cortical lesions; may become prominent in frontal/central regions due to

A number of EEG and qEEG studies have identified dysregulation of EEG activity following TBI, particularly in the alpha and theta frequency bands. Research has reported increases in theta and decreased alpha power and/or decreased coherence and asymmetry in the moderate to severe TBI population (Bricolo, Turazzi, Faccioli, Odorizzi, Sciarretta, et al, 1978). Similarly, Tebano, Cameroni, Gallozzi, Liozzo, Palazzino, et al, (1998) have demonstrated EEG dysregulation following mild TBI. They reported a shift in the alpha frequency and high beta. Thatcher, Walker, Gerson and Geisler (1989), analysed 608 mild TBI. Findings suggested that there was; 1) increased coherence and decreased phase in frontal and frontal-temporal regions; 2) decreased power differences between anterior and posterior cortical regions; and 3) reduced alpha power in posterior cortical regions. The practice of electroencephalography biofeedback aims to normalise the dysregulation of cortical activity following TBI.

2.1.1.4.2 Application of Electroencephalography Biofeedback

An emerging and promising new treatment approach for TBI is Electroencephalography (EEG) biofeedback training. The therapeutic application of EEG biofeedback is often referred to as Neurofeedback or Neurotherapy. EEG biofeedback is an operant conditioning procedure which aims to modify and normalise dysregulated EEG patterns. Importantly, Thatcher (1999) highlights that the exact physiological foundations of this process are not yet well understood. The biofeedback processes involves the recording and immediate feedback of brain waves (EEG) by electrodes detecting EEG activity through the patient's scalp. As described by Hughes (1994) the EEG is transmitted from the electrodes to: 1) amplifiers -

amplification is required given the EEG rhythms are only microvolts in amplitude; 2) filters, often expressed as time-constants - at times very slow or very fast (artifactual) rhythms need filtering out; and 3) through to a computer monitor - where the EEG feedback is presented to the patient in the form of a game.

This feedback procedure commonly involves rewarding the patient for a transient increase over threshold of EEG activity within a frequency band requiring enhancement, with concurrent inhibition of activity over threshold of the frequency band disproportionately dominant (Othmer, Othmer, & Kaiser, 1999). The rewarding and inhibiting of certain EEG frequencies is observed by the patient on a computer monitor in the form of visual and auditory feedback. The visual feedback (computer graphics) and auditory feedback (e.g. music) change in response to changes in the patient's EEG. The patient is instructed to attempt to produce and maintain the positive feedback. That is, they need to keep the computer graphics moving and the music playing, representing the concurrent and simultaneous increase and inhibition of the appropriate frequencies being trained. Conversely, when the patient produces dysregulated EEG activity all positive feedback is ceased (computer graphics cease to move, and music stops playing). The patient's raw EEG signal is continuously monitored by the clinician to set and alter the threshold of reward contingencies (Othmer et al, 1999). Over time, during this operant conditioning procedure the individual learns to modify the amplitude, frequency, or coherency of the electrophysiological dynamics of their own brain (Thatcher, 1999).

2.1.1.4.3 Historical Perspective of Electroencephalography Biofeedback

Electroencephalography biofeedback research emerged during the end of the 1960s. A number of studies demonstrated the ability to control brain wave rhythms through

operant conditioning and biofeedback techniques in the epileptic population through the suppression of Sensory Motor Rhythm (SMR), (12-15 Hz), (Sterman & Wywicka, 1967; Sterman et al, 1969; Sterman & Friar, 1972; Sterman, Macdonald & Stone, 1974; Sterman & Macdonald, 1978; & Sterman & Shouse, 1980). Similarly, Lubar and Bahler (1976) found that through EEG biofeedback training, epileptics where able to enhance SMR frequency and suppress theta slow wave frequency. Following this, it was discovered that patients demonstrated increased attentiveness, focus, and concentration. Lubar and Shouse (1976), and Lubar and Lubar, (1984), found that improvements in attention and reduced distractibility in academic settings resulted in increased school performance and grades.

This past research has built a foundation for EEG biofeedback to be utilised as a treatment for a number of varying disorders. When reviewing a mix of well controlled (including recent preliminary double-blind placebo controlled designs), poorly controlled, large studies, single and multiple case studies, the areas receiving the most attention which demonstrate some level of efficacy include: *attention deficit hyperactivity disorder* (Rossiter & La Vaque, 1995; Radvanski, Wadhwani, Sabo, & Vergara, 2001; Monastra, Monastra & George, 2002; Fuchs, Birbaumer, Lutzenberger, Gruzelier, & Kaiser, 2003; DeBeus, Ball, DeBeus, & Herrington, 2004; & Lévesque, Beauregard, & Mensour, 2006), *learning disabilities* (Lubar, Gross, Shively, & Mann, 1990; Tansey, 1991; & Orlando & Rivera, 2004), *and substance addictions* (Peniston, & Kulkovsky, 1989; Peniston, & Kulkovsky, 1990; Bodenhamer-Davis, Callaway, & DeBeus, 2003; & Burkett, Cummins, Dickson, & Skolnick, 2004). Importantly, recent research has begun to demonstrate functional brain changes following EEG biofeedback. Compared to controls, fMRI detected the normalisation of the anterior cingulate cortex functioning and improved impulse

regulation following EEG biofeedback in a sample of children with attention deficit hyperactivity disorder (ADHD), (Lévesque et al, 2006).

Recently, preliminary evidence in single case study designs has begun to emerge in the effectiveness of EEG biofeedback as a treatment for a variety of psychiatric conditions. A single case study (Thomas & Sattlberger, 1997) reported a significant reduction in anxiety symptomatology following 15 sessions of EEG biofeedback. At a three year follow up all clinical scales on the Minnesota Multiphasic Personality Inventory – Second Edition (MMPI-2) were within the normal range. Similarly, Baehr, Rosenfeld & Baehr (1997) illustrated the effectiveness of using alpha asymmetry EEG biofeedback to alleviate depressive symptomatology in two depressed patients. A significant reduction in depressive symptomatology and changes in personality factors was evident following EEG biofeedback on the clinical scales on the MMPI. Hammond (2003) demonstrated that following the implementation of EEG biofeedback in two patients with obsessive compulsive disorder, a reduction was observed in obsessive compulsive behaviour, depressive, anxious, and somatic symptomatology, as measured by the Yale-Brown Obsessive-Compulsive Scale and MMPI-2. Patients maintained their improvements at 13 and 15 months follow-ups, respectively. Similarly, further research conducted by Hammond (2005) provided further evidence for the efficacy of treating depression with EEG biofeedback. Of eight patients 77.8% made significant reductions in their depressive symptomatology following an average of 20.75 thirty-minute sessions of EEG biofeedback.

A substantial number of studies are reporting successful treatment outcomes following EEG biofeedback for a diverse range of disorders. There are many

similarities between the symptoms being treated within these disorders and the sequelae following TBI. For example, people with TBI more commonly experience attentional problems, learning difficulties, depression, anxiety, and in some cases epilepsy. Therefore, the diverse body of research outlined would suggest that EEG biofeedback may be effectively applied in the treatment of TBI sequelae.

2.1.1.4.4 The Effectiveness of Electroencephalography Biofeedback in TBI

An increasing, but small body of research has provided some preliminary support for the effectiveness of EEG biofeedback in the treatment for various TBI sequelae (Walker, Norman, & Weber, 2002; Hammond, 2003; Hopkins & Sterman, 2003; Sterman, 2003; & Hammond, 2004).

One of the earliest clinical reports to demonstrate anecdotal evidence of the effectiveness of EEG biofeedback in the TBI population was conducted by Ayers (1987). Following the treatment of 250 TBI patients, Ayers reported that the patients experienced an increase in energy, improvements in short-term memory, and a decrease in depressive symptomatology, anger issues, and headaches. This evidence is based on an accumulation of clinical TBI cases and not on a well controlled and designed study. Combating some of the methodological issues from Ayers previous work, Ayers (1993) conducted a well controlled study to measure the effectiveness of EEG biofeedback in the rehabilitation of brain injury. Subjects, who were three years post injury, were randomly assigned into two groups, the EEG biofeedback treatment group (N=6), and the comparison psychotherapy group (N=6). The EEG biofeedback group continued to receive psychotherapy in addition to the experimental treatment. Results indicated no changes pre-post assessment in the psychotherapy only group.

Conversely, improvements were noted on formal and self-report measures of mood, anger, and anxiety symptomatology in the EEG biofeedback group.

Anecdotal and clinical case studies have continued to provide preliminary support for the effectiveness of EEG Biofeedback in TBI. Byers (1995) presented a case study of a 58 year old patient six years post mild TBI with persistent post concussion symptoms. Following 31 sessions of EEG biofeedback, a general reduction in amplitude was observed through qEEG. Neuropsychological assessment revealed significant improvements in the patient's ability to utilise fluid intelligence, working memory, cognitive flexibility, problem-solving ability, and verbal fluency. No mood or personality changes were evident pre-post therapy as measured by the Beck Depression Inventory and MMPI – 2. Additionally, the patient also received 12 sessions of cognitive strategy training and was medicated with antidepressants (Prozac) throughout the course of treatment. Subsequently, some level of change may be attributable to these factors. In a larger clinical study, Hoffman et al (1996) collated outcome data from two separate clinics specialising in EEG biofeedback for brain injury patients. Outcome data was collected for 50 mild TBI patients, most of which were beyond the spontaneous recovery period. Following an average of 40 treatment sessions, a significant improvement in most symptoms (clustered into physical, emotional, and cognitive) was reported.

A more recent multiple single case study (Salerno, 1997) examined the effectiveness of EEG biofeedback in 10 TBI subjects, ranging from mild to severe in severity, at least 13 months post injury. Findings indicated changes in the alpha and SMR frequencies on qEEG, statistically significant improvements in attention-concentration, sleeping difficulties, and mood. Although self-reported measures

generally indicated improvements in memory, this was not supported by statistically significant results on formal testing. Similarly, another case study has provided evidence for the effectiveness of EEG biofeedback in the treatment of physical and functional abilities (Ayers, 1999). Ayers presented a case of a 17 year old patient nine years post severe brain injury, who was wheelchair bound, with severe expressive speech difficulties, and had extreme spasticity. Following 50 sessions of EEG biofeedback the patient was much clearer in articulation and functioning independently.

Some recent case studies have demonstrated that EEG biofeedback may be effective in reducing neurological sequelae following TBI. Hopkins and Sterman (2003) implemented an intense EEG biofeedback program on a patient who was greater than one year post multiple TBIs, with post traumatic epilepsy following the most recent brain injury. Anticonvulsant medication failed to control the seizure activity. Following one month of therapy, the patient experienced a cessation of seizures and was able to withdraw from medications. Similarly, Sterman (2003) applied EEG biofeedback on a TBI patient with a history of incapacitating exertion-induced headaches subsequent to a motor vehicle accident. Following qEEG guided training, results indicated normalisation of EEG activity, significant symptom reduction in headache logs, and the patient returned to gainful employment. In addition to the neurological problems already addressed, a partially blinded case study (Hammond, 2004) evaluated the rehabilitation of balance and urinary incontinence following brain injury. It was evident on pre-post measures that balance improved in all patients. Improvements were commonly noted by the patients within three sessions, and the total treatment generally ranged from 10 to 15 sessions. One patient, blinded to the potential effects of EEG biofeedback on urinary incontinence, demonstrated a

reduction in the frequency of incontinent events. The frequency of incontinence declined from three or four accidents daily, to only an infrequent accident, as reported by the patient and her mother.

Larger studies have been conducted in the evaluation of EEG biofeedback. Walker et al (2002) designed a non-controlled study which examined the functional outcomes of EEG biofeedback in a mild TBI population. Twenty-six patients with post-concussive symptoms, three to 70 months post injury, received 40 treatment sessions. Activities of daily living (e.g. work, social and/or leisure activities) were measured during and following treatment by a Global Improvement Score. A significant improvement was noted in 88% of the patients, with positive changes occurring most frequently in self-reported headaches and memory loss or confusion. Participants gainfully employed prior to treatment all reported that they were able to return to work following treatment. Similar to the previous study design described, a non-controlled four-part study was conducted by Bounias, Laibow, Bonaly, & Stubblebine (2001), Laibow, Stubblebine, Sandground, & Bounias (2001), Bounias, Laibow, Stubblebine, Sandground, & Bonaly (2002), Laibow, Stubblebine, Sandground, & Bounias (2002). Over the course of these studies, Bounias and colleagues investigated the effectiveness of EEG biofeedback within a sample of 27 brain injured patients under drug free conditions. They devised a classification of clinical syndromes including: 1) motor; 2) language; 3) cognitive; 4) psychosocial; 5) pain-related; 6) neuropsychiatric; and 7) metabolic. Patients were reported to be unambiguously distributed in all classes except for the metabolic class. The percentage of symptoms eliminated following treatment provided an index of improvement or rehabilitation rate. After a number of sessions (ranging between 23 to 132), the TBI subjects' average improvement or rehabilitation rates were: 77% for motor functions; 87% for

cognitive functions; 77% for psychosocial functioning; 80% for pain difficulties; and 67% for neuropsychiatric symptomatology, with a range of class improvements from 59% up to 87%. They also demonstrated normalisation in frequency bands as detected by qEEG. It was concluded that EEG biofeedback can be used successfully to treat patients with brain injury.

An increasing number of well controlled studies have emerged in the literature over the past five years. Tinius and Tinius (2000) evaluated the effectiveness of simultaneous EEG biofeedback and cognitive retraining over 20 session within a mild TBI group (N=16) and ADHD group (1N=13), compared to normal controls (N=15) not receiving treatment. It was unclear and not reported whether they controlled for natural recovery in the mild TBI group. Their findings demonstrated significant improvement in sustained attention, response accuracy, and a significant decrease in self-reported neuropsychological symptoms in both the mild TBI and ADHD groups compared to controls. Given cognitive remediation techniques were used simultaneously during EEG biofeedback treatment, it can only be concluded that the concurrent use of each treatment was effective and not EEG biofeedback alone. Research has also evaluated the use of EEG biofeedback during the early phases of recovery. A study conducted by Keller (2001) applied EEG biofeedback to 12 moderate TBI subjects during the spontaneous recovery period, compare to a well matched control group of nine patients receiving standard computerised training. After ten sessions the EEG biofeedback group made significant improvements in attentional functioning as compared to the control group. This suggested that EEG biofeedback may be a useful tool in the rehabilitation of the early recovery phase following TBI.

Schoenberger, Shiflett, Esty, Ochs, & Matheis (2001) designed a controlled study which investigated the effectiveness of 25 EEG biofeedback sessions on 12 TBI subjects ranging in severity from mild to moderately-severe, at least 12 months post injury. The subjects were randomly assigned to either the treatment group or a wait list control group which received treatment following a waiting period. In comparison to the wait list control group, significant improvements were demonstrated in self-reported depression, fatigue, and on a range of problematic symptomatology. Significant improvements in cognitive functioning were also reported including: working memory, immediate memory of new material, and retention of information. Treatment gains were maintained from post-treatment to follow-up assessment and in some cases further improvement was observed. Additionally, Schoenberger and colleagues reported interesting clinical findings within their TBI sample. Following EEG biofeedback, a number of subjects were able to reduce and in some cases cease medications (generally pain medications). Most participants were reported to have experienced meaningful improvements in occupational and social functioning.

Further controlled research has been conducted by Thornton (2002). Thornton compared four brain injured patients who underwent qEEG guided EEG biofeedback for memory rehabilitation, to a qEEG database obtained from 59 subjects during auditory memory tasks. All subjects were at least one year post brain injury. The therapy protocols attempted to normalise specific EEG variables associated with memory functioning that deviated from the control group. Following 55 sessions, case one improved 94% in memory functioning; case two improved 61% following 16 sessions; case three demonstrated 110% improvement following 15 sessions; and case four made 28% improvement in memory function following 21 double sessions,

as compared to the qEEG database control group. These memory improvements were maintained at one month to one year following the withdrawal of the treatment.

2.1.1.4.5 Limitations of Electroencephalography Biofeedback

Electroencephalography Biofeedback research bears similar limitations and methodological issues as that of cognitive rehabilitation. Some studies evaluating EEG biofeedback, although using formal cognitive measures, have failed to use formal functional outcome measures (e.g. return to work, and cognitive, emotional, and behavioural functioning in every day life). This raises the question of the ecological validity of EEG biofeedback, questioning the ability of the patients to generalise change in EEG activity to everyday life. In addition, the ecological validity and generalisability of neuropsychological measures to the functional environment has been questioned (Bowman, 1996; Teasdale et al, 1997; Oddy et al, 1999; & Wilson, 2002). Furthermore, there is a great lack in follow-up data following the rehabilitation of TBI using EEG biofeedback. Of the research reviewed only two studies (Schoenberger et al, 2001; & Thornton, 2002) included follow-up data. This makes it difficult to accurately ascertain if patients are maintaining the reported improvements once the treatment is withdrawn.

With the exception of a few studies, the great majority of the research within the EEG biofeedback field for TBI appears to be anecdotal, based on clinical reports, or have used poorly controlled designs. A number of studies have failed to implement well controlled designs (Ayers, 1987; Byers, 1995; Hoffman et al, 1996; Salerno, 1997; Ayers, 1999; Bounias et al, 2001; Laibow et al, 2001; Bounias et al, 2002; Laibow et al, 2002; Walker et al, 2002; Hopkins & Sterman, 2003; & Sterman, 2003). Furthermore, some well controlled studies evaluating the effectiveness of EEG

biofeedback have been compromised by the concurrent use of other treatments (Tinius & Tinius, 2000). Most of the research conducted in the field has accounted for the natural recovery period following TBI. However, some studies continue to include subjects less than six months post TBI (Hoffman et al, 1996; & Walker et al, 2002). Finally research has failed to control for the effect of perceived self-efficacy on the rehabilitation outcome, and has not used placebo designs, or sham controlled studies. As previously noted, the ethical principles of implementing such methods have been questioned (La Vaque & Rossiter, 2001). Furthermore, it would be very difficult to maintain limited awareness of this treatment within a placebo group.

2.2 SUMMARY OF REHABILITATION APPROACHES

The outcomes of the rehabilitation approaches discussed are compromised by a number of environmental and patient characteristics. Sohlberg and Mateer (2001) highlight a number of factors which can profoundly influence the rehabilitation outcome, including: premorbid functioning; personality; social support; and environmental demands, just to name a few. Ponsford (2004) reports that negative prognostic indicators for the recovery of TBI include: a history of learning difficulties, low intelligence, unemployment, lower socioeconomic status, substance abuse and psychiatric disorders. Lack of awareness and poor motivation are characteristics of some TBI patients which compromise the effectiveness of rehabilitation (Alderman, 2004). Sohlberg and Mateer (2001) indicate that patients who lack self-awareness may not acknowledge changes in their functioning, or the need for treatment, and are often resistant to rehabilitation activities. Mood and behaviour disorders, commonly reported following TBI, can be detrimental to the effectiveness of a patient's rehabilitation. Mood disorders can impact on the TBI patient's motivation levels and commitment to rehabilitation (Sohlberg & Mateer,

2001). Such characteristics which may be evident following TBI may also create methodological issues when evaluating rehabilitation techniques. Research has often used self-reported questionnaires to evaluate outcomes. Individuals who lack self awareness may not acknowledge changes occurring due to treatment. Furthermore, a lack of self awareness and motivation, and mood disturbance may impact on the patient's commitment to rehabilitation. Inclusion of such patients in rehabilitation research may further compromise the outcomes.

In reviewing the cognitive rehabilitation and EEG biofeedback literature, potential efficacy exists for some specific methods in cognitive rehabilitation, and there is some preliminary evidence supportive of EEG biofeedback (in the remediation of cognitive, emotional, behavioural, and neurological sequelae). Research in the cognitive rehabilitation domain has more consistently included follow-up reviews than in the EEG biofeedback field. Long-term follow-up reviews are imperative in the measurement of efficacy of the rehabilitation approach. Compared to cognitive rehabilitation studies, EEG biofeedback research has more frequently excluded TBI patients during the natural recovery phase. Therefore, the outcome data is more likely to represent the effects of the treatment rather than natural recovery.

Despite these observed differences, both rehabilitation approaches have comparable methodological issues compromising the validity of the results. In particular, both domains of research include a large number of anecdotal findings, clinical single case studies, poorly controlled studies, and non-blinded placebo controlled studies. Subsequently, this leaves very few studies with Class I evidence - that is, evidence provided by one or more, well-designed, prospective, blinded, controlled clinical studies (Thatcher, 2000). Overall, there does appear to be efficacy in a small number

of well designed studies which applied formulaic methods in cognitive rehabilitation, and some preliminary evidence for the application of EEG biofeedback.

Chapter 3.

RATIONALE

3.1 STUDY RATIONALE

The literature reviewed in the previous chapter highlighted a number of areas in the rehabilitation of TBI that require further study. An issue of particular note was the degree to which the efficacy of each rehabilitation approach was compromised by inadequate methodological designs. The relevant efficacy of different treatments is best established by directly comparing different treatment approaches, however this has rarely occurred (particularly following EEG biofeedback). There has been little research investigating the effectiveness of EEG biofeedback including severe TBI populations. Cognitive rehabilitation research, on the other hand, has largely examined the application of very specific techniques within the TBI population, despite the need for a holistic approach targeting not only cognition, but emotional, and behavioural sequelae. Finally, there is limited evidence or published studies reporting on the cerebral electrophysiological dynamics following cognitive rehabilitation. The current study will aim to address some of these issues.

3.2 AIMS OF RESEARCH

The aim of the present study was to investigate the effectiveness of cognitive rehabilitation and EEG biofeedback as treatments for TBI. Research has identified some efficacy for the use of specific cognitive rehabilitation methods for the treatment of TBI, and there is some preliminary evidence for the effectiveness of EEG biofeedback. By comparing each treatment approach the study aims to determine if one therapy is more effective than the other in treating the cognitive, emotional, and behavioural sequelae of TBI.

Research has also demonstrated that normalisation of the electrophysiology of the brain occurs following EEG biofeedback. However, little research has demonstrated, or even measured the normalisation in EEG (using qEEG) following cognitive rehabilitation. Subsequently, the study also aimed to determine if one therapy was more effective than the other in normalising the electrophysiological dynamics of the brain.

3.3 HYPOTHESES

3.3.1 Hypothesis One

EEG biofeedback will be more effective than cognitive rehabilitation in the treatment of cognitive, emotional, and behavioural sequelae following TBI.

The literature reviewed has demonstrated some preliminary evidence that EEG biofeedback may assist in the rehabilitation of a broad range of sequelae following TBI, including, cognitive, emotional, behavioural, neurological, and physiological. The efficacy of specific cognitive rehabilitation methods has been demonstrated to assist in the remediation of specific cognitive functions, but this approach has less frequently measured and demonstrated changes in other domains (e.g. emotional, behavioural, neurological, and physiological functioning). Consequently, it is hypothesised that EEG biofeedback will be more effective than cognitive rehabilitation in the treatment of cognitive, emotional, and behavioural sequelae following TBI.

3.3.2 Hypothesis Two

EEG biofeedback will be more effective than cognitive rehabilitation in normalising the dysregulated electrophysiology of the TBI brain.

Electroencephalography biofeedback has been demonstrated to be an effective and direct method of normalising dysregulated cerebral electrophysiology in TBI individuals. On the contrary, cognitive rehabilitation does not directly aim to achieve EEG normalisation, and has not yet been substantially demonstrated in the literature. Therefore, it is hypothesised that EEG biofeedback will be more effective than cognitive rehabilitation in normalising the dysregulated electrophysiology of the TBI brain.

Chapter 4.

METHODOLOGY

4.1 PARTICIPANTS

Participants were recruited from a number of brain injury support organisations (Head Way, Bear In Mind, and Liaise). A total of 29 participants expressed an interested in participating in the study, however, only 10 participants met the selection criteria. Of the 19 participants excluded from the study: five were outside the ages of 18 to 50 years; four were less than one year from the time of closed traumatic brain injury; a number had pre-morbid neurological disorders and brain injuries not traumatic in nature – three had a cerebrovascular accident, two had cerebral tumours, and one had epilepsy; one indicated the presence of a pre-morbid learning difficulty; and three reported serious pre-morbid psychological/psychiatric problems. A total of 10 participants signed the consent forms and commenced participation in the study. Two participants chose to discontinue participation during the initial assessment due to the long distance and frequency of travelling required in order to attend the treatment programs. One participant was deemed unsuitable for the study following completion of the initial assessment, given belated reports of a psychiatric illness (schizophrenia). One participant completed the initial assessment, first treatment program (cognitive rehabilitation), and the post first treatment assessment, however ceased participation prior to commencement of the second treatment program due to changes in their personal circumstances.

A total of six participants completed the research project. The participants consisted

of four males and two females, with a mean age of 48.7 years (range of 45 to 51 years), a mean education of 9.3 years (range of 4 to 12 years), and a mean time since injury of 16.7 years (range 1.4 to 47 years). The mean severity was "very severe" (range of moderate to extremely severe); with a mean estimated pre-morbid intelligence of 102 (ranging from 91.1 to 113.8); and a mean Full Scale Intelligence Quotient of 98 (ranging from 63 to 118). All participants had also sustained additional orthopaedic injuries at the time of the TBI. During the current research, all participants were living independently.

4.2 MEASURES

The assessment materials consisted of: one measure of intellectual functioning, Wechsler Adult Intelligence Scale –Third Edition (WAIS-III); one measure of pre-morbid functioning, National Adult Reading Test- Revised (NART-R); ten measures of cognitive functioning, 1) Tests of Variables of Attention (TOVA); 2) Paced Auditory Serial-Addition Test (PASAT); 3) Rey Auditory Verbal Learning Test (RAVLT); 4) Rey Complex Figure; 5) Symbol Search; 6) The Speed and Capacity of Language Processing Test (SCOLP); 7) Controlled Oral Work Association Test (COWAT); 8) Trail Making Test (TMT); and five questionnaires: 1) Beck Depression Inventory- Second Edition (BDI-II); 2) State Trait Anxiety Inventory- Second Edition (STAI-II); 3) State Trait Anger Expression Inventory-Second Edition (STAXI-II); 4) Neurobehavioural Rating Scale; and 5) demographic questionnaire. The materials also included a Plain Language Statement (Appendix 1) and Informed Consent Form for participants (Appendix 2)

4.2.1 The Informed Consent Form

Prior to commencement in the project the participants were required to sign the

informed consent form (See Appendix 2). The consent form was devised using the outline provided by the Department of Psychology Ethic Committee, Victoria University of Technology. The consent form provided participants with a plain language summary of the purpose, procedure, and aims of the study. In addition, it assured participants that every effort would be made to safeguard anonymity and confidential information. The consent form also outlined that a payment was required to be made prior to the commencement of the research project to cover the running costs of the qEEG. Finally, the consent form reminded participants that their participation was voluntary, and that they could withdraw at any time they desired.

4.2.2 The Demographic Information Questionnaire

This questionnaire required participants to provide the following demographic information: age, education level, time since brain injury, country of birth, current employment status and status prior to injury, length of coma and post traumatic amnesia (PTA), length of hospital stay, orthopaedic injuries, pre-morbid psychological and neurological functioning, and any current treatment (including medications) for TBI (Appendix 3). When possible, medical records were obtained to gain accurate measures of PTA and coma length following TBI. When it was not possible to obtain medical records, a close relative or significant other that was present at the time of injury provided an account of the coma length and PTA.

4.2.3 Cognitive Measures

4.2.3.1 General Intellectual Measures

4.2.3.1.1 National Reading Test- Revised

The National Adult Reading Test – Revised (NART-R) was developed by Blair and Spreen (1989) and it provided a standardised measure of pre-morbid intellectual ability. The NART-R is a reading test consisting of 61 irregularly spelled words, which the person is required to read. The following NART-R equations were used to predict estimated premorbid IQ; 1) estimated Verbal IQ = 128.7 – 0.89 X (NART-R errors); 2) estimated Performance IQ = 119.4 – 0.42 X (NART-R errors); and 3) estimated Full Scale IQ = 127.8 – 0.78 X (NART-R errors). Strong interscorer reliability, internal consistency (Blair & Spreen, 1989), and test-retest reliability (Raguet, Campbell, Berry, Schmitt, & Smith, 1996), have been established (see Appendix 4). However, research has demonstrated that the administration of the NART prior to one year post brain injury runs the risk of significantly underestimating pre-morbid intelligence (Riley & Simmonds, 2003). Conversely, when administered one year post injury there is significantly less error associated with the estimation of pre-morbid intelligence (Riley & Simmonds, 2003). Given the participants included in the study were all at least one year post brain injury, the NART-R was considered to be a relatively accurate measure of pre-morbid intelligence (allowing for those with literacy or reading problems pre-morbidly).

4.2.3.1.2 Wechsler Adult Intelligence Scale –Third Edition

The Wechsler Adult Intelligence Scale – Third Edition was developed and revised by Wechsler (1997) to provide a standardised measure of general intellectual functioning. The Australian standardised version was administered in the present

study. Test-retest reliability, split half reliability, criterion validity, construct validity have been established (Psychological Corporation, 1997).

4.2.3.2 Attention

4.2.3.2.1 Test of Variables of Attention

The Test of Variables of Attention (TOVA) is an objective and standardised computer-based continuous performance test which was designed to measure visual attention, in particular sustained attention, impulse control, and speed of processing (Greenberg & Waldman, 1993; & Greenberg, Kindschi, & Corman, 2000). The TOVA commences with a 2.5 minute practice session, followed by the test session which is 22.6 minutes in duration and is divided into four quarters. The visual test consisted of two easily discriminated geometric figures (one a target and the other a non-target) which were presented for a duration of 100msec, at fixed intervals of two seconds. The target figure consisted of a square with a hole at the top, while the non-target figure consisted of a square with a whole at the bottom, and were both centred on the computer screen. The targets and non-targets are pre-designed to appear at two different conditions: 1) Stimulus infrequent = 36 targets and 126 non-targets per quarter, lasting two quarters, and 2) Stimulus frequent = 126 targets and 36 non-targets per quarter, lasting two quarters (Leark, Dupuy, Greenberg, Corman, & Kindschi, 1996).

The participants received standardised instructions. Using a highly accurate (± 1msec) electronic microswitch, participants were instructed to push the switch as fast and accurately as possible when the target geometric figure appeared on the screen, and refrain from triggering the switch when the non-target was presented. The

TOVA measures the participant's performance on Omission Errors, Commission Errors, Response Time, and Variability. Omission errors occur when the participant fails to respond to the designated target and such errors are considered to be indicative of inattention. Commission errors occur when the participant responds (presses the switch) to the non-target and these errors are reported to be indicative of impulsivity or failure to inhibit a response. The response time is the average latency of the correct response, which is interpreted as a measure of speed of information processing. Finally, variability (standard deviation of mean correct response times) is considered a measure of consistency in response times (Weyandt, Mitzland, & Thomas, 2002).

The TOVA was normed on 2000 individuals, consisting of year by year norms for each gender from 4 to 19 years and grouped norms by gender from 20 to 80+ years. Reliability, validity, sensitivity, specificity, and standard error of measurement (SEM) have been established (Greenberg et al, 2000; & Leark, Wallace & Fitzgerald, 2004), (see Appendix 4). Given it is a non-language based test, it is reported to require no left-right discrimination or sequencing, have no appreciable practice effects, and it minimises the potential of the results by learning, cultural effects, and a learning disability (Greenberg et al, 2000). However, Leark et al (2004) identified that practice effects occurred at both 90 minute and one week test-retest intervals, but only for the commissions test.

4.2.3.2.2 Paced Auditory Serial Addition Test

The Paced Auditory Serial Additional Test (PASAT) was developed by Gronwall (1977) and is a serial addition task which measures the capacity and rate of information processing, and sustained and divided attention (Spreen & Strauss, 1998).

Spreen and Strauss (1998) indicated that the participant is required to comprehend auditory input, respond verbally, and inhibit encoding of the response while attending to the next stimulus in a series. The random delivery of 61 numbers from 1 to 9 is presented on a pre-recorded tape, in the same order, but at different speeds over four trials (at 2.4, 2.0, 1.6, & 1.2 second intervals). The subject is instructed to add pairs of numbers, whereby each number is added to the one that immediately precedes it (i.e. the second is added to the first, the third to the second, and so on). Approximately, 15 to 20 minutes were required to conduct the PASAT. Reliability and validity has been established for the PASAT (Egan, 1988; McCaffrey, Cousins, Westervelt, Martnowicz, Remick, et al, 1995; & Sherman, Strauss, & Spellacy, 1997), (see Appendix 4). McCaffrey et al (1995) noted practice effects with repeated administration. Therefore, alternative forms were used in the present study.

4.2.3.3 Memory

4.2.3.3.1 Rey Auditory Verbal Learning Test

Re-developed by Lezak (1983) the Rey Auditory Verbal Learning Test (RAVLT) was designed to assess verbal learning and memory (Rey, 1958). In particular, the RAVLT measures immediate memory span, new learning, susceptibility to interference, and recognition memory (Spreen & Strauss, 1998). The test consists of five learning trials, an interference list, a free recall test, followed by a 20 minute delayed recall test and finally a recognition test (Spreen & Strauss, 1998). Test re-test reliability, criterion validity, and the standard error of difference have been established (Spreen & Strauss, 1998; Moritz, Iverson & Woodward, 2003; & Lemay, Bedard, Rouleau, & Tremblay, 2004), (see Appendix 4). Research conducted by Lemay et al (2004) demonstrated that the RAVLT was a reliable instrument for

repeated neuropsychological testing providing alternate forms were used. In the present study, efforts to maximise reliability and to minimise practice effects were addressed by using alternate forms at each assessment.

4.2.3.3.2 Rey-Osterrieth Complex Figure Test

The Rey-Osterrieth Complex Figure Test (CFT) is a measure of visuospatial constructional ability and visual memory (Spreen & Strauss, 1998), designed by Rey (1941) with further development by Osterrieth (1944). The participants were required to copy the figure as carefully as possible while being timed (no time limit was imposed). Following three minutes and without prior warning the participants were then asked to reproduce the figure from memory (Spreen & Strauss, 1998). Reliability and validity have been established for the CFT (Delaney, Prevey, Cramer, & Mattson, 1992; Meyers & Meyers, 1995; & Tupler, Welsh, Asare-Aboagye, & Dawson, 1995). Spreen and Strauss (1998) report that practice effects occur with repeated administration of the same figure. In order to reduce the practice effects of the CFT the presentation of alternative forms (complex figures) was considered. However, research has demonstrated that alternative figures are not of equal difficulty and their use in a test-retest situation can be problematic (Delaney et al, 1992; & Gagnon, Awad, Mertens, & Messier, 2003). Therefore, the decision was made to re-administer the CFT on each testing session.

4.2.3.4 Speed of Information Processing

4.2.3.4.1 Symbol Search (WAIS-III)

Symbol search is a subtest from the WAIS-III, included in the Processing Speed Index, PIQ, and ultimately the FSIQ. Symbol search is reported to measure visual

processing speed, planning, and perceptual organisation (Kaufman & Lichtenberger, 1999). This subtest has a relatively strong test-retest reliability co-efficient of 0.79. Furthermore, the standard error of measurement for symbol search has been calculated and is reported to be ± 1.27 (The Psychological Corporation, 1997).

4.2.3.4.2 The Speed and Capacity of Language Processing Test

The Speed and Capacity of Language Test (SCOLP) is a measure of the efficiency of language comprehension. Specifically, the test used in the study was The Speed of Comprehension Test. Originally developed by Collins and Quillian (1969) this test measures the speed of language comprehension. The test consisted of 100 simple statements about the world, which include an equal proportion of true and false (silly) sentences. Following the administration of standardised instructions, the participants were required to quickly identify as many true and silly sentences as possible within two minutes. The total errors were tallied, and if the error score was less than 10% the error score was used to calculate a scaled score (The Medical Research Council, 1992). Reliability and validity have been established (The Medical Research Council, 1992), (see Appendix 4).

4.2.3.5 Executive Functions

4.2.3.5.1 Controlled Oral Word Association Test (Phonological and Semantic)

The Controlled Oral Word Association Test (COWAT) assesses phonological and semantic verbal fluency. The phonological fluency test administered was F.A.S., (Miller, 1984) and the semantic fluency test was the category of animals (Rosen, 1980). Reliability, the standard error of prediction, and validity have been demonstrated for both the phonological (F.A.S.) and semantic (Animals) verbal

fluency tests (Harrison, Buxton, Husain, & Wise, 2000; Vlaar & Wade, 2003; & Henry & Crawford, 2004), (see Appendix 4). Furthermore, research has demonstrated that at 12 month retesting of verbal fluency, no improvement on performance was evident (Basso, Bornstein, & Lang, 1999). Therefore, it was concluded that verbal fluency measures were not subject to significant practice effects, and the present study re-administered these measures at each testing session.

4.2.3.5.2 Trail Making Test

A test within the Halstead-Reitan Battery (Reitan & Wolfson, 1993), the Trail Making Test (TMT) consists of two parts (Trails A and Trails B) designed to measure speed of attention, sequencing, mental flexibility, complex visual scanning, motor functions (Spreen & Strauss, 1998) and in Trails B, cognitive flexibility (Kortte, Horner, & Windham, 2002). Participants were provided with standardised instructions. Part A required them to quickly connect 25 encircled numbers, which were randomly arranged on a page, in the correct order, while being timed. Part B required the participants to quickly and accurately connect 25 encircled numbers and letters in alternating order, while being timed. Reliability, the standard error of prediction, and validity have been demonstrated for both Trails A and B (Basso et al, 1999; Heaton, Temkin, Dikmen, Avitable, Taylor, et al, 2001; & Kortte et al, 2002), (see Appendix 4). Furthermore, Basso et al (1999) demonstrated that performance on Trails A and B did not significantly improve as a result of retesting at 12 months. Therefore, it was concluded that the tests were not subject to significant practice effects and these measures were re-administered on each testing session.

4.2.4 Emotional and Behavioural Measures

4.2.4.1 Beck Depression Inventory- Second Edition

Reported to be an effective screening tool for self-reported depression following TBI (Green, Felmingham, Baguley, Slewa-Younan, & Simpson, 2001), the Beck Depression Inventory - Second Edition (BDI-II), (Beck, Steer, Brown, 1996) is a measure of depression severity in individuals above the age of 13 years. The severity of depression is represented by the following scores: 0-13 as minimal; 14-19 as mild; 20-28 as moderate; and 29-63 as severe (Beck et al, 1996). Reliability and validity have been established for the BDI-II (Beck et al, 1996; & Sprinkle, Lurie, Insko, Atkinson, Jones, et al, 2002), (see Appendix 4).

4.2.4.2 State Trait Anxiety Inventory for Adults

The State Trait Anxiety Inventory (STAI) is a self-administered test designed to measure the severity of state (how the participant feels at the very moment of the test) and trait (how the participant generally feels) anxiety (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). The state anxiety (S-anxiety) and trait anxiety (T-anxiety) scales consist of 20 statements each. For each of the 20 items in the S-anxiety scale, participants were required to rate the intensity of their feelings on a four point scale: 1) not at all; 2) somewhat; 3) moderately so; or 4) very much so. In the T-anxiety scale participants were required to rate the frequency of their feelings on a four point scale: 1) almost never; 2) sometimes; 3) often; or 4) almost always. The participants were given the S-anxiety scale to complete first, followed by the T-anxiety scale. Reliability and validity were established by Spielberger et al, (1983), (see Appendix 4), however, more recent research has reported significant changes in the T-anxiety scale over time (Stanley, Novy, Bourland, Beck & Averill, 2001). Given that trait

anxiety is expected to remain stable over time, poor test-retest reliability of the T-anxiety scale suggests that this may not be a reliable measure. On the basis of these findings, caution is warranted in the interpretation of this measure.

4.2.4.3 State Trait Anger Expression Inventory-Second Edition

The State Trait Anger Expression Inventory – Second Edition (STAXI-2) is a self-administered test developed to provide an overall measure of the expression and control of anger, in particular, state and trait anger, and anger expression (Spielberger, 1999). The STAXI-2 consists of 57 items, with six scales, five subscales, and an Anger Expression Index (Spielberger, 1999). The State Anger Scale of 15 items required participants to rate the intensity of their anger at that very moment on four point scale ranging from *Not at all* to *Very much so*. The Trait Anger Scale of 10 items required the participants to rate how they generally feel on a four point scale ranging from *Almost never* to *Almost always*. Finally, the Anger Expression and Control Scale of 32 items, required participants to rate how they react when they get angry on a four point scale also ranging from *Almost never* to *Almost always* (Spielberger, 1999). The reliability and validity have been established (Bishop & Quah, 1998; & Spielberger, 1999), (see Appendix 4).

4.2.4.4 Neurobehavioural Rating Scale.

The Neurobehavioural Rating Scale (NRS), (Levin, High, Geothe, Sisson, Overall, et al, 1987) was developed to assess the neurobehavioural sequelae of brain injury (Spreen & Strauss, 1998). It consists of 27 items which are further divided into four factors including: 1) Cognitive/energy; 2) Metacognition; 3) Somatic/anxiety; and 4) Language. The participants and their significant others were required to rate the

presence or severity of neurobehavioural symptomatology on a seven point scale, ranging from *not present* to *extremely severe* (with the assistance of the examiner if needed). Levin et al (1987) demonstrated that subjects with differing levels of brain injury severity significantly differed on their performance of three factors: 1) Cognitive/energy; 2) Metacognition; and 3) Language. Therefore, this suggested that this measure distinguished well between differing injury severities, and would be a good measure of change in severity of neurobehavioural symptomatology following rehabilitation. Self-reported questionnaires, however, may not be an adequate source of information for this subject population (Sbordone, Seyranian, & Ruff, 1998). Consequently, the NRS was also given to a significant other (a partner, family member, or close friend) in addition to the clinician assisted participant's report.

4.2.5 Quantitative Electroencephalogram

The Quantitative Electroencephalogram (qEEG) hardware system used to measure brain wave activity was the Lexicore NeuroSearch-24 recording system. The Neurometric system (NxLink) developed by John, Prichep, and Easton (1988) was used to guide protocol choices during the EEG biofeedback training. The software used to conduct statistical analysis for each participant's pre-post treatment qEEGs, was the NeuroGuide database version 2.1.1., (Thatcher, 2005).

The qEEG was used to measure the changes in the electrophysiological dynamics of each participant's brain using a multichannel recording (19 scalp electrodes at standardised positions according to the International 10–20 system), with all scalp recordings referenced to linked ear lobes (see diagram in Appendix 4). Electrode impedances were maintained below 5 kilo-ohms (kΩ), and electrical conductance was assisted by electrolyte paste. If there was excessive eye or muscle artifact observed

during the 15 minute eyes-closed resting recording, the recording was paused until the EEG was relatively free from significant artifact, and then the recording re-commenced. All raw EEG data was artifacted and analysed by an experienced external EEG technician, who accepted 120 seconds of raw EEG for analysis in each condition. For each NeuroGuide analysis, split-half and test-retest reliabilities did not fall below 0.80.

Absolute power, relative power, symmetry, and coherence were computed in the following bandwidths: Delta: 0-4Hz, Theta: 4-8Hz, Alpha: 8-13Hz, Beta1: 13-32Hz, and Beta2: 32-64Hz. The qEEG software provided a statistical analysis whereby the participant's EEG was expressed as a deviation from the normative group in Z score units (standard deviations from the mean). The database allowed participant's EEG to be compared with a reference population. Pathology was assumed when the participant's EEG was one or more standard deviations from the mean (reference population). The EEG is represented in colour topographic maps (see example in Appendix 5). The reliability, sensitivity, specificity, and validity of the qEEG have been established (Thatcher, Walker, Gerson, & Geisler, 1989; Thatcher, Cantor, McAlaster, Geisler, & Krause, 1991; Kondacs & Szabo, 1999; & Thatcher, North, Curtin, Walker, Biver, et al, 2001), (see Appendix 4).

4.3 PROCEDURE

The study was a multiple single case, ABBA, cross over design. Given the significant variation of sequelae in the TBI population, it was difficult to acquire well matched groups of TBI participants. The difficulty in controlling for group differences in the TBI population was accounted for in this study through a multiple single subject design, whereby each individual served as their own control.

Participants with TBI underwent an initial assessment including a quantitative electroencephalograph (qEEG), a battery of neuropsychological tests (cognitive measures), emotional, and behavioural measures. The participants were then randomly assigned to commence in either one of the treatment groups, totalling 20 hours over a period of ten weeks (EEG biofeedback or cognitive rehabilitation). The brief battery of cognitive, emotional, and behavioural measures, and qEEG were then re-administered post treatment, measuring cognitive, emotional, behavioural, and brain wave pattern changes, respectively. Participants then commenced the second alternative treatment, also totalling 20 hours over ten weeks. Following this treatment period, participants were re-administered the brief battery of cognitive, emotional, behavioural, and qEEG measures to determine any changes in functioning. A final ten week follow-up assessment was then administered to determine any maintenance or loss of change following the cessation of treatments.

4.3.1 Electroencephalography (EEG) Biofeedback

The EEG biofeedback training consisted of twenty 60 minute sessions, occurring twice weekly over a ten week period. Individual protocols for EEG biofeedback were determined for each participant according to the qEEG results (highest z score deviations) and clinical presentation prior to commencement in this treatment. Given the available equipment, the EEG biofeedback trained abnormal Absolute and Relative power levels (as opposed to Asymmetry or Coherence) using a single scalp electrode placement, with a ground electrode on one ear and a reference electrode on the other. Through this operant conditioning procedure, the participants were taught to modify the electrophysiological dynamics of their brains. The biofeedback was provided through EEG Spectrum Neurocybernetics equipment.

4.3.2 Cognitive Rehabilitation

Cognitive rehabilitation consisted of twenty 60 minute sessions, occurring twice weekly over a ten week period. The cognitive rehabilitation was devised and constructed on principles outlined by Sohlberg and Mateer (2001). They indicated that the goals of cognitive rehabilitation may include improving cognitive and behavioural skills, compensating for cognitive and behavioural limitations, and assisting a client to understand and manage emotional reactions to changes in his or her functioning. It is described as an eclectic and holistic approach using a variety of techniques and strategies to improve abilities; to teach new and compensatory skills; to facilitate regulation of behaviour; and to modify negative or disruptive thoughts, feelings, and emotions (Sohlberg & Mateer, 2001), (for detailed outline, see Appendix 6).

Utilising Sohlberg and Mateer's principles, a general plan was established to facilitate in the delivery of cognitive rehabilitation, where each cognitive domain and emotional difficulty would be addressed over the ten week period (see Appendix 7). Given each participant presented with varying cognitive, emotional, and behavioural sequelae, the plan was modified accordingly, and tailored to suit the specific sequelae for each participant. Depending on each participant's cognitive, emotional, and behavioural presentation, the cognitive rehabilitation techniques used consisted of a variety of compensatory strategies, restorative strategies, and if emotional issues needed addressing, cognitive behavioural therapeutic techniques and behavioural management were used.

4.4 ASSESSMENT OF CHANGE FOLLOWING REHABILITATION

In order to determine the reliability of change in individual participants' test scores

following rehabilitation, measurement error and reliable change scores were reported where possible. Cotton, Crewther, and Crewther (2005) reported two types of error which can occur in a subject's observed test scores, including: systemic biases (e.g. practice effects) and random errors (e.g. incidental variation – changes in subjects' behaviour at each testing session). In order to address such error associated with test measurement, and determine the significance of change in a participant's observed score, the Standard Error of Measurement (SE_M), the Standard Error of Differences (SE_{DIFF}), and Standard Error of Prediction (SE_P) were applied. However, it is noted that such error measurements do not take into account practice effects (Basso et al, 1999; & Chelune, 2003).

The SE_M of a test provided confidence intervals (band of error) surrounding the subjects observed score (Chelune, 2003). If the subject's score fell outside the band of error it was less likely to reflect the effects of measurement error (McCaffrey, Duff, & Westervelt, 2000). The SE_M is computed with the following formula: $SE_M = SD\ (1 - r_{xx})^{1/2}$, where SD is the standard deviation of the test scores and r_{xx} represents the test's reliability coefficient (Chelune, 2003). The SE_{DIFF} is a method of calculating reliable change and is derived from the SE_M. Chelune (2003) reported that the SE_{DIFF} is the spread of the distribution of expected test-retest difference scores around a mean of zero (no difference). It is computed with the following formula: $SE_{DIFF} = (2\ (SE_M)^{\ 2})^{1/2}$, (Chelune, 2003). The SE_P is another method of calculating reliable change and represent the standard error of a retest score predicted from a baseline score in the following regression equation: $SE_P = SD_Y\ (1 - r_{12}^2)^{1/2}$, (Chelune, 2003). The SD_Y represents the standard deviation of scores during the second assessment interval, and r_{12} represents the correlation between test scores across the assessment intervals (Basso et al, 1999).

In the present study, in order to evaluate significant change in each participant, the previously described reliable change measures were implemented. However, calculations of estimating error and reliable change were not acquired from the present study's subject sample, and there was great difficulty in obtaining reliable change research findings for the TBI population. Therefore, calculations were acquired from studies which had computed these measurements from cognitive measures relevant to the current research, however, using normal populations (Basso et al, 1999; Harrison et al, 2000; Moritz et al, 2003; & Leark et al, 2004).

4.5 DATA ANALYSIS

Statistical Package For Social Sciences (Version 12) software was used to analyse the data. Histograms displaying descriptive statistics (means and standard deviations) and line graphs displaying the change in mean scores of each measure (pre-post treatments) were created to screen the results. These techniques assisted in the detection of outliers, non normality, and the heterogeneity of the sample. Non parametric tests were used as an alternative to parametric analysis given: **1)** the small sample size; **2)** the expected non-normality of distribution; **3)** that the distribution would change in nature over time; and **4)** transformation was deemed inappropriate: population characteristics would not be normal.

Quantitative EEGs were collected using Lexicor NeuroSearch-24 hardware. NeuroGuide software and NxLink software was used to analyse each participant's qEEG (absolute power) pre and post treatments. Inbuilt statistics within the NeuroGuide software allowed the application of parametric statistics by comparing both group qEEG data and individual qEEG data pre and post treatments. Using non-

parametric statistics, further evaluation of absolute power Z scores assisted in the determination of whether significant change was in the desired direction, towards the mean, indicative of EEG normalisation.

Chapter 5.

RESULTS

PART ONE

5.1 DATA SCREENING & ANALYSIS

During the data screening process, descriptive statistics detected a significant outlier. This outlier was significantly different from the remaining five participants, given he: had a moderate TBI; was only one year post injury; had limited education; and had an intellectual quotient within the Extremely Low range. The remaining five participants had endured severe TBIs (ranging from severe to extremely severe – determined according to PTA duration), were a number of years post injury (ranging from 2 to 47), had obtained a higher level of education, and their intellectual performance was significantly higher (Full Scale scores ranging from Low Average to High Average). Consequently, the outlier was removed from the final group analysis. All six individual participants' characteristics can be observed in Table 3. The group's (N=5) descriptive statistics (means and Standard deviations) can be observed in Table 4.

Table 4: *Individual Participants' characteristics.*

	Case 1	Case 2	Case 3*	Case 4	Case 5	Case 6
AGE	49.00	50.00	51.00	51.00	45.00	46.00
Years of education	10.00	8.00	4.00	12.00	10.00	12.00
Years since brain injury	22.00	47.00	1.40	2.00	23.00	5.00
Length of coma in DAYS	12.00	7.00	.00	1.00	42.00	.16
Length of PTA in DAYS	42.00	35.00	.16	21.00	38.00	3.00
Severity of brain injury	Extremely Severe	Extremely Severe	Moderate	Very Severe	Extremely Severe	Severe
Estimated pre-morbid IQ	109.00	98.94	91.14	113.76	101.28	99.72
Full Scale IQ	103.00	118.00	63.00	117.00	85.00	104.00
Verbal IQ	103.00	119.00	65.00	113.00	90.00	98.00
Performance IQ	102.00	113.00	67.00	121.00	80.00	113.00

NOTE:

* Outlier

Table 5: *Descriptive Statistics- Group Means and Standard Deviations.*

	N	Minimum	Maximum	Mean	Std. Deviation
AGE	5	45	51	48.2	2.58844
Years of education	5	8	12	10.4	1.67332
Years since brain injury	5	2	47	19.8	17.96385
Length of coma in DAYS	5	0.16	42	12.432	17.21375
Length of PTA in DAYS	5	3	42	27.8	15.95932
Severity of brain injury	5	4	6	5.4	0.89443
Estimated pre-morbid IQ	5	98.94	113.76	104.54	6.5215
Full Scale IQ	5	85	118	105.4	13.39029
Verbal IQ	5	90	119	104.6	11.58879
Performance IQ	5	80	121	105.8	15.92796

Prior to analysing any treatment effects, it was necessary to determine if the order of treatment affected the participants' performance on the neuropsychological, emotional, and behavioural measures administered. A non-parametric measure, the Mann-Whitney U test, was utilised to determine if any treatment order differences

were evident (2 participants received EEG biofeedback first and cognitive rehabilitation second, while 3 participants received the treatments in the reverse order). Where no treatment order effects were identified, a further non-parametric measure, the Wilcoxon test, was used to determine if any significant differences were evident between each treatment. The Bonferroni correction was then applied. As highlighted by Aron and Aron (1994) the Bonferroni procedure ensures a more stringent significance level for comparison so that the overall chance of any one of the comparisons being mistakenly significant is reasonably low.

The difference scores between pre-post assessments for each treatment were plotted in bar graphs for each measure. The trends of the difference scores between pre-post assessments for each treatment were analysed (the trend being defined as the tendency for one treatment to more consistently show larger difference scores pre-post assessment than the alternative treatment). Where possible, the standard error of measurement (SE_M), the standard error of differences (SE_{DIFF}), and/or the standard error of prediction (SE_P) were used to determine if any significant change in the participants performance post treatment was change likely to be associated with the rehabilitation, and not with measurement error.

Inbuilt parametric analyses (paired t-tests) were used to identify any significant change in the group and individual absolute power EEG data, pre and post treatments. Non-parametric analysis was not used given the parametric analysis was an inbuilt function of the qEEG software. The qEEG paired t-test results obtained from this software only supplied the p-values. Therefore, degrees of freedom and t-values were not reported in the present results. The qEEG analysis included further evaluation of the absolute power Z scores. The Z scores were used to determine if any statistically

significant change was in the desired direction (towards the mean), hence, suggestive of the normalisation of brain electrophysiology. Non-parametric analysis (Chi-square test) of the number of statistically significant Z score changes was use to determine if there was a significant difference between each treatment.

Given the significant variation within the TBI population of cognitive, emotional, and behavioural symptomatology, and cerebral electrophysiology, results were also explored for each individual participant.

5.2 INDIVIDUAL PARTICIPANT RESULTS

All participants were randomly assigned to complete each treatment, either commencing in EEG biofeedback (Treatment A) or Cognitive rehabilitation (Treatment B). They underwent neuropsychological, cognitive, emotional, behavioural, and qEEG assessment before and after each treatment, and at a ten week follow-up. Although no formal assessment was administered to measure functional changes, each participant's self reported change was collected. All participants attended appointments regularly. No serious psychological and/or neurological conditions were reported prior to their TBI. With the exception of occasional pain and nausea management medication use by Participant Three, all participants were not regularly taking any medications or receiving any other treatments during their participation in the present study.

5.2.1 PARTICIPANT ONE (P1): AB Design

Participant One (P1) was a 49 year old male, who presented as a very friendly, keen, and motivated man. An excellent level of rapport was established and maintained throughout the course of the program. He was 22 years post extremely severe TBI as a result of a motor vehicle accident (see Table 4). He reported orthopaedic injuries at the time of his accident, which included damage to the right eye causing impaired and double vision. He further reported anger management issues during the early years following his TBI, and ongoing depression and anxiety. Prior to his TBI he had completed 10 years of education, and owned / managed his own business. Following the TBI he maintained causal employment.

Participant One was randomly assigned to commence in the EEG biofeedback program first, followed by cognitive rehabilitation. Prior to commencement he was

estimated to have a pre-morbid IQ within the average range. On assessment, his Full Scale, Verbal and Performance IQ, were within the average range, consistent with estimated pre-morbid IQ. For participant one that fatigue was an issue during assessment sessions. He particularly reported being extremely fatigued during the post EEG Biofeedback (second) assessment.

5.2.1.1 EEG Biofeedback Program

Prior to EEG biofeedback, Participant One's qEEG topographic map indicated an overall reduction in absolute power for all frequency bands. An elevation in Theta relative power (across entire cortex, more prominent anteriorly), a less prevalent elevation (not greater than one standard deviation from the mean) in Delta and Beta relative power, and a reduction (not greater than one standard deviation from the mean) in Alpha relative power were observed. On the basis of these qEEG findings the EEG biofeedback protocols included: **1)** Fz: Inhibited Theta (4-7 Hz), rewarded Sensory Motor Rhythm (low Beta – 12-14 Hz), and inhibited Muscle activity/tension (see Appendix 9 for qEEG topographic map).

5.2.1.2 Cognitive Rehabilitation Program

Just prior to the commencement of cognitive rehabilitation, Participant One described a number of cognitive difficulties with memory, concentration/distractibility, planning, and organisation which were impacting on his functional capacity in every day life. Consequently, the cognitive rehabilitation plan consisted of various compensatory strategies which were collaboratively devised and employed to assist with his described difficulties. He did not report any emotional or behavioural difficulties at this time. Therefore, they were not addressed in this part of the

treatment program.

A number of compensatory strategies were implemented throughout the course of cognitive rehabilitation. Such strategies as diary training, utilising a kitchen whiteboard, and using a notepad were devised to assist with memory, planning and organisational difficulties. Further strategies were employed to assist in the effective monitoring and use of the diary and white board. Strategies included: making diary entries three times a day (associated with meal times); checking and revising the white board at the end of the day (associated with last diary check); and having a completion box on the white board which he would tick off once a task was complete. In order to prevent losing personal items around the house, a particular (special) place in the kitchen was designated where he would have to consistently return his keys, wallet, diary and other important personal items while at home.

Participant One's mobile phone alarm was used to assist in keeping him on task (e.g. prevent hours surfing on internet and not completing planned prioritised tasks). Upon commencement of an activity, he was required to set his alarm to alert himself when the allocated time for that activity was over. He could then move on to the next activity. It was also set to remind him to leave home for appointments.

5.2.1.3 Formal Neuropsychological Assessment Results

The trends of the results, as observed in the graphs (see Appendix 10), were analysed as described below. Despite increased fatigue during assessment, Participant One made greater improvements in attentional functioning following EEG biofeedback as compared to cognitive rehabilitation. Significant reductions can be observed in all measures of the T.O.V.A (omission errors, commission errors, response time, and

variability) following EEG biofeedback, with only one significant improvement in variability following cognitive rehabilitation. Improvement in performance on the PASAT is also noted only following EEG biofeedback. He maintained all significant improvements in attentional functioning at the ten week follow-up assessment.

With respect to memory, most improvement was observed following cognitive rehabilitation. A significant improvement in verbal memory (RAVLT – total score) performance was noted following cognitive rehabilitation. Small improvements in verbal delayed recall and verbal recognition were also noted following cognitive rehabilitation and not EEG biofeedback. However, decreases in recognition memory would not normally be expected. On further examination of the results, it is possible that fatigue levels may have impacted on concentration were likely to have influenced his performance. No improvements were noted in visual recall following either therapy. Participant one did not maintain his improved performance in memory functioning at the ten week follow-up assessment.

Participant One made improvements in speed of information processing tasks following EEG biofeedback, but not cognitive rehabilitation. Following EEG biofeedback, his performance on symbol search and response time (T.O.V.A.) significantly improved, and his performance on language and comprehension processing speed also improved. He maintained improvements only on the response time of the T.O.V.A. at the ten week follow-up assessment.

In tests of executive functioning, Participant One showed improvements only following EEG biofeedback. Improved performance following EEG biofeedback was noted in COWAT (both FAS and animals) and in both Trails A and Trails B.

However, significance was not reached in FAS. Participant one continued to maintain his improved performance in all measures at the ten week follow-up assessment, reaching significance in FAS and Trails A measures.

5.2.1.4 Formal Emotional and Behavioural Assessment Results

Participant One reported reductions in depression and anxiety symptomatology following both EEG biofeedback and cognitive rehabilitation. However, much larger reductions were reported following EEG biofeedback. Reductions were continued to be reported at the ten week follow-up assessment. Comparable reductions in anger symptomatology were reported following both treatments, however little change was maintained at the ten week follow-up assessment. Furthermore, P1 reported great reductions in overall symptomatology reporting on the Neurobehavioural Rating Scale (NRS), but only following EEG biofeedback. This was maintained at the ten week follow-up assessment. However, his significant other (friend) reported an increase in symptomatology on the NRS following EEG biofeedback, no change at all following cognitive rehabilitation, and significant increase at ten week follow-up assessment. The reliability of the significant other report is questionable. The significant other was becoming disgruntled at having to complete the same questions on multiple occasions. On the final occasion it took some persuasion for her to complete the NRS.

5.2.1.5 Self-Reported Functional Changes

Participant One reported a number of functional changes during the course of each treatment program. During the EEG biofeedback program P1 reported that he felt his concentration had improved and that he generally felt better in every day life.

Functionally, at the end of the ten week cognitive rehabilitation program, P1 was independently using each strategy implemented. He reported being able to better manage his cognitive difficulties by using these strategies.

5.2.1.6 Quantitative Electroencephalogram Results

5.2.1.6.1 Post EEG biofeedback

Following 20 sessions of EEG biofeedback paired t-tests revealed a number of significant changes in the absolute power of all frequency bands. Significant changes (P-values) in absolute power can be observed in topographic maps (Figure 2). The P-values can be observed numerically in Table 6, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

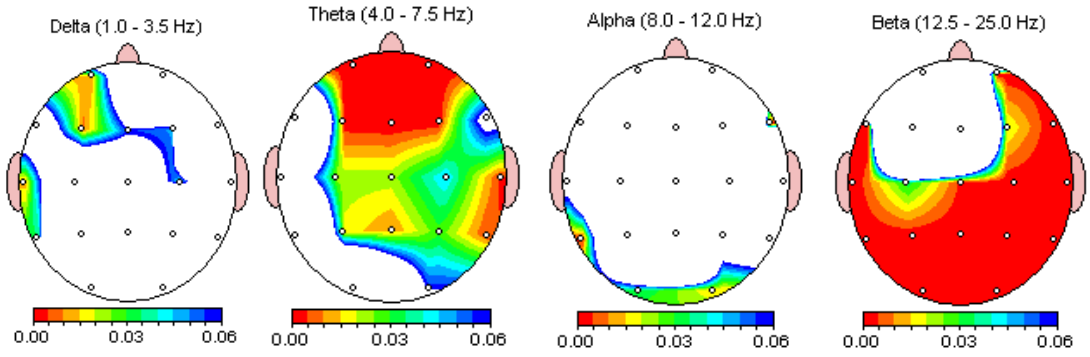

Figure 2: *Participant one- Topographic maps: Statistically Significant change (P-values) in absolute power following EEG biofeedback*

Table 6: *P1 - Statistically Significant change (P-Values) in absolute power following EEG biofeedback*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA	
FP1	0.014	0.002	0.163	0.689	FP2
F3	0.008	0.001	0.781	0.754	F4
C3	0.167	0.023	0.313	0.033	C4
P3	0.409	0.019	0.247	0.000	P4
O1	0.644	0.190	0.035	0.000	O2
F7	0.107	0.242	0.431	0.000	F8
T3	0.012	0.103	0.082	0.000	T4
T5	0.034	0.231	0.003	0.000	T6

Intrahemispheric: RIGHT

DELTA	THETA	ALPHA	BETA
0.246	0.000	0.153	0.001
0.049	0.001	0.978	0.021
0.041	0.045	0.886	0.001
0.588	0.028	0.100	0.000
0.626	0.057	0.015	0.000
0.736	0.073	0.000	0.000
0.128	0.004	0.794	0.000
0.552	0.006	0.129	0.000

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.056	0.001	0.788	0.423
Cz	0.131	0.023	0.196	0.004
Pz	0.159	0.010	0.631	0.000

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 7 demonstrates significant changes in the absolute power. Sixteen of the 19 sites progressed towards the mean in the absolute power of Delta, with six sites reaching statistical significance. Progression towards the mean can be observed at all sites across the cortex for the absolute power of Theta, however four of the 19 sites were not significant. Movement towards the mean can be observed in eight of the 19 sites for the absolute power of Alpha, however statistical significance was not reached. Significant movement away from the mean was observed at four sites for Alpha. The absolute power of Beta demonstrates a statistically significant change approaching the mean at only one site, and significant change away from the mean at 15 of the 19 sites.

Table 7: *P1 - Change in Z scores for Delta, Theta, Alpha and Beta pre & post EEG biofeedback.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-1.27	-0.93**	-1.83	-1.40**	-1.60	-1.64	-1.21	-1.26
Fp2	-1.28	-1.04	-1.31	-1.04**	-1.32	-1.34	-0.77	-0.89*
F3	-2.17	-1.78**	-2.39	-1.94**	-1.85	-1.76	-1.41	-1.40
F4	-2.14	-1.90**	-2.46	-1.90**	-1.73	-1.66	-1.21	-1.36*
C3	-2.23	-1.96	-2.58	-2.21**	-2.11	-1.98	-1.25	-1.42*
C4	-2.22	-2.11**	-2.50	-2.11**	-1.79	-1.76	-1.20	-1.43*
P3	-2.25	-2.08	-2.38	-2.05**	-1.77	-1.80	-1.29	-1.53*
P4	-1.97	-2.05	-2.38	-2.05**	-1.55	-1.61	-1.23	-1.46*
O1	-1.82	-1.96	-1.97	-1.77	-1.76	-1.89*	-1.59	-1.92*
O2	-1.76	-1.51	-1.83	-1.51**	-1.61	-1.75*	-1.45	-1.80*
F7	-1.15	-0.84	-1.53	-1.46	-1.59	-1.61	-0.95	-0.63**
F8	-1.13	-1.31	-1.58	-1.31**	-1.17	-1.38*	-0.06	-0.41*
T3	-2.00	-1.55**	-1.91	-1.79	-1.91	-2.02	-0.92	-1.39*
T4	-1.89	-1.75	-2.21	-1.75**	-1.81	-1.75	-1.15	-1.42*
T5	-2.07	-1.84**	-1.72	-1.58	-1.52	-1.77*	-0.60	-1.68*
T6	-1.92	-1.63	-1.94	-1.63**	-1.57	-1.64	-1.45	-1.73*
Fz	-1.89	-1.87	-2.37	-1.87**	-1.73	-1.64	-1.25	-1.32
Cz	-2.43	-2.48	-2.94	-2.48**	-2.05	-1.93	-1.23	-1.46*
Pz	-2.37	-2.29	-2.68	-2.29**	-1.85	-1.83	-1.43	-1.69*

NOTE: *Change towards '0' representing a normalisation in EEG.*

*** Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.1.6.2 Post Cognitive Rehabilitation

Following 20 sessions of Cognitive Rehabilitation paired t-tests revealed significant changes in the absolute power of Delta, Theta, Alpha and Beta. Significant changes in absolute power can be observed in topographic maps (Figure 3). The P-values can be observed numerically in Table 8, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

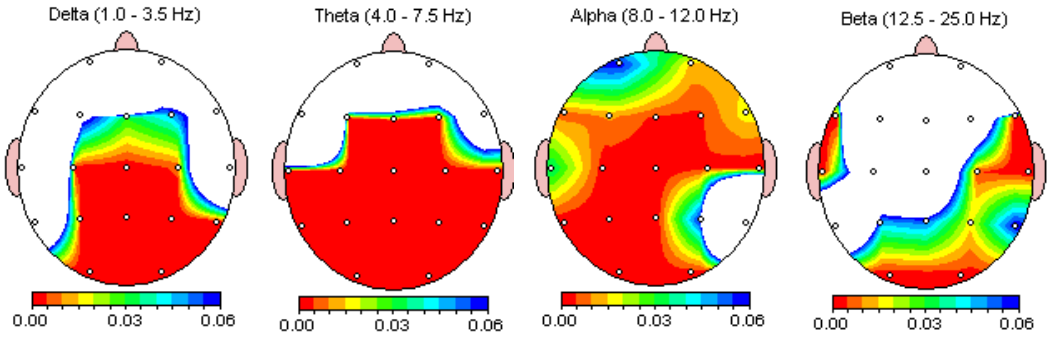

Figure 3: *P1 - Topographic maps - Statistically Significant change (P-values) in absolute power following Cognitive Rehabilitation*

Table 8: *P1 - Statistically Significant change (P-Values) in absolute power following Cognitive Rehabilitation*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA
FP1	0.882	0.458	0.058	0.134
F3	0.070	0.003	0.011	0.638
C3	0.002	0.000	0.001	0.117
P3	0.001	0.000	0.000	0.047
O1	0.002	0.000	0.000	0.000
F7	0.394	0.765	0.007	0.000
T3	0.498	0.000	0.035	0.002
T5	0.177	0.000	0.000	0.174

Intrahemispheric: RIGHT

	DELTA	THETA	ALPHA	BETA
FP2	0.143	0.255	0.014	0.743
F4	0.046	0.000	0.004	0.136
C4	0.006	0.000	0.004	0.005
P4	0.000	0.000	0.060	0.017
O2	0.000	0.000	0.003	0.003
F8	0.085	0.157	0.018	0.000
T4	0.247	0.001	0.000	0.000
T6	0.000	0.000	0.462	0.060

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.030	0.002	0.003	0.123
Cz	0.000	0.000	0.000	0.171
Pz	0.001	0.000	0.000	0.054

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 9 demonstrates significant changes in the absolute power of each frequency band. Progression towards the mean can be observed in 17 of the 19 sites in the absolute power of Delta, with 11 of these sites reaching statistical significance. Movement towards the mean can be observed at 18 of the 19 sites for the absolute power of Theta, with 15 sites reaching statistical significance. Similarly, 18 of the 19 sites

progressed towards the mean for the absolute power of Alpha, with 15 statistically significant, and at one site statistically significant change away from the mean can be observed. Seventeen of the 19 sites have changed towards the mean for the absolute power of Beta, with 11 reaching statistical significance, and one site changed away from the mean.

Table 9: *P1 - Change in Z scores for Delta, Theta, Alpha and Beta pre & post Cognitive Rehabilitation.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-0.70	-0.73	-1.17	-1.29	-1.55	-1.43	-1.11	-1.22
Fp2	-1.24	-1.10	-1.13	-1.02	-1.34	-1.17**	-0.88	-0.85
F3	-1.73	-1.27	-1.82	-1.39**	-1.59	-1.35**	-1.11	-1.05
F4	-1.89	-1.36**	-2.11	-1.54**	-1.67	-1.39**	-1.29	-1.10
C3	-1.96	-1.35**	-2.14	-1.42**	-1.71	-1.31**	-1.11	-0.93
C4	-1.88	-1.26**	-2.21	-1.63**	-1.66	-1.45**	-1.42	-1.11**
P3	-2.13	-1.47**	-1.97	-1.14**	-1.45	-1.02**	-1.31	-1.13**
P4	-1.97	-1.20**	-2.12	-1.40**	-1.31	-1.13	-1.45	-1.24**
O1	-2.13	-1.51**	-1.68	-0.94**	-1.67	-1.21**	-1.92	-1.28**
O2	-2.13	-1.14**	-1.61	-0.81**	-1.52	-1.11**	-1.88	-1.54**
F7	-0.99	-1.17	-1.24	-1.23	-1.24	-1.52*	-0.10	-1.13*
F8	-1.06	-0.77	-1.41	-1.26	-1.46	-1.19**	-0.83	-0.49**
T3	-1.65	-1.42	-1.69	-1.26**	-1.68	-1.46**	-1.19	-0.87**
T4	-1.56	-1.37	-1.99	-1.58**	-1.89	-1.60**	-1.71	-1.25**
T5	-1.79	-1.46	-1.49	-0.90**	-1.49	-1.01**	-1.42	-1.28
T6	-2.05	-1.38**	-1.70	-1.22**	-1.50	-1.42	-1.91	-1.79
Fz	-1.58	-1.06**	-1.82	-1.35**	-1.54	-1.30**	-1.19	-1.00**
Cz	-1.99	-1.13**	-2.38	-1.53**	-1.71	-1.45**	-1.18	-1.02**
Pz	-2.10	-1.36**	-2.21	-1.37**	-1.49	-1.18**	-1.46	-1.31**

NOTE: *Change towards '0' representing a normalisation in EEG.*

** *Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.1.6.3 Final Follow-up Assessment

From the commencement of the program (initial assessment-prior to first treatment) to the final follow-up assessment, paired t-tests revealed some significant changes in the absolute power of Delta, Theta, Alpha and Beta. Significant changes in absolute

power can be observed in topographic maps (Figure 4). The P-values can be observed numerically in Table 10, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

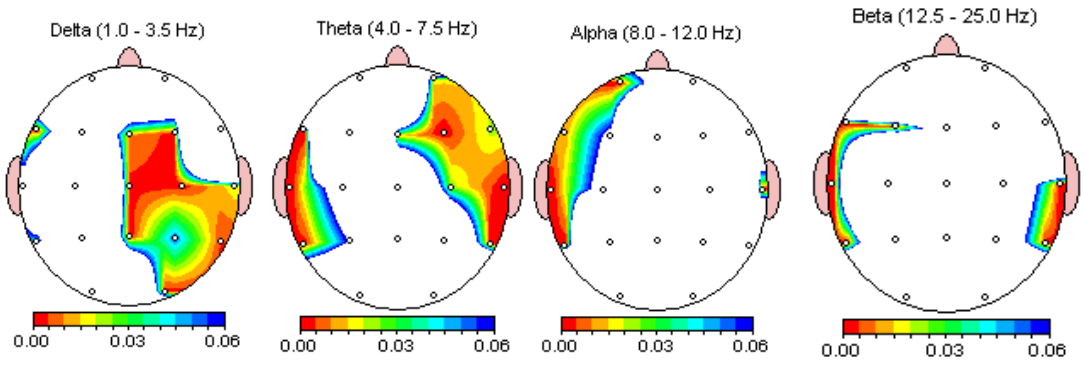

Figure 4: *P1 - Topographic maps - Statistically Significant change (P-values) in absolute power between initial and final Assessment*

Table 10: *P1 - Statistically Significant change (P-Values) in absolute power between initial and final Assessment*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA	
FP1	0.254	0.751	0.001	0.837	FP2
F3	0.196	0.808	0.072	0.002	F4
C3	0.535	0.097	0.083	0.609	C4
P3	0.314	0.064	0.687	0.252	P4
O1	0.484	0.183	0.994	0.419	O2
F7	0.003	0.000	0.013	0.000	F8
T3	0.089	0.000	0.000	0.000	T4
T5	0.043	0.002	0.001	0.000	T6

Intrahemispheric: RIGHT

DELTA	THETA	ALPHA	BETA
0.216	0.016	0.184	0.476
0.003	0.000	0.778	0.108
0.002	0.023	0.970	0.156
0.051	0.218	0.679	0.142
0.002	0.419	0.816	0.331
0.655	0.021	0.174	0.722
0.018	0.000	0.002	0.001
0.005	0.002	0.418	0.003

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.010	0.017	0.549	0.110
Cz	0.003	0.223	0.871	0.589
Pz	0.000	0.909	0.564	0.527

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 11 demonstrates significant changes in the absolute power of each frequency band.

Progression towards the mean can be observed in 14 of the 19 sites for the absolute power of Delta, with 7 of these sites reaching statistical significance. Two sites demonstrated significant change away from the mean. Movement towards the mean can be observed at 12 of the 19 sites for the absolute power of Theta, with 6 sites reaching statistical significance. Three sites demonstrated significant change away from the mean. Seven of the 19 sites progressed towards the mean for the absolute power of Alpha, with only one site reaching statistically significance, and four sites showed statistically significant change away from the mean. Thirteen of the 19 sites changed towards the mean for the absolute power of Beta, with four reaching statistical significance, and two sites significantly changed away from the mean.

Table 11: *P1 - Change in Z scores for Delta, Theta, Alpha and Beta between initial and final Assessment.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-1.27	-1.55	-1.83	-1.79	-1.60	-1.77*	-1.21	-1.19
Fp2	-1.28	-1.22	-1.31	-1.19**	-1.32	-1.36	-0.77	-0.75
F3	-2.17	-2.09	-2.39	-2.40	-1.85	-1.90	-1.41	-1.17**
F4	-2.14	-1.70**	-2.46	-2.02**	-1.73	-1.68	-1.21	-1.06
C3	-2.23	-2.13	-2.58	-2.74	-2.11	-2.18	-1.25	-1.29
C4	-2.22	-1.61**	-2.50	-2.15**	-1.79	-1.78	-1.20	-1.07
P3	-2.25	-2.02	-2.38	-2.51	-1.77	-1.76	-1.29	-1.40
P4	-1.97	-1.58	-2.38	-2.13	-1.55	-1.56	-1.23	-1.16
O1	-1.82	-1.69	-1.97	-2.07	-1.76	-1.72	-1.59	-1.63
O2	-1.76	-1.31**	-1.83	-1.63	-1.61	-1.55	-1.45	-1.39
F7	-1.15	-1.75*	-1.53	-2.09*	-1.59	-1.67*	-0.95	-0.32**
F8	-1.13	-1.17	-1.58	-1.28**	-1.17	-1.26	-0.06	-0.03
T3	-2.00	-2.26	-1.91	-2.63*	-1.91	-2.37*	-0.92	-1.64*
T4	-1.89	-1.49**	-2.21	-1.51**	-1.81	-1.55**	-1.15	-0.93**
T5	-2.07	-2.34*	-1.72	-2.05*	-1.52	-1.73*	-0.60	-1.49*
T6	-1.92	-1.41**	-1.94	-1.54**	-1.57	-1.48	-1.45	-1.27**
Fz	-1.89	-1.66**	-2.37	-2.11**	-1.73	-1.75	-1.25	-1.14
Cz	-2.43	-1.81**	-2.94	-2.71	-2.05	-2.05	-1.23	-1.22
Pz	-2.37	-1.66**	-2.68	-2.56	-1.85	-1.87	-1.43	-1.43

NOTE: *Change towards '0' representing a normalisation in EEG.*

*** Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.1.7 Results Summary of Participant One

Although increased fatigue was reported by Participant One, formal testing revealed improvements in attention, speed of information processing, and executive functioning following EEG biofeedback, but not cognitive rehabilitation. He maintained improvements in all of these domains at a ten week follow-up assessment. He demonstrated improvements in memory functioning following cognitive rehabilitation, and not EEG biofeedback. However, he failed to maintain this improvement at the ten week follow-up assessment. Improvements in depression, anxiety, anger, and overall neurobehavioural symptomatology were reported by P1 following both treatments. However, larger reductions in self reported depression, anxiety, and neurobehavioural symptomatology followed EEG biofeedback. These were maintained at the ten week follow-up. Conversely, the significant other's report on neurobehavioural symptomatology was not consistent with the subject's self reports. This was likely due to difficulty in maintaining co-operation with the significant other. Everyday functional changes were reported by P1 following both treatments. Participant One made a number of significant changes towards normalisation in his EEG following EEG biofeedback (particularly in the frequencies trained: Delta – Theta). Significant changes towards normalisation continued across a greater number of sites and frequencies following cognitive rehabilitation. He continued to show some significant change towards normalisation at the ten week follow-up assessment.

5.2.2 PARTICIPANT TWO (P2): AB design

Born in Malta, Participant Two (P2) was a 50 year old male, who presented as a very friendly, jovial, and boisterous man. Excellent rapport was established and maintained throughout the course of the program. He was 47 years post extremely severe TBI as a result of a fall (see Table 4). His family reported orthopaedic injuries (broken bones) at the time of his accident. He further reported years of ongoing anxiety and anger management issues following his TBI (throughout childhood, adolescence, and adulthood). He has completed eight years of education and following the TBI has maintained full-time employment.

Participant Two was randomly assigned to commence in the EEG biofeedback program first, followed by cognitive rehabilitation. He obtained an estimated IQ within the average range on a formal reading test. On neuropsychological assessment, however, he demonstrated a high average performance on Full Scale, Verbal, and Performance IQ.

5.2.2.1 EEG Biofeedback Program

Prior to EEG biofeedback, Participant Two's qEEG topographic maps indicated an overall reduction in absolute power for all frequency bands. An elevation in Alpha relative power (across entire cortex, more prominent anteriorly) and a reduction (not greater than one standard deviation from the mean) in Theta and Beta relative power were observed. An increase in right posterior (P4) power asymmetry was observed within the Delta and Theta range. On the basis of these qEEG findings the EEG biofeedback protocols included: **1)** P4: Inhibited Delta and Theta (2-7 Hz), rewarded Sensory Motor Rhythm (low Beta – 12-14 Hz), and inhibited Muscle activity/tension

(see Appendix 9 for qEEG topographic map).

5.2.2.2 Cognitive Rehabilitation Program

Participant Two described a number of cognitive (attentional, memory, planning, and organisation), emotional (anxiety), and behavioural (anger) difficulties, which were impacting on his functional capacity in every day life. Consequently, the cognitive rehabilitation plan consisted of cognitive behavioural techniques and various external and internal compensatory strategies, which were employed to assist with his described difficulties.

Cognitive behavioural strategies were employed to assist with Participant Two's difficulties with anxiety and aggression. These issues were addressed first as it was likely they were having an impact on his cognitive function. Relaxation and breathing strategies were taught, and practiced regularly outside of rehabilitation sessions with the assistance of a tape recorded session. In addition, a number of anger management strategies were implemented including: substituting the behaviour with a different behaviour; delaying tactics (e.g. counting to ten); and anticipatory avoidance strategies (identifying the antecedent to an aggravating situation and learning to avoid putting himself in such a situation). In order to identify the antecedents, P2 maintained a journal, whereby he recorded all situations which aggravated him and how he responded to the situation (verbal and/or physical aggression). Participant Two's perception of situations which made him angry was also addressed and challenged. In this way, he had opportunities to attempt to perceive or think about aggravating situations differently.

Additionally, a number of compensatory strategies were implemented throughout the

course of cognitive rehabilitation to address attentional, memory, planning, and organisation difficulties. Such strategies as writing up a step-by-step plan with time lines, and using a notepad to create lists were devised to assist with planning and organisational difficulties. In order to further assist in preventing losing personal items around the house, a particular (special) place was selected where he would have to consistently return his keys, wallet, and other important personal items while at home. Internal compensatory strategies (e.g. practicing visual and/or auditory cues, and practicing a person's name) were implemented to assist with recalling a person's name and word finding. Self-instruction strategies were devised to assist with redirecting his concentration when in conversation and while driving. In addition to this, a bright symbol (figurine) was placed in his car within his visual field as a reminder to pay attention while driving. Furthermore, to better manage his distractibility, all auditory and visual distractions were expected to be minimised when he was performing an activity requiring concentration.

5.2.2.3 Formal Neuropsychological Assessment Results

The trends of the results, as observed in the graphs (see Appendix 10), were analysed as described below. Participant Two made variable improvements in attentional functioning following each treatment. Significant reductions, that is, improvements in performance, can be observed in the T.O.V.A (commission errors) following EEG biofeedback, and in the T.O.V.A. (response time) following cognitive rehabilitation. Improvement in performance on the PASAT is noted following both treatments, however greater improvement followed EEG biofeedback. Improved performance was only maintained in the T.O.V.A. commission errors and the PASAT at the ten week follow-up assessment.

Similar improvements were observed with verbal and visual memory following both treatments. Equal improvement in verbal and visual delayed recall was observed following both treatments. Non-significant improvement in verbal memory (RAVLT – total score) performance was noted following EEG biofeedback compared to cognitive rehabilitation. At the ten week follow-up assessment P2 maintained his improved performance to some degree on the RAVLT (total recall) and his performance on delayed visual recall made further gains.

On measures of speed of information processing, P2 showed consistent improvements in performance following cognitive rehabilitation. Following EEG biofeedback, his performance on language and comprehension processing speed greatly improved compared to cognitive rehabilitation. Participant Two's performance in language and comprehension processing speed made further gains at the ten week follow-up assessment.

In tests of executive functioning, P2 showed significant variability. His performance on COWAT (FAS) significantly declined following EEG biofeedback, with no significant changes evident following each treatment on COWAT (animals) and Trails A. However, his performance on Trails B following EEG biofeedback significantly improved. No improvement in the performance on Trails B post cognitive rehabilitation was evident, but P2 made further gains in his performance on Trails B at the ten week follow-up assessment.

5.2.2.4 Formal Emotional and Behavioural Assessment Results

Participant Two reported reductions in depression and anxiety symptomatology following both EEG biofeedback and cognitive rehabilitation. However, much larger

reductions in symptoms were reported following EEG biofeedback. Reductions continued to be reported at the ten week follow-up assessment. Comparable reductions in anger symptomatology were reported following both treatments. With the exception of a continued reduction in trait anger, other anger symptomatology returned to pre-treatment levels at the ten week follow-up assessment. Participant Two and his significant other (wife) both reported large reductions in overall symptom reporting on the Neurobehavioural Rating Scale (NRS), but only following EEG biofeedback. No improvement was reported following cognitive rehabilitation. At the ten week follow-up assessment, participant two indicated that the reductions in symptom reporting were maintained, while his significant other reported further gains.

5.2.2.5 Self-Reported Functional Changes

Participant Two reported a number of functional changes during the course of each treatment program. During the EEG biofeedback program, P2 indicated that he was feeling more alert, quicker in his thoughts, and was less forgetful around the house. Furthermore, he reported a reduction in the frequency and intensity of aggression in heated situations, and felt a general sense of calm. His significant other confirmed these reports. By the end of the ten week cognitive rehabilitation program, P2 was independently using most strategies implemented. Changes he indicated as important were: greater concentration while driving his truck at work, continued practice of breathing and relaxation strategies, and better control over his behavioural response to anger provoking situations.

5.2.2.6 Quantitative Electroencephalogram Results

5.2.2.6.1 Post EEG biofeedback

Following 20 sessions of EEG biofeedback paired t-tests revealed a number of significant changes in the absolute power of all frequency bands. Significant changes (P-values) in absolute power can be observed in topographic maps (Figure 5). The P-values can be observed numerically in Table 12, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

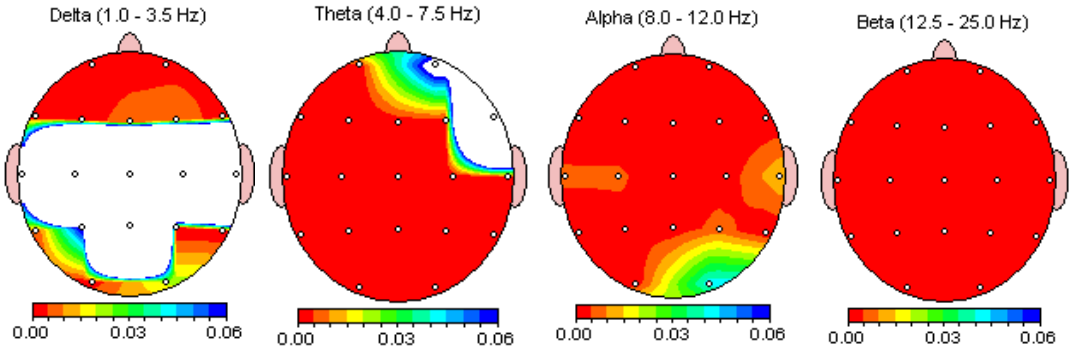

Figure 5: *P2 - Topographic maps: Statistically Significant change (P-values) in absolute power following EEG biofeedback.*

Table 12: *P2 - Statistically Significant change (P-Values) in absolute power following EEG biofeedback*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA
FP1	0.000	0.002	0.001	0.000
F3	0.001	0.000	0.001	0.000
C3	0.681	0.000	0.006	0.000
P3	0.065	0.000	0.003	0.000
O1	0.002	0.000	0.002	0.000
F7	0.000	0.000	0.000	0.000
T3	0.114	0.000	0.006	0.000
T5	0.006	0.001	0.001	0.000

Intrahemispheric: RIGHT

	DELTA	THETA	ALPHA	BETA
FP2	0.002	0.073	0.001	0.000
F4	0.010	0.000	0.001	0.000
C4	0.584	0.000	0.001	0.000
P4	0.000	0.000	0.008	0.000
O2	0.022	0.000	0.044	0.000
F8	0.002	0.391	0.001	0.000
T4	0.626	0.005	0.014	0.000
T6	0.000	0.000	0.001	0.000

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.009	0.000	0.002	0.000
Cz	0.801	0.000	0.001	0.000
Pz	0.922	0.000	0.001	0.000

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 13 demonstrates significant changes in the absolute power. Eighteen of the 19 sites progressed towards the mean for the absolute power of Delta, with 11 sites reaching statistical significance, and in one site significant movement away from the mean could be observed. Progression towards the mean can be observed at all sites across the cortex for the absolute power of Theta, with 17 of the 19 sites reaching statistical significance. Statistically significant movement towards the mean can be observed in all of the 19 sites for the absolute power of Alpha and Beta.

Table 13: *P2 - Change in Z scores for Delta, Theta, Alpha and Beta pre & post EEG biofeedback.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-2.74	-1.97**	-2.95	-2.38**	-1.01	-0.60**	-1.58	-1.13**
Fp2	-1.58	-1.26**	-1.50	-1.37	-0.80	-0.57**	-0.94	-0.71**
F3	-2.81	-1.85**	-3.18	-2.28**	-0.99	-0.58**	-1.43	-0.80**
F4	-3.01	-2.20**	-3.27	-2.55**	-0.87	-0.47**	-1.35	-0.58**
C3	-2.06	-1.55	-2.78	-2.03**	-1.06	-0.76**	-1.19	-0.65**
C4	-2.35	-1.88	-2.85	-2.08**	-0.89	-0.52**	-1.15	-0.27**
P3	-2.47	-1.84	-2.91	-2.12**	-1.31	-1.01**	-1.47	-0.89**
P4	-1.49	-1.92*	-2.56	-1.84**	-1.02	-0.70**	-1.14	-0.59**
O1	-2.15	-1.39**	-2.70	-1.79**	-1.36	-1.11**	-1.91	-1.35**
O2	-1.88	-1.17**	-2.05	-1.34**	-1.12	-0.94**	-1.59	-1.04**
F7	-2.77	-1.64**	-2.56	-1.75**	-0.92	-0.49**	-1.40	-0.98**
F8	-2.14	-1.57**	-1.82	-1.67	-0.55	-0.24**	-0.75	-0.12**
T3	-2.19	-1.64	-2.35	-1.76**	-1.09	-0.77**	-1.42	-1.04**
T4	-1.93	-1.80	-1.59	-1.24**	-0.62	-0.33**	-0.84	-0.30**
T5	-2.06	-1.39**	-2.48	-1.94**	-1.48	-1.20**	-1.79	-1.24**
T6	-1.83	-1.08**	-1.74	-1.22**	-1.01	-0.71**	-1.17	-0.63**
Fz	-2.47	-1.78**	-3.17	-2.42**	-0.95	-0.58**	-1.30	-0.69**
Cz	-2.30	-1.78	-3.24	-2.23**	-1.04	-0.66**	-1.22	-0.49**
Pz	-2.59	-2.12	-3.10	-2.18**	-1.24	-0.93**	-1.54	-0.90**

NOTE: *Change towards '0' representing a normalisation in EEG.*

*** Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.2.6.2 Post Cognitive Rehabilitation

Following 20 sessions of cognitive rehabilitation paired t-tests revealed some areas of

significant change in the absolute power of Delta, Alpha and Beta. Significant changes in absolute power can be observed in topographic maps (Figure 6). The P-values can be observed numerically in Table 14, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

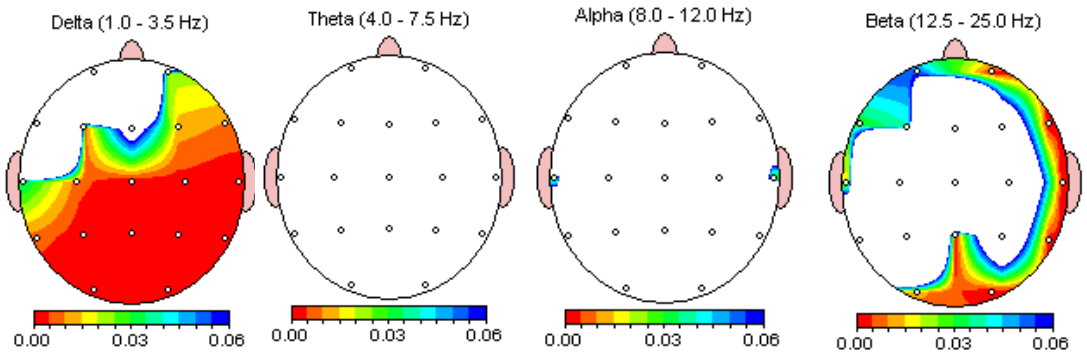

Figure 6: *P2 - Topographic maps - Statistically Significant change (P-values) in absolute power following Cognitive Rehabilitation*

Table 14: *P2 - Statistically Significant change (P-Values) in absolute power following Cognitive Rehabilitation*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA
FP1	0.650	0.196	0.533	0.054
F3	0.009	0.719	0.451	0.039
C3	0.007	0.708	0.594	0.964
P3	0.001	0.743	0.149	0.555
O1	0.000	0.098	0.191	0.012
F7	0.840	0.413	0.805	0.031
T3	0.036	0.457	0.026	0.018
T5	0.007	0.547	0.251	0.230

Intrahemispheric: RIGHT

	DELTA	THETA	ALPHA	BETA
FP2	0.022	0.133	0.254	0.002
F4	0.013	0.278	0.361	0.122
C4	0.003	0.863	0.401	0.175
P4	0.000	0.991	0.768	0.130
O2	0.003	0.949	0.745	0.000
F8	0.010	0.207	0.187	0.000
T4	0.000	0.759	0.026	0.000
T6	0.000	0.845	0.522	0.001

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.078	0.699	0.423	0.469
Cz	0.000	0.738	0.895	0.938
Pz	0.001	0.280	0.170	0.000

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 15 demonstrates significant changes in the absolute power of Delta, Alpha and Beta. No

change is evident in Theta. Progression away from the mean can be observed in 18 of the 19 sites in the absolute power of Delta, with 16 of these sites reaching statistical significance. Movement towards the mean can be observed at 12 of the 19 sites for the absolute power of Alpha, however only one of these sites reached statistical significance. One site for Alpha demonstrated statistically significant change away from the mean. Fifteen of the 19 sites progressed towards the mean for the absolute power of Beta, with nine sites reaching statistical significance, and one site showed statistically significant change away from the mean.

Table 15: *P2 - Change in Z scores for Delta, Theta, Alpha and Beta pre & post Cognitive Rehabilitation.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-2.23	-2.37	-2.46	-2.53	-0.73	-0.64	-1.51	-1.40
Fp2	-1.45	-1.61*	-1.42	-1.51	-0.71	-0.61	-1.05	-0.94**
F3	-2.28	-2.57*	-2.68	-2.55	-0.72	-0.63	-1.27	-1.17**
F4	-2.52	-2.96*	-2.79	-3.03	-0.62	-0.52	-1.23	-1.13
C3	-2.01	-2.34*	-2.34	-2.24	-0.89	-0.90	-1.21	-1.27
C4	-2.05	-2.54*	-2.39	-2.52	-0.70	-0.63	-0.90	-0.83
P3	-2.12	-2.49*	-2.22	-2.15	-1.10	-1.15	-1.35	-1.33
P4	-1.77	-2.30*	-2.22	-2.28	-0.94	-0.92	-1.16	-1.07
O1	-0.44	-1.99*	-1.57	-1.85	-1.12	-1.17	-1.80	-1.61**
O2	-1.63	-2.04*	-1.71	-1.68	-1.00	-0.94	-1.66	-1.32**
F7	-2.09	-1.97	-1.84	-1.54	-0.43	-0.45	-0.54	-0.46**
F8	-1.82	-2.21*	-1.92	-2.10	-0.45	-0.29	-0.57	-0.13**
T3	-1.64	-1.85*	-1.57	-1.58	-0.69	-0.90*	-1.01	-1.21*
T4	-1.65	-2.42*	-1.80	-1.89	-0.68	-0.46**	-0.92	-0.46**
T5	-1.68	-1.91*	-1.69	-1.62	-1.12	-1.16	-1.48	-1.47
T6	-1.66	-2.26*	-1.77	-1.80	-1.08	-1.02	-1.56	-1.30**
Fz	-2.19	-2.43	-2.64	-2.70	-0.68	-0.59	-1.23	-1.19
Cz	-1.91	-2.57*	-2.55	-2.58	-0.77	-0.75	-1.10	-1.13
Pz	-1.43	-2.37*	-2.55	-2.44	-1.13	-1.07	-1.80	-1.41**

NOTE: *Change towards '0' representing a normalisation in EEG.*

*** Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.2.6.3 Final Follow-up Assessment

From the commencement of the program (initial assessment-prior to first treatment) to the final follow-up assessment, paired t-tests revealed some significant changes in

the absolute power of Delta, Theta, Alpha and Beta. Significant changes in absolute power can be observed in topographic maps (Figure 7). The P-values can be observed numerically in Table 16, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

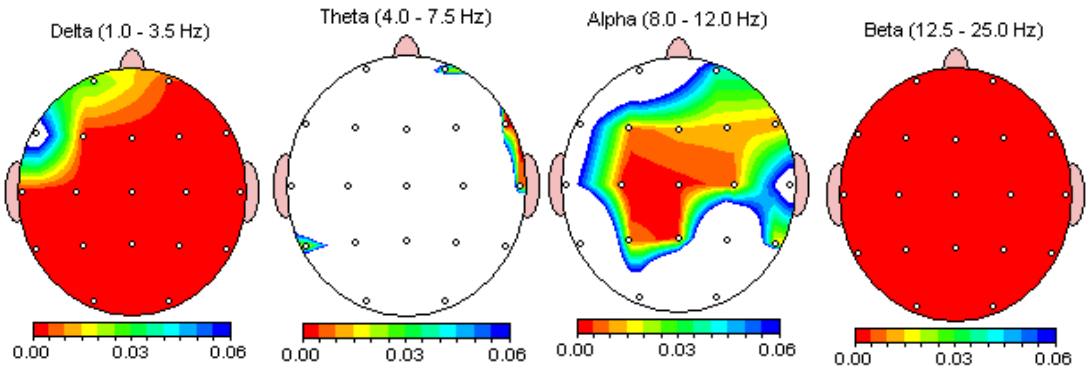

Figure 7: *P2 - Topographic maps - Statistically Significant change (P-values) in absolute power between initial and final Assessment*

Table 16: *P2 - Statistically Significant change (P-Values) in absolute power between initial and final Assessment*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA
FP1	0.026	0.308	0.115	0.000
F3	0.000	0.893	0.008	0.000
C3	0.000	0.727	0.000	0.000
P3	0.001	0.127	0.008	0.000
O1	0.000	0.387	0.103	0.000
F7	0.076	0.513	0.074	0.000
T3	0.000	0.184	0.071	0.000
T5	0.000	0.014	0.206	0.000

Intrahemispheric: RIGHT

	DELTA	THETA	ALPHA	BETA
FP2	0.004	0.016	0.038	0.000
F4	0.001	0.831	0.013	0.000
C4	0.000	0.692	0.007	0.000
P4	0.000	0.876	0.188	0.000
O2	0.000	0.969	0.415	0.000
F8	0.000	0.000	0.010	0.000
T4	0.000	0.008	0.079	0.000
T6	0.000	0.409	0.015	0.000

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.000	0.891	0.012	0.000
Cz	0.005	0.095	0.003	0.000
Pz	0.000	0.239	0.003	0.000

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 17 demonstrates significant changes in the absolute power of all frequencies.

Progression away from the mean can be observed all 19 sites for the absolute power of Delta, with 18 of these sites reaching statistical significance. Movement towards the mean can be observed at seven of the 19 sites for the absolute power of Theta, however only one of these sites reached statistical significance. Three sites for Theta demonstrated statistically significant change away from the mean. Four of the 19 sites were observed to progress towards the mean for the absolute power of Alpha, however none of these were statistically significant. Ten of the 19 sites demonstrated change away from the mean for Alpha, with seven sites reaching statistical significance. Statistically significant progression towards the mean can be observed in all 19 sites for the absolute power of Beta.

Table 17: *P2 - Change in Z scores for Delta, Theta, Alpha and Beta between initial and final Assessment.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-2.74	-3.14*	-2.95	-3.09	-1.01	-1.10	-1.58	-1.25**
Fp2	-1.58	-1.83*	-1.50	-1.66*	-0.80	-0.93*	-0.94	-0.83**
F3	-2.81	-3.52*	-3.18	-3.26	-0.99	-1.14*	-1.43	-0.91**
F4	-3.01	-3.60*	-3.27	-3.36	-0.87	-1.06*	-1.35	-0.84**
C3	-2.06	-3.16*	-2.78	-2.81	-1.06	-1.27*	-1.19	-0.80**
C4	-2.35	-3.26*	-2.85	-2.82	-0.89	-1.02*	-1.15	-0.39**
P3	-2.47	-3.07*	-2.91	-2.75	-1.31	-1.35*	-1.47	-1.00**
P4	-1.49	-2.67*	-2.56	-2.59	-1.02	-1.02	-1.14	-0.66**
O1	-2.15	-3.00*	-2.70	-2.67	-1.36	-1.21	-1.91	-1.54**
O2	-1.88	-2.52*	-2.05	-2.14	-1.12	-1.03	-1.59	-1.17**
F7	-2.77	-3.13	-2.56	-2.55	-0.92	-0.92	-1.40	-0.62**
F8	-2.14	-2.81*	-1.82	-2.27*	-0.55	-0.77*	-0.75	-0.32**
T3	-2.19	-2.95*	-2.35	-2.30	-1.09	-1.10	-1.42	-0.96**
T4	-1.93	-2.66*	-1.59	-1.86*	-0.62	-0.68	-0.84	0.10**
T5	-2.06	-2.73*	-2.48	-2.24**	-1.48	-1.34	-1.79	-1.37**
T6	-1.83	-2.33*	-1.74	-1.88	-1.01	-0.85**	-1.17	-0.61**
Fz	-2.47	-3.17*	-3.17	-3.21	-0.95	-1.11*	-1.30	-0.92**
Cz	-2.30	-2.82*	-3.24	-3.06	-1.04	-1.20*	-1.22	-0.68**
Pz	-2.59	-3.19*	-3.10	-2.97	-1.24	-1.33*	-1.54	-1.04**

NOTE: *Change towards '0' representing a normalisation in EEG.*

** *Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.2.7 Results Summary of Participant Two

Participant Two demonstrated variable improvements in all cognitive domains following both treatments. However, at the ten week follow-up assessment, only improvements made following EEG biofeedback were maintained, and/or further gains were made. Improvements in depression, anxiety, anger, and overall neurobehavioural symptomatology were reported by P2 following both treatments. However, greater improvements in self reported depression, anxiety, and neurobehavioural symptomatology were indicated followed EEG biofeedback. These were maintained at the ten week follow-up. Importantly, the significant other's report on neurobehavioural symptomatology was consistent with the participants self reports. Furthermore, functional gains in every day life following both treatments were reported by P2 and confirmed by his significant other. Participant Two made a number of significant changes towards EEG normalisation in each frequency band following EEG biofeedback. Significant changes towards normalisation were minimal following cognitive rehabilitation (mostly evident in the Beta frequency). He continued to show some significant change towards normalisation at the ten week follow-up assessment, particularly in the Beta frequency range.

5.2.3 PARTICIPANT THREE (P3): AB design *(Outlier eliminated from group results)*

Born in Croatia, Participant Three (P3) was a 51 year old male, who presented as a very distressed and unhappy man. Establishing rapport with P3 was extremely difficult. He indicated that until he commenced the cognitive rehabilitation program he had little respect for the researcher. He reported cultural, age, and in particular gender, as barriers to the therapeutic relationship. With greater personal interaction during the cognitive rehabilitation program, Participant Three demonstrated greater motivation as compared to the EEG biofeedback program. According to medical records P3 was one year and four months post moderate TBI as a result of a direct insult to the occipital region of the head by a steel reinforced rubber hose (see Table 4). No other orthopaedic injuries were sustained. Prior to his TBI he had completed four years of education, and was employed in a supervisory role onsite in construction. Following the TBI he has been unemployed and receives Workcover benefits.

Participant Three was randomly assigned to commence in the EEG biofeedback program first, followed by cognitive rehabilitation. His estimated pre-morbid IQ rated in the Average range, was significantly stronger than his measured IQ. His Full Scale, Verbal, and Performance IQ, were rated within the Extremely Impaired range. It should be noted that headaches, vertigo, and nausea were exacerbated during assessment sessions, and greatly affected his performance at all assessment sessions.

5.2.3.1 EEG biofeedback Program

Participant Three was randomly assigned to commence in the EEG biofeedback

program first. Prior to EEG biofeedback, P3's qEEG topographic maps indicated an overall reduction in absolute power for all frequency bands. An elevation in Theta and less significantly in Delta relative power was observed. A reduction in Alpha and Beta relative power were also observed. A right sided elevation in all frequencies was observed in power asymmetry. On the basis of these qEEG findings the EEG biofeedback protocols included: **1)** C4: Inhibited Delta and Theta (2-7 Hz), rewarded Sensory Motor Rhythm (low Beta – 12-14 Hz), and inhibited Muscle activity/tension (see Appendix 9 for qEEG topographic map).

5.2.3.2 Cognitive Rehabilitation Program

Participant Three described a variety of disabling symptomatology, including cognitive (memory, concentration, planning, and organisation), emotional (anxiety and depression), and somatic (dizziness, headaches, vertigo, muscle tension, and nausea) difficulties which were impacting significantly on his functional capacity in every day life. Participant Three was referred to the research project by his doctor following his unsuccessful participation in a hospital based rehabilitation program to assist with his somatic concerns. Therefore, the cognitive rehabilitation plan consisted of cognitive behavioural therapy and various external compensatory strategies which were collaboratively devised and employed to assist with his described difficulties.

Cognitive behavioural techniques were used to address Participant Three's difficulties with anxiety and depression. Participant Three's emotional difficulties commenced one year following injury, indicating that they were secondary, or a consequence of, the reduction in quality of life (including his inability to return to work) due to his ongoing cognitive and somatic difficulties. Relaxation and breathing techniques were

taught, and a guided imagery relaxation tape was provided for daily practice. Despite formal testing and observation, P3 was reluctant to acknowledge he had depression. One of the aims of the cognitive rehabilitation program was to increase his understanding of his emotional difficulties, and how they might impact on his cognitive functioning.

Participant Three responded well to external compensatory methods to assist with his cognitive and somatic difficulties. Sticky notes with messages placed around the house, a diary, clock alarms, a white board for daily tasks, and note pads for forward planning were all implemented to assist with memory, planning and organisational difficulties. He had difficulty sustaining attention and fatigued easily. Taking frequent rest breaks during activities, alternating activities, and reducing all visual and auditory distractions while performing a task were strategies implemented. Participant Three reported dizziness/vertigo and the subsequent nausea to be the most debilitating symptoms following his TBI. These symptoms were elicited by movement, such as being surrounded by moving people in a shopping centre and trying to watch live soccer games. Taking frequent breaks while at the soccer, and visiting supermarkets during non-peak shopping were strategies used to reduce the amount of stimulation to which he was exposed.

5.2.3.3 Formal Neuropsychological Assessment Results

The trends of the results, as observed in the graphs (see Appendix 10), were analysed as described below. Participant Three's performance on measures of attentional functioning more consistently improved following cognitive rehabilitation. Significant reductions, that is, improvements in performance can be observed in the T.O.V.A. (omissions, commission errors, and variability) and in the PASAT

following cognitive rehabilitation. Conversely, a significant increase in T.O.V.A. omissions and commissions, and poorer performance on the PASAT was observed following EEG biofeedback. The T.O.V.A. response time increased following cognitive rehabilitation. Improved performance was only maintained in the T.O.V.A. variability at the ten week follow-up assessment. Participant Three's performance on all other attentional measures significantly worsened at the ten week follow-up assessment.

A decline in P3's memory performance in both verbal and visual domains was observed across all measures following both treatments. This decline in performance was also evident at the ten week follow-up assessment. Similarly, his performance on speed of processing tasks consistently declined following both treatments, and this trend continued at the ten week follow-up assessment.

On tests of executive functioning, Participant Three demonstrated significant variability. No significant change was evident on P3's performance on the COWAT (FAS and animals). His showed significant improvements on both Trails A and B following cognitive rehabilitation, and a significant deterioration in his performance was observed following EEG biofeedback. He did not maintain his performance, and in fact performed significantly worse on all measures at the ten week follow-up assessment.

5.2.3.4 Formal Emotional and Behavioural Assessment Results

Participant Three reported reductions in state anxiety symptomatology following cognitive rehabilitation, and maintained these at the ten week follow-up assessment. Conversely, on all other behavioural measures (depression, trait anxiety, and anger)

P3 reported increases in symptomatology following both treatments and at the ten week follow-up assessment. Consistent with this reporting, both P3 and his significant other (wife) reported a worsening or no change in overall symptom reporting on the Neurobehavioural Rating Scale (NRS), following both treatments and at the ten week follow-up.

5.2.3.5 Self-Reported Functional Changes

Participant Three reported a small number of functional changes during the course of cognitive rehabilitation, but not during EEG biofeedback**.** Following cognitive rehabilitation P3 continued to use the relaxation and breathing strategies, took frequent breaks, and went out in public during non-peak hour. He indicated that he felt all the strategies were helping him cope better in every day life. Unfortunately, not all techniques were successfully implemented and used independently by P3. Importantly, a lack of family support during the course of the rehabilitation made it difficult to both implement and maintain a number of external compensatory strategies.

5.2.3.6 Quantitative Electroencephalogram Results

5.2.3.6.1 Post EEG biofeedback

Following 20 sessions of EEG biofeedback paired t-tests revealed a number of significant changes in the absolute power of all frequency bands. Significant changes (P-values) in absolute power can be observed in topographic maps (Figure 8). The P-values can be observed numerically in Table 18, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

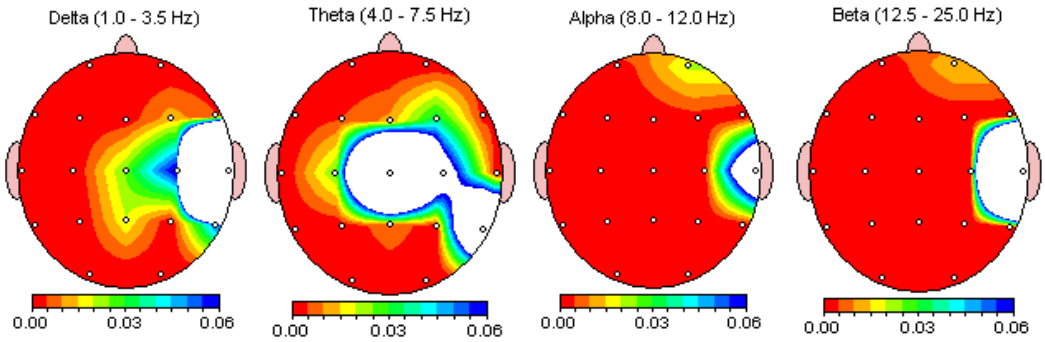

Figure 8: *P3 – Topographic maps: Statistically Significant change (P-values) in absolute power following EEG biofeedback.*

Table 18: *P3 – Statistically Significant change (P-Values) in absolute power following EEG biofeedback*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA
FP1	0.000	0.000	0.000	0.000
F3	0.000	0.005	0.000	0.000
C3	0.000	0.026	0.000	0.000
P3	0.000	0.000	0.000	0.000
O1	0.000	0.000	0.000	0.000
F7	0.000	0.000	0.000	0.000
T3	0.000	0.000	0.000	0.000
T5	0.000	0.000	0.000	0.000

Intrahemispheric: RIGHT

	DELTA	THETA	ALPHA	BETA
FP2	0.000	0.001	0.021	0.015
F4	0.013	0.035	0.002	0.000
C4	0.063	0.080	0.000	0.000
P4	0.000	0.001	0.000	0.000
O2	0.000	0.000	0.000	0.000
F8	0.005	0.003	0.000	0.000
T4	0.777	0.002	0.117	0.592
T6	0.052	0.161	0.000	0.000

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.002	0.012	0.000	0.000
Cz	0.026	0.271	0.000	0.000
Pz	0.020	0.010	0.000	0.000

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 19 demonstrates significant changes in the absolute power. All 19 sites progressed towards the mean in the absolute power of Delta, with 17 of the 19 sites reaching statistical significance. Progression towards the mean can be observed at 17 of 19 sites across the cortex for the absolute power of Theta, with 14 sites reaching statistical significance. Two sites for Theta significant changed away from the mean.

Seventeen of the 19 sites for the absolute power of Alpha significantly progress towards the mean and significant movement away from the mean was observed in only one site. Statistically significant movement towards the mean can be observed in 16 of the 19 sites for the absolute power of Beta, with only two sites significantly progressing away from the mean.

Table 19: *P3 – Change in Z scores for Delta, Theta, Alpha and Beta pre & post EEG biofeedback.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-1.67	-1.04**	-1.37	-1.08**	-1.91	-1.40**	-1.30	-0.56**
Fp2	-1.08	-0.68**	-1.03	-0.85**	-1.37	-1.25**	-0.66	-0.56**
F3	-1.99	-1.30**	-1.22	-0.66**	-1.80	-1.30**	-1.49	-0.64**
F4	-1.86	-1.48**	-1.30	-0.94**	-1.65	-1.43**	-1.23	-0.72**
C3	-2.05	-1.29**	-0.96	-0.47**	-1.91	-1.30**	-1.34	-0.43**
C4	-1.79	-1.49**	-1.12	-0.85	-1.67	-1.30**	-1.52	-0.54**
P3	-2.31	-1.36**	-1.27	-0.56**	-1.80	-1.12**	-1.77	-0.48**
P4	-1.70	-0.73**	-1.11	-0.55**	-1.38	-0.91**	-1.56	-0.69**
O1	-1.58	-0.53**	-1.11	-0.04**	-1.53	-0.84**	-2.03	-0.47**
O2	-1.63	-0.54**	-0.99	-0.12**	-1.64	-0.91**	-1.98	-0.64**
F7	-1.66	-0.58**	-1.61	-0.45**	-1.91	-0.98**	-0.68	0.06**
F8	-1.37	-1.13**	-1.18	-1.41*	-1.20	-1.53*	0.11	-0.23*
T3	-2.42	-0.44**	-1.62	-0.13**	-1.96	-0.68**	-0.40	0.61*
T4	-1.62	-1.45	-1.00	-1.38*	-1.40	-1.52	-0.87	-0.88
T5	-2.14	-0.60**	-1.57	-0.18**	-2.16	-0.94**	-2.50	-0.02**
T6	-1.28	-0.88	-0.80	-0.63	-1.16	-0.78**	-1.60	-0.91**
Fz	-1.63	-1.19**	-1.10	-0.64**	-1.68	-1.35**	-1.42	-0.74**
Cz	-1.73	-1.36**	-0.93	-0.69	-1.80	-1.38**	-1.62	-0.72**
Pz	-2.02	-1.53**	-1.29	-0.83**	-1.76	-1.21**	-1.87	-0.87**

NOTE: *Change towards '0' representing a normalisation in EEG.*

** *Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.3.6.2 Post Cognitive Rehabilitation

Following 20 sessions of cognitive rehabilitation paired t-tests revealed some areas of significant change in the absolute power across all frequency bands. Significant changes in absolute power can be observed in topographic maps (Figure 9). The P-values can be observed numerically in Table 20, with red values representing a

significant increase in absolute power, and blue values indicating a significant decrease.

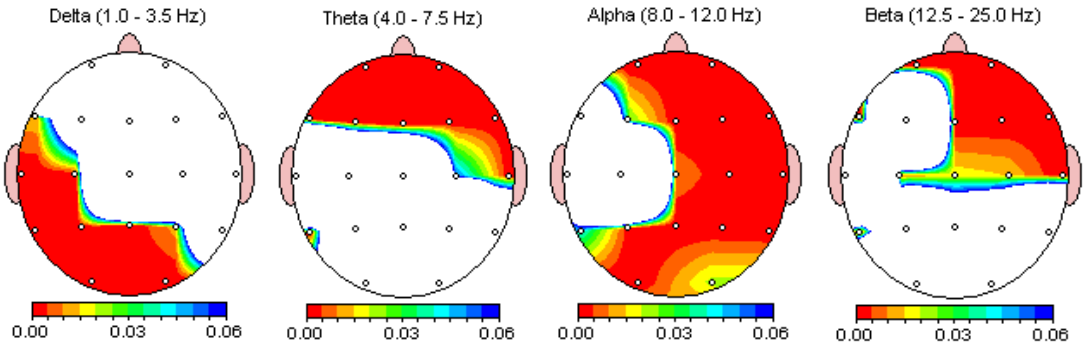

Figure 9: *P3 – Topographic maps – Statistically Significant change (P-values) in absolute power following Cognitive Rehabilitation*

Table 20: *P3 – Statistically Significant change (P-Values) in absolute power following Cognitive Rehabilitation*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA	
FP1	0.892	0.000	0.001	0.002	FP2
F3	0.167	0.001	0.026	0.668	F4
C3	0.000	0.378	0.795	0.006	C4
P3	0.000	0.317	0.001	0.291	P4
O1	0.000	0.094	0.000	0.498	O2
F7	0.014	0.005	0.269	0.001	F8
T3	0.000	0.849	0.110	0.752	T4
T5	0.000	0.000	0.041	0.000	T6

Intrahemispheric: RIGHT

DELTA	THETA	ALPHA	BETA
0.680	0.000	0.000	0.000
0.285	0.000	0.000	0.000
0.448	0.054	0.000	0.015
0.012	0.828	0.000	0.190
0.000	0.202	0.022	0.067
0.899	0.000	0.000	0.000
0.760	0.001	0.000	0.000
0.284	0.263	0.000	0.417

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.993	0.001	0.000	0.000
Cz	0.775	0.355	0.009	0.018
Pz	0.000	0.242	0.000	0.139

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 21 demonstrates significant changes in the absolute power across all frequency bands. Progression towards the mean can be observed in 15 of the 19 sites in the absolute power of Delta, with eight of these sites reaching statistical significance. One site for

Delta demonstrated significant change away from the mean. Six sites in the absolute power of Theta were observed to move towards the mean, with only one site reaching statistical significance. Thirteen sites in Theta progressed away from the mean and eight of these reached statistical significance. Significant movement towards the mean can be observed in only one of the 19 sites for the absolute power of Alpha. Sixteen of the 19 sites for Alpha progressed away from the mean and 15 of these reached statistical significance. Five of the 19 sites were observed to move towards the mean for the absolute power of Beta, with two sites reaching statistical significance. Thirteen Beta sites showed change away from the mean, with nine sites reaching statistical significance.

Table 21: *P3 – Change in Z scores for Delta, Theta, Alpha and Beta pre & post Cognitive Rehabilitation.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-1.25	-1.18	-1.72	-2.24*	-1.89	-2.15*	-1.66	-1.86*
Fp2	-0.83	-0.80	-1.00	-1.34*	-1.43	-1.71*	-0.98	-1.14*
F3	-2.28	-2.06	-1.33	-1.86*	-1.84	-1.96*	-1.77	-1.77
F4	-1.97	-2.07	-1.23	-2.06*	-1.70	-2.01*	-1.49	-1.74*
C3	-2.42	-1.70**	-0.93	-1.04	-1.83	-1.83	-1.14	-1.30*
C4	-1.93	-1.83	-0.99	-1.36	-1.60	-1.82*	-1.27	-1.01**
P3	-2.53	-1.82**	-1.21	-1.09	-1.51	-1.72*	-1.41	-1.42
P4	-2.06	-1.71**	-1.13	-1.19	-1.19	-1.60*	-1.46	-1.53
O1	-1.63	-1.04**	-0.83	-0.56	-0.94	-1.31*	-1.57	-1.50
O2	-1.55	-0.95**	-0.63	-0.48	-0.90	-1.14*	-1.61	-1.50
F7	-0.99	-1.30*	-1.21	-1.61*	-1.76	-1.84	-0.97	-1.13*
F8	-0.85	-0.96	-1.03	-1.52*	-1.39	-1.79*	-0.42	-1.01*
T3	-2.28	-1.37**	-1.35	-1.31	-1.69	-1.60	-0.18	-0.02
T4	-1.59	-1.71	-1.19	-1.64*	-1.28	-1.87*	0.22	-0.55*
T5	-2.00	-1.42**	-1.34	-0.88**	-1.64	-1.54**	-1.92	-0.88**
T6	-1.70	-1.55	-0.97	-1.19	-0.97	-1.52*	-1.79	-1.85
Fz	-1.96	-1.89	-1.19	-1.76*	-1.70	-1.96*	-1.67	-1.86*
Cz	-2.05	-1.93	-0.84	-1.05	-1.66	-1.81*	-1.58	-1.73*
Pz	-2.37	-1.82**	-1.37	-1.23	-1.52	-1.81*	-1.74	-1.80

NOTE: *Change towards '0' representing a normalisation in EEG.*

** *Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.3.6.3 Final Follow-up Assessment

From the commencement of the program (initial assessment-prior to first treatment) to the final follow-up assessment, paired t-tests revealed some significant changes in the absolute power of Delta, Theta, Alpha and Beta. Significant changes in absolute power can be observed in topographic maps (Figure 10). The P-values can be observed numerically in Table 22, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

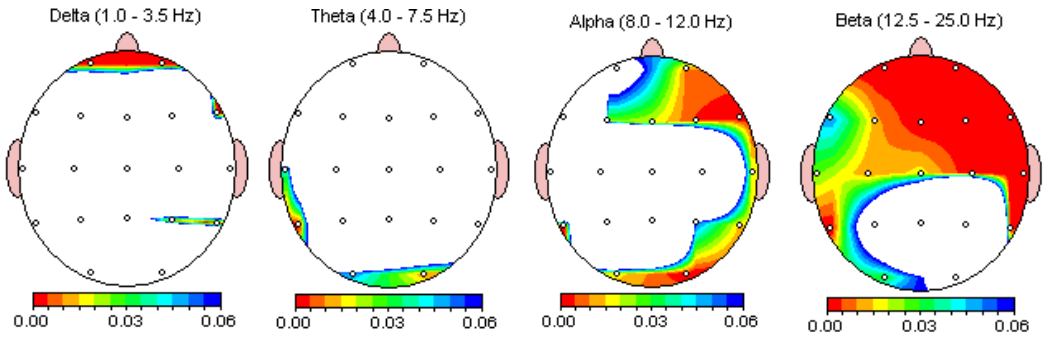

Figure 10: *P3 – Topographic maps – Statistically Significant change (P-values) in absolute power between initial and final Assessment*

Table 22: *P3 – Statistically Significant change (P-Values) in absolute power between initial and final Assessment*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA
FP1	0.001	0.514	0.092	0.000
F3	0.439	0.131	0.025	0.011
C3	0.878	0.465	0.984	0.010
P3	0.739	0.369	0.591	0.112
O1	0.986	0.050	0.017	0.030
F7	0.239	0.327	0.614	0.049
T3	0.077	0.039	0.724	0.025
T5	0.316	0.001	0.000	0.000

Intrahemispheric: RIGHT

	DELTA	THETA	ALPHA	BETA
FP2	0.000	0.931	0.008	0.000
F4	0.721	0.533	0.005	0.000
C4	0.667	0.066	0.360	0.000
P4	0.012	0.288	0.055	0.894
O2	0.876	0.010	0.000	0.085
F8	0.000	0.429	0.000	0.000
T4	0.714	0.560	0.019	0.000
T6	0.000	0.178	0.015	0.003

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.342	0.072	0.019	0.000
Cz	0.425	0.139	0.904	0.017
Pz	0.102	0.406	0.537	0.254

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 23 demonstrates significant changes in the absolute power of each frequency. Progression away from the mean can be observed in nine of the 19 sites for the absolute power of Delta, with one site reaching statistical significance. Movement towards the mean can be observed at eight sites for Delta, with four of these sites reaching statistical significance. Twelve of the 19 sites for the absolute power of Theta demonstrated change towards the mean, reaching statistical significant at three sites. Fourteen of the 19 sites were observed to move away from the mean for the absolute power of Alpha, with seven of these sites reaching statistical significance. Progression towards the mean can be observed in Alpha at five sites, reaching statistical significance at three sites. Ten of the 19 sites demonstrated change away from the mean in the absolute power of Beta, with nine sites reaching statistical significance. Movement towards the mean can be observed at nine of the 19 sites for Beta, with six of these sites reaching statistical significance.

Table 23: *P3 – Change in Z scores for Delta, Theta, Alpha and Beta between initial and final Assessment.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-1.67	-0.93**	-1.37	-1.79	-1.91	-2.01	-1.30	-1.61*
Fp2	-1.08	-0.64**	-1.03	-1.05	-1.37	-1.47*	-0.66	-0.77*
F3	-1.99	-2.08	-1.22	-1.46	-1.80	-2.00*	-1.49	-1.64*
F4	-1.86	-1.86	-1.30	-1.41	-1.65	-1.86*	-1.23	-1.51*
C3	-2.05	-2.08	-0.96	-0.89	-1.91	-1.97	-1.34	-1.05**
C4	-1.79	-1.83	-1.12	-0.88	-1.67	-1.79	-1.52	-0.71**
P3	-2.31	-2.34	-1.27	-1.12	-1.80	-1.79	-1.77	-1.58
P4	-1.70	-1.36**	-1.11	-0.96	-1.38	-1.56	-1.56	-1.52
O1	-1.58	-1.58	-1.11	-0.81	-1.53	-1.39**	-2.03	-1.86**
O2	-1.63	-1.54	-0.99	-0.60**	-1.64	-1.25**	-1.98	-1.85
F7	-1.66	-1.22	-1.61	-1.47	-1.91	-1.94	-0.68	-0.84*
F8	-1.37	-0.93**	-1.18	-1.08	-1.20	-1.52*	0.11	-0.59*
T3	-2.42	-2.04	-1.62	-1.40**	-1.96	-1.95	-0.40	0.04**
T4	-1.62	-1.75	-1.00	-1.08	-1.40	-1.55*	-0.87	-0.04**
T5	-2.14	-1.96	-1.57	-1.13**	-2.16	-1.75**	-2.50	-1.34**
T6	-1.28	-1.86*	-0.80	-0.95	-1.16	-1.43*	-1.60	-1.74*
Fz	-1.63	-1.76	-1.10	-1.37	-1.68	-1.91*	-1.42	-1.83*
Cz	-1.73	-1.88	-0.93	-0.74	-1.80	-1.88	-1.62	-1.77*
Pz	-2.02	-2.24	-1.29	-1.17	-1.76	-1.83	-1.87	-1.96

NOTE: *Change towards '0' representing a normalisation in EEG.*

** *Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.3.7 Results Summary of Participant Three

Overall, Participant Three displayed a deterioration or little change in performance on many measures of cognitive functioning following each treatment. Participant Three's increased somatic concerns (vertigo and nausea) during each assessment session greatly impacted on his performance. However, consistent improvements on attentional measures following cognitive rehabilitation were observed. With the exception of his performance on the T.O.V.A variability, he did not maintain any improvements at the ten week follow-up assessment, and often his performance deteriorated further. With the exception of state anxiety, all other self-reported behavioural measures indicated worsening or no change in symptomatology following

both treatments, and at ten week follow-up assessment. This was consistent in the significant other's report on neurobehavioural symptomatology. Although not indicated on formal assessment, P3 reported some improvement in his functional capacity of everyday life following cognitive rehabilitation, but not EEG biofeedback. Despite these results, P3 made a number of significant changes towards EEG normalisation in each frequency band following EEG biofeedback. Significant changes towards normalisation were less frequent following cognitive rehabilitation. He continued to demonstrate some significant change towards normalisation at the ten week follow-up assessment.

Nevertheless, in view of the issues that needed to be addressed, following the completion of the research program, P3 was referred for additional psychological therapy, to a vestibular rehabilitation program, and also was linked into two brain injury support groups.

5.2.4 PARTICIPANT FOUR (P4): BA design

Participant Four (P4) was 51 year old male, who presented as a very friendly, flamboyant, and motivated man. Excellent rapport was established and maintained throughout the course of the program. As reported by medical records, he was two years post very severe TBI as a result of a motorcycle accident (see Table 4). He reported orthopaedic injuries at the time of his accident, which included damage to his vertebrae. The orthopaedic injuries resulted in ongoing pain management issues. Prior to his TBI he had completed 12 years of education, and owned / managed his own business. Following the TBI he receives the disability support pension and maintains some casual self-employment.

Participant Four was randomly assigned to commence in the cognitive rehabilitation program first, followed by EEG biofeedback. He was estimated to have a pre-morbid IQ within the high average range and his Verbal and Full Scale IQs were consistent with his pre-morbid IQ, however his Performance IQ fell within the Superior range. It should be noted that severe back pain and his subsequent need to frequently move during testing (from sitting, to standing, to lying on the floor, etc) might have impacted on his performance across assessment sessions.

5.2.4.1 Cognitive Rehabilitation Program

Participant Four described a number of cognitive difficulties with memory, concentration/distractibility, problem-solving, planning, and organisation, which were impacting on his functional capacity in every day life. The cognitive rehabilitation plan consisted of various compensatory strategies which were collaboratively devised and employed to assist with his described difficulties. He did not report any

significant emotional or behavioural difficulties at this time. Therefore, they were not addressed in this part of the treatment program.

Various compensatory strategies were implemented throughout the course of cognitive rehabilitation. Strategies to assist with memory, planning and organisation included: diary and white board training, utilising a planner on his mobile phone and personal computer, using the alarm on his mobile phone, financial planning, and carrying a notepad and pen at all times (to jot down 'to do' lists and important notes). In order to further assist in preventing losing personal items around the house, a particular (special) place in the kitchen was determined where he would have to consistently return his keys, wallet, diary and other important personal items while at home. A cork board was attached to the wall above this special place, where all bills and important notices were to be pinned. Additionally, P4 frequently approached problem-solving in an impulsive manner and reported difficulty in obtaining his goals. Therefore, step-by-step guidelines, discouraging impulsive responses and providing structure were implemented. Further strategies were employed to assist with difficulties in sustaining attention and distractibility. He was required to take frequent breaks when concentrating on a task. This was supplemented by setting an alarm upon commencement of a break, reminding him to return to task if he became distracted. By the end of the ten week cognitive rehabilitation program, P4 was independently using most of the strategies implemented.

5.2.4.2 EEG biofeedback Program

Prior to EEG biofeedback, Participant Four's qEEG topographic maps indicated a strong elevation in Theta absolute and relative power (anteriorly), a less prominent elevation in Alpha absolute and relative power (within one standard deviation from

the mean), and a reduction Delta and Beta absolute and relative power. On the basis of these qEEG findings the EEG biofeedback protocols included: **1)** Fz: Inhibited Theta (4-7 Hz), rewarded Sensory Motor Rhythm (low Beta – 12-14 Hz), and inhibited Muscle activity/tension (see Appendix 9 for qEEG topographic map).

5.2.4.3 Formal Neuropsychological Assessment Results

The trends of the results, as observed in the graphs (see Appendix 10), were analysed as described below. Participant Four made variable improvements in attentional functioning following each treatment. Significant improvement in his performance was observed in the T.O.V.A (commission errors) following EEG biofeedback, and in the T.O.V.A. (variability) following cognitive rehabilitation. Improvement in his performance on the PASAT was noted following EEG biofeedback. Improved performance was only maintained in the T.O.V.A. variability and the PASAT at the ten week follow-up assessment. Despite not improving his performance following either treatment on the T.O.V.A. (response time), significant improvement was noted at the ten week follow-up. The variation noted in his concentration across different assessment sessions is likely to be a reflection of pain management difficulties, and his subsequent need to frequently move during testing (from sitting, to standing, to lying on the floor, etc).

Variable improvements were also observed with verbal and visual memory following both treatments. Comparable improvements in visual delayed recall can be observed following both treatments. Significant improvement in verbal memory (RAVLT – total score) performance was noted following cognitive rehabilitation, but not EEG biofeedback. Participant Four maintained his improved performance on the RAVLT (total recall) and made further gains on delayed visual recall at the ten week follow-up

assessment. Although no improvement was noted following each treatment for auditory delayed memory recall, he demonstrated improvement at the final ten week follow-up.

On measures of speed of information processing, Participant Four showed more consistent improvements in performance following cognitive rehabilitation than EEG biofeedback. Despite improvement following cognitive rehabilitation, greater improvement in his performance on language and comprehension processing speed was evident following EEG biofeedback. Participant Four's performance made further gains on all measures at the ten week follow-up assessment.

Participant Four displayed significant variability in tests of executive functioning. No significant changes were evident following each treatment on COWAT (animals and FAS). His performance on Trails A significantly deteriorated following cognitive rehabilitation, and although his performance on Trails B improved following both treatments, however this was not significant. Although not significant, P4 made further gains in his performance on all executive measures at the ten week follow-up assessment. Again, his fluctuating concentration due to pain and subsequent movement was likely to create variability in his performance across assessments.

5.2.4.4 Formal Emotional and Behavioural Assessment Results

Participant Four reported reductions in depression and anxiety symptomatology following both EEG biofeedback and cognitive rehabilitation. However, much larger reductions in symptoms were reported following EEG biofeedback. Improvements continued to be reported at the ten week follow-up assessment. Variable changes in anger symptomatology were reported following both treatments, with small

reductions in state anger, and a small increase in symptom reporting of trait anger and anger expression. Reduced state anger and anger expression was evident at the ten week follow-up assessment. Participant Four reported minimal reductions in overall symptom reporting on the Neurobehavioural Rating Scale (NRS) following both treatments at the ten week follow-up. His significant other (sister) reported small improvements following cognitive rehabilitation, but not following EEG biofeedback or at the ten week follow-up.

5.2.4.5 Self-Reported Functional Changes

Participant Four reported a number of functional changes during the course of each treatment**.** Following cognitive rehabilitation most strategies were successfully implemented and used independently by P4. He reported being able to better organise his day, and reduce his time on work tasks by utilising the problem solving strategies taught. However, there were some difficulties in successfully implementing compensation strategies around the home. Participant Four's mother, who had sustained a recent severe TBI, returned home from rehabilitation during his participation in the research program. Unfortunately, this complicated the successful implementation of strategies. During the course of EEG biofeedback, P4 reported "less fogginess", the ability to think more clearly, and a general improvement in memory. He also reported a significant decrease in back pain. However this was only brief as his back pain resumed when he displaced his already injured spinal disk.

5.2.4.6 Quantitative Electroencephalogram Results

5.2.4.6.1 Post Cognitive Rehabilitation

Following 20 sessions of cognitive rehabilitation paired t-tests revealed some areas of significant change in the absolute power of Delta, Theta, Alpha, and Beta. Significant changes in absolute power can be observed in topographic maps (Figure 11). The P-values can be observed numerically in Table 24, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

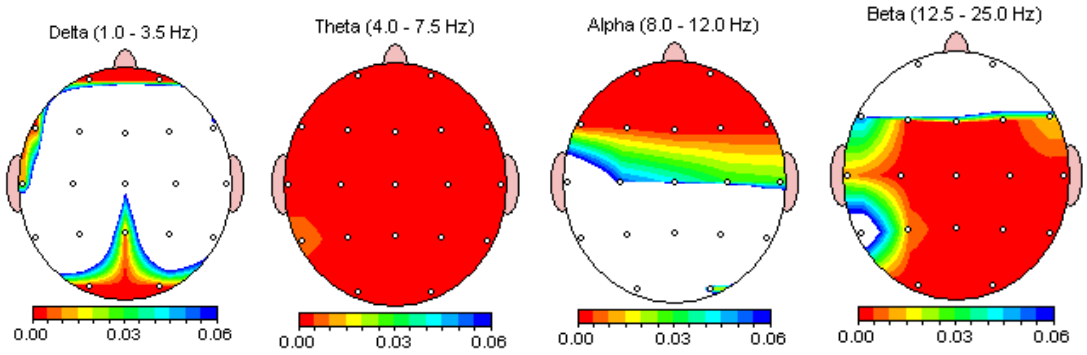

Figure 11: *P4 - Topographic maps - Statistically Significant change (P-values) in absolute power following Cognitive Rehabilitation*

Table 24: *P4 - Statistically Significant change (P-Values) in absolute power following Cognitive Rehabilitation*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA	
FP1	0.004	0.000	0.001	0.668	FP2
F3	0.424	0.000	0.005	0.000	F4
C3	0.405	0.001	0.054	0.000	C4
P3	0.212	0.001	0.915	0.008	P4
O1	0.002	0.000	0.499	0.000	O2
F7	0.004	0.001	0.003	0.049	F8
T3	0.015	0.002	0.111	0.011	T4
T5	0.275	0.008	0.455	0.088	T6

Intrahemispheric: RIGHT

DELTA	THETA	ALPHA	BETA
0.000	0.000	0.001	0.340
0.709	0.000	0.002	0.000
0.256	0.000	0.030	0.000
0.195	0.000	0.753	0.000
0.003	0.000	0.011	0.000
0.069	0.000	0.001	0.016
0.324	0.000	0.027	0.000
0.084	0.000	0.274	0.000

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.859	0.000	0.002	0.000
Cz	0.070	0.000	0.043	0.000
Pz	0.006	0.000	0.970	0.000

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 25 demonstrates significant changes in the absolute power of each frequency. Progression towards the mean can be observed in six of the 19 sites for the absolute power of Delta, with four sites reaching statistical significance. Movement away from the mean can be observed at thirteen sites for Delta, with three sites reaching statistical significance. Eighteen of the 19 sites for the absolute power of Theta demonstrated statistically significant change towards the mean, while one site reached statistical significance moving away from the mean. Eleven of the 19 sites were observed to move away from the mean in the absolute power of Alpha, with nine of these sites reaching statistical significance. Progression towards the mean can be observed in Alpha at eight sites, and reached statistical significance at two sites. Movement away from the mean is evident for the absolute power of Beta in 13 of the 19 sites, with 11 sites reaching statistical significance. Six Beta sites approached the mean, with five reaching statistical significance.

Table 25: *P4 - Change in Z scores for Delta, Theta, Alpha and Beta pre & post Cognitive Rehabilitation.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-1.62	-1.05**	0.35	-0.30**	-0.29	-0.54*	-0.63	-0.67
Fp2	-1.24	-0.85**	-0.04	-0.39*	-0.39	-0.57*	-0.51	-0.54
F3	-1.84	-1.75	0.59	-0.04**	-0.00	-0.26*	-0.27	-0.58*
F4	-1.91	-1.99	0.82	-0.15**	-0.02	-0.35*	-0.27	-0.55*
C3	-1.87	-2.03	0.65	0.13**	0.23	0.06	-0.01	-0.39*
C4	-1.70	-1.92	0.74	-0.15**	0.01	-0.21*	-0.20	-0.56*
P3	-1.57	-1.82	1.00	0.45**	0.52	0.54	0.48	0.30**
P4	-1.75	-1.85	0.66	-0.08**	0.22	0.14	0.07	-0.22*
O1	-0.30	-0.73*	2.51	1.49**	1.28	1.22	1.03	0.84**
O2	-1.12	-1.47*	1.35	0.40**	0.78	0.56**	0.26	-0.06**
F7	-1.73	-1.18**	0.54	-0.01**	-0.20	-0.41*	-0.53	-0.68*
F8	-1.57	-1.16	0.65	-0.14**	-0.11	-0.40*	-0.23	-0.36*
T3	-1.60	-1.32**	0.72	0.23**	0.56	0.41	0.11	-0.03**
T4	-1.42	-1.56	0.74	-0.01**	0.12	-0.10**	-0.38	0.47*
T5	-0.59	-0.73	1.78	1.20**	1.30	1.39	1.07	0.98
T6	-1.66	-1.82	0.42	-0.18**	0.23	0.09	-0.29	-0.51*
Fz	-1.62	-1.70	0.70	-0.07**	0.02	-0.29*	-0.18	-0.47*
Cz	-1.63	-1.94	0.85	0.10**	0.07	-0.15*	0.45	-0.21**
Pz	-1.75	-2.28*	0.68	-0.00**	0.14	0.09	0.14	-0.21*

NOTE: *Change towards '0' representing a normalisation in EEG.*

** *Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.4.6.2 Post EEG biofeedback

Following 20 sessions of EEG biofeedback paired t-tests revealed a number of significant changes in the absolute power of all frequency bands. Significant changes (P-values) in absolute power can be observed in topographic maps (Figure 12). The P-values can be observed numerically in Table 26, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

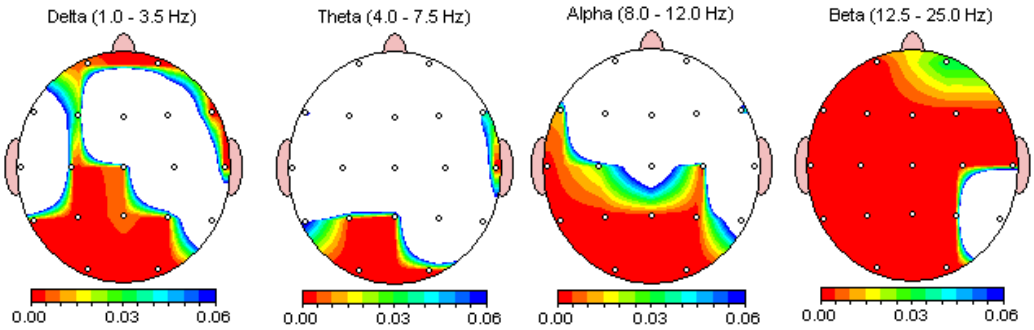

Figure 12: *P4 - Topographic maps: Statistically Significant change (P-values) in absolute power following EEG biofeedback.*

Table 26: ***P4 - Statistically Significant change (P-Values) in absolute power following EEG biofeedback***

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA	
FP1	0.007	0.200	0.221	0.000	FP2
F3	0.025	0.169	0.570	0.000	F4
C3	0.004	0.447	0.025	0.000	C4
P3	0.000	0.000	0.000	0.000	P4
O1	0.000	0.000	0.000	0.000	O2
F7	0.140	0.048	0.012	0.000	F8
T3	0.530	0.868	0.002	0.000	T4
T5	0.001	0.063	0.000	0.000	T6

Intrahemispheric: RIGHT

DELTA	THETA	ALPHA	BETA
0.000	0.460	0.248	0.035
0.207	0.950	0.893	0.000
0.474	0.464	0.002	0.000
0.000	0.456	0.000	0.000
0.000	0.000	0.000	0.000
0.000	0.036	0.034	0.003
0.001	0.000	0.874	0.000
0.200	0.114	0.098	0.763

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.903	0.087	0.162	0.000
Cz	0.006	0.608	0.107	0.000
Pz	0.008	0.000	0.000	0.000

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 27 demonstrates significant changes in the absolute power. Of six sites demonstrating progression towards the mean in the absolute power of Delta, only three sites reached statistical significance. Movement away from the mean in Delta was observed in 13 of the 19 sites, with 10 of these sites reaching statistical significance. Progression towards the mean can be observed at 10 of the 19 sites for the absolute power of Theta, with four sites reaching statistical significance. Movement away from the

mean can be observed in nine of the 19 sites for Theta, however statistical significance was reached at only three sites. Movement towards the mean was observed at 11 sites for the absolute power of Alpha, reaching statistical significance at five sites. Alpha demonstrated change towards the mean in eight sites, with statistically significant change at five sites. Statistically significant change approaching the mean for the absolute power of Beta can be observed in ten of the 19 sites, and change away from the mean in Beta is evident at nine of the 19 sites, reaching significance at eight sites.

Table 27: *P4 - Change in Z scores for Delta, Theta, Alpha and Beta pre & post EEG biofeedback.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-0.99	-1.43*	-0.04	0.15	-0.32	-0.22	-0.69	-0.35**
Fp2	-0.89	-1.16*	-0.22	-0.18	-0.42	-0.39	-0.54	-0.48**
F3	-1.57	-1.85*	0.15	0.30	-0.10	-0.02	-0.52	-0.09**
F4	-1.72	-1.84	0.40	0.34	-0.09	-0.08	-0.43	-0.15**
C3	-1.49	-1.92*	0.39	0.50	0.22	0.33*	-0.19	0.24*
C4	-1.72	-1.58	0.52	0.36	-0.07	0.15*	-0.23	0.04**
P3	-1.35	-0.57**	0.90	1.93*	0.74	1.42*	0.70	1.85*
P4	-1.69	-1.26**	0.39	0.53	0.20	0.51*	0.09	0.46*
O1	-0.49	-1.66*	1.92	0.56**	1.05	-0.06**	0.82	-0.11**
O2	-1.19	-1.84*	0.86	0.23**	0.58	-0.13**	0.07	-0.33*
F7	-1.27	-1.33	0.13	0.41*	-0.29	-0.02**	-0.78	-0.21**
F8	-0.98	-1.83*	0.38	0.05**	-0.15	-0.36*	-0.29	-0.51*
T3	-1.52	-1.57	0.41	0.44	0.41	0.16**	-0.09	0.58*
T4	-1.25	-1.69*	0.56	-0.32**	0.10	-0.02	0.38	-0.29**
T5	-0.01	-0.60*	2.00	1.83	1.59	0.88**	1.47	0.85**
T6	-1.57	-1.29	0.15	0.08	0.03	0.02	-0.37	-0.43
Fz	-1.48	-1.37	0.31	0.50	-0.04	0.08	-0.34	0.08**
Cz	-1.56	-1.92*	0.65	0.64	0.05	0.19	0.18	0.56*
Pz	-1.85	-1.48**	0.43	1.11*	0.19	0.82*	0.13	1.01*

NOTE: *Change towards '0' representing a normalisation in EEG.*

** *Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.4.6.3 Final Follow-up Assessment

From the commencement of the program (initial assessment-prior to first treatment)

to the final follow-up assessment, paired t-tests revealed some significant changes in the absolute power of Delta, Theta, Alpha and Beta. Significant changes in absolute power can be observed in topographic maps (Figure 13). The P-values can be observed numerically in Table 28, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

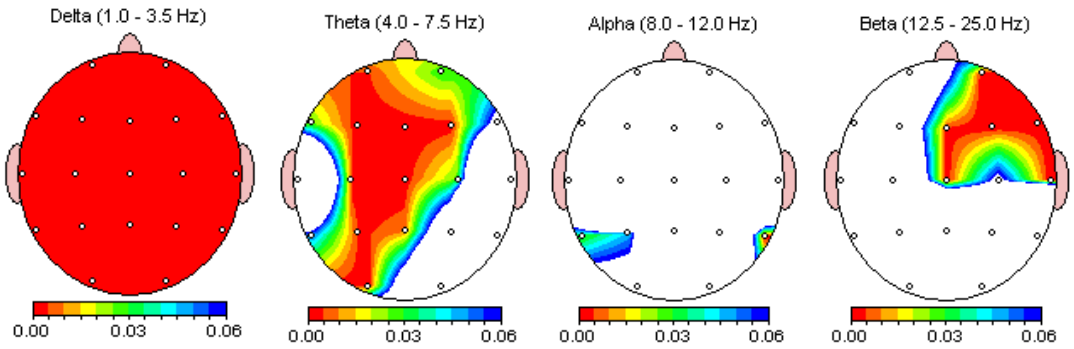

Figure 13: *P4 - Topographic maps - Statistically Significant change (P-values) in absolute power between initial and final Assessment*

Table 28: *P4 - Statistically Significant change (P-Values) in absolute power between initial and final Assessment*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA	
FP1	0.000	0.005	0.141	0.139	FP2
F3	0.000	0.001	0.548	0.107	F4
C3	0.000	0.001	0.733	0.166	C4
P3	0.000	0.004	0.046	0.917	P4
O1	0.000	0.002	0.082	0.788	O2
F7	0.000	0.025	0.603	0.093	F8
T3	0.000	0.223	0.167	0.659	T4
T5	0.000	0.050	0.028	0.181	T6

Intrahemispheric: RIGHT

DELTA	THETA	ALPHA	BETA
0.000	0.029	0.186	0.001
0.000	0.002	0.479	0.001
0.000	0.027	0.676	0.068
0.000	0.113	0.253	0.203
0.000	0.182	0.066	0.548
0.000	0.070	0.241	0.000
0.000	0.303	0.261	0.000
0.000	0.691	0.000	0.770

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.000	0.000	0.318	0.003
Cz	0.000	0.002	0.934	0.000
Pz	0.000	0.008	0.170	0.346

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 29

demonstrates significant changes in the absolute power of each frequency. Statistically significant progression towards the mean can be observed in 11 of the 19 sites for the absolute power of Delta, with significant change away from the mean in eight sites. Movement towards the mean can be observed at 18 of the 19 sites for the absolute power of Theta, with 11 sites reaching statistical significance. Only one site for Theta demonstrated statistically significant change away from the mean. One of three sites moving towards the mean reached statistical significance for the absolute power of Alpha, and two of 16 sites observed to be progressing away from the mean reached statistical significance. Progression towards the mean can be observed in the absolute power of Beta at seven sites, reaching statistical significance at two sites. Movement away from the mean can be observed at 11 of the 19 sites for Beta, with four of these sites reaching statistical significance.

Table 29: *P4 - Change in Z scores for Delta, Theta, Alpha and Beta between initial and final Assessment.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-1.62	0.97**	0.35	0.00**	-0.29	-0.41	-0.63	-0.74
Fp2	-1.24	0.16**	-0.04	-0.22*	-0.39	-0.48	-0.51	-0.65*
F3	-1.84	1.20**	0.59	0.15**	-0.00	-0.06	-0.27	-0.43
F4	-1.91	1.40**	0.82	0.26**	-0.02	-0.13	-0.27	-0.54*
C3	-1.87	1.76**	0.65	0.25**	0.23	0.24	-0.01	-0.16
C4	-1.70	1.73*	0.74	0.31**	0.01	0.03	-0.20	-0.36
P3	-1.57	1.61*	1.00	0.63**	0.52	0.63**	0.48	0.48
P4	-1.75	1.40**	0.66	0.31	0.22	0.25	0.07	-0.04
O1	-0.30	2.25*	2.51	2.10**	1.28	1.37	1.03	1.02
O2	-1.12	2.10*	1.35	1.18	0.78	0.81	0.26	0.24
F7	-1.73	1.54**	0.54	0.36**	-0.20	-0.13	-0.53	-0.47
F8	-1.57	1.54**	0.65	0.37	-0.11	-0.25	-0.23	-0.64*
T3	-1.60	2.60*	0.72	0.71	0.56	0.63	0.11	0.14
T4	-1.42	2.41*	0.74	0.53	0.12	0.17	-0.38	0.02**
T5	-0.59	2.39*	1.78	1.47	1.30	1.43*	1.07	1.23
T6	-1.66	2.11*	0.42	0.32	0.23	0.35*	-0.29	-0.33
Fz	-1.62	0.90**	0.70	0.12**	0.02	-0.12	-0.18	-0.41*
Cz	-1.63	1.24**	0.85	0.31**	0.07	0.03	0.45	0.02**
Pz	-1.75	1.16**	0.68	0.26**	0.14	0.21	0.14	0.03

NOTE: *Change towards '0' representing a normalisation in EEG.*

*** Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.4.7 Results Summary of Participant Four

Overall, Participant Four showed great variability in change following each treatment across all cognitive domains. He tended to maintain his performance in all cognitive domains at the ten week follow-up assessment and often made further gains even when no improvement and/or change was evident following each treatment alone. However, his variable performance across each assessment was likely to be attributable to his back pain and consequent frequent movement impacting on his concentration during testing. Improvements in depression, anxiety, and state anger were evident following each treatment. However, larger reductions in self reported depression, state anxiety, and state anger were evident following EEG biofeedback. These were maintained at the ten week follow-up assessment. Despite these changes, minimal neurobehavioural symptomatology changes were reported by P4. Conversely, the significant other's report on neurobehavioural symptomatology indicated some improvement following cognitive rehabilitation, which was not maintained at the ten week follow-up. Furthermore, functional gains in everyday life following both treatments were reported by P4. He made a number of significant changes towards normalisation in his EEG following both EEG biofeedback (across all frequencies) and cognitive rehabilitation (in particular, within the Theta range). He continued to display some significant change towards normalisation at the ten week follow-up assessment.

5.2.5 PARTICIPANT FIVE (P5): BA Design

Participant Five (P5) was a very friendly and motivated, 45 year old female, who had significant expressive speech difficulties. Excellent rapport was established and maintained throughout the course of the program. She was 23 years post extremely severe TBI as a result of a motor vehicle accident (see Table 4). She reported orthopaedic injuries at the time of the accident, which included broken ribs, right shoulder and elbow, and a pieced lung. Preceding the TBI she had completed 10 years of education, and maintained full-time employment. Following the TBI P5's primary income has been the disability support pension.

Participant five was randomly assigned to commence in the cognitive rehabilitation program first, followed by EEG biofeedback. Consistent with her estimated pre-morbid IQ, Participant Five's Verbal Scale IQ was within the Average range. However, her Performance and Full Scale IQ fell within the low average range. Importantly, P5 reported difficulties with fatigue, particularly when attempting to concentrate over long periods. Although frequent breaks were implemented during testing, P5 fatigued quickly during each assessment session. Furthermore, a number of circumstances beyond her control caused great emotional distress impacting on her assessment performance, particularly during the final ten week follow-up assessment session.

5.2.5.1 Cognitive Rehabilitation Program

Prior to the commencement of cognitive rehabilitation, Participant Five described a number of cognitive difficulties with attention/concentration, and language (expressive speech and word finding) which were impacting on her functional

capacity in everyday life. Participant Five also reported significant levels of depressive and anxiety symptomatology. Consequently, the cognitive rehabilitation plan commenced with cognitive behavioural therapy strategies to address the emotional issues, followed by various compensatory strategies which were collaboratively devised and employed to assist with her described cognitive difficulties.

Relaxation and breathing strategies were taught and supplemented with a pre-recorded tape assisting Participant Five to practice relaxation at home. Over the ten weeks of treatment P5's perception of her experiences in life following her TBI were explored and challenged. A number of homework tasks were provided to help her view her experiences in a different way. Participant Five reported becoming easily upset, but was not able to identify the triggers. Hence, she was given homework to assist in identifying emotional triggers. Once a number of emotional triggers were identified, P5 was able to practice better control in her response to the trigger. Furthermore, P5 found it difficult to recall positive experiences and events that occur in her life on a daily basis. Therefore, she commenced a journal to note down daily positive experiences and events, which she could reflect on at later dates. A number of personal tragedies and stressors (family death and relationship issues) during the course of this program impacted on each session for P5. Consequently, grief counselling was also factored into this part of the treatment process.

Following her TBI, Participant Five self-implemented a number of effective compensatory strategies to assist with memory, planning, and organisation difficulties. She reported that she continues to use these strategies effectively. A number of additional compensatory strategies were implemented throughout the

course of cognitive rehabilitation to address other cognitive domains. To assist with attention, concentration, and fatigue difficulties, P5 was instructed to take frequent breaks on any tasks that she had identified as problematic (e.g. while reading and/or watching long movies). In particular, P5 reported difficulties with concentration while driving long distances. Therefore, in addition to taking frequent breaks when driving, P5 was required to reduce all other distractions within the car (e.g. Radio/stereo off, no talking to passengers, no talking on mobile phone– even with hands free). Multiple strategies were implemented to assist P5 with word finding difficulties, in particular the recall of names. Visual imagery was practiced to assist with learning to associate visual features of a person to their name. In addition, when P5 was first introduced to a person she was to practice the persons name in their conversation.

5.2.5.2 EEG Biofeedback Program

Prior to EEG biofeedback, Participant Five's qEEG topographic maps indicated a strong elevation in Alpha absolute and relative power (across the entire cortex), and a reduction Delta, Theta, and Beta absolute and relative power. On the basis of these qEEG findings the EEG biofeedback protocols included: **1)** Cz: Inhibited Alpha (8-11 Hz), rewarded Sensory Motor Rhythm (low Beta – 12-14 Hz), and inhibited Muscle activity/tension (see Appendix 9 for qEEG topographic map).

5.2.5.3 Neuropsychological Assessment Results

The trends of the results, as observed in the graphs (see Appendix 10), were analysed as described below. Participant Five made a number of improvements in attentional functioning following each treatment. Her performance improved following both

treatments on the T.O.V.A (omission and commission errors), but this was not significant change. Significant improvements were evident on the T.O.V.A. (variability) following both treatments and on the T.O.V.A. (response time) following cognitive rehabilitation. A small improvement in her performance on the PASAT was only noted following cognitive rehabilitation. Participant Five's performance was maintained and further gains were made on all measures of attention at the ten week follow-up assessment. The variability in her attentional performance is likely attributable to her increased fatigue during assessment sessions.

Participant Five showed improvements across all verbal memory measures consistently following EEG biofeedback. Improved performance on delayed visual memory was only evident following cognitive rehabilitation. She maintained her improved performance only on delayed visual recall at the ten week follow-up assessment.

On measures of speed of information processing, P5 displayed more consistent improvements in performance following cognitive rehabilitation than EEG biofeedback. Participant Five's performance made further gains on all measures at the ten week follow-up assessment.

Participant Five demonstrated significant variability in tests of executive functioning. Her performance on COWAT (animals) significantly improved following cognitive rehabilitation, with no significant changes evident following each treatment on COWAT (FAS). Her performance on Trails A significantly improved following cognitive rehabilitation, while her performance on Trails B significantly improved following EEG biofeedback. Participant Five continued to maintain and/or make

further gains in her performance on all executive functioning measures at the ten week follow-up assessment.

5.2.5.4 Formal Emotional and Behavioural Assessment Results

Participant Five reported reductions in depression, anxiety, anger, and overall neurobehavioural symptomatology following both EEG biofeedback and cognitive rehabilitation. However, reductions were more consistent and generally greater across all measures following cognitive rehabilitation compared to EEG biofeedback. Improvements were only maintained in anger expression and the neurobehavioural symptomatology reporting at the ten week follow-up assessment. However, on the day of the ten week follow-up assessment personal circumstances (unrelated to the research program) caused great emotional distress. Participant Five's significant other (mother) reported a reduction in overall neurobehavioural symptomatology following only EEG biofeedback and not cognitive rehabilitation, with positive change maintained at the ten week follow-up review.

5.2.5.5 Self-Reported Functional Changes

Participant Five reported a number of functional changes during the course of each treatment. Following cognitive rehabilitation most techniques were successfully implemented and used independently by P5. However, P5 attributed improvements in emotional functioning to her improved functional capacity in every day life. She indicated a greater ability in identifying emotional triggers, and a sense of control over her emotional lability. Generally, P5 indicated that she felt much better, had fewer migraines, and increased energy. Participant Five's significant other confirmed these reports. During EEG biofeedback P5 reported a number of changes in her

cognition. Participant Five indicated that she felt more alert and focused, she was clearer and quicker in her thinking and decision making on every day tasks. Furthermore, she reported improvements in her sleep, less fatigue, a continued reduction in migraines, and a reduction in the severity of menstrual pain.

5.2.5.6 Quantitative Electroencephalogram Results

5.2.5.6.1 Post Cognitive Rehabilitation

Following 20 sessions of cognitive rehabilitation paired t-tests revealed some areas of significant change in the absolute power of Delta, Theta, Alpha, and Beta. Significant changes in absolute power can be observed in topographic maps (Figure 14). The P-values can be observed numerically in Table 30, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

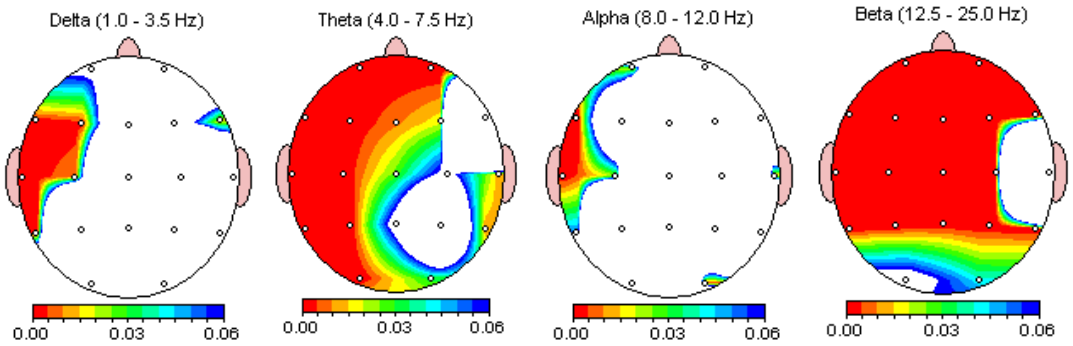

Figure 14: *P5 - Topographic maps - Statistically Significant change (P-values) in absolute power following Cognitive Rehabilitation*

Table 30: *P5 - Statistically Significant change (P-Values) in absolute power following Cognitive Rehabilitation*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA
FP1	0.066	0.000	0.019	0.000
F3	0.002	0.000	0.204	0.000
C3	0.010	0.000	0.023	0.000
P3	0.694	0.000	0.972	0.000
O1	0.279	0.000	0.924	0.074
F7	0.000	0.000	0.000	0.000
T3	0.000	0.000	0.000	0.000
T5	0.001	0.000	0.051	0.000

Intrahemispheric: RIGHT

	DELTA	THETA	ALPHA	BETA
FP2	0.161	0.000	0.486	0.000
F4	0.096	0.018	0.411	0.000
C4	0.486	0.069	0.878	0.000
P4	0.196	0.177	0.310	0.000
O2	0.637	0.033	0.005	0.041
F8	0.016	0.983	0.268	0.000
T4	0.203	0.013	0.013	0.999
T6	0.286	0.012	0.825	0.001

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.165	0.009	0.862	0.000
Cz	0.472	0.027	1.000	0.000
Pz	0.196	0.074	0.439	0.000

NOTE: *RED represents a significant increase. BLUE represents a significant decrease*.

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 31 demonstrates significant changes in the absolute power of each frequency. Progression away from the mean can be observed at 17 of the 19 sites for the absolute power of Delta, with five sites reaching statistical significance. Of the two sites demonstrating movement towards the mean for Delta, one site reached statistical significance. Seventeen of the 19 sites for the absolute power of Theta demonstrate change away from the mean, with 14 reaching statistical significance. Two sites were observed to move towards the mean for Theta, but neither reached significance. Change away from the mean was observed at 11 sites in the absolute power of Alpha, with statistical significance change occurring at four sites. Of seven sites noted to be progressing towards the mean in Alpha, only two were statistically significant. Progression away from the mean can be observed in the absolute power of Beta at 17 sites, reaching statistical significance at 15 sites. Statistically significant movement towards the mean can be observed at two of the 19 sites for Beta.

Table 31: *P5 - Change in Z scores for Delta, Theta, Alpha and Beta pre & post Cognitive Rehabilitation.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-1.06	-1.43	-1.11	-1.93*	-0.02	-0.32*	0.03	-0.40*
Fp2	-1.02	-1.13	-0.92	-1.22*	-0.28	-0.36	-0.11	-0.55*
F3	-1.12	-1.75*	-1.62	-2.14*	-0.15	-0.42	-0.34	-1.13*
F4	-1.62	-1.93	-2.20	-2.43*	-0.25	-0.28	-0.62	-1.15*
C3	-0.95	-1.55*	-1.33	-2.15*	-0.09	-0.37*	-0.17	-0.86*
C4	-1.18	-1.41	-1.73	-2.05	-0.45	-0.47	-0.62	-1.21*
P3	-1.19	-1.40	-0.97	-1.60*	-0.00	-0.06	-0.07	-0.50*
P4	-0.56	-0.81	-0.39	-0.59	-0.14	-0.01	-0.45	-0.74*
O1	-0.69	-1.07	-0.65	-1.21*	0.16	0.16	-0.12	-0.23
O2	-0.49	-0.72	-0.17	-0.45*	0.09	0.39*	-0.24	-0.09**
F7	-0.49	-1.46*	-0.53	-1.43*	0.26	-0.30*	1.22	-0.37**
F8	-1.98	-1.62**	-1.85	-1.63	-0.22	-0.10	0.07	-0.38*
T3	-0.31	-1.45*	-0.34	-1.52*	0.43	-0.15**	0.83	1.10*
T4	-1.87	-1.71	-2.02	-1.63	-0.54	-0.27**	0.75	0.93
T5	-0.56	-1.23*	-0.63	-1.49*	0.16	-0.02	0.10	-0.38*
T6	-0.87	-1.09	-0.69	-1.03*	-0.35	-0.27	-0.68	-0.89*
Fz	-1.26	-1.54	-1.90	-2.16*	-0.24	-0.36	-0.46	-1.17*
Cz	-1.30	-1.48	-1.95	-2.29*	-0.36	-0.45	-0.50	-1.12*
Pz	-0.95	-1.23	-0.97	-1.30	-0.24	-0.16	-0.50	-0.89*

NOTE: *Change towards '0' representing a normalisation in EEG.*

** *Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.5.6.2 Post EEG biofeedback

Following 20 sessions of EEG biofeedback paired t-tests revealed a number of significant changes in the absolute power of all frequency bands. Significant changes (P-values) in absolute power can be observed in topographic maps (Figure 15). The P-values can be observed numerically in Table 32, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

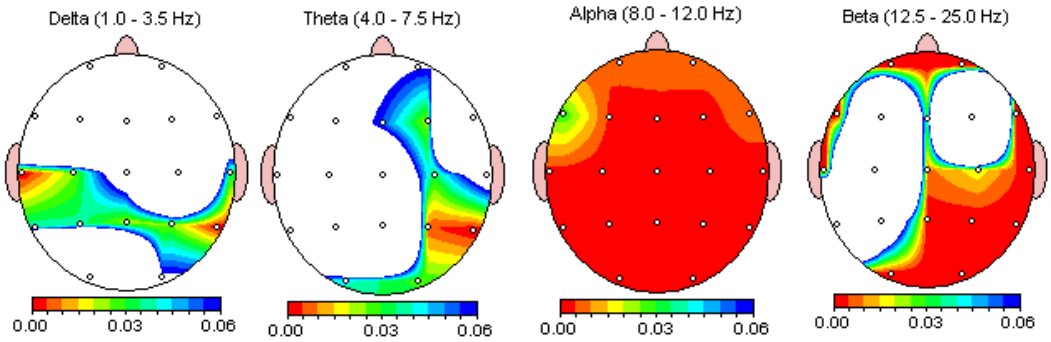

Figure 15: *P5 - Topographic maps: Statistically Significant change (P-values) in absolute power following EEG biofeedback.*

Table 32: *P5 - Statistically Significant change (P-Values) in absolute power following EEG biofeedback*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA
FP1	0.391	0.077	0.006	0.000
F3	0.342	0.080	0.003	0.316
C3	0.025	0.204	0.001	0.579
P3	0.037	0.267	0.000	0.130
O1	0.423	0.042	0.000	0.000
F7	0.399	0.398	0.028	0.000
T3	0.000	0.083	0.003	0.000
T5	0.039	0.166	0.002	0.400

Intrahemispheric: RIGHT

	DELTA	THETA	ALPHA	BETA
FP2	0.188	0.060	0.008	0.000
F4	0.737	0.022	0.001	0.641
C4	0.293	0.032	0.000	0.019
P4	0.024	0.006	0.000	0.000
O2	0.062	0.027	0.000	0.000
F8	0.140	0.521	0.010	0.000
T4	0.034	0.080	0.000	0.001
T6	0.001	0.000	0.000	0.000

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.221	0.054	0.002	0.032
Cz	0.088	0.119	0.000	0.008
Pz	0.021	0.404	0.000	0.002

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 33 demonstrates significant changes in the absolute power of each frequency. Statistically significant progression away from the mean can be observed at all 19 sites for the absolute power of Delta, with eight sites reaching statistical significance. Movement away from the mean can be observed at 18 of the 19 sites for the absolute power of Theta, with five sites reaching statistical significance. Only one site for the absolute power of Theta demonstrated statistically significant change towards the

mean. Statistically significant change away from the mean was observed at 13 sites in the absolute power of Alpha, with significant progression towards the mean evident at six sites. Progression towards the mean can be observed in the absolute power of Beta at nine sites, reaching statistical significance at six sites. Movement away from the mean can be observed at nine of the 19 sites for Beta, with eight sites reaching statistical significance.

Table 33: *P5 - Change in Z scores for Delta, Theta, Alpha and Beta pre & post EEG biofeedback.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-1.64	-1.71	-1.61	-1.86	-0.09	-0.37*	-0.75	0.18**
Fp2	-1.03	-1.22	-0.99	-1.08	-0.25	-0.43*	-0.61	-0.32**
F3	-2.03	-2.14	-1.88	-2.20	-0.10	-0.45*	-0.99	-0.87
F4	-1.92	-1.92	-2.01	-2.45*	-0.04	-0.40*	-1.11	-0.99
C3	-1.64	-1.96*	-1.81	-1.95	0.06	-0.28*	-0.82	-0.82
C4	-1.62	-1.76	-1.55	-1.93*	-0.06	-0.50*	-1.06	-1.21*
P3	-1.53	-1.96*	-1.15	-1.27	0.29	-0.07**	-0.43	-0.50
P4	-0.75	-1.12*	0.19	-0.32*	0.28	-0.15**	-0.45	-0.71*
O1	-0.88	-1.06	-0.46	-0.70*	0.76	0.22**	-0.10	-0.23*
O2	-0.31	-0.63	0.63	0.28**	0.91	0.46**	0.21	-0.02**
F7	-1.65	-1.75	-1.39	-1.53	-0.06	-0.26*	-0.30	0.40*
F8	-1.63	-1.81	-1.34	-1.38	-0.03	-0.22*	-0.69	0.00**
T3	-1.43	-1.91*	-1.30	-1.46	0.27	0.01**	0.47	1.01**
T4	-1.38	-1.78*	-1.35	-1.62	-0.10	-0.42*	-0.16	0.14**
T5	-1.27	-1.63*	-1.08	-1.21	0.33	0.09**	-0.36	-0.28
T6	-0.86	-1.44*	-0.18	-0.84*	0.09	-0.43*	-0.49	-0.75*
Fz	-1.68	-1.82	-1.86	-2.16	-0.08	-0.42*	-0.98	-1.08*
Cz	-1.74	-2.02	-2.00	-2.26	-0.03	-0.46*	-0.97	-1.15*
Pz	-1.20	-1.69*	-0.96	-1.08	0.16	-0.27*	-0.72	-0.90*

NOTE: *Change towards '0' representing a normalisation in EEG.*

** *Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.5.6.3 Final Follow-up Assessment

From the commencement of the program (initial assessment-prior to first treatment) to the final follow-up assessment, paired t-tests revealed some significant changes in the absolute power of Delta, Theta, Alpha and Beta. Significant changes in absolute

power can be observed in topographic maps (Figure 16). The P-values can be observed numerically in Table 34, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

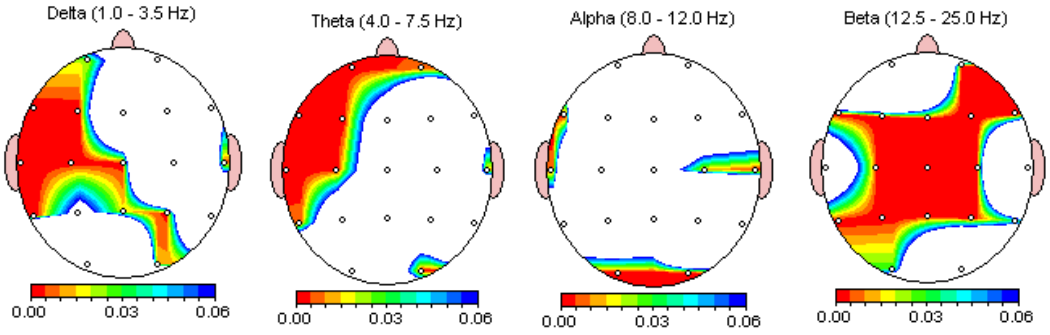

Figure 16: *P5 - Topographic maps - Statistically Significant change (P-values) in absolute power between initial and final Assessment*

Table 34: *P5 - Statistically Significant change (P-Values) in absolute power between initial and final Assessment*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA
FP1	0.020	0.000	0.141	0.640
F3	0.000	0.001	0.939	0.000
C3	0.000	0.002	0.683	0.000
P3	0.082	0.168	0.209	0.002
O1	0.928	0.291	0.000	0.029
F7	0.000	0.000	0.000	0.000
T3	0.000	0.000	0.008	0.159
T5	0.000	0.008	0.169	0.001

Intrahemispheric: RIGHT

	DELTA	THETA	ALPHA	BETA
FP2	0.184	0.008	0.951	0.000
F4	0.240	0.343	0.179	0.000
C4	0.617	0.574	0.013	0.000
P4	0.003	0.189	0.774	0.000
O2	0.012	0.002	0.000	0.359
F8	0.084	0.126	0.165	0.000
T4	0.006	0.013	0.002	0.625
T6	0.322	0.527	0.628	0.035

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.261	0.128	0.353	0.000
Cz	0.002	0.155	0.105	0.000
Pz	0.016	0.120	0.202	0.000

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 35 demonstrates significant changes in the absolute power of each frequency. Progression away from the mean can be observed at 16 of the 19 sites for the absolute

power of Delta, with nine sites reaching statistical significance. Of the three sites demonstrating movement towards the mean for Delta, two sites reached statistical significance. Sixteen of the 19 sites for the absolute power of Theta demonstrate change away from the mean, with eight reaching statistical significance. Three sites were observed to move towards the mean for Theta, but only one reached significance. Change towards the mean was observed at eight sites in the absolute power of Alpha, with statistical significance change occurring at three sites. Of eleven sites noted to be progressing away from the mean in Alpha, only three were statistically significant. Statistically significant progression away from the mean can be observed in the absolute power of Beta at 14 sites, with movement towards the mean evident at five sites, and reaching statistical significance at only one site.

Table 35: *P5 - Change in Z scores for Delta, Theta, Alpha and Beta between initial and final Assessment.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-1.06	-1.42*	-1.11	-1.64*	-0.02	-0.23	0.03	0.01
Fp2	-1.02	-1.10	-0.92	-1.03*	-0.28	-0.35	-0.11	-0.39*
F3	-1.12	-1.81*	-1.62	-1.97*	-0.15	-0.26	-0.34	-0.70*
F4	-1.62	-1.92	-2.20	-2.30	-0.25	-0.24	-0.62	-0.99*
C3	-0.95	-1.74*	-1.33	-1.78*	-0.09	-0.17*	-0.17	-0.49*
C4	-1.18	-1.37	-1.73	-1.84	-0.45	-0.33	-0.62	-1.05*
P3	-1.19	-1.61	-0.97	-1.26	-0.00	0.04	-0.07	-0.27*
P4	-0.56	-1.09*	-0.39	-0.75	-0.14	-0.17	-0.45	-0.75*
O1	-0.69	-0.82	-0.65	-0.55	0.16	0.46*	-0.12	-0.26*
O2	-0.49	-0.02**	-0.17	0.21*	0.09	0.51*	-0.24	-0.17
F7	-0.49	-1.30*	-0.53	-1.33*	0.26	-0.15**	1.22	-0.27**
F8	-1.98	-1.67	-1.85	-1.46	-0.22	-0.14	0.07	-0.33*
T3	-0.31	-1.31*	-0.34	-1.21*	0.43	0.12**	0.83	0.75
T4	-1.87	-1.57**	-2.02	-1.64**	-0.54	-0.31**	0.75	0.69
T5	-0.56	-1.23*	-0.63	-1.08*	0.16	0.21	0.10	-0.10*
T6	-0.87	-1.02	-0.69	-0.87	-0.35	-0.38	-0.68	-0.84*
Fz	-1.26	-1.51	-1.90	-1.99	-0.24	-0.28	-0.46	-1.03*
Cz	-1.30	-1.94*	-1.95	-2.12	-0.36	-0.33	-0.50	-1.03*
Pz	-0.95	-1.33*	-0.97	-1.35	-0.24	-0.17	-0.50	-0.77*

NOTE: *Change towards '0' representing a normalisation in EEG.*

*** Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.5.7 Results Summary of Participant Five

Overall, Participant Five demonstrated a number of improvements following each treatment across all cognitive domains. Both treatments were effective in improving attentional functioning. EEG biofeedback appeared to more effective in improving verbal memory and cognitive flexibility, while cognitive rehabilitation was more effective in improving visual memory, speed of information processing, and semantic verbal fluency. Participant Five maintained and/or made further gains in her performance across all cognitive domains at the ten week follow-up assessment. It is noted that fatigue impacted on each assessment session, and was likely to have influenced the results, causing some variability in her performance. Both treatments were effective in improving emotional and neurobehavioural symptomatology. However, greater change was more consistently observed following cognitive rehabilitation. Change was only maintained in anger expression and the neurobehavioural symptomatology reporting at the ten week follow-up assessment. However, unforeseen circumstances (unrelated to the research program) caused great emotional distress and impacted on her emotional state during this final follow-up assessment. Despite greater emotional changes reported by P5 following cognitive rehabilitation, her significant other reported greater change occurring following EEG biofeedback. Importantly, functional gains in everyday life following both treatments were reported by P5. Participant Five made significant changes towards normalisation in her EEG following both EEG biofeedback in the frequencies trained (Alpha and Beta), however little change towards normalisation was evident following cognitive rehabilitation and at the ten week follow-up assessment.

5.2.6 PARTICIPANT SIX (P6): BA Design

Participant Six (P6) was a very friendly and timid 46 year old female. She was 5 years post severe TBI as a result of a water skiing accident (see Table 4). Excellent rapport was established and maintained throughout the course of the program. She reported orthopaedic injuries including broken ribs and a shattered elbow. Prior to the TBI she had completed 12 years of education, and maintained very part-time self-employment (working from home for husband's business), which she continued to maintain following the accident.

Participant Six was randomly assigned to commence in the cognitive rehabilitation program first, followed by EEG biofeedback. A formal reading test estimated that she had a pre-morbid IQ within the average range. Her Verbal and Full Scale IQ were consistent with her estimated pre-morbid IQ, however her Performance IQ fell within the high average range. Participant Six reported ongoing difficulties with fatigue. Although frequent breaks were implemented during the assessment sessions, P6 often reported feeling fatigued and this impacted on her performance.

5.2.6.1 Cognitive Rehabilitation Program

Prior to the commencement of cognitive rehabilitation, Participant Six described a number of cognitive difficulties with memory, attention/concentration, and word finding which were impacting on her functional capacity in every day life. Fatigue was reported to exacerbate her difficulties. Participant Six also reported significant levels of anxiety symptomatology and features consistent with obsessive compulsive behaviours. The cognitive rehabilitation plan commenced with cognitive behavioural therapy strategies to address the emotional and behavioural issues, followed by

various compensatory strategies which were collaboratively devised and employed to assist with her described cognitive difficulties.

Participant Six reported becoming anxious when leaving home alone and visiting public places (most commonly in shopping centres). Heightened anxiety was also reported when she felt overloaded with multiple activities needing completion. Participant Six indicated that an increase in anxiety resulted in a number of obsessive and compulsive behaviours, such as repetitively doing activities in excess (self-washing behaviour – difficulty leaving shower) and constant counting of almost anything. To address generalised anxiety, relaxation and breathing strategies were taught and supplemented with a pre-recorded tape assisting P6 to practice breathing at home and when out in public places. When showering, an alarm sounded after 15 minutes requiring her to leave the shower and reduce excessive washing. Distractions were used to reduce her compulsive counting. Participant Six used her MP3 player when leaving the house to listen to music. Singing along to songs in her mind assisted in the reduction of counting. This was also used to reduce her anxiety while in social settings. Listening and focussing on the music while out in public places allowed P6 to gradually increase the length of time in which she left the house over the ten week period.

Such strategies as diary training, utilising a kitchen whiteboard, and using a notepad were devised to assist with memory, planning and organisational difficulties. Such difficulties were greatly impacting on her ability to complete her administration duties for her husband's business. She was required to make diary entries three times a day (associated with meal times); and also check and revise the white board at the end of the day. Her family were involved in leaving messages for her in a particular

section of the white board. In order to further assist in preventing losing personal items around the house, a special place in the kitchen was determined where she would have to consistently return her diary, handbag, and other important personal items while at home. Participant Six frequently became distracted around the house, and rarely completed a task (e.g. house left half vacuumed and washing remaining in machine for days). A number of timers with alarms were implemented to remind her to return to an incomplete task. At the time the alarm sounded, a voice recorder was used to remind her of the previous task she now had to return to, and record the task she was now leaving. Frequent breaks were recommended to assist with fatigue and concentration.

5.2.6.2 EEG Biofeedback Program

Prior to EEG biofeedback, Participant Six's qEEG topographic maps indicated an elevation in Alpha absolute and relative power (across entire cortex, more prominent anteriorly), an elevation in Theta absolute power (across entire cortex) with a slight elevation posteriorly in Theta relative power, and a reduction in Beta absolute and relative power (across entire cortex). On the basis of these qEEG findings the EEG biofeedback protocols included: **1)** Cz: Inhibited Alpha amplitude (8-11 Hz), rewarded Sensory Motor Rhythm (low Beta – 12-14 Hz), and inhibited Muscle activity/tension. This protocol was used for 35 minutes of each session. **2)** Cz: Inhibited Theta amplitude (4-7 Hz), rewarded Sensory Motor Rhythm (low Beta – 12-14 Hz), and inhibited Muscle activity/tension. This protocol was used for the final 10 minutes of each session (see Appendix 9 for qEEG topographic map).

5.2.6.3 Formal Neuropsychological Assessment Results

The trends of the results, as observed in the graphs (see Appendix 10), were analysed as described below. Participant Six made more consistent improvements in attentional functioning following EEG biofeedback as compared to cognitive rehabilitation. Her performance significantly declined on all T.O.V.A. measures following cognitive rehabilitation. Significant improvements were evident on the T.O.V.A. (response time and variability) following EEG biofeedback. Small improvements in P6's performance on the PASAT were noted following both treatments. Participant Six's performance was maintained on the T.O.V.A. (response time and variability) and the PASAT at the ten week follow-up assessment.

Participant Six demonstrated equal improvement in her performance on delayed visual memory following both treatments. She maintained this improved performance at the ten week follow-up assessment. Although no improvement was noted on all other verbal memory tasks following each treatment, improvements were noted on verbal memory tasks at the ten week follow-up.

On measures of speed of information processing, P6 showed more consistent improvements in performance following EEG biofeedback compared to cognitive rehabilitation. Participant Six's performance made further gains on all measures at the ten week follow-up assessment.

Participant Six exhibited significant variability on tests of executive functioning. Her performance on COWAT (FAS) improved following both treatments, however the change was only significant following cognitive rehabilitation. No significant changes were evident following each treatment on COWAT (animals). Following

EEG biofeedback her performance on Trails A significantly declined, while her performance on Trails B made a small non-significant improvement. This variability in P6's performance is likely to be attributable to reduced concentration as a result of increased fatigue. She did not maintain her improved performance on Trails B. Participant Six continued to maintain her improved performance only on COWAT (FAS) at the ten week follow-up assessment. Similarly, practice effects were not likely to solely explain her improved performance on COWAT (FAS).

5.2.6.4 Formal Emotional and Behavioural Assessment Results

Participant Six reported reductions in depression, anxiety, and anger symptomatology following both treatments. Improvements in depression were greater following EEG biofeedback, while improvements in state anxiety and anger expression were greater following cognitive rehabilitation. Participant Six reported greater reductions in neurobehavioural symptomatology following cognitive rehabilitation, while her significant other (husband) reported a greater reduction following only EEG biofeedback. All self-reported and significant other reports of emotional and neurobehavioural improvements were maintained at the ten week follow-up assessment.

5.2.6.5 Self-Reported Functional Changes

Participant Six reported a number of functional changes during the course of each treatment. By the end of the ten week cognitive rehabilitation program, P6 was independently using most strategies implemented. Participant Six reported being able to better manage and complete her house duties (cleaning, washing, vacuuming, etc). Due to the successful implementation of planning and organisational strategies, she

reported a reduction in her anxiety towards her administration duties for her husbands business. Participant Six indicated that she was working faster, more efficiently, and better managing her administration duties. With the implementation of the MP3 player, she indicated that she was able to remain within a shopping centre for longer periods before becoming anxious. Furthermore, she reported some improvement in her ability to manage her obsessive compulsive behaviours. During EEG biofeedback P6 reported improvement in her sleep quality, a cessation of panic in shopping centres, and a general reduction in anxiety, greater motivation, increased energy, a continued reduction in obsessive compulsive behaviours, and an improvement in her overall wellbeing. During the final two weeks of the EEG biofeedback, P6 felt greater confidence and commenced applying for additional part-time employment.

5.2.6.6 Quantitative Electroencephalogram Results

5.2.6.6.1 Post Cognitive Rehabilitation

Following 20 sessions of Cognitive Rehabilitation paired t-tests revealed some significant changes in the absolute power. Significant changes in absolute power can be observed in topographic maps (Figure 17). The P-values can be observed numerically in Table 36, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

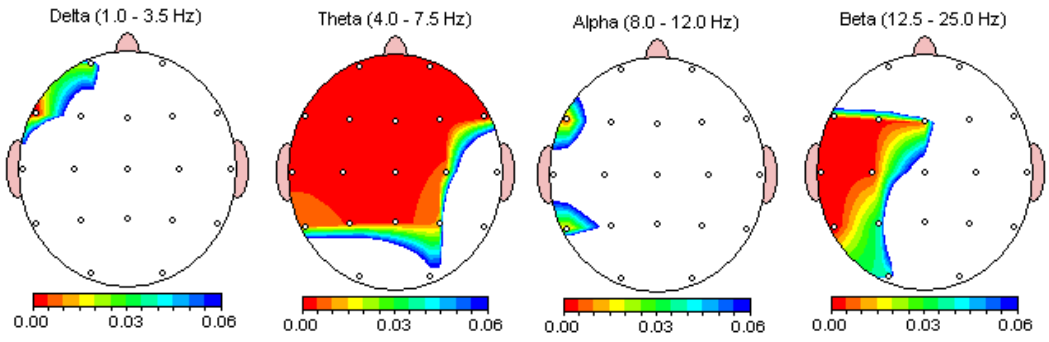

Figure 17: *P6 Topographic maps - Statistically Significant change (P-values) in absolute power*

following Cognitive Rehabilitation.

Table 36: *P6 - Statistically Significant change (P-Values) in absolute power following Cognitive Rehabilitation*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA
FP1	0.020	0.000	0.086	0.309
F3	0.106	0.000	0.138	0.002
C3	0.643	0.000	0.150	0.003
P3	0.064	0.004	0.077	0.039
O1	0.277	0.217	0.328	0.035
F7	0.000	0.000	0.007	0.000
T3	0.106	0.002	0.101	0.000
T5	0.915	0.010	0.014	0.000

Intrahemispheric: RIGHT

	DELTA	THETA	ALPHA	BETA
FP2	0.439	0.000	0.223	0.857
F4	0.433	0.000	0.394	0.259
C4	0.771	0.006	0.226	0.296
P4	0.891	0.011	0.076	0.891
O2	0.759	0.070	0.115	0.580
F8	0.100	0.000	0.172	0.856
T4	0.081	0.247	0.697	0.684
T6	0.662	0.690	0.722	0.951

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.088	0.000	0.151	0.006
Cz	0.278	0.001	0.115	0.088
Pz	0.612	0.001	0.086	0.345

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 37 demonstrates a progression towards the mean in the absolute power of Delta at eight of the 19 sites, however none of these reached statistical significance. Movement away from the mean was identified at 11 sites in Delta, with two sites reaching statistical significance. Statistically significant change away from the mean was evident at 15 of the 19 sites in the absolute power of Theta, with two sites progressing towards the mean but not reaching statistical significance. Seventeen of the 19 sites were observed to move away from the mean in the absolute power of Alpha, with only two sites reaching statistical significance, and two sites demonstrating non-significant change towards the mean, however neither reached statistical significance. Movement away from the mean in the absolute power of Beta was evident at 12 sites, with eight reaching statistical significance, and five sites demonstrated non-

significant change towards the mean.

Table 37: *P6 - Change in Z scores for Delta, Theta, Alpha and Beta pre & post Cognitive Rehabilitation.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-1.16	-1.29*	-0.36	-0.78*	-0.05	-0.19	-0.33	-0.40
Fp2	-0.88	-0.87	-0.36	-0.51*	-0.26	-0.31	-0.44	-0.43
F3	-0.69	-0.89	-0.12	-0.62*	0.01	-0.17	-0.35	-0.57*
F4	-0.70	-0.72	-0.15	-0.58*	0.05	-0.07	-0.45	-0.51
C3	-0.31	-0.37	0.11	-0.22*	0.02	-0.15	-0.09	-0.34*
C4	-0.31	-0.30	0.04	-0.12*	0.02	-0.10	-0.26	-0.30
P3	-0.47	-0.09	-0.20	-0.33*	-0.28	-0.45	-0.27	-0.37*
P4	-0.27	-0.16	-0.07	-0.17*	-0.17	-0.35	-0.33	-0.27
O1	0.10	0.34	0.36	0.36	-0.24	-0.35	-0.42	-0.53*
O2	0.38	0.54	0.86	0.73	0.07	-0.13	-0.28	-0.35
F7	-0.73	-1.13*	0.02	-0.56*	0.07	-0.20*	0.09	-0.33*
F8	-0.54	-0.67	0.14	-0.22*	0.11	0.01	-0.06	-0.05
T3	-0.81	-0.95	-0.09	-0.39*	-0.12	-0.40	-0.33	-0.82*
T4	-0.50	-0.67	-0.12	-0.12	-0.13	-0.10	-0.53	-0.49
T5	-0.36	-0.31	-0.25	-0.48*	-0.50	-0.76*	-0.49	-0.94*
T6	-0.22	-0.17	-0.10	-0.05	-0.36	-0.46	-0.53	-0.50
Fz	-0.54	-0.73	-0.22	-0.72*	-0.01	-0.21	-0.41	-0.61*
Cz	-0.40	-0.18	-0.08	-0.29*	0.02	-0.19	-0.09	-0.19
Pz	-0.45	-0.31	-0.21	-0.34*	-0.21	-0.39	-0.33	-0.33

NOTE: *Change towards '0' representing a normalisation in EEG.*

** *Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.6.6.2 Post EEG Biofeedback

Following 20 sessions of EEG biofeedback paired t-tests revealed a number of significant changes in the absolute power of Theta, Alpha, and Beta. Significant changes in absolute power can be observed in topographic maps (Figure 18). The P-values can be observed numerically in Table 38, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

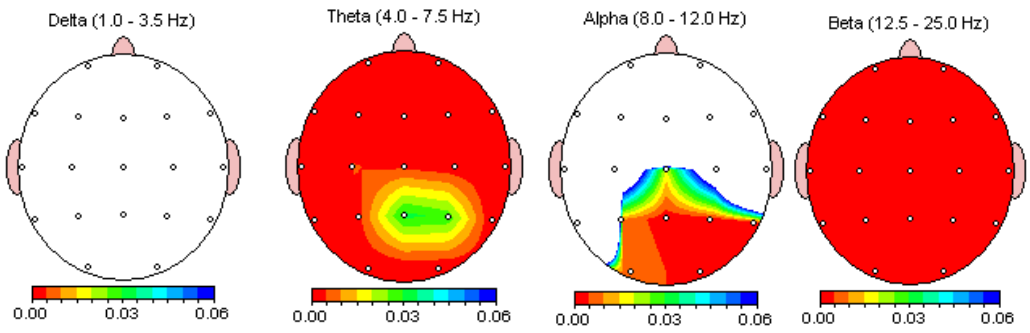

Figure 18: *P6 Topographic maps - Statistically Significant change (P-values) in absolute power following EEG biofeedback.*

Table 38: *P6 - Statistically Significant change (P-Values) in absolute power following EEG biofeedback*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA
FP1	0.316	0.000	0.290	0.000
F3	0.845	0.001	0.926	0.000
C3	0.898	0.005	0.117	0.000
P3	0.462	0.004	0.009	0.000
O1	0.343	0.000	0.010	0.000
F7	0.158	0.001	0.538	0.000
T3	0.633	0.000	0.222	0.002
T5	0.745	0.000	0.897	0.000

Intrahemispheric: RIGHT

	DELTA	THETA	ALPHA	BETA
FP2	0.887	0.000	0.509	0.000
F4	0.757	0.001	0.820	0.000
C4	0.641	0.001	0.094	0.000
P4	0.283	0.029	0.000	0.000
O2	0.122	0.000	0.000	0.001
F8	0.073	0.000	0.364	0.000
T4	0.308	0.000	0.323	0.000
T6	0.646	0.000	0.000	0.000

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.774	0.001	0.704	0.000
Cz	0.350	0.004	0.019	0.000
Pz	0.557	0.032	0.002	0.002

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 39 demonstrates no significant change in the absolute power of Delta. Statistically significant progression towards the mean in the absolute power of Theta was observed at 11 sites, and significant chance away from the mean was evident at eight sites. Nine of the 19 sites demonstrated movement towards the mean in the absolute power of Alpha, however none of these reached statistical significance. Statistically

significant movement away from the mean for Alpha was evident at seven sites. Significant change towards the mean in the absolute power of Beta was demonstrated at eight sites, with significant progression away from the mean at 11 sites.

Table 39: *P6 - Change in Z scores for Delta, Theta, Alpha and Beta pre & post EEG biofeedback.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-0.96	-1.55	-0.29	-0.66*	0.11	-0.01	-0.25	0.22**
Fp2	-0.75	-1.02	-0.30	-0.49*	-0.15	-0.18	-0.45	-0.23**
F3	-0.53	-1.04	-0.00	-0.35*	0.24	0.18	-0.20	0.17**
F4	-0.39	-1.08	0.15	-0.43*	0.29	0.25	-0.36	0.05**
C3	-0.21	-0.74	0.36	-0.05**	0.48	0.37	0.17	0.49*
C4	-0.02	-0.65	0.47	-0.14**	0.38	0.40	-0.02	0.37*
P3	-0.22	-0.82	0.37	-0.13**	0.13	0.19*	0.16	0.44*
P4	0.05	-0.54	0.41	-0.01**	0.12	0.36*	0.03	0.35*
O1	0.46	-0.19	1.31	0.30**	0.12	0.36*	-0.11	0.27*
O2	0.73	0.02	1.61	0.78**	0.28	0.69*	-0.06	0.30*
F7	-0.71	-1.41	0.06	-0.13*	0.18	0.12	-0.03	0.38*
F8	-0.41	-1.05	0.28	-0.23**	0.26	0.24	-0.11	0.25*
T3	-0.85	-1.43	0.32	-0.14**	0.22	0.09	-0.26	-0.06**
T4	-0.35	-0.91	0.24	-0.36*	0.11	0.12	-0.73	-0.18**
T5	-0.16	-0.59	0.44	-0.14**	-0.08	-0.01	-0.12	0.22*
T6	0.13	-0.15	0.47	0.10**	-0.09	0.23*	-0.44	0.20**
Fz	-0.42	-0.98	-0.05	-0.49*	0.23	0.19	-0.25	0.07**
Cz	-0.18	-0.87	0.27	-0.30*	0.35	0.41*	0.38	0.57*
Pz	-0.19	-0.75	0.26	-0.17**	0.12	0.25*	0.16	0.35*

NOTE: *Change towards '0' representing a normalisation in EEG.*

** *Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.6.6.3 Final Follow-up Assessment

From the commencement of the program (initial assessment-prior to first treatment) to the final follow-up assessment, paired t-tests revealed some significant changes in the absolute power of Delta, Theta, Alpha and Beta. Significant changes in absolute power can be observed in topographic maps (Figure 19). The P-values can be observed numerically in Table 40, with red values representing a significant increase in absolute power, and blue values indicating a significant decrease.

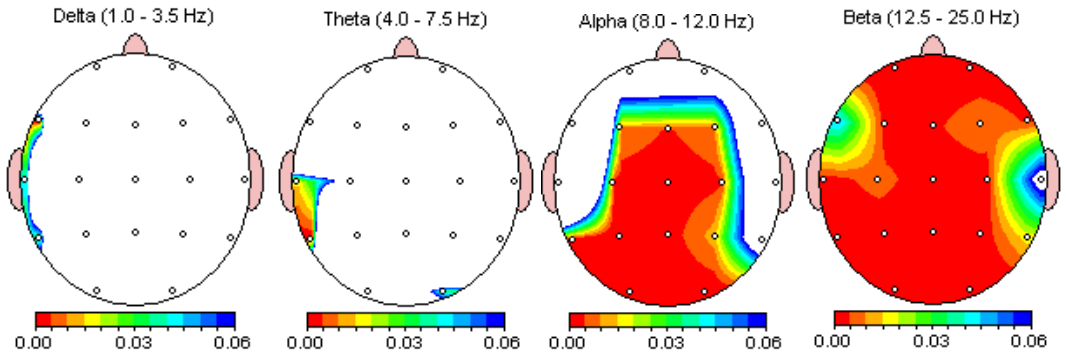

Figure 19: *P6 Topographic maps - Statistically Significant change (P-values) in absolute power Between the initial assessment and final follow-up.*

Table 40: *P6 - Statistically Significant change (P-Values) in absolute power between the initial and final follow-up assessment*

Intrahemispheric: LEFT

	DELTA	THETA	ALPHA	BETA
FP1	0.448	0.092	0.101	0.000
F3	0.715	0.132	0.010	0.000
C3	0.802	0.076	0.002	0.008
P3	0.472	0.761	0.000	0.000
O1	0.140	0.192	0.000	0.001
F7	0.000	0.375	0.305	0.046
T3	0.043	0.016	0.275	0.002
T5	0.032	0.001	0.000	0.000

Intrahemispheric: RIGHT

	DELTA	THETA	ALPHA	BETA
FP2	0.363	0.095	0.093	0.000
F4	0.713	0.157	0.009	0.010
C4	0.779	0.397	0.004	0.001
P4	0.997	0.533	0.012	0.000
O2	0.620	0.032	0.001	0.000
F8	0.629	0.083	0.129	0.004
T4	0.948	0.960	0.133	0.070
T6	0.276	0.361	0.089	0.018

Intrahemispheric: CENTER

	DELTA	THETA	ALPHA	BETA
Fz	0.816	0.173	0.004	0.004
Cz	0.313	0.353	0.001	0.000
Pz	0.529	0.353	0.002	0.000

NOTE: *RED represents a significant increase. BLUE represents a significant decrease.*

Further evaluation of the Z scores enabled the determination of whether the significant change was towards the mean (zero '0' representing the mean). Table 41 demonstrates a progression towards the mean in the absolute power of Delta at 15 of the 19 sites, with two sites reaching statistical significance. Of three sites moving away from the mean for Delta, only one reached statistical significance. Movement away from the mean was identified at 13 sites for the absolute power of Theta, with

two sites reaching statistical significance. Five of the 19 sites were observed to progress towards the mean for Theta, with one site reaching statistical significance. Of the nine sites observed to move towards the mean in the absolute power of Alpha, five sites reached statistical significance. Change away from the mean was evident at nine sites of Alpha, reaching statistical significance at seven sites. Movement towards the mean in the absolute power of Beta was evident at 14 sites, with 13 sites reaching statistical significance, and three sites demonstrated significant change away from the mean.

Table 41: *P6 - Change in Z scores for Delta, Theta, Alpha and Beta between initial and final Assessment.*

	DELTA		THETA		ALPHA		BETA	
Site	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**
Fp1	-1.16	-0.99	-0.36	-0.57	-0.05	0.05	-0.33	0.01**
Fp2	-0.88	-0.79	-0.36	-0.46	-0.26	-0.19	-0.44	-0.23**
F3	-0.69	-0.52	-0.12	-0.38	0.01	0.18*	-0.35	-0.00**
F4	-0.70	-0.44	-0.15	-0.40	0.05	0.23*	-0.45	-0.22**
C3	-0.31	-0.31	0.11	-0.21	0.02	0.29*	-0.09	0.14*
C4	-0.31	-0.05	0.04	-0.05	0.02	0.26*	-0.26	0.07**
P3	-0.47	-0.33	-0.20	-0.18	-0.28	0.07**	-0.27	0.07**
P4	-0.27	-0.15	-0.07	-0.12	-0.17	0.05**	-0.33	0.09**
O1	0.10	0.32	0.36	0.65	-0.24	0.12**	-0.42	-0.16**
O2	0.38	0.49	0.86	1.12*	0.07	0.47*	-0.28	0.18**
F7	-0.73	0.04**	0.02	0.00	0.07	0.17	0.09	-0.02**
F8	-0.54	-0.45	0.14	-0.04	0.11	0.17	-0.06	0.14*
T3	-0.81	-1.12*	-0.09	-0.42*	-0.12	-0.07	-0.33	-0.62*
T4	-0.50	-0.38	-0.12	-0.06	-0.13	-0.02	-0.53	-0.41
T5	-0.36	-0.08**	-0.25	0.15**	-0.50	0.14**	-0.49	-0.06**
T6	-0.22	-0.21	-0.10	-0.10	-0.36	-0.24	-0.53	-0.29**
Fz	-0.54	-0.34	-0.22	-0.43	-0.01	0.19*	-0.41	-0.14**
Cz	-0.40	-0.14	-0.08	-0.16	0.02	0.30*	-0.09	0.32*
Pz	-0.45	-0.30	-0.21	-0.24	-0.21	0.06**	-0.33	0.05**

NOTE: *Change towards '0' representing a normalisation in EEG.*

*** Significant Change towards normalisation (0).*

**Significant Change away from normalisation (0).*

5.2.6.7 Results Summary of Participant Six

Overall, Participant Six showed a number of improvements following each treatment

across all cognitive domains. However, it appears that cognitive improvements were made more consistently following EEG biofeedback then cognitive rehabilitation, particularly in attentional functioning and speed of information processing. Importantly, at the ten week follow-up assessment P6 maintained and/or made further gains in her performance in all cognitive domains, even when improvements were not evident following individual treatments. However, her increased fatigue during each assessment session was likely to have had some impact on her performance and consequently the results. Both treatments were effective in improving emotional symptomatology, with greater improvements in depression following EEG biofeedback, and in anxiety and anger following cognitive rehabilitation. Participant Six reported greater reductions in neurobehavioural symptomatology following cognitive rehabilitation, while her significant other reported greater reductions following EEG biofeedback. All self-reported and significant other reports of emotional and neurobehavioural improvements were maintained at the ten week follow-up assessment. Participant Six reported a number of significant functional gains following each treatment. Finally, P6 made significant changes towards normalisation in her EEG following EEG biofeedback in the frequencies trained (Theta and Beta), however no significant change towards normalisation was observed following cognitive rehabilitation. Significant change towards normalisation was evident across all frequency bands at the ten week follow-up assessment, particularly in the Alpha and Beta range.

RESULTS

PART TWO

5.3 GROUP RESULTS

5.3.1 Examination of Treatment Order

Mann-Whitney U tests were used to determine if the treatment order affected the participants' performance on cognitive, emotional, and behavioural measure. There were no significant differences in treatment order for any measure (see Appendix 11).

5.3.2 Neuropsychological Assessment Results

See Appendix 12 for Wilcoxon descriptive statistics.

5.3.2.1 Attention

5.3.2.1.1 Test of Variables of Attention

5.3.2.1.1.1 Omissions

The Wilcoxon test revealed no statistically significant difference between the two treatments ($Z = -0.74$, $p = 0.46$). The group's difference scores between pre-post assessments for each treatment are highlighted in Figure 20. The SE_M for the TOVA omissions is ± 5.61 (Leark, et al, 2004). The bar graph suggests that two participants (1 & 4) were able to reduce the amount of omission errors following EEG biofeedback, however only one participant produced clinically significant change. These two participants (1 & 4) also demonstrated clinically significant change between the initial and final follow-up assessment. Two participants (3 & 4) were able to reduce omission errors following cognitive rehabilitation, but the change was

not significant. For participant 5, omission errors significantly increased following cognitive rehabilitation. Three (1, 3, & 4) of the five participants demonstrated reductions in omission errors between their initial assessment and final follow-up assessment, however, only two (1 & 4) reached clinical significance. Overall, the trend of the graph would suggest that the participants' level of clinically significant change in omission errors post treatments, and at the 10 week follow-up, was quite variable.

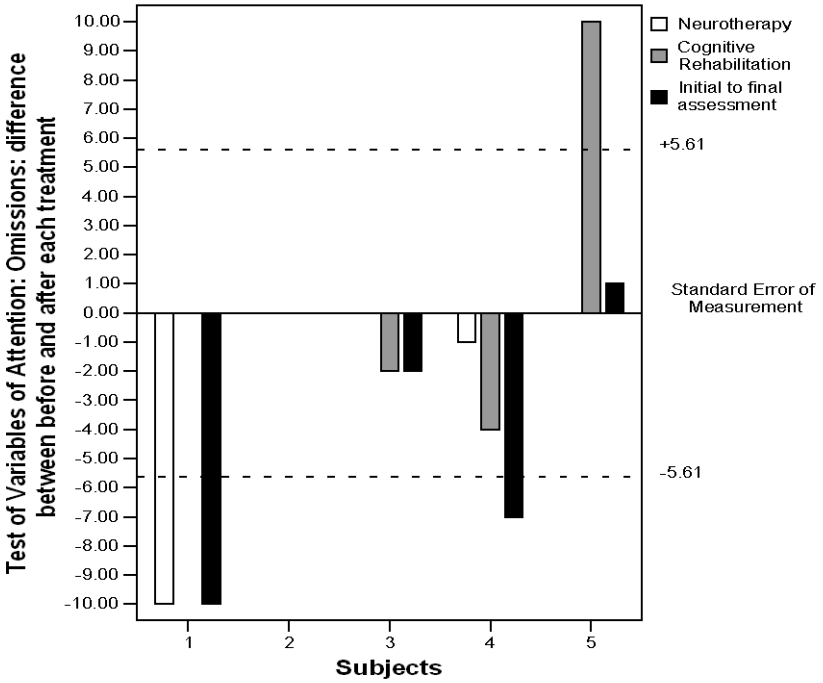

Figure 20: *TOVA (omissions) - Difference between pre-post assessments following each treatment.*

5.3.2.1.1.2 Commissions

The Wilcoxon test revealed no statistically significant difference between the two treatments (Z = -1.48, p = 0.14). The difference scores between pre-post assessments for each treatment are highlighted in Figure 21. The SE_M for the TOVA commissions is ± 7.65 (Leark, et al, 2004). The bar graph suggests that four of the five participants (1, 2, 3, & 4) were able to reduce the amount of commission errors following EEG

biofeedback, with three producing clinically significant change. One participant (5) increased in commission errors following EEG biofeedback, but this was not significant. Following cognitive rehabilitation two participants (2 & 5) significantly increased in commission errors, and two (3 & 4) demonstrated non-significant reductions in commission errors. Three (1, 2, & 4) of the five participants demonstrated reductions in commission errors between their initial assessment and final follow-up assessment. However, only two (1 & 2) reached clinical significance. Overall, the trend of the graph would suggest that EEG biofeedback as compared to cognitive rehabilitation was more consistent in producing clinically significant change, and more effective in reducing commission errors.

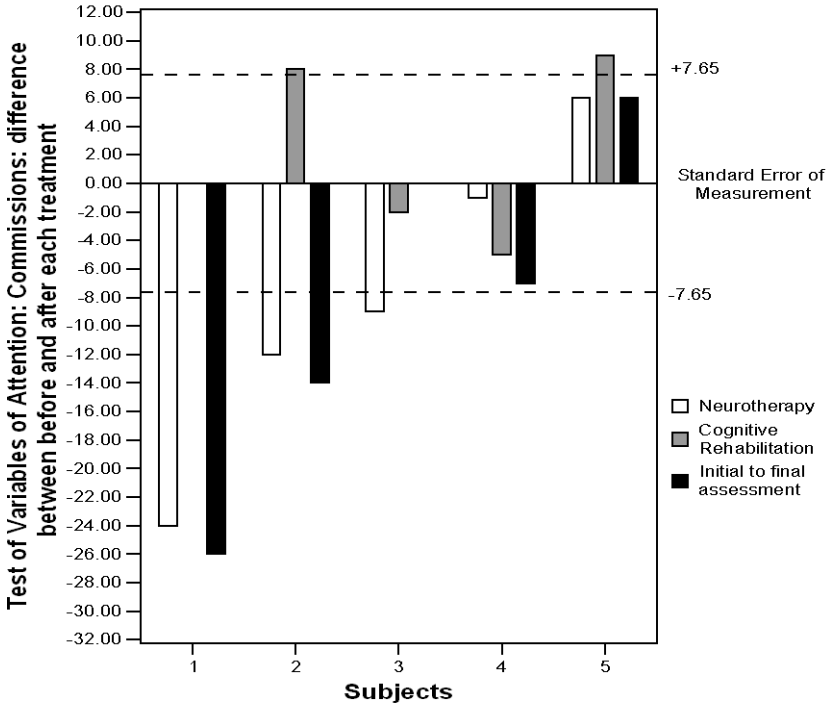

Figure 21: *TOVA (commissions) - Difference between pre-post assessments following each treatment.*

5.3.2.1.1.3 Response Time

The Wilcoxon test revealed no statistically significant difference between the two

treatments (Z = -0.41, p = 0.69). The difference scores between pre-post assessments for each treatment are highlighted in Figure 22. The SE_M for the TOVA response time is ± 6.87 (Leark, et al, 2004). The bar graph suggests that following EEG biofeedback two participants (1 & 5) made clinically significant improvements in their response times, and two (3 & 4) significantly slowed in their response. Similarly, following cognitive rehabilitation two participants (2 & 4) made clinically significant improvements, and two (3 & 5) significantly slowed. Four out of the five (1, 3, 4, & 5) made clinically significant improvements in response time between their initial assessment and final follow-up assessment. Overall, the trend of the graph would suggest that the participants' level of clinically significant change in response time post treatments was quite variable. However, most of the participants made clinically significant improvements between the initial assessment and final follow-up assessment.

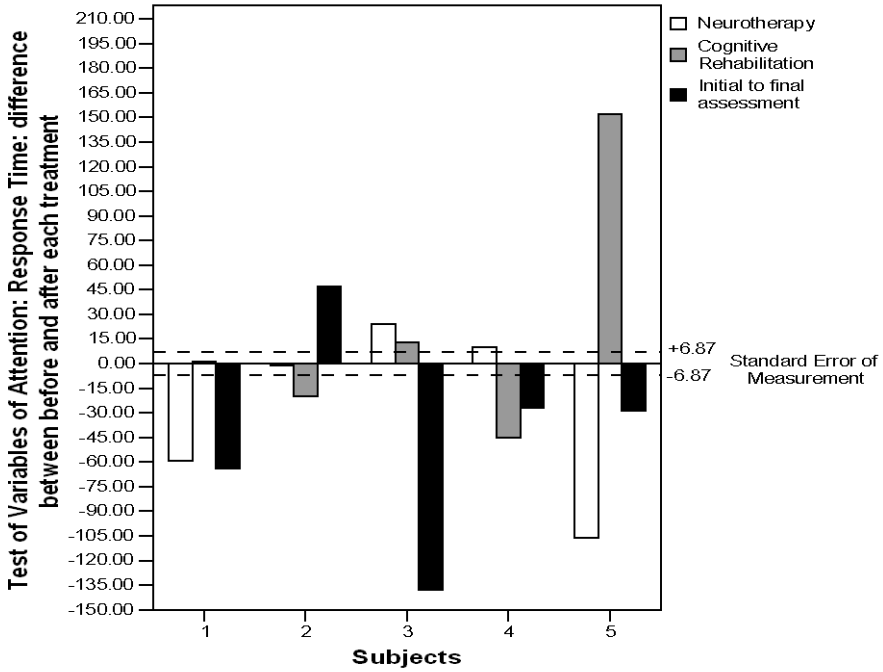

Figure 22: *TOVA (response time) - Difference between pre-post assessments following each treatment.*

5.3.2.1.1.4 Variability

Wilcoxon revealed no statistically significant difference between the two treatments ($Z = -0.67$, $p = 0.5$). The difference scores between pre-post assessments for each treatment are highlighted in Figure 23. The SE_M for TOVA variability is ± 5.41 (Leark, et al, 2004). The bar graph suggests that following EEG biofeedback, four out of the five participants (1, 3, 4, & 5) reduced their variability, with three reaching clinically significant change. Similarly, following cognitive rehabilitation four out of the five participants (1, 2, 3, & 4) demonstrated a reduction in variability, and three of these were clinically significant. Four out of the five (1, 3, 4, & 5) made clinically significant reductions in variability between their initial assessment and final follow-up assessment. Overall, the trend of the graph would suggest that both treatments were consistent and effective in reducing the amount of variability in the participants' performance. A majority of the participants made clinically significant improvements between the initial assessment and final follow-up assessment.

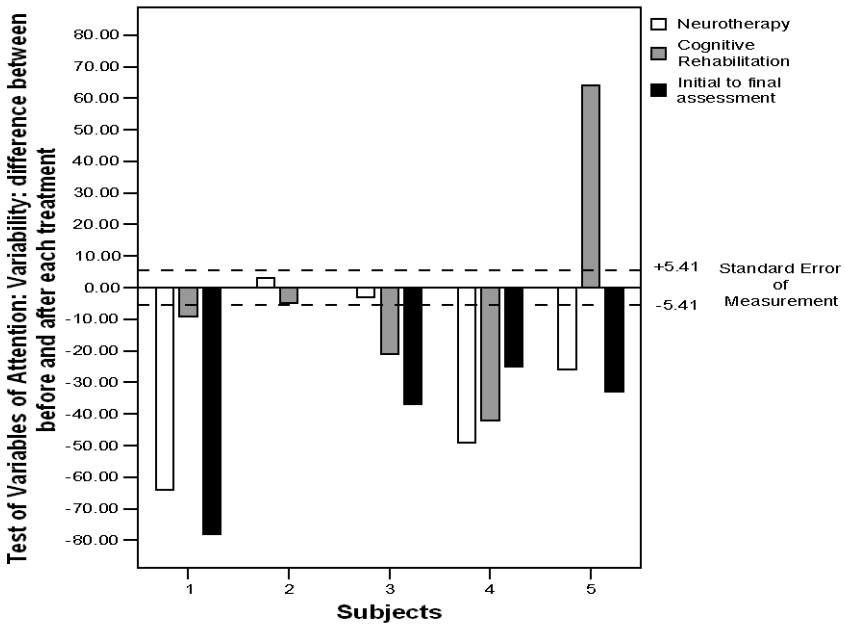

Figure 23: *TOVA (variability) - Difference between pre-post assessments following each treatment.*

5.3.2.1.1.5 Paced Auditory Serial Addition Test

The Wilcoxon test revealed no statistically significant difference between the two treatments (Z = -0.41, p = 0.69). The difference scores between pre-post assessments for each treatment are highlighted in Figure 24. No measurement error scores were available for the PASAT. The bar graph suggests that four of the five participants (1, 2, 3, & 5) made improvements in their performance following EEG biofeedback, and one participant (4) did not. Three participants (2, 4, & 5) made improvements following cognitive rehabilitation, and the two (1 & 3) did not. Four of the five participants (2, 3, 4, & 5) demonstrate improvements between their initial assessment and final follow-up. Overall, the trend of the graph would suggest that most of the participants' performances improved consistently following both treatments. Most of the participants also made improvements between the initial assessment and final follow-up assessment.

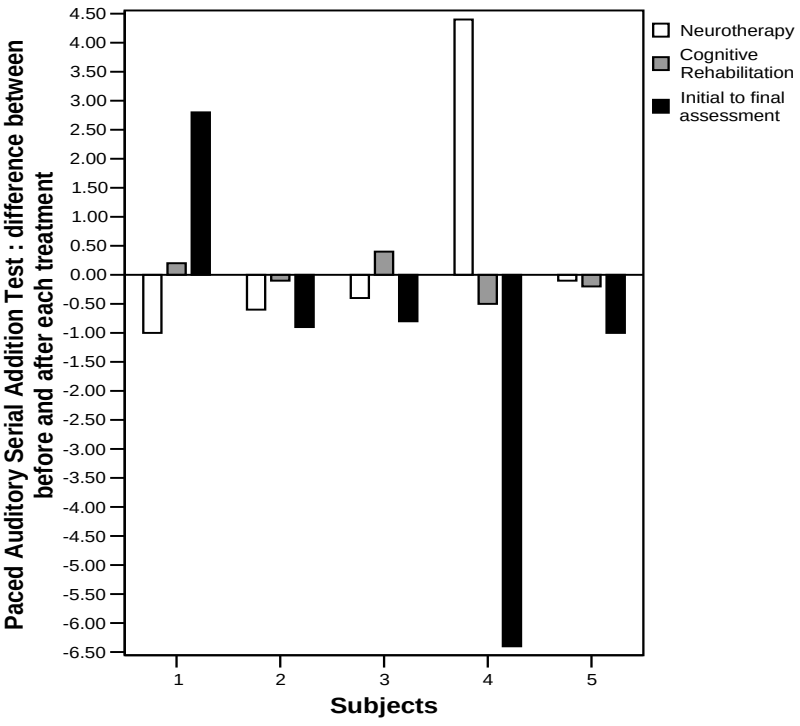

Figure 24: *PASAT - Difference between pre-post assessments following each treatment.*

5.3.2.2 Memory

5.3.2.2.1 Rey Auditory Verbal Learning Test

5.3.2.2.1.1 Total Recall

The Wilcoxon test revealed no statistically significant difference between the two treatments ($Z = -1.08$, $p = 0.28$). The difference scores between pre-post assessments for each treatment are highlighted in Figure 25. Moritz et al (2003) reported the SE_M to be 3.22 at time one, and 4.5 at time two. The SE_{DIFF} was 5.54. The bar graph suggests that two participants (2 & 4) made improvements in the total number of words recalled following EEG biofeedback (one reaching clinical significance), and three participants (1, 3, &, 5) declined in their performance (two reaching clinical significance). Two participants (1 & 3) demonstrated clinically significant improvements in total word recall following cognitive rehabilitation, and two participants (2 & 4) demonstrated a non-significant decline. Three of the five participants (2, 3, & 5) made improvements in their performance between initial and final follow-up assessment (two reaching clinical significance). Overall, the trend of the graph would suggest that the participants' level of change post treatments and at the ten week follow-up was quite variable.

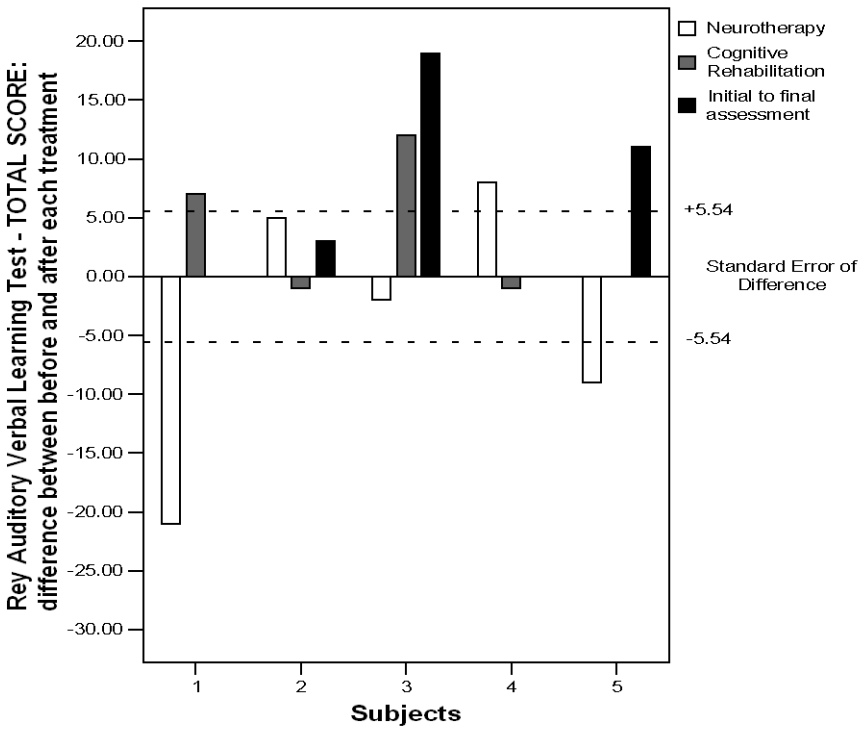

Figure 25: *RAVLT (Total Recall) - Difference between pre-post assessments following each treatment.*

5.3.2.2.1.2 Delayed Recall

The Wilcoxon test revealed no statistically significant difference between the two treatments (Z = -0.18, p = 0.85). The difference scores between pre-post assessments for each treatment are highlighted in Figure 26. No measurement error scores were available for the RAVLT (delayed recall). The bar graph suggests that following both treatments two participants were able to recall more words following, and two participants' performance declined. When analysing the change between initial and final follow-up assessments, one participant (1) declined in their performance, and two (3 & 5) improved in their delayed word recall ability. Overall, the trend of the graph would suggest that the participant's level of change post treatments and at ten week follow-up was variable.

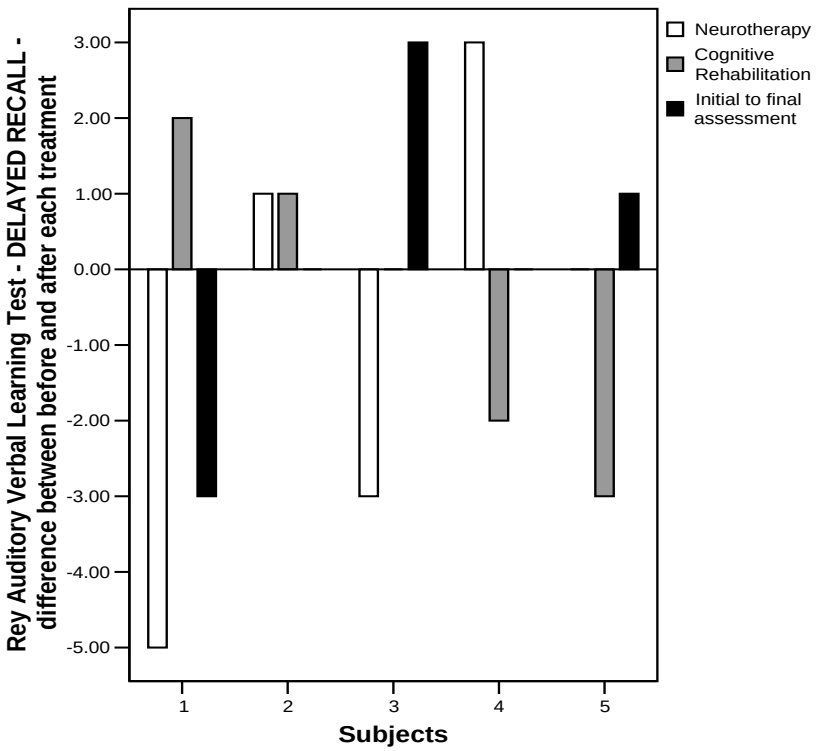

Figure 26: *RAVLT (Delayed Recall) - Difference between pre-post assessments following each treatment.*

5.3.2.2.1.3 Recognition

The Wilcoxon test revealed no statistically significant difference between the two treatments (Z = -0.55, p = 0.58). The difference scores between pre-post assessments for each treatment are highlighted in Figure 27. No measurement error scores were available for the RAVLT (recognition). The bar graph suggests that following EEG biofeedback three participants' (3, 4, & 5) recognition improved, one participant's (1) performance declined, and one (2) demonstrated no change. Following cognitive rehabilitation, only one participant (1) showed improvement in recognition ability, one participant's (5) performance declined, and no change was detected in the remaining participants (2, 3, & 4). Change was consistently detected in recognition ability between the initial and final follow-up assessment in four of the five participants. Overall, the trend of the graph is suggestive of only small improvements

in recognition performance. These changes are more consistently observed following EEG biofeedback compared to cognitive rehabilitation, and occur in the majority of participants between the initial and final follow-up assessment.

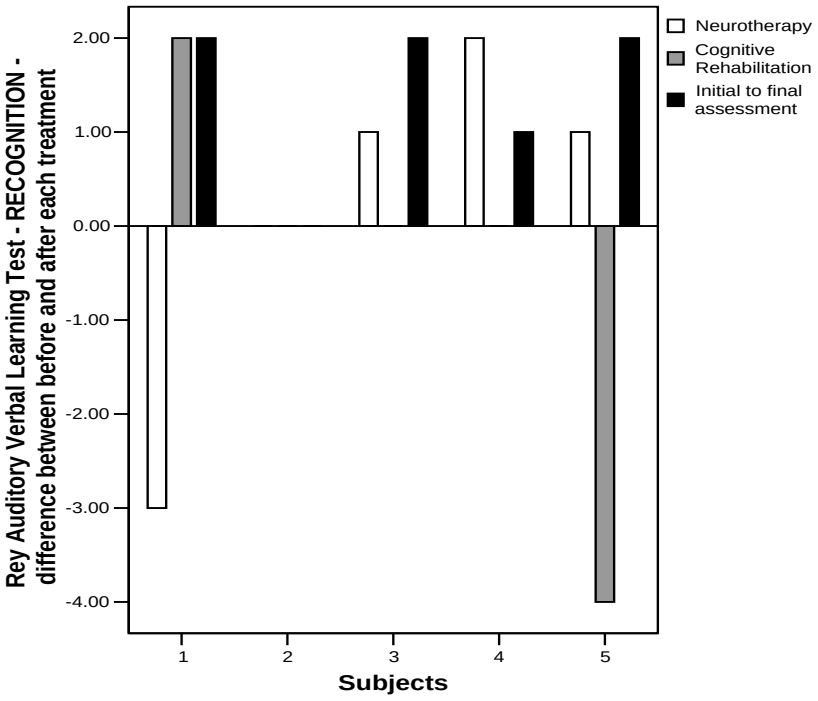

Figure 27: *RAVLT (Recognition) - Difference between pre-post assessments following each treatment.*

5.3.2.2.2 Rey-Osterrieth Complex Figure

5.3.2.2.2.1 Copy

The Wilcoxon test revealed no statistically significant difference between the two treatments (Z = -1.63, p = 0.10). The difference scores between pre-post assessments for each treatment are highlighted in Figure 28. No measurement error scores were available for the RCF copy. The bar graph is suggestive of change in four of the five participants (1, 2, 3, & 5) following EEG biofeedback, whereby their ability to copy the RCF declined. Only one participant (4) made small improvements following EEG biofeedback. Following cognitive rehabilitation three participants (1, 3, & 5)

made improvements in their copy of the RCF. Performances declined in participants (1, 2, 4, & 5) between the initial and final follow-up assessment. Overall, the trend of the graph suggests that improvements were more consistently identified following cognitive rehabilitation compared to EEG biofeedback, however there was a consistent decline in the participants' performance between initial and final follow-up assessments. Furthermore, all change noted was small.

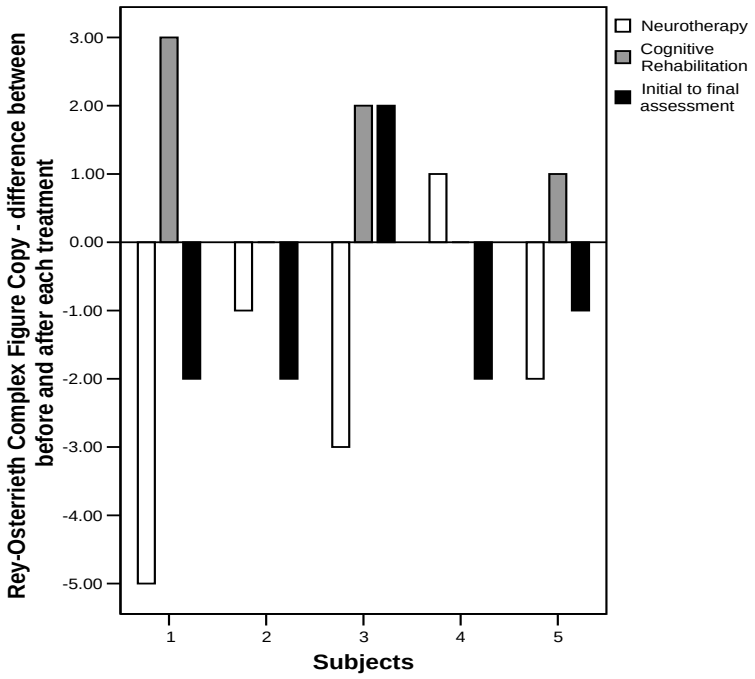

Figure 28: *Rey-Osterrieth Complex Figure (Copy) - Difference between pre-post assessments following each treatment.*

5.3.2.2.2.2 Delayed Recall (3 minute)

The Wilcoxon test revealed no statistically significant difference between the two treatments (Z = -1.07, p = 0.29). The difference scores between pre-post assessments for each treatment are highlighted in Figure 29. No measurement error scores were available for the RCF delayed recall. The bar graph suggests that three participants (2, 3, & 5) were able to recall more detail following EEG biofeedback, while two

participants' (1 & 4) performance declined. Four participants (2, 3, 4, & 5) improved in their visual recall following cognitive rehabilitation, with only one participant's (1) performance declining. These four participants (2, 3, 4, & 5) all demonstrated greater improvements between the initial and final follow-up assessment. Overall, the trend of the graph would suggest that participants' performance in visual memory improved following both treatments, with more consistent improvements noted following cognitive rehabilitation. Most of the participants also made further gains at the final follow-up assessment.

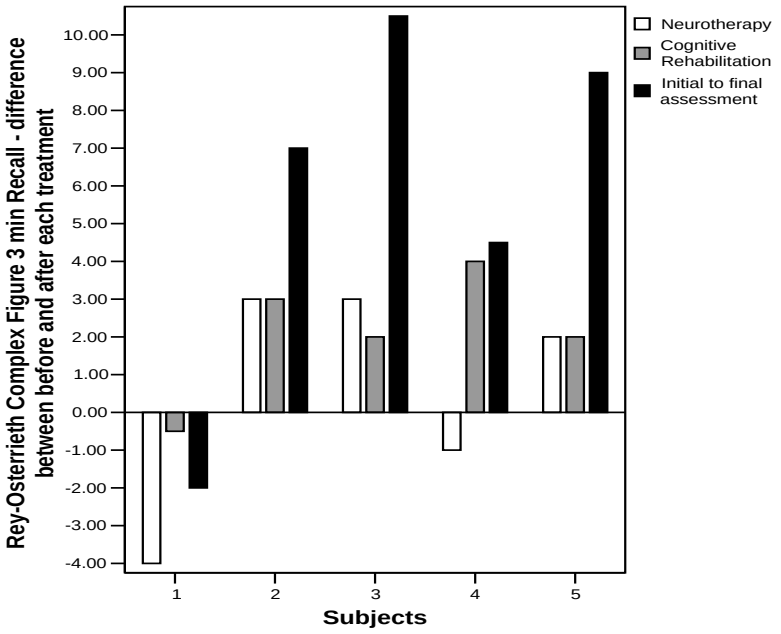

Figure 29: *Rey-Osterrieth Complex Figure (Recall) - Difference between pre-post assessments following each treatment.*

5.3.2.3 Speed of Information Processing

5.3.2.3.1 Symbol Search

The Wilcoxon test revealed no statistically significant difference between the two treatments (Z = -0.41, p = 0.69). The difference scores between pre-post assessments

for each treatment are highlighted in Figure 30. The SE_M for symbol search is ± 1.27 *(The Psychological Corporation, 1997).* The bar graph suggests that following EEG biofeedback three participants' (3, 4, & 5) speed of processing slowed, however this was only clinically significant in two cases. One participant (1) made a clinically significant improvement following EEG biofeedback. Following cognitive rehabilitation two participants' (1 & 2) performance slowed, the change in one of these was clinically significant. Three participants (3, 4, &, 5) improved following cognitive rehabilitation, this change being clinically significant in two participants. Two participants (3 & 5) demonstrated clinically significant improvements between the initial and final follow-up assessments. Overall, the trend of the graph suggests that the participants' level of change post treatments and at the final follow-up was quite variable.

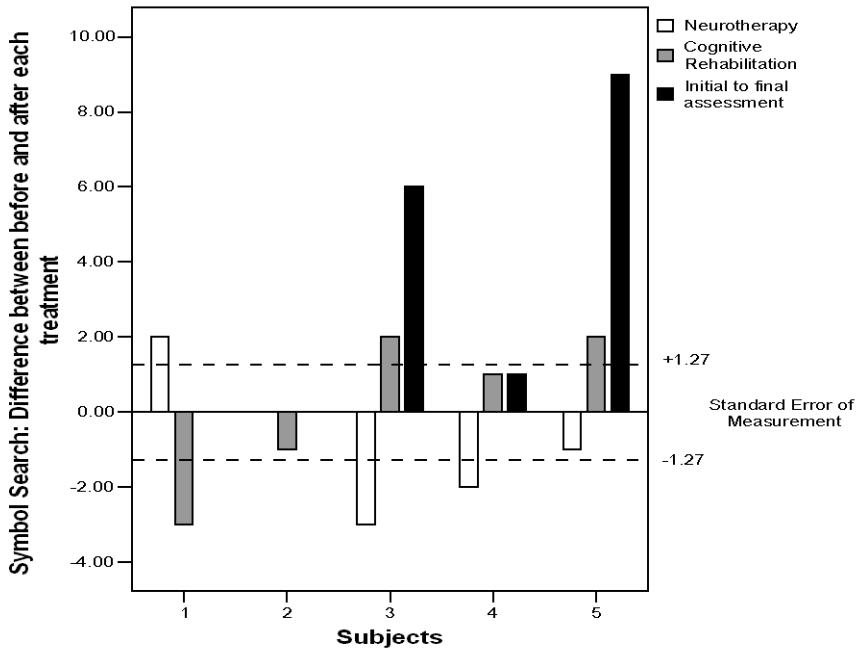

Figure 30: *Symbol Search - Difference between pre-post assessments following each treatment.*

5.3.2.3.2 Speed and Capacity of Language Processing Test

The Wilcoxon test revealed no statistically significant difference between the two treatments (Z = -1.36, p = 0.18). The difference scores between pre-post assessments for each treatment are highlighted in Figure 31. No measurement error scores were available for the SCOLP. The bar graph suggests that following EEG biofeedback all five participants made improvements in their speed of language processing. Following cognitive rehabilitation three participant's (2, 3, & 4) performance improved, however two participants' (1 & 5) performance declined. Four of the five participants appeared to have made even greater gains at the final follow-up assessment. Overall, the trend of the graph suggests that EEG biofeedback yielded more consistent improvements in speed of language comprehension than cognitive rehabilitation. The amount of change observed at the final follow-up assessment is greater than in the two treatments alone.

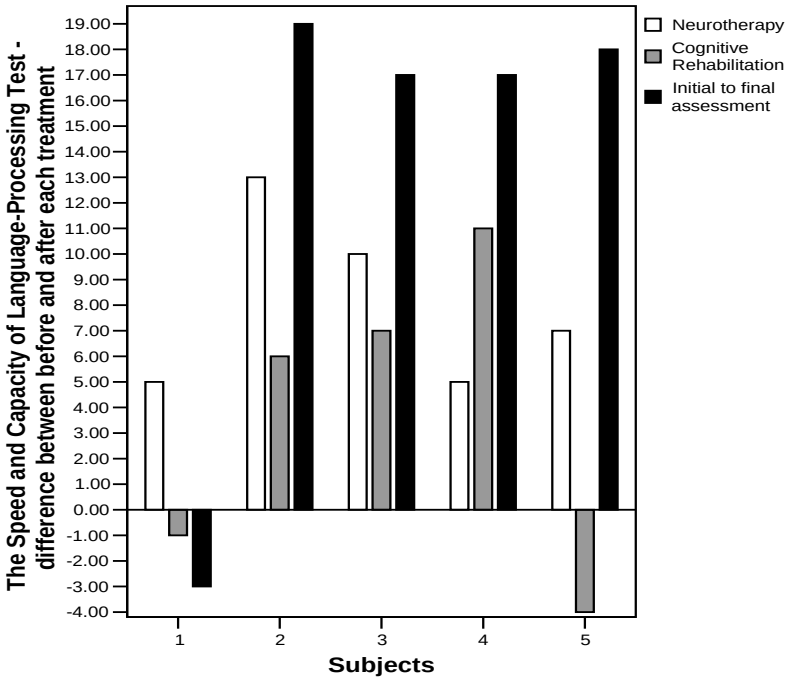

Figure 31: *SCOLP - Difference between pre-post assessments following each treatment.*

5.3.2.4 Executive Functioning

5.3.2.4.1 Controlled Oral Word Association Test

5.3.2.4.1.1 Phonemic – FAS

The Wilcoxon test revealed no statistically significant difference between the two treatments ($Z = -0.73$, $p = 0.47$). The difference scores between pre-post assessments for each treatment are highlighted in Figure 32. Harrison et al (2000) reported on the SE_P for the phonemic fluency of FAS (± 7.15). The bar graph suggests that following EEG biofeedback two participants (1 & 5) made non-significant improvements in word production, and one participant's (2) performance significantly declined. Following cognitive rehabilitation two participants demonstrated improvements (2 & 5), with one being clinically significant, and two participants (1 & 3) demonstrated a non-significant decline. All five participants improved in their ability to produce words between the initial and final follow-up assessments, with two (1 & 5) reaching clinical significance. Overall, the trend of the graph suggests that the participants' level of change post treatments was quite variable. However, all five participants' performances made further gains through out the course of the program (between initial and final assessment).

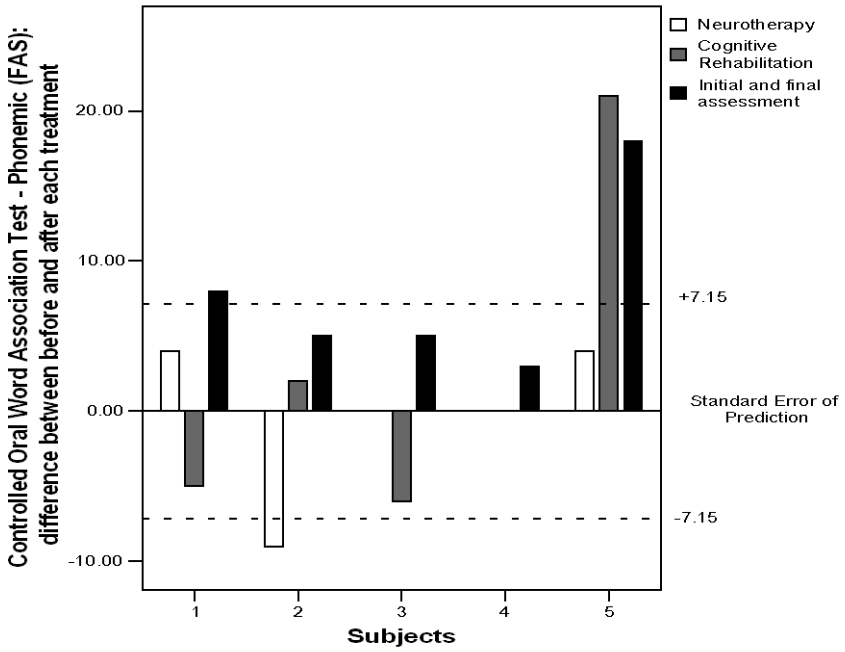

Figure 32: *COWAT (FAS) - Difference between pre-post assessments following each treatment.*

5.3.2.4.1.2 Semantic – Animals

The Wilcoxon test revealed no statistically significant difference between the two treatments (Z = -0.54, p = 0.59). The difference scores between pre-post assessments for each treatment are highlighted in Figure 33. Harrison et al (2000) reported on the SE_P for the semantic category of animals (± 4.33). The bar graph suggests that following EEG biofeedback three participants (1, 2, & 5) made improvements (one reaching clinical significance), and two participants (3 & 4) demonstrated a non-significant decline. Two participants (2 & 4) made improvements following cognitive rehabilitation (one reaching clinical significance), and the performance in one participant (1) significantly declined. All participants' performances improved between initial and final follow-up assessments, but only one participant demonstrated clinically significant change. Overall, the trend of the graph suggests that the participants' level of change post treatments was quite variable. However, all five participants made further gains at the final follow-up assessment.

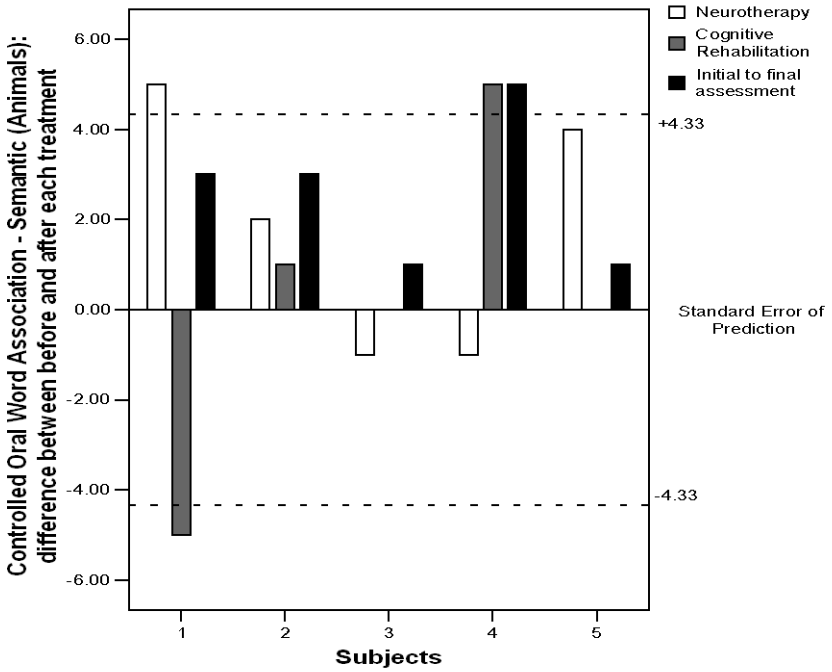

Figure 33: *COWAT (animals) - Difference between pre-post assessments following each treatment.*

5.3.2.4.2 Trail Making Test

5.3.2.4.2.1 Part A

The Wilcoxon test revealed no statistically significant difference between the two treatments (Z = -0.41, p = 0.69). The difference scores between pre-post assessments for each treatment are highlighted in Figure 34. Basso et al (1999) demonstrated a SE_P of ± 6.80 for Trails A. The bar graph suggests that following EEG biofeedback two participants (1 & 3) improved in their speed of processing (one reaching clinical significance), however two participants (4 & 5) significantly slowed in their performance. Following cognitive rehabilitation the performance of three participants (2, 4, & 5) improved (one reaching clinical significance), and two participants (1 & 3) declined (one reaching clinical significance). Four of the five participants' performances improved between the initial and final follow-up assessment, and two of these reached clinical significance. Overall, the trend of the graph suggests that the

participants' level of change post treatments was quite variable. However, most participants made further gains by the final follow-up assessment.

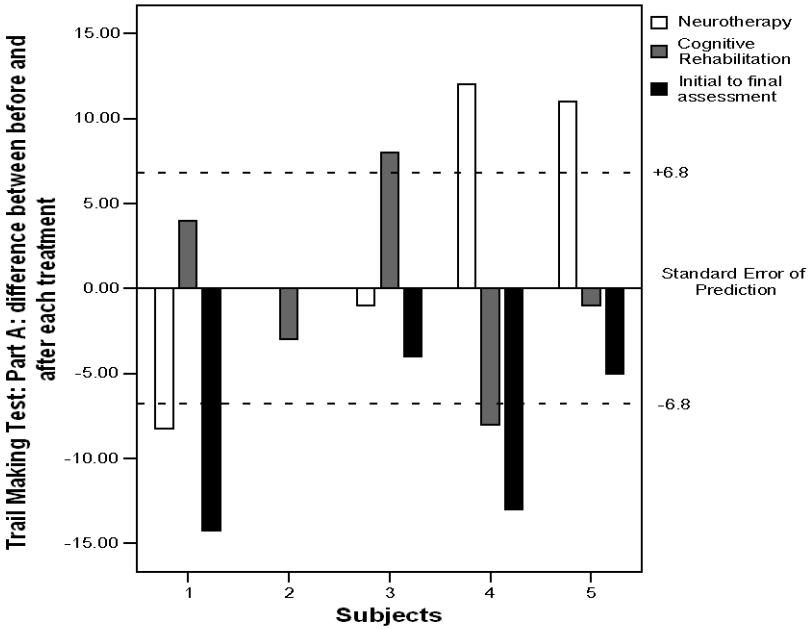

Figure 34: *TRAILS (Part A) - Difference between pre-post assessments following each treatment.*

5.3.2.4.2.2 Part B

The Wilcoxon test revealed a statistically significant difference ($p < .05$) between the two treatments ($Z = -2.02$, $p = 0.04$). However, when applying the Bonferroni correction no significant difference ($p > 0.0025$) could be identified. The difference scores between pre-post assessments for each treatment are highlighted in Figure 35. Basso et al (1999) demonstrated a SE_P of ± 14.85 for Trails B, and Heaton et al (2001) reported a SE_{DIFF} of ±18.6 for Trails B. The trend of the bar graph demonstrates that all five participants demonstrated improvements following EEG biofeedback, with three participants reaching clinical significance. Following cognitive rehabilitation three participants (2, 3, & 5) made non-significant improvements, and two participants' (1 & 4) performances declined (one reaching clinical significance). Four of the five participants' (1, 2, 3, & 4) performances

improved from the commencement to completion of the program. Overall, EEG biofeedback was more consistent and effective than cognitive rehabilitation at improving performance on the Trails B. A majority of the participants' made further gains at the follow-up assessment.

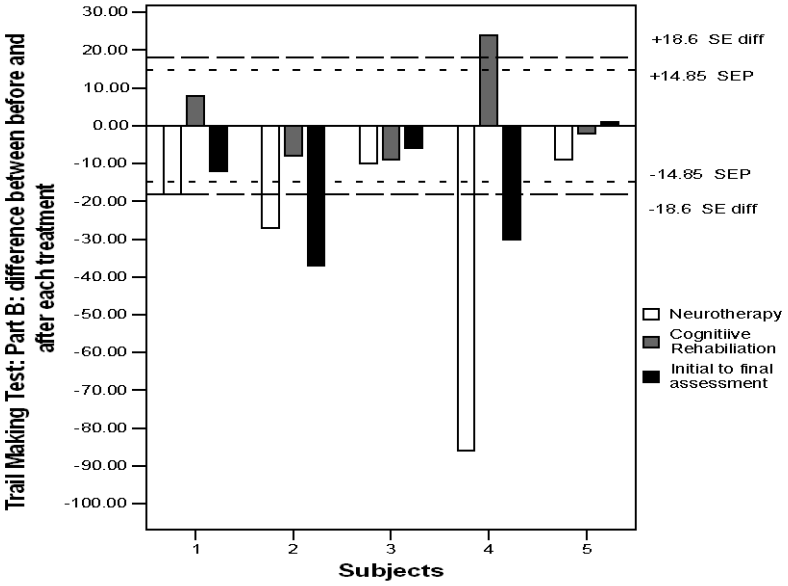

Figure 35: *TRAILS (Part B) - Difference between pre-post assessments following each treatment.*

5.3.3 Emotional and Behavioural Assessment Results

See Appendix 12 for Wilcoxon descriptive statistics.

5.3.3.1 Beck Depression Inventory – Second Edition

The Wilcoxon test revealed no statistically significant difference between the two treatments ($Z = -1.48$, $p = 0.14$). The difference scores between pre-post assessments for each treatment are highlighted in Figure 36. The bar graph suggests that all participants were able to reduce depressive symptomatology following both treatments. However, on close examination a greater reduction in symptom reporting can be observed following EEG biofeedback as compared to cognitive rehabilitation. Four of the five participants also made substantial reductions in depressive symptomatology at the final follow-up assessment. Overall, the trend of the graph suggests that both treatments were effective in reducing depressive symptomatology, with greater reductions observed in most participants following EEG biofeedback. Most participants reported less depressive symptoms at the final ten week follow-up assessment.

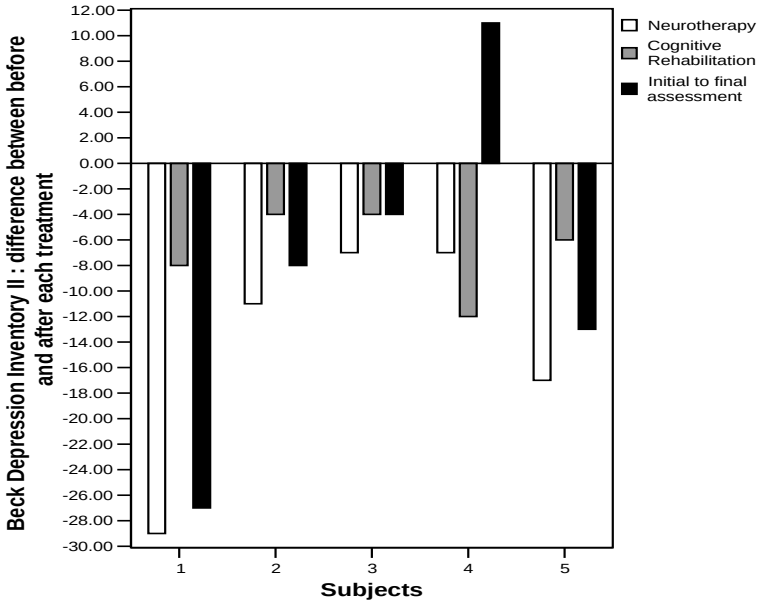

Figure 36: *BDI-II - Difference between pre-post assessments following each treatment.*

5.3.3.2 State Trait Anxiety Inventory

5.3.3.2.1 State anxiety

The Wilcoxon test revealed no statistically significant difference between the two treatments (Z = -0.96, p = 0.34). The difference scores between pre-post assessments for each treatment are highlighted in Figure 37. The bar graph suggests that reductions in state anxiety can be observed in all participants following EEG biofeedback, and in four of the five participants following cognitive rehabilitation. Four of the five participants reduced state anxiety at the final follow-up assessment. Overall, the trend of the graph suggests that both treatments were equally effective in reducing state anxiety, and most participants continued to report reductions in anxiety at the final follow-up.

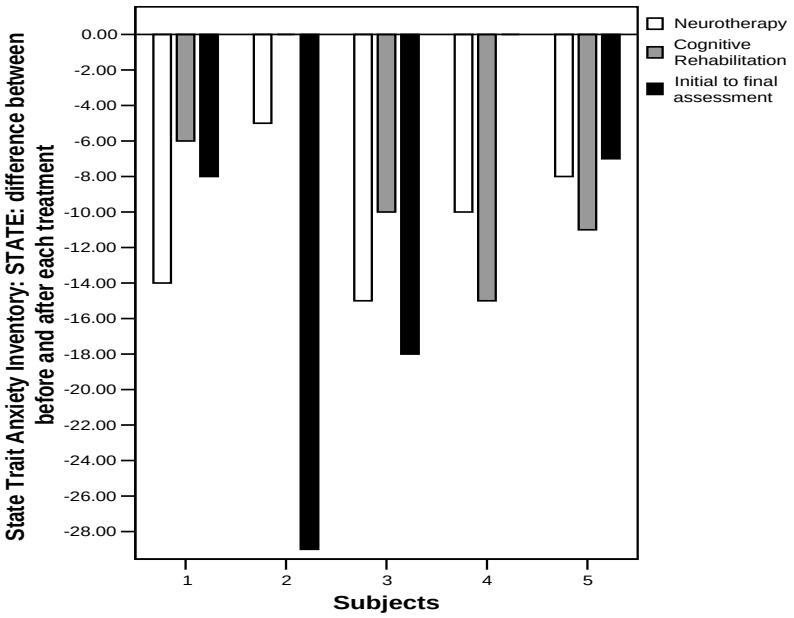

Figure 37: *STAI (State) - Difference between pre-post assessments following each treatment.*

5.3.3.2.2 Trait anxiety

The Wilcoxon test revealed no statistically significant difference between the two treatments (Z = -0.14, p = 0.89). The difference scores between pre-post assessments for each treatment are highlighted in Figure 38. The bar graph suggests that reductions in trait anxiety can be observed in all participants following cognitive rehabilitation, and in four of the five participants following EEG biofeedback. Four of the five participants reduced trait anxiety at the final follow-up assessment. Overall, the trend of the graph suggests that both treatments were equally effective in reducing trait anxiety, and a majority of the participants continued to report reductions at the follow-up assessment.

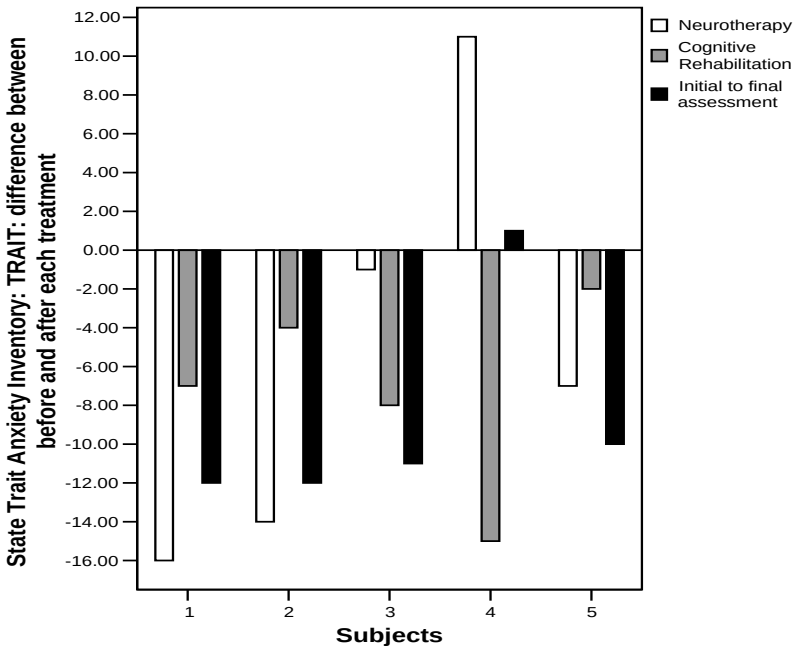

Figure 38: *STAI (Trait) - Difference between pre-post assessments following each treatment.*

5.3.3.3 State Trait Anger Expression Inventory-Second Edition

5.3.3.3.1 State Anger

The Wilcoxon test revealed no statistically significant difference between the two

treatments (Z = -0.37, p = 0.71). The difference scores between pre-post assessments for each treatment are highlighted in Figure 39. The bar graph suggests that a reduction in state anger was observed in three of the participants (1, 3, & 4) following EEG biofeedback, and also in three of the participants (2, 3, &, 4) following cognitive rehabilitation. One participant (3) demonstrated a reduction and one (4) an increase in state anger at the final follow-up assessment. Overall, the trend of the graph suggests that both treatments were effective in reducing state anger, but little change was demonstrated at the final follow-up.

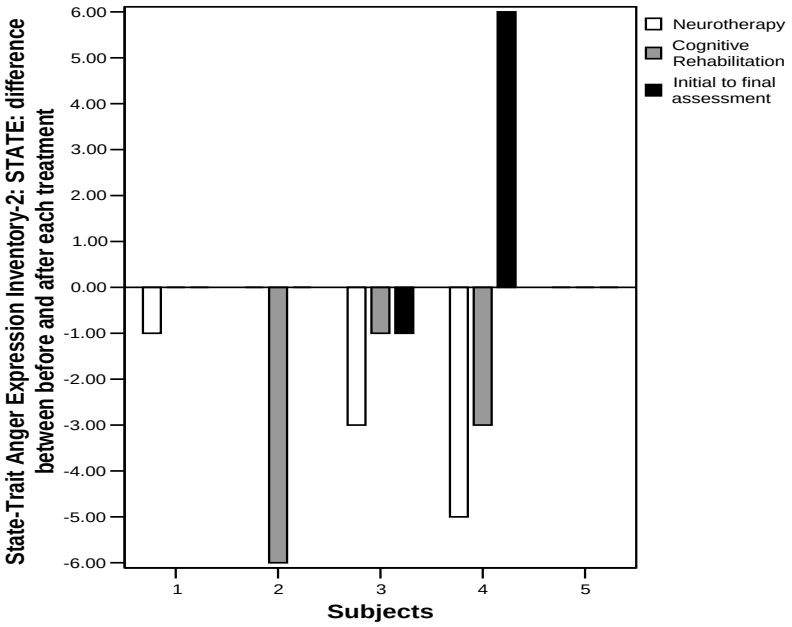

Figure 39: *STAXI-II (State) - Difference between pre-post assessments following each treatment.*

5.3.3.3.2 Trait Anger

The Wilcoxon test revealed no statistically significant difference between the two treatments (Z = -0.73, p = 0.47). The difference scores between pre-post assessments for each treatment are highlighted in Figure 40. The bar graph suggests that a reduction in trait anger was observed in three participants following both treatments. Small increases in trait anger were observed in two participants (3 & 4) following

EEG biofeedback and in one participant (3) following cognitive rehabilitation. At the ten week follow-up assessment, two participants (2 & 5) had reduced trait anger, while one participant (3) had increased trait anger. Overall, the trend of the graph suggests that both treatments were effective in reducing trait anger in a majority of participants, but change was not consistently maintained at follow-up.

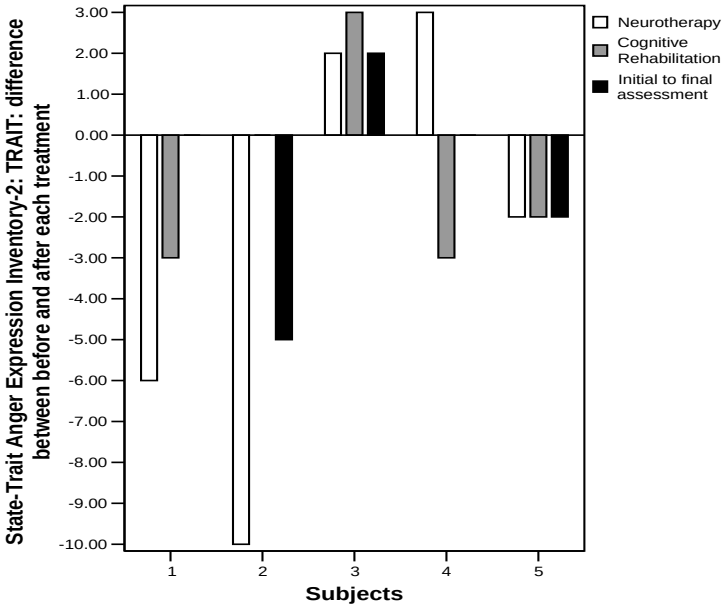

Figure 40: *STAXI-II (Trait) - Difference between pre-post assessments following each treatment.*

5.3.3.3.3 Anger Expression Index

The Wilcoxon test revealed no statistically significant difference between the two treatments ($Z = -0.81$, $p = 0.42$). The difference scores between pre-post assessments for each treatment are highlighted in Figure 41. The trend of the bar graph demonstrates improvements in the control of anger expression in four of the participants (1, 2, 4, & 5) following both EEG biofeedback and cognitive rehabilitation. At the final follow-up assessment, four of the participants (1, 3, 4, & 5) demonstrated better control of their anger expression. Overall, the trend of the graph suggests that both treatments were effective in improving the control of anger

expression, and this was maintained at the final follow-up assessment.

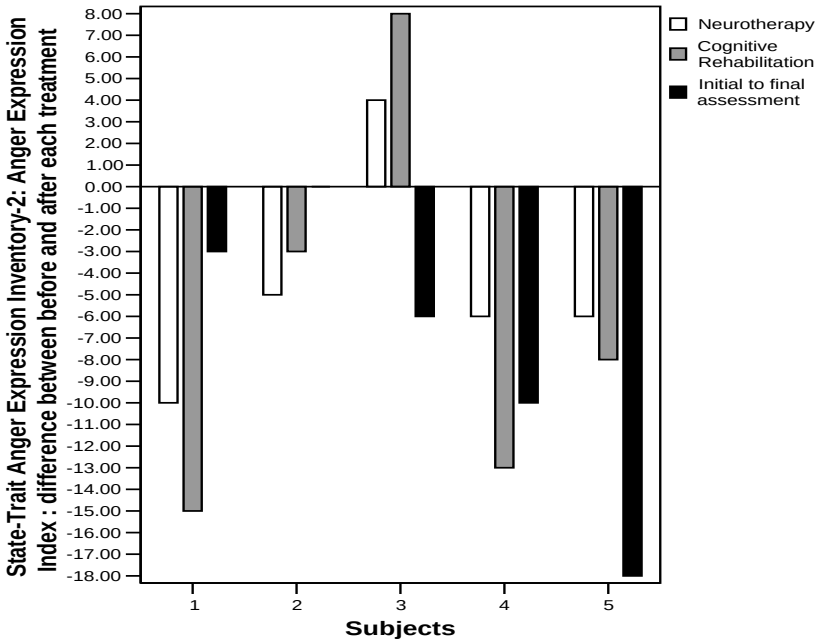

Figure 41: *STAXI-II (Anger Expression Index) - Difference between pre-post assessments following each treatment.*

5.3.3.4 Neurobehavioural Rating Scale.

5.3.3.4.1 Participant Reports

The Wilcoxon test revealed no statistically significant difference between the two treatments (Z = -0.41, p = 0.69). The difference scores between pre-post assessments for each treatment are highlighted in Figure 42. The bar graph suggests that following both treatments, three participants reported reductions in neurobehavioural symptoms, while two participants reported an increase in symptoms. However, on close examination a greater reduction in symptom reporting can be observed following EEG biofeedback as compared to cognitive rehabilitation. All participants reported reductions in neurobehavioural symptomatology at the final follow-up assessment. Overall, the trend of the graph suggests that both treatments were

effective in reducing self-reported neurobehavioural symptomatology, with a slightly greater reductions following EEG biofeedback. All participants continued to report less neurobehavioural symptoms at the final follow-up.

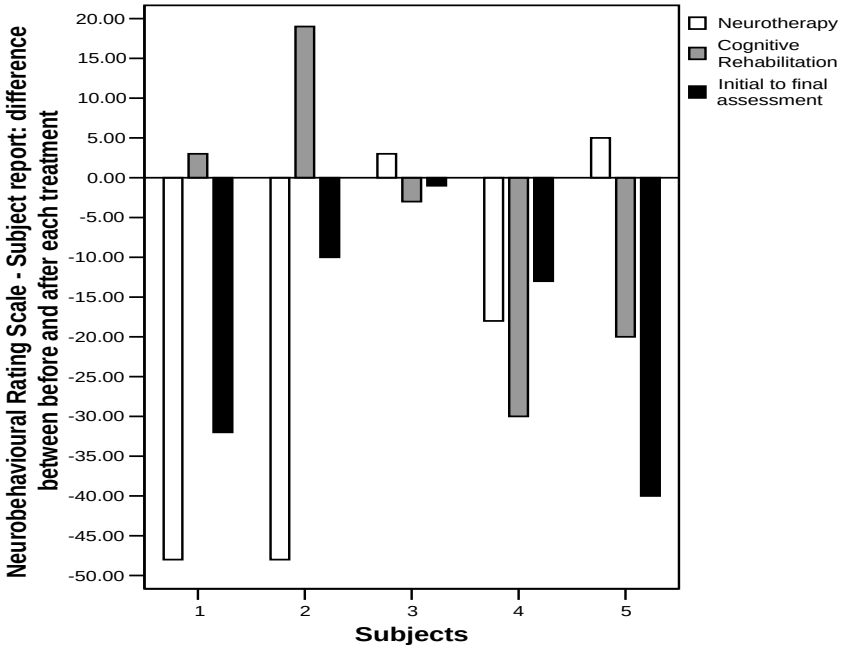

Figure 42: *NRS (Participant Report) - Difference between pre-post assessments following each treatment.*

5.3.3.4.2 Next of Kin (Significant Other) Reports

The Wilcoxon test revealed no statistically significant difference between the two treatments (Z = -0.54, p = 0.59). The difference scores between pre-post assessments for each treatment are displayed in Figure 43. The bar graph suggests that following EEG biofeedback three of the five participants' (2, 4, & 5) significant others reported a reduction in neurobehavioural symptomatology, while two (1 & 3) reported an increase in symptomatology. Following cognitive rehabilitation only one participant's (3) significant other reported a reduction in symptoms, while in three participants (2, 4, & 5), an increase was reported. At the final follow-up assessment, three participants' significant others (2, 4, & 5) reported a reduction in symptoms,

while two (1 & 3) reported an increase in symptomatology. Overall, the trend of the graph suggests that significant others observed a greater reduction in neurobehavioural symptomatology following EEG biofeedback compared to cognitive rehabilitation. At the ten week follow-up, only significant others who reported reductions following EEG biofeedback, continued to report neurobehavioural reductions.

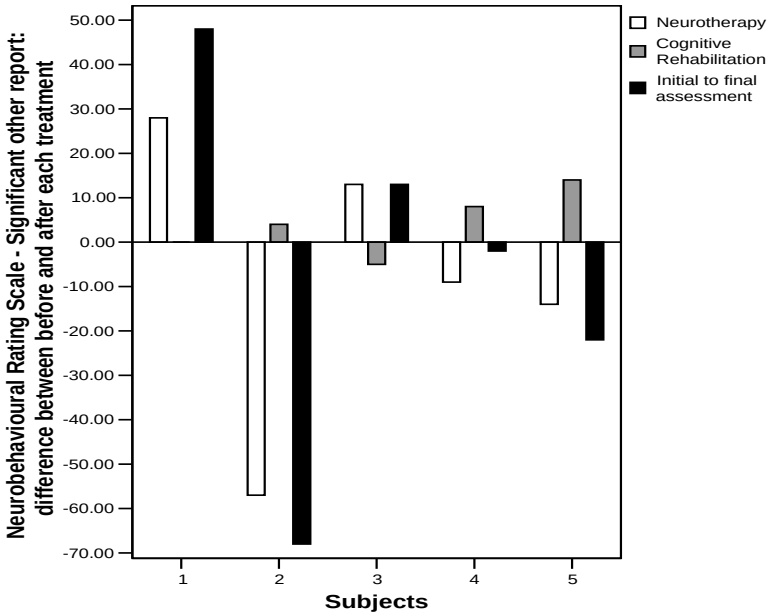

Figure 43: *NRS (Next of Kin) - Difference between pre-post assessments following each treatment.*

5.3.4 Group Quantitative Electroencephalogram Results

The group qEEG results must be interpreted with caution. Although the parametric analysis (paired t-tests) was run, it was difficult to accurately interpret change within the group. This was due to the heterogeneity in each participant's cerebral electrophysiological dynamics, which was observed on the initial qEEG assessments. Given such heterogeneity existed, different treatment protocols were provided in EEG biofeedback. It is noted that the software package used to analyse the results did not supply the degrees of freedom and t-values. Hence, only p-values are reported in the results. Despite the caution warranted in the paired t-test analysis, further non-parametric analysis (Chi-square test) using the significant change of absolute power Z scores (including all frequency bands) provided an accurate measure in the direct comparison of qEEG data for each treatment.

5.3.4.1 Pre and post EEG biofeedback

Following 20 sessions of EEG biofeedback paired t-tests revealed a statistically significant ($p < 0.01$) increase in the absolute power of Beta at F7 ($p = 0.004$). No statistically significant change was identified in the absolute power of Delta, Theta or Alpha. Significant change in absolute power can be observed in the topographic maps (Figure 44).

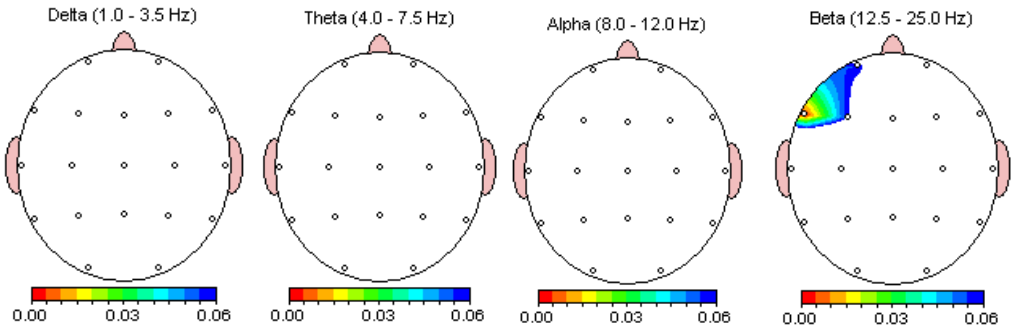

Figure 44: *Topographic maps - Statistically Significant change (P-values) in absolute power following*

EEG biofeedback for both treatment groups.

5.3.4.2 Pre and Post Cognitive Rehabilitation

Following 20 sessions of cognitive rehabilitation paired t-tests revealed a statistically significant ($p < 0.05$) reduction in the absolute power of Theta at Fp1 ($p = 0.031$), and in Alpha at F7 ($p = 0.036$). A statistically significant ($p < 0.05$) increase in the absolute power was observed in Beta at T4 ($p = 0.044$). No significant change was identified in the absolute power of Delta. Significant change in absolute power can be observed in the topographic maps (Figure 45).

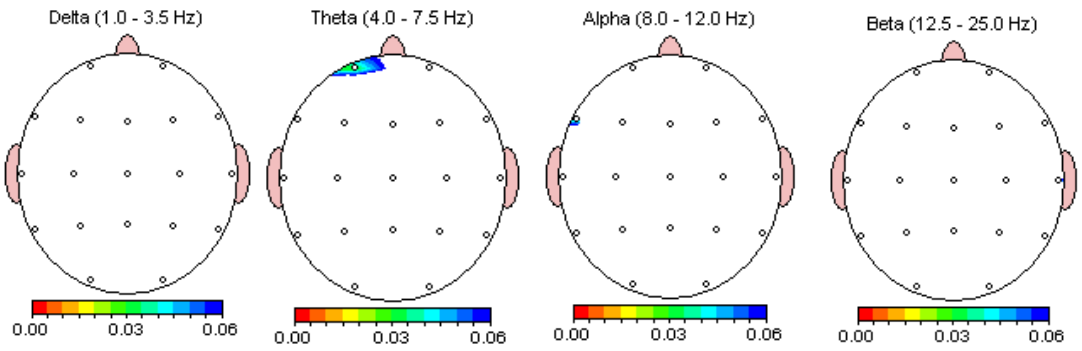

Figure 45: *Topographic maps - Statistically Significant change (P-values) in absolute power following Cognitive Rehabilitation for both treatment groups.*

5.3.4.3 Comparison between initial and final Assessment

In order to measure the amount of change from commencement to completion (follow-up assessment) of the research program paired t-tests were run. This revealed a statistically significant ($p < 0.05$) decrease in the absolute power of Theta at C3 ($p = 0.048$), and a statistically significant ($p < 0.05$) increase of Alpha at O1 ($p = 0.041$) and T6 ($p = 0.045$). No significant change was identified in the absolute power of Delta or Beta. Significant change in absolute power can be observed in the topographic maps (Figure 46).

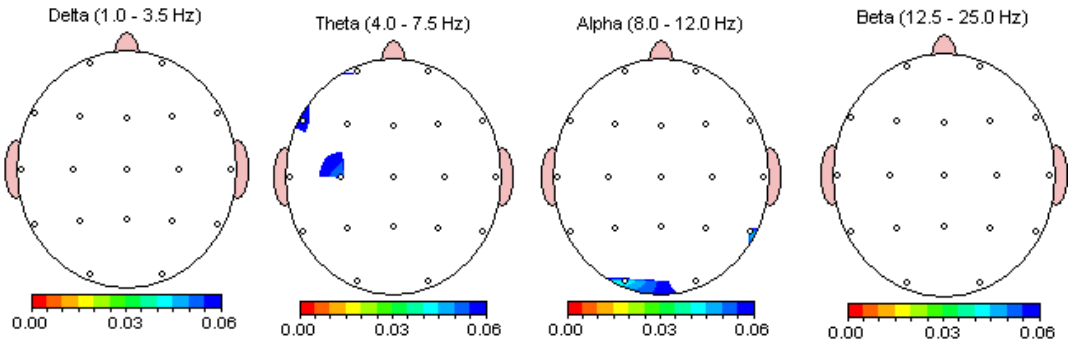

Figure 46: *Topographic maps - Statistically Significant change (P-values) in absolute power between initial and final assessments for both treatment groups.*

5.3.4.4 Treatment Comparison of the Absolute Power Z Score Change

Chi-square analysis of the number of statistically significant absolute power Z score changes, revealed a significant difference ($p < 0.01$) between EEG biofeedback and cognitive rehabilitation ($\chi^2 = 13.1$, $df = 2$, $p = .0014$). Electroencephalograph biofeedback compared to cognitive rehabilitation showed greater normalisation of the EEG (see Table 42 for Chi-square test).

Table 42 ***Chi-square: number of statistically significant absolute power Z score changes following each treatment.***

ACTUAL	Towards Normalisation	Away from Normalisation	No Significant Change		
EEG Biofeedback	142	107	207	456	50.0%
Cognitive Rehab	96	109	251	456	50.0%
	238	216	458	912	

EXPECTED	Towards Normalisation	Away from Normalisation	
EEG Biofeedback	119.0	108.0	229.0
Cognitive Rehab	119.0	108.0	229.0

Chi-square probability =	0.0014
Chi-square =	13.13

5.3.5 Summary of the Group Results

The group results are summarised in the following tables. The tables display a tally of the number of participants whose performance significantly improved or declined on each individual measure of cognitive, emotional, and behavioural functioning, following each treatment. The results summarise the group's overall performance in each cognitive domain, and overall emotional/behavioural functioning. The group qEEG results, displays the absolute power Z scores (including all frequency bands) which either significantly normalised, or significantly moved away from normalisation, following each treatment.

5.3.5.1 Summary of the Group Neuropsychological Results

In summary, EEG biofeedback was more effective than cognitive rehabilitation in improving attentional functioning (see Table 43) and overall cognition (see Table 47). When excluding the individual analysis of each cognitive test, minimal differences between each treatment can be observed in the remaining cognitive domains. Greater improvements (as compared to a decline in performance) can be observed at the final follow-up assessment for all cognitive domains.

Table 43: ***Number of participants whose Attentional performance significantly improved or worsened***

	Post EEG biofeedback		**Post Cognitive Rehabilitation**		**Final Follow-up**	
MEASURE	**improved**	**worsened**	**improved**	**worsened**	**improved**	**worsened**
Omission	1	0	0	1	2	0
Commission	3	0	0	2	2	0
Response time	2	2	2	2	4	1
Variability	3	0	3	1	4	0
PASAT	4	1	3	2	4	1
ATTENTION SCORE	**13**	**3**	**8**	**8**	**16**	**2**

Table 44: ***Number of participants whose Memory performance significantly improved or worsened***

	Post EEG biofeedback		Post Cognitive Rehabilitation		Final Follow-up	
MEASURE	**improved**	**worsened**	**improved**	**worsened**	**improved**	**worsened**
RAVLT total	1	2	2	0	2	0
RAVLT delayed	2	2	2	2	2	1
RAVLT recog.	3	1	1	1	4	0
Rey Figure recall	3	2	4	1	4	1
MEMORY SCORE	**9**	**7**	**9**	**4**	**12**	**2**

Table 45: ***Number of participants whose Speed of Information Processing performance significantly improved or worsened***

	Post EEG biofeedback		Post Cognitive Rehabilitation		Final Follow-up	
MEASURE	**improved**	**worsened**	**improved**	**worsened**	**improved**	**worsened**
Symbol Search	1	2	2	1	2	0
SCOLP	5	0	3	2	4	1
SPEED SCORE	**6**	**2**	**5**	**3**	**6**	**1**

Table 46: ***Number of participants whose Executive Functioning performance significantly improved or worsened***

	Post EEG biofeedback		Post Cognitive Rehabilitation		Final Follow-up	
MEASURE	**improved**	**worsened**	**improved**	**worsened**	**improved**	**Worsen**
FAS	0	1	1	0	2	0
Animals	1	0	1	1	1	0
Trails A	1	2	1	1	2	0
Trails B	3	0	0	1	2	0
EXECUTIVE SCORE	**5**	**3**	**3**	**3**	**7**	**0**

Table 47: ***Number of participants whose overall cognitive performance significantly improved or worsened.***

	Post EEG biofeedback		Post Cognitive Rehabilitation		Final Follow-up	
TOTALS	**improved**	**worsened**	**improved**	**worsened**	**improved**	**worsened**
Attention	13	3	8	8	16	2
Memory	9	7	9	4	12	2
Speed	6	2	5	3	6	1
Executive	5	3	3	3	7	0
COGNITIVE SCORE	**33**	**15**	**25**	**18**	**41**	**5**

5.3.5.2 Summary of the Group Emotional and Behavioural Results

Comparable improvements on self-reported emotional, behavioural, and neurobehavioural measures were evident following both treatments and at the final follow-up assessment.

Table 48: ***Number of participants whose overall self-reported emotional and behavioural functioning significantly improved or worsened.***

	Post EEG biofeedback		Post Cognitive Rehabilitation		Final Follow-up	
	improved	**worsened**	**improved**	**worsened**	**improved**	**worsened**
Depression	5	0	5	0	4	1
State Anxiety	5	0	4	0	4	0
State Anger	3	0	3	0	1	1
Anger Expression	4	1	4	1	4	0
Neurobehavioural	3	2	2	2	5	0
EMOTIONAL-BEHAVIOURAL SCORE	**20**	**3**	**18**	**3**	**18**	**2**

5.3.5.3 Summary of the Group Quantitative EEG Results

Overall, EEG biofeedback was more effective than cognitive rehabilitation in achieving the normalisation of dysregulated cerebral EEG. As can be observed in Table 49, a greater number of sites across all frequencies showed significant normalisation (compared to a significant shift away from normalisation) following EEG biofeedback. On the contrary, more sites showed a significant shift away from normalisation following cognitive rehabilitation. Similarly, at the final follow-up assessment the group showed a significant shift away from normalisation at more sites, compared to significant normalisation.

Table 49: ***Absolute power Z scores (including all frequency bands) demonstrating either the number which significantly normalised, or significantly shifted away from normalisation, following each treatment.***

	Post EEG biofeedback		**Post Cognitive Rehabilitation**		**Final Follow-up**	
Participant	**improved**	**worsened**	**improved**	**worsened**	**Improved**	**worsened**
One	22	19	52	2	20	11
Two	66	1	10	18	21	31
Three	22	27	29	24	25	15
Four	13	34	5	38	7	34
Five	19	26	0	27	22	14
TOTAL	**142**	**107**	**96**	**109**	**95**	**105**

NOTE: Significant improvement = towards normalisation

Significant worsening = away from normalisation

Chapter 6.

DISCUSSION

6.1 EFFICACY OF EEG BIOFEEDBACK AND COGNITIVE REHABILITATION AS TREATMENTS FOR TRAUMATIC BRAIN INJURY

A number of published studies have explored the effectiveness of cognitive rehabilitation and EEG biofeedback separately, as treatments for various sequelae following TBI. Although treatment efficacy is best established by comparing different treatment techniques, most previous studies in the EEG biofeedback field have failed to compare this relatively new technique to well established techniques being employed within the TBI population. Despite using a small sample size, the present study is one of the first studies to directly compare EEG biofeedback with a well established and widely used rehabilitation technique within the TBI population. Within the EEG biofeedback literature, the present study is one of the first to use a more severe TBI population.

Furthermore, within the cognitive rehabilitation literature, the current study is one of the first to use a more holistic approach to treating TBI. That is, the cognitive rehabilitation program was designed to address not only cognitive difficulties, but also directly treat emotional and behavioural sequelae of TBI. Unlike the previous cognitive rehabilitation research (which often uses a formulaic rehabilitation design), the present study implemented a treatment program tailored to the individual, which was ecologically valid and consistent with treatments being proved by clinicians in real life. Finally, the current study used TBI participants who were a substantial

number of years following injury. This patient population is not frequently explored in rehabilitation research.

6.1.1 Rehabilitation of Cognitive Sequelae

It was hypothesised that EEG biofeedback would be more effective than cognitive rehabilitation in the treatment of cognitive sequelae following TBI. The group results demonstrate support for this hypothesis only on tasks requiring visual information processing skills and complex attentional control. Overall, the group tended to perform better following EEG biofeedback (compared to cognitive rehabilitation) in speed of language and comprehension, response accuracy (impulsivity), and mental control / attentional shifting. Improvements in these specific cognitive functions, in particular speed of language and comprehension, have not been well documented or formally measured in the previous EEG biofeedback literature for the TBI population. Support for improved response accuracy in the TBI population following EEG biofeedback has been demonstrated by Tinius and Tinius (2000). However, their results were confounded by the TBI participants concurrently receiving cognitive rehabilitation. Consistent with the improved mental control/attentional shifting identified in the present study, Byers (1995) also revealed improvement in the cognitive flexibility of the TBI population following EEG biofeedback. Importantly, in the present study, all changes were maintained by the majority of participants at the follow-up assessment, with further gains made in speed of language and comprehension.

Despite the limited availability of research within the TBI population, the present findings are consistent with EEG biofeedback research which has examined other areas of neurological dysfunction, in particular ADHD. A number of recent studies

have reported significant improvements in complex attentional control and impulse regulation following EEG biofeedback (Rossiter & La Vaque, 1995; Radvanski et al, 2001; Monastra et al, 2002; & Fuchs et al, 2003). Most recently, a published study by Lévesque et al (2006) also demonstrated improvement in the impulse regulation of children with attentional difficulties following EEG biofeedback. Not only did their findings suggest improvement in response inhibition, the fMRI demonstrated the normalisation of the anterior cingulate cortex functioning following EEG biofeedback, compared to controls. Using fMRI (Soeda et al, 2005) and SPECT (Goethals et al, 2004) studies have demonstrated impaired functioning in the anterior cingulate cortex of TBI patients compared to normal controls when performing response inhibition tasks. Furthermore, Goethals et al (2004) indicated that slowed speed of processing in TBI during a response inhibition task suggests difficulty with resistance to distractions. Based on their findings and the findings of the current study, it may be plausible to speculate that the improvements in complex attentional processes, response inhibition, and improved speed of language and comprehension demonstrated in the present study, are interrelated and may be the result of normalising anterior cingulate cortex functioning following EEG biofeedback.

In contrast to expectations, there were no significant improvements in the group's ability to sustain their attention following either treatment. When examining the results on an individual basis, only one participant (P1) made significant improvements in sustained attention following EEG biofeedback, and one participant (P3) following cognitive rehabilitation. Despite these findings, only P1 maintained the improvement at the follow-up. Contrary to the present results, recent previous research has demonstrated improvement in sustained attention following TBI using EEG biofeedback (Tinius & Tinius, 2000) and in cognitive rehabilitation (Palmese &

Raskin, 2000; Sohlberg et al, 2000; & Stathopoulou & Lubar 2004). However, it must be noted that a number of participants had difficulties with fatigue (P1, P5, & P6), chronic pain (P4), and physiological / somatic concerns (P3), which impacted on their ability to sustain attention during assessment sessions.

Contrary to the hypothesis, the results of the present study indicated that neither treatment was more effective than the other in improving verbal memory functioning, with great variability between participants' performances. This is in contrast with previous research (Schoenberger, et al 2001; & Thornton, 2002) whereby verbal memory improvements were reported in TBI participants following EEG biofeedback. Furthermore, these studies demonstrated the maintenance of memory improvements following the withdrawal of the treatment. Again, external influences during assessment sessions (i.e., fatigue and pain) resulted in fluctuating attention in a majority of participants, and consequently one would expect variability in the memory results.

Conflicting with the expectations of the present study, improvements in visual memory (although small) were more consistently observed following cognitive rehabilitation compared to EEG biofeedback. Further gains were made at the follow-up assessment. It must be noted that the limited training sites (predominantly in central and frontal regions) used within the EEG biofeedback program may have accounted for the lack of significant improvement in visual memory. However, given a majority of participants made gradual improvement at each testing session, following both treatments, and further gains were made at the final follow-up assessment, it would not be unreasonable to consider the possibility of practice effects. As highlighted by Spreen and Strauss (1998), practice effects occur with

repeated administration of the same figure. Additionally, no reliable change indices were obtained for this measure of visual memory, leaving the significance of this result questionable.

Contrary to the findings of Byers (1995), which demonstrated significant improvements in executive functioning (including verbal fluency) following EEG biofeedback, the participants' performances on measures of executive functioning failed to support the hypothesis. The group did not perform better on executive measures following EEG biofeedback, and performed in a variable manner irrespective of treatment. Despite this, significant improvements and further gains were noted at the final follow-up assessment.

Overall, the group tended to make further gains in all cognitive domains, with the exception of verbal memory, at the final follow-up assessment. This even occurred in many cases where little or no change in cognitive function was detected following individual treatments. Consistent with the work of Sohlberg and Mateer (2001), these findings suggest that collectively implementing different approaches is more effective in achieving the best outcome than each treatment given in isolation.

Given there were multiple presentations of each test, the contribution of practice effects must be considered as a possible explanation of this continued improvement. However, if practice effects mediated the change and not the actual treatment, it would be expected that a gradual improvement at each testing session might be observed following each treatment condition. This was not the case. Despite varying changes following each treatment program (e.g. improved performance following Treatment A, and then reduced performance following Treatment B) further gains

were often still made at follow-up. With the exception of visual memory, it was unlikely that practice effects solely mediated change. Alternatively, the improvements demonstrated at the final follow-up assessment may also be explained by a phenomenon often reported following EEG biofeedback, where, once the treatment is removed, the electrophysiological dynamics of the brain continue to improve. Consequently, as reported by past research, cognitive changes not only are maintained, but further gains may be made over time (Thornton, 2002).

Not only may the individual characteristics of each participant (e.g. fatigability and pain) have contributed to the great variability noted in cognitive performance following both treatments, but also the neuropsychological tests may not have been sensitive enough to detect the relevant changes experienced by the participants. A great majority of the past research evaluating the effectiveness of rehabilitation in TBI has used functional measures, and limited formal cognitive assessment. This is generally due to the questionable ecological validity and generalisability of neuropsychological measures to the functional environment (Bowman, 1996). Oddy et al, (1999) indicated that measures of impairment, particularly neuropsychological tests, provide valuable information for formulating realistic goals for cognitive rehabilitation, but are not suitable for the measurement of outcome. Commonly, significant functional improvements may exist, despite the lack of significant changes on formal cognitive measures (Teasdale et al, 1997; & Wilson, 2002).

On the other hand, the present study may not have observed global improvements in cognitive functioning following EEG biofeedback given the limited number of sessions provided. Previous studies have demonstrated cognitive improvements in milder TBI populations following 31 sessions (Byers, 1995) and 40 sessions

(Hoffman et al, 1996; & Walker et al, 2002). A case study, which demonstrated a successful outcome in severe TBI, applied 50 sessions (Ayers, 1999). The current study only provided 20 EEG biofeedback sessions in a severe TBI population, and this may not have been adequate to produce global changes in cognitive functioning.

6.1.2 Rehabilitation of Emotional and Behavioural Sequelae

It was hypothesised that EEG biofeedback would be more effective than cognitive rehabilitation in the treatment of emotional and behavioural sequelae following TBI. Overall results demonstrate some support for the hypothesis, with respect to changes in depressive symptomatology. Although it must be noted that large reductions in self-reported depressive symptomatology were evident following both treatment programs, greater reductions were more consistently observed following EEG biofeedback compared to cognitive rehabilitation. These findings are consistent with a number of studies demonstrating reductions on self-reported measures of depression in TBI following EEG biofeedback (Ayers, 1987; Ayers, 1993; Salerno, 1997; & Schoenberger, et al 2001) and in treating depression in non–TBI populations (Baehr et al, 1997; & Hammond, 2003). Additionally, further gains were made in the majority of the group at the final follow-up assessment.

Although consistent improvements in anxiety and anger symptomatology were observed following EEG biofeedback, contrary to expectations, comparable improvements in this symptomatology were also observed following cognitive rehabilitation. It must be noted that during cognitive rehabilitation, cognitive behavioural therapy techniques were utilised to address emotional and behavioural difficulties. Therefore, the cognitive behaviour techniques were equally effective as EEG biofeedback in reducing reported anxiety and anger. This reduction was only

maintained at the follow-up assessment in state anxiety and anger expression, but not in state anger. The present results were not consistent with research conducted by Ayers (1993). Ayers demonstrated improvements on formal and self-report measures of anger and anxiety symptomatology following EEG biofeedback, but not following psychotherapy**.** On the contrary, the current findings are consistent with previous research whereby improvement in anxiety symptomatology has been demonstrated in TBI following both treatments given in isolation, EEG biofeedback (Ayers, 1993; & Ayers 1987) and cognitive behavioural therapy (Williams et al, 2003; & Williams, Evans, & Wilson, 2003). Similarly, the present findings are consistent with past research which has demonstrated the effective management of anger expression in TBI following both EEG biofeedback (Ayers, 1987) and cognitive behavioural therapy (Medd & Tate, 2000).

It has long been established in the literature that TBI patients and their significant others display inconsistent perceptions of the TBI individual's neurobehavioural functioning (Braun, Baribeau, & Ethier, 1988). Hence, given this inconsistency and the reduced insight often reported following severe TBI (Sbordone et al, 1998) both the participants and their significant other were required to report on changes in overall neurobehavioural symptomatology. The group results were consistent with the present study's expectations, whereby the TBI group more consistently reported a greater reduction in neurobehavioural symptomatology following EEG biofeedback than cognitive rehabilitation. The group's significant others' results were consistent with the participants' reporting. In fact, significant others more consistently reported neurobehavioural reductions following EEG biofeedback compared to cognitive rehabilitation than the participants' self-reporting. These results are inconsistent with previous research which has identified under-reporting of cognitive, emotional, and

behavioural symptoms in TBI individuals compared to their significant others (Sbordone et al, 1998).

However, some inconsistencies in symptom reporting between the participants and significant others became evident at the final follow-up assessment. At the follow-up, all participants within the group reported reductions in neurobehavioural symptomatology, regardless of which treatment was reported to be more effective. In contrast, only the significant others that had previously reported reductions in neurobehavioural symptomatology following EEG biofeedback continued to report reductions at the final follow-up assessment.

When examining the individual participants' self-reporting and their significant others' reporting of neurobehavioural symptomatology, as identified in previous research (Braun et al, 1988; & Sbordone et al, 1998), there was not always consistency. However, there were some clear explanations for the inconsistencies. In particular, Participant One's significant other was becoming disgruntled with their ongoing involvement in the study, and needed much encouragement to complete the neurobehavioural symptomatology report accurately. Other difficulties also arose, whereby Participant Four's significant other had minimal contact with the participant during the duration of the study, and there was an increased difficulty for them to accurately identify any changes in neurobehavioural functioning.

The changes in emotional and behavioural measures identified in the present study may be explained by multiple factors, ranging from functional brain changes, aspects of the therapeutic process (e.g. patient motivation and therapeutic relationship), perceived self efficacy, and external factors. Problematic mood and emotional

functioning (in particular depression) have been associated with functional changes in the anterior cingulate cortex (Rogers, Kasai, Koji, Fukuda, Iwanami, et al, 2004). Although not using TBI populations, very recent studies have reported changes in the anterior cingulate cortex following both EEG biofeedback (Lévesque et al, 2006) and CBT (Straube et al, 2006). In the current study the findings suggest, it may be plausible to consider that both treatments contributed towards improvement in anterior cingulate cortex functioning, and subsequently mood. In this group, improvement in anterior cingulate cortex functions (complex attentional control and response inhibition) were only observed following EEG biofeedback, and greater reductions in depressive symptomatology were identified following EEG biofeedback as compared to cognitive behavioural techniques. This suggests, particularly given greater consistency evident following EEG biofeedback in the improvement of functions associated with this brain structure, that EEG biofeedback may be more effective in improving the functioning of the anterior cingulate cortex than cognitive behavioural therapy. At the very least, both treatments may ultimately normalise the anterior cingulate cortex functioning, but EEG biofeedback may be able to achieve this result more quickly.

On the other hand, as improvements were generally observed on all measures of emotion and behaviour following both treatments, other influences must be considered as contributing towards these changes, including the therapeutic process (e.g. the participants' expectation of the therapy, their attitude, motivation, co-operation, and the therapeutic relationship or alliance) and perceived self efficacy. With the exception of Participant Three, excellent motivation, co-operation, rapport, and therapeutic relationships were established between the therapist and each participant. Furthermore, each participant participating in the study expected that the

treatments would improve their functioning. Therefore, one could argue that any improvements noted in these participants could be due to the effectiveness of these therapeutic factors and high levels of perceived self efficacy.

Participant Three provides a good example of the impact the relationship alliance and perceived self efficacy may have on the outcome of rehabilitation. During the beginning of the research project, due to cultural, age, and gender differences, P3's perception of both the therapist and initial treatment was poor. This greatly contributed towards his reduced motivation and co-operation during the EEG biofeedback program, and ultimately impacted on his outcomes. His performance either did not change or significantly deteriorated during the course of this treatment. Once the second treatment (cognitive rehabilitation) had commenced P3 altered his perception of the therapist and treatment, demonstrated greater motivation and co-operation, and consequently the therapeutic alliance improved. Improvements in his cognitive performance and self-reporting on emotional and behavioural measures were more consistently observed following this second treatment which he may have perceived as more relevant and practically based.

6.1.3 Functional Outcomes

Despite the present study yielding inconsistent findings across formal cognitive measures, according to subjective reports, cognitive, emotional, behavioural, and physiological improvements occurred at a functional level. Unfortunately these outcomes were not formally assessed. It appears that changes in functional activities of everyday life may be of greater value in determining change than the formal cognitive measures. In the present study, the consistency between the participants' and significant others' reporting implied that the participants' self-reports of

functional change were likely to be accurate. Therefore, it would seem that any positive functional changes reported by the participants must be considered in the context of successful rehabilitation. As highlighted by Bedard et al (2003) the ultimate index of a successful rehabilitation is the extent to which individuals with TBI resume their pre-injury lifestyle and activities.

Although functional outcomes were not formally measured in the present study, during the course of each treatment program participants' reports of any functional change was noted. A number of functional outcomes were reported following each treatment, indicating that both forms of rehabilitation were effective in producing positive functional changes.

On the other hand, self-reported improvements in cognitive functions were more consistently noted following EEG biofeedback than cognitive rehabilitation. This was particularly evident in relation to attention/concentration, where participants (P1, P2, P4, & P5) reported feelings of being "more alert and focused", "less foggy", and "thinking clearer" during every day tasks requiring concentration. This was consistent with the group results which demonstrated improvements on formal neuropsychological measures of complex attentional functioning. Also consistent with the group neuropsychological assessment results, Participants Two and Five reported improvements in their speed of thinking following EEG biofeedback, but no participants reported changes in these areas following cognitive rehabilitation.

Contrary to the results of the formal cognitive assessment, Participants Two and Four reported less forgetfulness following EEG biofeedback, with no participants reporting memory changes following cognitive rehabilitation. Subject reports of improved

functional memory following TBI have also been demonstrated following EEG biofeedback (Walker et al, 2002). Similar to the present findings, Salerno (1997) found that TBI subjects reported functional memory changes, however statistically significant results on formal measures of memory were not evident.

Previous research has demonstrated that although computer based rehabilitation may result in improved cognitive functioning on formal testing, unlike the utility of compensatory strategies, TBI individuals fail to generalise their cognitive improvements from computer based rehabilitation to every day life settings (Niemann et al, 1990). This does not appear to be the case in the present study. As can be observed, a number of functional changes in cognition were reported following EEG biofeedback.

In contrast to other cognitive domains, two participants (P4 & P6) reported improvements in their planning and organisational abilities within every day activities following cognitive rehabilitation. No improvements were reported by participants following EEG biofeedback. This is consistent with previous studies where external compensation strategies similar to the strategies implemented in the present study, such as the use of palmtop / electronic devices (Kim et al, 1999; & Kim et al, 2000) and diaries (Ownsworth & McFarland, 1999; & Fleming et al, 2005) have demonstrated effectiveness in managing executive difficulties following TBI.

With the exception of changes in sleep quality, all other improvements in physiological functioning were generally reported following both treatments. A number of studies have reported physiological changes following EEG biofeedback. However, physiological functioning does not appear to have been measured following

cognitive rehabilitation. Consistent with previous research (Salerno, 1997) improved sleep quality was reported by Participant Five and Six only following EEG biofeedback. Following both treatments, improvements were reported by P5 and P6 in fatigability and energy levels. These findings support the results of previous studies following EEG biofeedback (Ayers, 1987; & Schoenberger et al, 2001). A consistent finding following EEG biofeedback in the treatment of TBI was a reduction in pain, in particular headaches (Ayers, 1987; Bounias et al, 2001; Laibow et al, 2001; Bounias et al, 2002; Laibow et al, 2002; Walker et al, 2002; & Sterman, 2003). The present study supported these findings, as a reduction in headaches / migraines, back pain, and menstrual pain was reported by participants following EEG biofeedback. One participant also reported a reduction in headaches following cognitive rehabilitation. Contrary to previous research (Hammond, 2004) which demonstrated improved balance following EEG biofeedback, no improvements were reported in P3's balance/vertigo difficulties following either treatment.

For participants where emotional and behavioural difficulties applied, functional improvements in anxiety (e.g. reports of being able to leave the house for longer periods without panicking) and anger (e.g. a reduction in the number and intensity of outbursts in every day situations) were reported following both treatments. Improvements in anxiety are consistent with previously published studies following EEG biofeedback (Thomas & Sattlberger, 1997; & Hammond, 2003) and cognitive rehabilitation – utilising cognitive behavioural techniques (Carney et al, 1999; & Williams et al, 2003). Furthermore, similar to past research (Hammond, 2003; & Williams et al, 2003), a reduction in Participant Six's obsessive compulsive behaviours following both treatments reduced their impact on her everyday life.

Importantly, most participants reported maintaining a number of the compensatory strategies and cognitive behavioural techniques following the withdrawal of the cognitive rehabilitation program. Although they did not specify improvements in specific cognitive functioning, they all reported being able to better manage their cognitive and emotional difficulties. Those that did not continue to maintain most strategies implemented, in particular P3, experienced circumstances out of their control which impacted on the successful implementation (i.e. un-cooperative and unsupportive family members).

6.1.4 Normalisation of Dysregulated Electrophysiology in Traumatic Brain Injury.

It was expected that EEG biofeedback would be more effective than cognitive rehabilitation in normalising dysregulated EEG following TBI. This would be expected given EEG biofeedback directly aims to normalise EEG activity, whereas cognitive rehabilitation does not. When reviewing the group results, it was difficult to determine the clinical significance of the changes within the group EEG activity, due to the significant variation in the electrophysiological dynamics between each participant, and subsequent differing EEG training protocols.

In light of this, a significant increase in Beta activity for the group was observed following both treatments, while a significant decrease in Theta and Alpha frequencies was observed only following cognitive rehabilitation. Significant reductions in Theta and increases in Alpha were noted at the ten week follow-up assessment. Interestingly, Beta was the one frequency which was consistently applied in all six participants' EEG biofeedback protocols. In all participants, through positive reinforcement, the Beta frequency was expected to increase.

However, the frequencies which received inhibitory training varied between participants. Therefore, similar group changes in these frequencies would not be expected following EEG biofeedback.

More clinically meaningful results can be obtained by reviewing the EEG data on a multiple single-case study basis. Through this process the normalisation of the EEG can be ascertained as compared to simply reporting on significant increases and decreases in frequencies which do not necessarily represent normalisation. In examining the individual EEG results, evidence for the effectiveness of EEG biofeedback in the normalisation of EEG, as compared to cognitive rehabilitation, was unambiguous. All six individual participants made statistically significant changes towards normalisation in the electrophysiological dynamics of their brain following EEG biofeedback. Further statistical analysis of the grouped individual EEG results (directly comparing each treatment) also provided statistical support for this hypothesis. This is consistent with research (Byers, 1995; Salerno, 1997; Bounias et al, 2001; Laibow et al, 2001; Bounias et al, 2002; Laibow et al, 2002; & Sterman, 2003) who all demonstrated normalisation in EEG following EEG biofeedback in TBI.

Despite these findings, although Participant One demonstrated normalisation in EEG activity following EEG biofeedback, greater normalisation across more sites and frequencies was noted following cognitive rehabilitation. One other participant (P4) demonstrated relatively comparable normalisation following both treatments. Although little research has evaluated qEEG following cognitive rehabilitation, this result is consistent with research conducted by Stathopoulou and Lubar (2004). Following the implementation of a cognitive retraining program, five TBI participants

demonstrated improvements in EEG activity, establishing a link between cognitive rehabilitation and EEG changes. However, it should be noted that P1 received EEG biofeedback prior to cognitive rehabilitation. There is some evidence to suggest that when EEG biofeedback has ceased, patients tend to continue to improve in their cognitive functioning over time (Thornton, 2002). Hence, it may be possible that improvements following the cessation of treatment may be due to the continuing normalisation of dysregulated EEG. Therefore, in this particular participant it may also be argued that the greater improvement may have been due to the effect of the initial EEG biofeedback treatment.

If this were the case, it would be expected that normalisation of EEG activity would be consistent with improved cognitive, emotional, and behavioural performance following EEG biofeedback. In examining the group results, following EEG biofeedback, the normalisation of EEG activity may be considered consistent with improved performance (on formal measures and self-reports) in visual information processing skills (complex attentional control, response inhibition, and speed of language and comprehension) and in emotional functioning (namely, depression). However, on further evaluation of the individual participants, the validity of this relationship is questionable.

For example, following EEG biofeedback, Participant One made significant improvements in his EEG and on formal measures of attentional functioning, speed of information processing, and executive functioning. Hence, one could interpret a cause-and-effect relationship between normalisation of EEG and improvements in these areas of cognitive functioning. Despite these findings, following cognitive rehabilitation P1 demonstrated greater EEG normalisation across more sites and

frequencies than following EEG biofeedback. However, little improvement on formal cognitive tests was demonstrated. Similarly, disparities can be observed between P3's EEG and neuropsychological outcomes. Participant Three demonstrated either no change or further decline on formal testing of cognitive, emotional, and behavioural functioning following EEG biofeedback. In addition, he did not report any functional changes following this treatment. Despite this, his EEG demonstrated significant normalisation across all frequencies following EEG biofeedback.

Furthermore, most participants maintained cognitive, emotional, and behavioural improvements and/or made further gains at the final ten week assessment. However, this is not entirely consistent with the qEEG results at the ten week follow-up assessment. Most participants continued to demonstrate some level of significant normalisation in EEG at the final follow-up assessment, but normalisation was consistently noted at fewer sites than directly following treatments. Consequently, although greater normalisation in EEG was evident following EEG biofeedback compared to cognitive rehabilitation, the clinical significance of this result remains questionable. The present results may suggest a relationship between the normalising EEG activity and visual information processing, however some individual inconsistencies contradict this finding. Therefore, it remains unclear whether normalisation in EEG activity is related to positive changes in cognitive, emotional, and behavioural functioning.

6.2 METHODOLOGICAL ISSUES

Important methodological issues must be raised in the present study. Firstly, the most significant issue was the very small sample size used in the study. Given, the level of

commitment and length of time participants were required to maintain participation, it was difficult to acquire and maintain TBI participants in the treatment programs. Furthermore, obtaining participants within the strict criteria implemented limited the availability of participants able to partake.

The small sample size resulted in a study which had limited statistical power to detect group differences. Therefore, examination of the trends of the results was warranted. To compensate for the error of administering the same test on multiple occasions, and to strengthen the significance of the interpretation of results, where possible reliable change indices were obtained. However, the change indices used in the present study were not based on research using TBI populations and were not administered at the exact test re-test time intervals as the present study, and were based on change index measures for normal populations. Test re-test intervals greatly varied between different studies. As a result, the findings of the present study must be considered cautiously given the use of a very small sample and the difficulties in obtaining completely accurate and reliable change indices.

Secondly, caution is also warranted as factors such as practice effects on cognitive measures, the therapeutic relationship, and degree of perceived self efficacy were not well controlled for, and may have confounded the current results.

Thirdly, this study did not include formal assessment or outcome measures of functional changes. Given that the ecological validity and generalisability of neuropsychological measures to the functional environment has been questioned, formal neuropsychological tests may not be sensitive enough to detect the relevant changes experienced by the participants. It appears that changes in functional

activities of everyday life may be of greater value in determining change than formal cognitive measures.

Finally, in the context of the current research, the EEG biofeedback program was relatively limited compared to this treatment as provided in clinical practice. Clinically, patients are not restricted to 20 sessions, nor are they restricted to training only EEG amplitudes, and at only one of 19 sites. As evident in the literature review, Coherence training (in addition to amplitude training) has been demonstrated to be an effective form of EEG biofeedback in the TBI population. Time commitments and EEG equipment availability compromised the ecological validity of the EEG biofeedback program, and potentially limited its efficacy in the present study.

6.3 IMPLICATIONS FOR FUTURE RESEARCH IN TRAUMATIC BRAIN INJURY REHABILITATION

As the present study was very small, the statistical power and analysis was limited, only allowing preliminary findings. Future research assessing the efficacy of EEG biofeedback in the TBI population should continue to directly compare this treatment to a widely and commonly used treatment (e.g. cognitive rehabilitation) but using a larger sample size. In particular, it will be important for future research to demonstrate the efficacy of EEG biofeedback by directly comparing it with other well established treatments.

Future research will also need to carefully evaluate the appropriate use of outcome measures in the TBI population. Outcome measures in the present study mostly included formal neuropsychological measures, however the current literature has identified that measuring outcomes using formal neuropsychological assessment is

not always consistent with functional changes in every day life. Measures of functional change are generally a better indicator of an individual's ability to function in everyday life, and level of return to pre-injury functioning. Consequently, using formal neuropsychological measures may not be the most appropriate method of measuring outcomes following rehabilitation in TBI.

Another important avenue for future research in this field would be to evaluate the appropriateness of qEEG assessment as an outcome measure. The present study identified inconsistencies in the normalisation of EEG activity (as measured by the qEEG) and improvements in cognitive, emotional, and behavioural functioning. Future studies need to evaluate the ability of the qEEG to generalise EEG normalisation to the patient's neuropsychological functioning and functional changes in every day life.

Given the possible impact of outside influences on the therapeutic process (influence of therapeutic relationship and participant's individual characteristics), placebo controlled studies should be designed to limit this effect. However, the difficulty in maintaining the participant's limited awareness during both treatments (EEG biofeedback or cognitive rehabilitation) is acknowledged. Further research should also examine and control for the beliefs and expectations in relation to the outcome of these rehabilitation approaches. Perceived self efficacy may not only have an impact on rehabilitation in general, but may have greater influence on one approach as compared to another.

In clinical practice, TBI patients receiving EEG biofeedback often receive both training at a greater number of placement/electrode sites, and experience a greater

number of treatment sessions, as compared to the present study. Furthermore, given its increasing efficacy, training EEG coherence (in addition to training EEG amplitude) following TBI is becoming more commonly used in clinical practice. Therefore, to improve the ecological validity of EEG biofeedback, future research should account for the aforementioned limitations.

Finally, recent fMRI studies have provided some preliminary insight into the brain regions likely to normalise following the interventions under discussion. However, functional imaging studies, assessing functional brain changes following these rehabilitation methods, have not yet been applied in the TBI population. Further empirical studies are required to validate possible change in the anterior cingulate cortex in TBI following EEG biofeedback and cognitive rehabilitation in TBI.

6.4 CONCLUSIONS

Overall, the present study's findings provide preliminary support for the efficacy of EEG biofeedback in the rehabilitation of a broad range of sequelae following chronic severe TBI. The study's results further our knowledge by demonstrating support for the effectiveness of EEG biofeedback (compared to cognitive rehabilitation) in the treatment of specific cognitive difficulties, namely visual information processing skills (complex attentional control, response inhibition, speed of language and comprehension) and depression. These changes in cognition were consistent across formal cognitive measures and self-reported functional changes. Additionally, support for the effectiveness of cognitive rehabilitation (compared to EEG biofeedback) was demonstrated in the treatment of visual memory. Support for the ecological validity of the latter approach was further established by the successful implementation and utilisation of cognitive and behavioural strategies.

The present study also provides tentative support for the efficacy of both treatment methods, and in particular, applying a holist and ecologically valid approach to cognitive rehabilitation (utilising cognitive behavioural techniques), to effectively treat emotional and behavioural sequelae following chronic TBI. This was supported by both self-reported formal emotional/behavioural measures and through self-reported everyday functional changes.

The findings also add to the present literature by providing support for the efficacy of EEG biofeedback (compared to cognitive rehabilitation) in the normalisation of dysregulated EEG activity. However, individual participant inconsistencies between the normalisation of EEG activity and the direction of cognitive change, contradict this finding. Therefore, although EEG biofeedback was clearly effective in normalising dysregulated EEG, it remains unclear whether normalisation in EEG activity can be generalised to changes in cognitive, emotional, and behavioural functioning following TBI.

The present research outcomes, which suggest improved complex attentional control, response inhibition, speed of language processing, and depression, following EEG biofeedback, are a collection of functions associated with the functioning of the anterior cingulate cortex. It may be plausible to speculate that in the present study EEG biofeedback may have normalised this brain region, resulting in a specific collection of positive cognitive and emotional changes. This would be consistent with the findings of recent research following cognitive behavioural therapy which also demonstrated the normalisation of anterior cingulate cortex functioning. Therefore, the treatments in this study may have also contributed towards improved brain function, resulting in the further gains in functioning reported at the final

follow-up assessment.

Importantly, in the present study many further gains in cognitive, emotional, and behavioural functioning were exhibited at the final follow-up assessment. Ultimately, this implies that the combination of both rehabilitation approaches appears to elicit the most favourable outcome compared to either one applied alone.

Finally, the study's findings indicate that significant changes in cognitive, emotional, and behavioural difficulties can be effected in individuals with severe TBI many years after their brain injury. This finding has important implications for the management and treatment of brain injured individuals in the longer term.

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Appendices

APPENDIX 1:

Plain Language Statement – For Participants

VICTORIA UNIVERSITY

DEPARTMENT OF PSYCHOLOGY

Invitation to Participate in a Research Study

My name is Joanne Stephens, I am undertaking studies for a Doctorate in Clinical Neuropsychology at Victoria University. Part of my studies involves a research project. This is supervised by Dr Peter Dowling, Department of Psychology at Victoria University in collaboration with Kerrin Braithwaite and Jacques Duff, at the Behavioural Neurotherapy Clinic.

I am undertaking research on the effectiveness of two treatment programs, neurotherapy and cognitive rehabilitation on traumatic brain injury. Cognitive rehabilitation is well established and is a widely used treatment for traumatic brain injury. There is emerging evidence that neurotherapy can also effectively treat cognitive, behavioural and emotional difficulties following traumatic brain injury. Therefore, the research aims to study the effectiveness of these two treatments.

Participants in the study have an initial assessment involving the administration of a quantitative electroencephalography (QEEG) and a selection of neuropsychological tests. The QEEG is used to measure the electrical activity in your brain, or brain wave patterns. If the assessment suggests that either of these treatments are appropriate, you will be offered one of treatment programs for ten weeks, neurotherapy or cognitive rehabilitation. After this treatment the QEEG and selection of neuropsychological tests will be given again to measure the benefits of the treatment.

To partly cover the cost of using the equipment, a total fee of $150 will apply and this is payable prior to starting in the research study. This fee covers all assessments and the treatment program. If the treatment is not considered appropriate after the initial assessment, the fee will be refunded in full. However, no refund is available once the treatment program has commenced. It is important for participants to attend all of the treatment sessions and the evaluation sessions to ensure the full benefits of the treatment.

Your participation will enable me to gather valuable information on the effectiveness of the treatment programs, neurotherapy and cognitive rehabilitation. By participating, you will have the opportunity to work towards improving your functioning following the traumatic brain injury. Information and data obtained during this study will be treated as confidential by the researchers. The research thesis and any research papers arising from this study will not contain any person's name or any other information that will allow individual people to be identified.

If you would like the opportunity to participate, please complete the consent forms and return them in the reply paid envelopes within the next week. Should you have any concerns regarding the manner in which this research project is conducted, please do not hesitate to inform the researchers directly, or the Victoria University Human Research Ethics Committee (tel. 9688 4710).

Results will be available at the end of the project from the Department of Psychology. If you have any queries you can contact myself or Peter Dowling on 9365 2556.

Thanking you in anticipation

Joanne Stephens

APPENDIX 2:

INFORMED CONSENT FORM

VICTORIA UNIVERSITY

DEPARTMENT OF PSYCHOLOGY

Consent Form for Participants Involved in Research

Study Title: The Effectiveness of Neurotherapy and Cognitive Rehabilitation as a Treatment of Traumatic Brain Injury

INFORMATION

We would like to invite you to be a part of a study looking at the effectiveness of two treatment programs for traumatic brain injury, namely neurotherapy and cognitive rehabilitation. This project is a collaborative study between Victoria University Psychology Department and the Behavioural Neurotherapy Clinic. It aims to determine the effectiveness of both forms of treatment for the cognitive, emotional and behavioural difficulties that can occur after of traumatic brain injury.

You will be given an electroencephalography (EEG) technique called quantitative electroencephalography (QEEG) and a selection of neuropsychological tests to assess your functioning after brain injury. Following the initial assessment you will be assigned to commence in one of the treatment programs for ten weeks, neurotherapy or cognitive rehabilitation. At the end of this treatment period, the QEEG and neuropsychological tests will be re-administered to assess the benefits of the treatment.

During the course of the QEEG and neurotherapy, you may be exposed to some minimal discomfort when sensors are placed on your scalp and ears and occasionally cleaning of the skin can be mildly irritating. The sensor placements will be consistently placed on the same location on the scalp and ears. Your skin will be cleaned and prepared with EEG materials and exposed to EEG paste regularly. This is a standard and widely accepted procedure by electroencephalography clinicians. During the course of the study, we will be asking about your experiences since the brain injury and we would be available to discuss any concerns if you wish to do so.

At the end of the project you will receive a report on the study. The research thesis and any research papers arising from this study will not contain any person's name or any other information that would allow individual participants to be identified.

CERTIFICATION BY PARTICIPANT

I,_____

of _____

certify that I am at least 18 years old and that I am voluntarily giving my consent to participate in the study entitled: The Effectiveness of Neurotherapy and Cognitive Rehabilitation in the Treatment of Traumatic Brain Injury', being conducted at Victoria University and the Behavioural Neurotherapy Clinic by Joanne Stephens, Dr. Peter Dowling,, Kerrin Braithwaite, and Jacques Duff.

I certify that the objectives of the study, together with any risks to me associated with the procedures to be carried out in the study, have been fully explained to me by Joanne Stephens and that I freely consent to participation involving the use of these procedures (as listed below).

Procedures:

Administration of quantitative electroencephalography (QEEG) and a selection of neuropsychological tests to determine severity of traumatic brain injury. Completion of one of the treatment programs, neurotherapy or cognitive rehabilitation. The QEEG assessment and battery of neuropsychological tests will be administered before and after each treatment program to evaluate progress.

I certify that I have had the opportunity to have any questions answered and that I understand that I can withdraw from this study at any time and that this withdrawal will not jeopardise me in any way.

I have been informed that a fee of $150 is payable prior to starting the initial assessment phase. This fee is refundable if I am not included in the treatment phase of the study but is not refundable otherwise.

I have been informed that the information I provide will be kept confidential.

Signed: **.....}** **Date: .....**

Any queries about your participation in this project may be directed to the researcher (Joanne Stephens, ph. 9848 9100). If you have any queries or complaints about the way you have been treated, you may contact the Secretary, University Human Research Ethics Committee, Victoria University of Technology, PO Box 14428 MCMC, Melbourne, 8001 (telephone no: 03-9688 4710).

APPENDIX 3:

Demographic Information Questionnaire

Demographic information

1. Country of birth: _____

Country of Parents Birth - Mother: _____

- Father: _____

2. Date of Birth: _____ Your age in Years: _____

Please tick appropriate answer/box:

3. YOUR HIGHEST EDUCATION LEVEL:

- Attended primary school: ☐
- Completed primary school: ☐
- Attended secondary school: ☐
- Completed secondary school: ☐
- Attended a tertiary institution: ☐
- Completed a TAFE course ☐
- Completed a degree course: ☐
- Completed postgraduate studies: ☐

Grade Level when finished education: _____

---

4. EMPLOYMENT STATUS PRIOR TO BRAIN INJURY:

- Unemployed: ☐
- Disability Support Pension: ☐
- Casual Employment ☐
- Part-time Employment ☐
- Full-time Employment ☐
- Student ☐

5. CURRENT EMPLOYMENT STATUS:

- Unemployed: ☐
- Disability Support Pension: ☐
- Casual Employment: ☐
- Part-time Employment: ☐
- Full-time Employment: ☐
- Student: ☐

Length of time in current employment status since brain injury: _____

6. Number of years since Traumatic Brain Injury: _____

Year (Date) of Traumatic Brain Injury: _____ / _____ /_____

---

7. PERIOD OF LOSS OF CONCIOUSNESS OR COMA:

No loss of consciousness: ☐

****Number of hours unconscious or in a coma:*** _____

OR

****Number of days unconscious or in a coma:*** _____

OR

****Number of weeks unconscious or in a coma:*** _____

---

8. TIME SINCE FIRST MEMORY FOLLOWING BRAIN INJURY:

*For example, you may NOT have lost consciousness, but you may not remember any events for a number of minutes to hours after the brain injury.

*If you DID lose consciousness and/or were in a coma, it may have been a number of hours/days/weeks after the coma that you regained your memory. This is called Post Traumatic Amnesia.

No Loss of memory: ☐

* ***Number of hours before first memory:*** _____

OR

* ***Number of days before first memory:*** _____

OR

* ***Number of weeks before first memory:*** _____

9. How long was your stay in hospital:

* ***Number of hours you spent in hospital*** _____

OR

* ***Number of days you spent in hospital*** _____

OR

* ***Number of weeks you spent in hospital*** _____

---

10. At the time of your brain injury, did you have any other orthopaedic injuries:

Yes ☐ No ☐

If yes, explain: _____

_____

_____

_____

_____

---

11. Did you have any psychological and / or neurological conditions prior to the brain injury:

(For example, depression, anxiety, substance addiction, attentional disorder, learning difficulties, brain tumour, brain haemorrhage, brain aneurysm, epilepsy, etc)

Yes ☐ No ☐

If yes, explain: _____

_____

_____

_____

_____

12. Did you develop any psychological and / or neurological conditions following the brain injury:

(For example, depression, anxiety, substance addiction, attentional disorder, learning difficulties, brain tumour, brain haemorrhage, brain aneurysm, epilepsy, etc)

Yes ☐ No ☐

If yes, explain: _____

_____

_____

_____

13. Are you receiving any current treatment for your brain injury:

Yes ☐ No ☐

If yes, explain: _____

_____

_____

_____

**** Are you taking any medications ?***

If so, please list them: _____

_____

_____

_____

APPENDIX 4:

Reliability and Validity of Measures Used

NATIONAL ADULT READING TEST - REVISED

Blair & Spreen, (1989)

- Interscorer reliability = 0.99
- Internal consistency = 0.94

Raguet et al (1996)

- Test-retest reliability in normal adults = 0.92

TEST OF VARIABLES OF ATTENTION

Leark et al (2004)

- Test-retest reliability coefficients at 90 minute intervals

  Omission = .70 Commission = .78

  Response time = .84 Variability = .87

- SEM at a 90 minute interval

  Omission = 8.22 Commission = 7.03

  Response time = 6.00 Variability =5.41

- Test-retest reliability coefficients at one week intervals:

  Omission = .86 Commission = .74

  Response time = .79 Variability = .87

- SEM at a one week interval

  Omission = 5.61 Commission = 7.65

  Response time = 6.87 Variability = 5.41

Greenberg et al (2000)

- Sensitivity = correctly identified attentional disorders 84% of the time
- Specificity = correctly identified normal individual 89% of the time

PACED AUDITORY SERIAL ADDITION TEST

Egan (1988)

- Split half reliability = .96

McCaffrey et al (1995)

- Test-retest reliability coefficient at seven day interval = .93
- Test-retest reliability coefficient at ten day interval = .97

Sherman et al (1997)

- Construct validity with measures of focused attention

  Digit Symbol = .35

  Stroop = -.35 and of

- Construct validity with measures of capacity and encoding

  Arithmetic = .49,

  Consonant Trigrams = .31

- Construct validity with measures of reaction time

  Visual and auditory reaction time = -.13

(Hence, the results suggest that there was substantial overlap between other measures so of attention, but not with processing speed).

REY AUDITORY VERBAL LEARNING TEST

Lemay et al (2004)

- Test-retest reliability coefficients for total recall = range of .72 to .78
- Test-retest reliability coefficients for immediate recall = range of .67 to .76
- Test-retest reliability coefficients for delayed recall = range of .71 to .81

Moritz et al (2003)

- Standard error of measurement (time one) = 3.22
- Standard error of measurement (time two) = 4.5
- Standard error of difference = 5.54.

REY-OSTERRIETH COMPLEX FIGURE TEST

Delaney et al (1992)

- Inter-rater reliability = .91

Meyers & Meyers (1995).

- Test-retest reliability co-efficients for immediate recall = .76
- Test-retest reliability co-efficients for delayed recall =.89

THE SPEED & CAPACITY OF LANGUAGE PROCESSING TEST

The Medical Research Council (1992).

- Test-retest reliability coefficients = range of .84 to .87.
- Parallel form (form A and B) reliability = .93.
- Construct validity with other measures:

  Category generation = .52

  Colour naming =.56

  Categorisation test = .55

CONTROLLED ORAL WORD ASSOCIATION TEST (Phonological and Semantic)

Harrison et al (2000)

- Test-retest reliability coefficients for FAS = .82
- Test-retest reliability coefficients for animals = .68
- Standard error of prediction for FAS = 7.15
- standard error of prediction for animals = 4.33

Vlaar & Wade (2003).

- Inter-observer reliability for FAS total score = .90

Henry & Crawford (2004)

TRAIL MAKING TEST Part A & Part B

Basso et al (1999)

- Test-retest reliability coefficients for Trails A = .38
- Test-retest reliability coefficients for Trails B = .64
- Standard error of prediction for Trails A = 6.80
- Standard error of prediction for Trails B = 14.85

Heaton et al (2001)

- Test-retest reliability coefficient for Trails B = .72
- Standard error of difference for Trails B = 18.6

Kortte et al (2002)

- Validity - correlations with other measures:

  WCST (percentage perseveration) for Trails A = .51

  WCST (percentage perseveration) for Trails B = .59

  COWAT for Trails A = .30

  COWAT for Trails B = .35

  WAIS-R Digit Span for Trails A = .22

  WAIS-R Digit Span for Trails B = .27

  CVLT (total score) for Trails A = .52

  CVLT (total score) for Trails B = .53

BECK DEPRESSION INVENTORY – SECOND EDITION

Beck et al (1996)

- Test-retest reliability coefficient = .93
- Construct validity – correlations with:

  BDI (original version) = .93

- Convergent validity - correlations with:

  Beck Hopelessness Scale = .68

The scale for suicidal ideation = .37

Beck Anxiety Inventory = .60

Hamilton Psychiatric Rating Scale for Depression = .71

Hamilton Rating Scale for Anxiety = .47

Sprinkle et al (2002)

- Test-retest reliability coefficient = .96
- Criterion validity - correlations

  Structured clinical interview for DSM-IV Axis I = .83
- Sensitivity (correctly identifying depressive mood) = 84%
- False-positive rate = 18%

STATE TRAIT ANXIETY INVENTORY ADULT VERSION

Spielberger et al (1983)

- test-retest reliability coefficients for T-anxiety:

  One hour interval (males = .84 and females = .76)

  20 day interval (males = .86 and females = .76)

  104 day interval (males = .73 and females = .77)
- test-retest reliability coefficients for S-anxiety:

  One hour interval (males = .33 and females = .16)

  20 day interval (males = .54 and females = .27)

  104 interval (males = .33 and females = .31)

STATE TRAIT ANGER EXPRESSION INVENTORY – SECOND EDITION

Bishop & Quah (1998)

- Test-retest reliability coefficients for Trait Anger = .74
- Test-retest reliability coefficients for Anger In = .82
- Test-retest reliability coefficients for Anger Out = .80

- Test-retest reliability coefficients for Anger Expression = .88
- Test-retest reliability coefficients for State Anger scale = .01

Spielberger (1999)

- Concurrent validity - correlations with:

    Buss-Durkee Hostility Inventory = range of .66 to .71

    MMPI Hostility scale = range of .43 to .59

    MMPI Overt Hostility Scale = range of.27 to .32

QUANTITATIVE ELECTROENCEPHALOGRAM

Kondacs & Szabo, 1999).

- Test-retest interval of 25-62 months in total absolute power = .78

Thatcher et al (1989)

- Discriminant accuracy in the detection of mild TBI = greater than 90%

Thatcher et al (2001)

- Discriminant analysis between mild and severe TBI groups showed:

    Classification accuracy = 96.39%

    Sensitivity = 95.45%

    Specificity = 97.44%

Thatcher et al (1991)

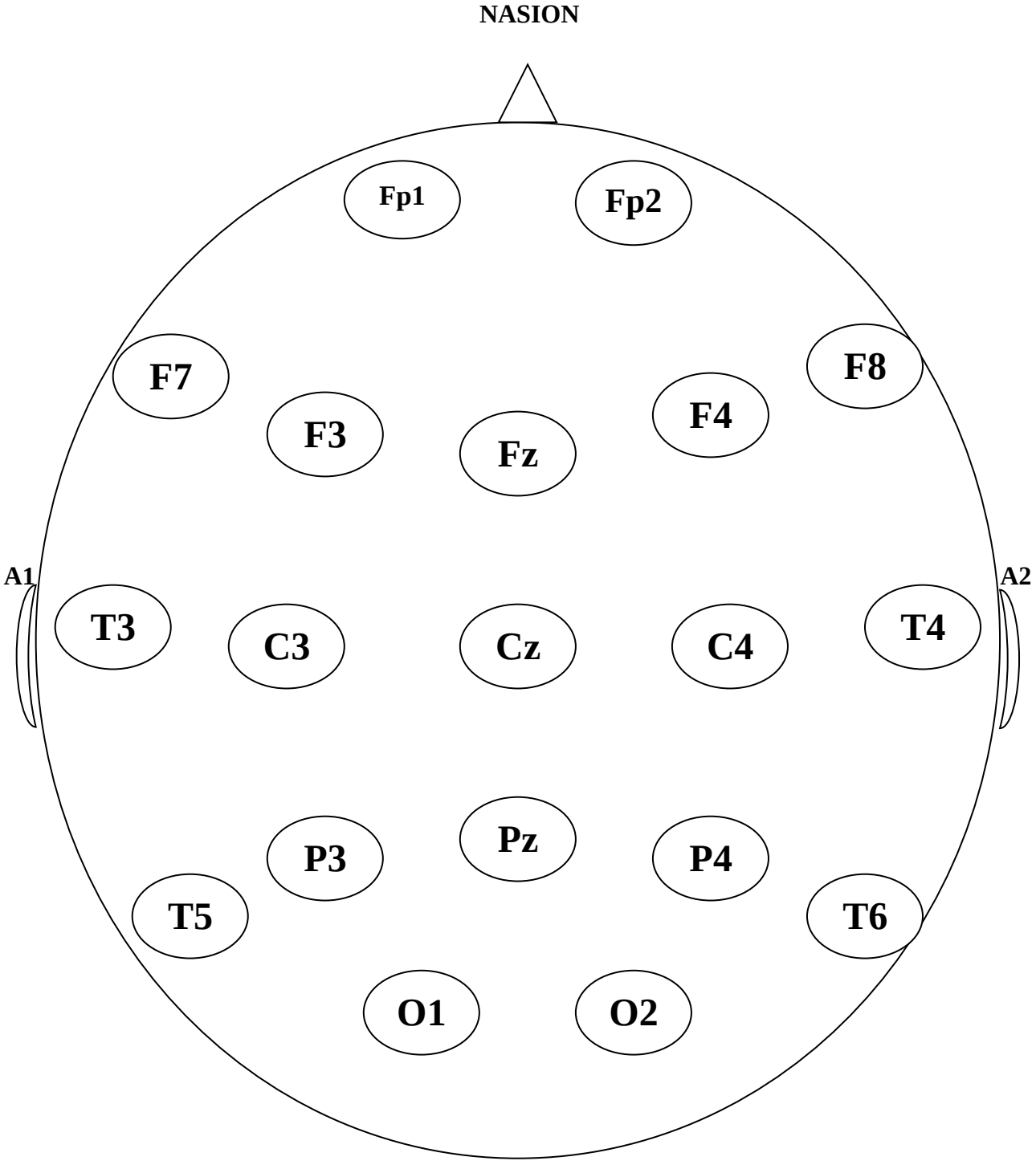
- Best predictors of functional outcome in neurotrauma subjects was a combination of qEEG and GCS accounting for 74.65% of the variance, which exhibited a discriminant accuracy between good outcome and death of 95.8%

APPENDIX 5:

International 10 – 20 System of Electrode Placement

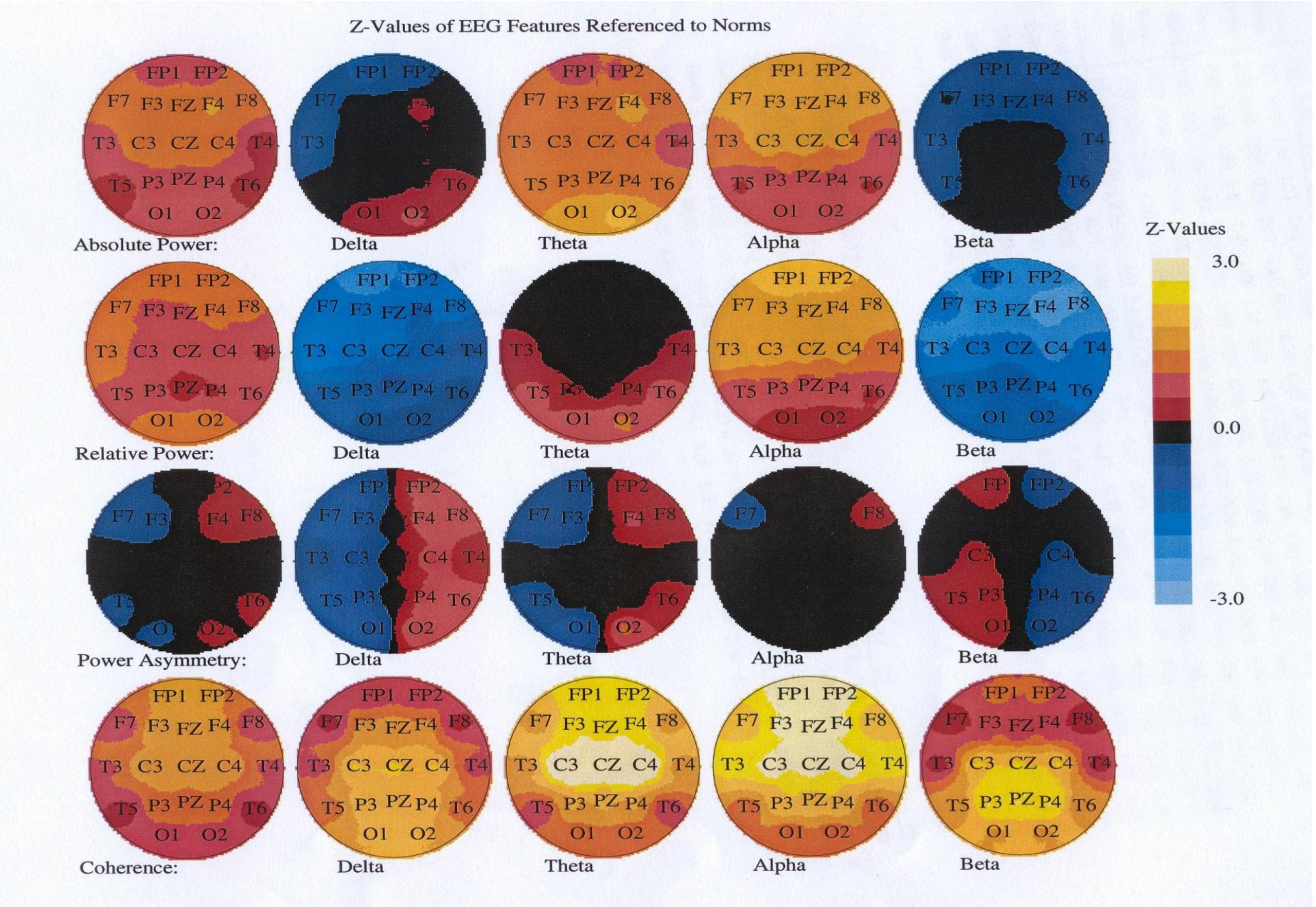
International 10-20 System of Electrode Placement

Fp1 & Fp2	= Prefrontal	Fz	= Frontal midline
F3 & F4	= Frontal	Cz	= Central Vertex
F7 & F8	= Anterior Temporal	Pz	= Parietal Midline
C3 & C4	= Central		
T3 & T4	= Mid-Temporal		
T5 & T6	= Posterior Temporal		
P3 & P4	= Parietal		
O1 & O2	= Occipital		



APPENDIX 6:

Example of Quantitative Electroencephalogram (qEEG): Coloured Topographic Map


Z-Values of EEG Features Referenced to Norms
Absolute Power:
Relative Power:
Power Asymmetry:
Coherence:
Delta
Theta
Alpha
Beta
Z-Values
3.0
0.0
-3.0

APPENDIX 7:

Principles of Cognitive Rehabilitation

Sohlberg and Mateer (2001) Page 21

Principles of Cognitive Rehabilitation

- Cognitive rehabilitation is informed by medical and neuropsychological diagnosis, but is based on an ever-evolving formulation of the individual client's needs and his or her problems and strengths from physical, cognitive, emotional, and social perspectives.
- Cognitive rehabilitation requires a sound therapeutic alliance among the therapist, client, and family members or other caregivers.
- Cognitive rehabilitation emphasizes collaboration and active participation.
- Cognitive rehabilitation is goal-oriented and, while problem-focused, builds on strengths.
- Cognitive rehabilitation has a primary focus on education, with an emphasis on empowerment, self-control, and self-sufficiency.
- Cognitive rehabilitation sessions are structured, and treatment plans and activities are developed with reference to both assessment results and current performance data.
- Cognitive rehabilitation goals may include improving cognitive and behavioural skills, compensating for cognitive and behavioural limitations, and assisting a client to understand and manage emotional reactions to changes in his or her functioning.
- Cognitive rehabilitation assists clients in achieving a more accurate understanding of their strengths and limitations, and in adjusting to injury-related changes in functioning and in life circumstances.
- Cognitive rehabilitation is eclectic: It uses a variety of techniques and strategies to improve abilities: to teach new and compensatory skills; to facilitate regulation of behaviour; and to modify negative or disruptive thoughts, feelings, and emotions.
- Cognitive rehabilitation seeks to understand each client's previous lifestyle, including abilities, goals, values, relationships, values, roles, personality, and behavioural patterns.
- Cognitive rehabilitation is responsive to changing theories and technologies.
- Cognitive rehabilitation professionals recognise and respond to the need to evaluate objectively the effectiveness of interventions.
- Team-based cognitive rehabilitation offers the advantage of seeing a problem or opportunity from a number of related but distinct professional perspectives.

APPENDIX 8:

Cognitive Rehabilitation Plan

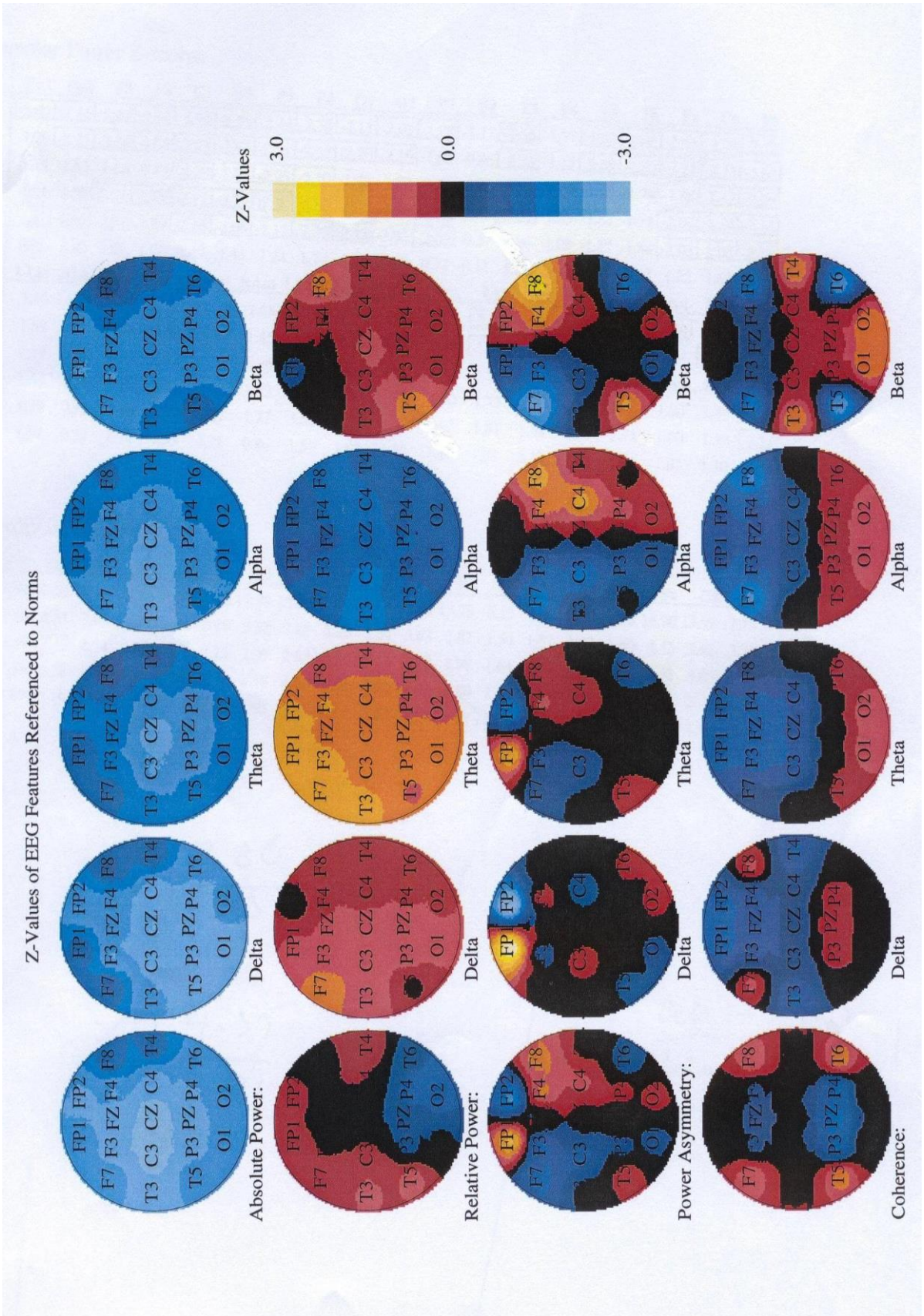
Cognitive Rehabilitation Plan

Pre Therapy:	History collection & problem list collecting Assessment : (Two week period)
Week 1:	Commence Cognitive Rehabilitation Feedback about assessment Develop a problem list and prioritise problems Agreement in a plan with the client
Week 2:	Memory strategies
Week 3:	Review memory strategies Attention and information processing strategies
Week 4:	Review attention and information processing strategies Executive function strategies
Week 5:	Review memory, attention and executive functions strategies Half way progress review – 'Are we on track ?' Revise Plan
Week 6:	Emotional issues – how are they impacting on cognitive functioning
Week 7 to 9:	Based on reviewed plan from week 5
Week 10:	Review: recapping on strategies Determine what strategies are working Determine how the subject might progress and build on current strategies Future plan

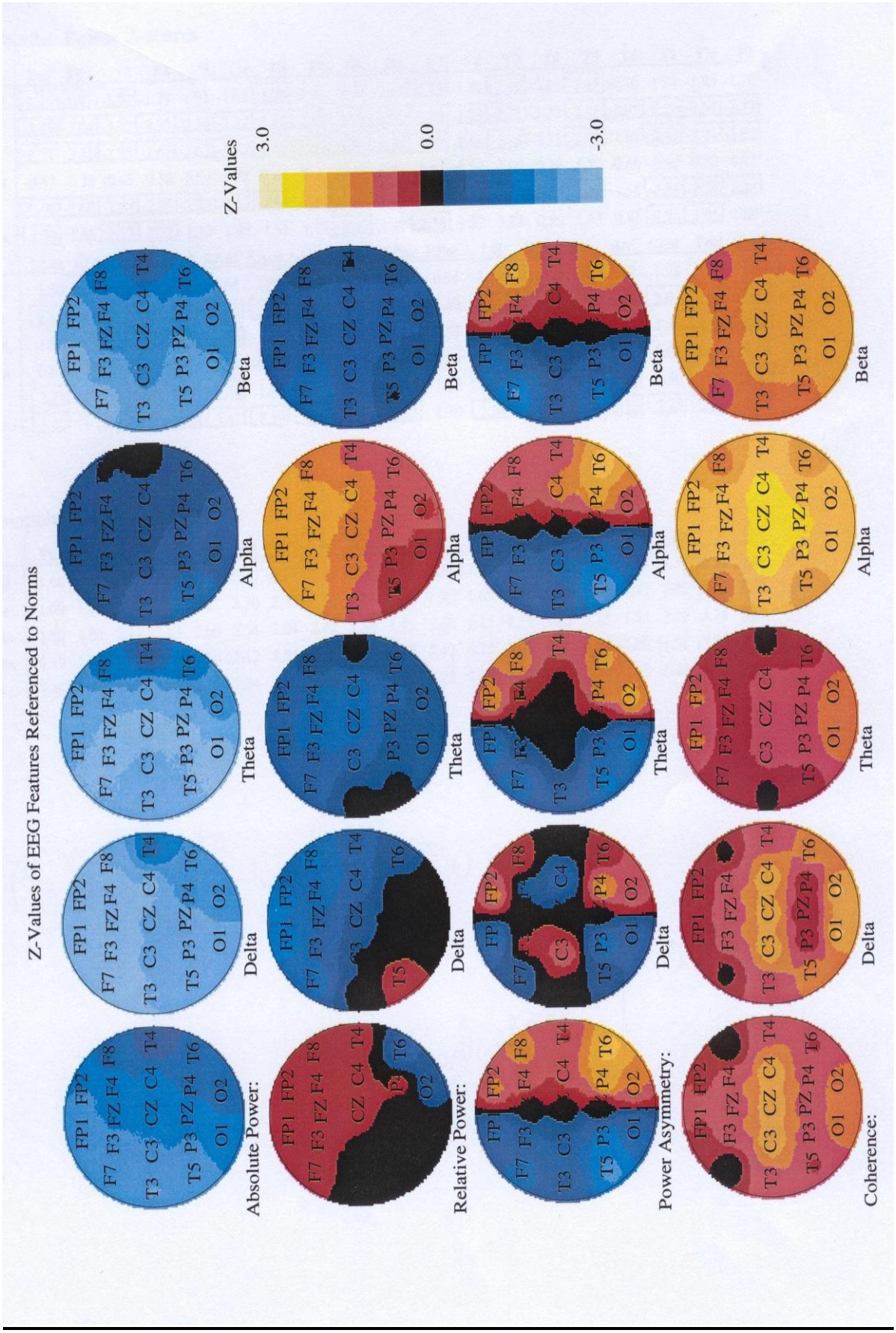
APPENDIX 9:

Pre EEG Biofeedback qEEG Topographic Maps

PARTICIPANT ONE

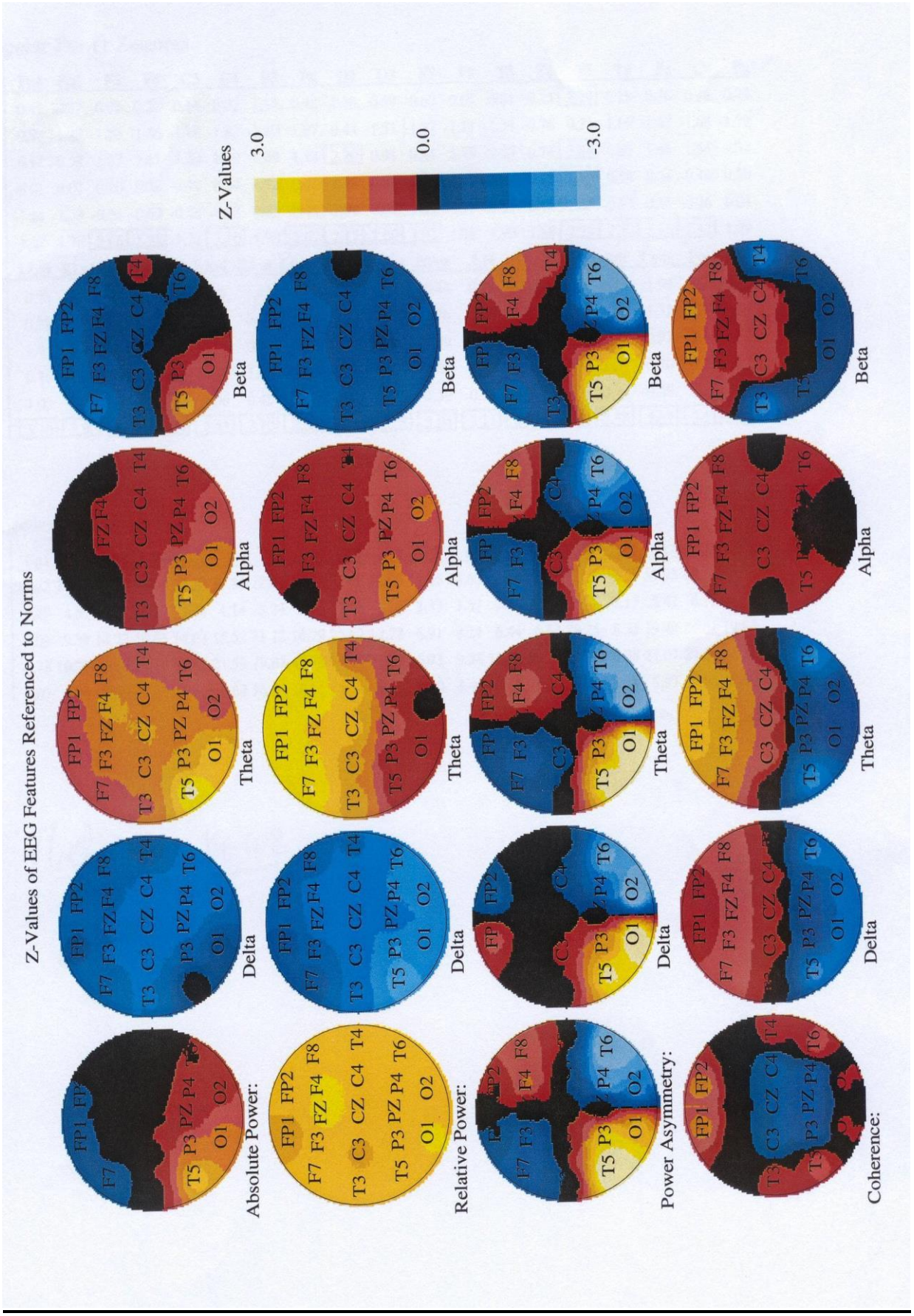


PARTICIPANT TWO

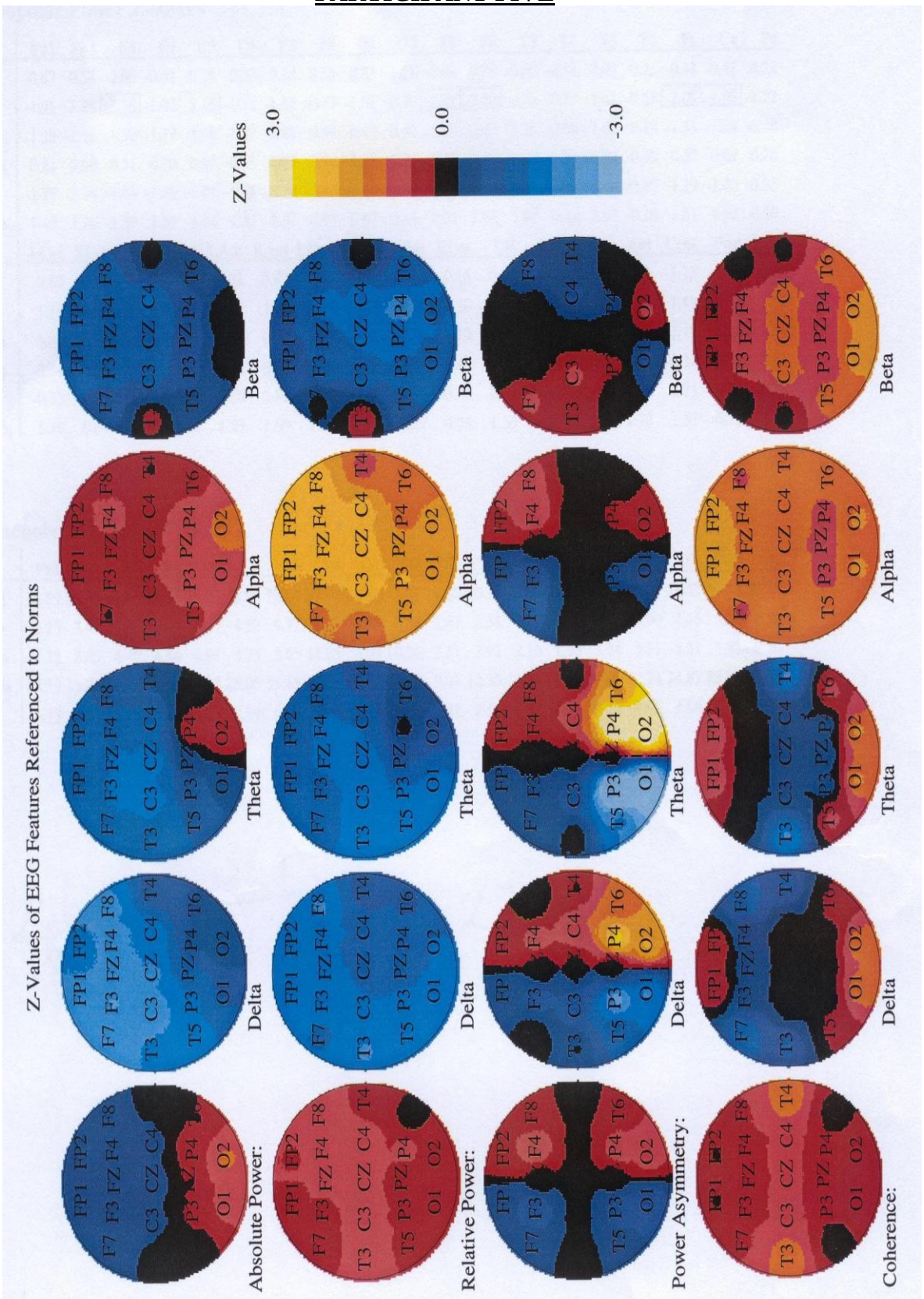


PARTICIPANT THREE

PARTICIPANT FOUR



PARTICIPANT FIVE

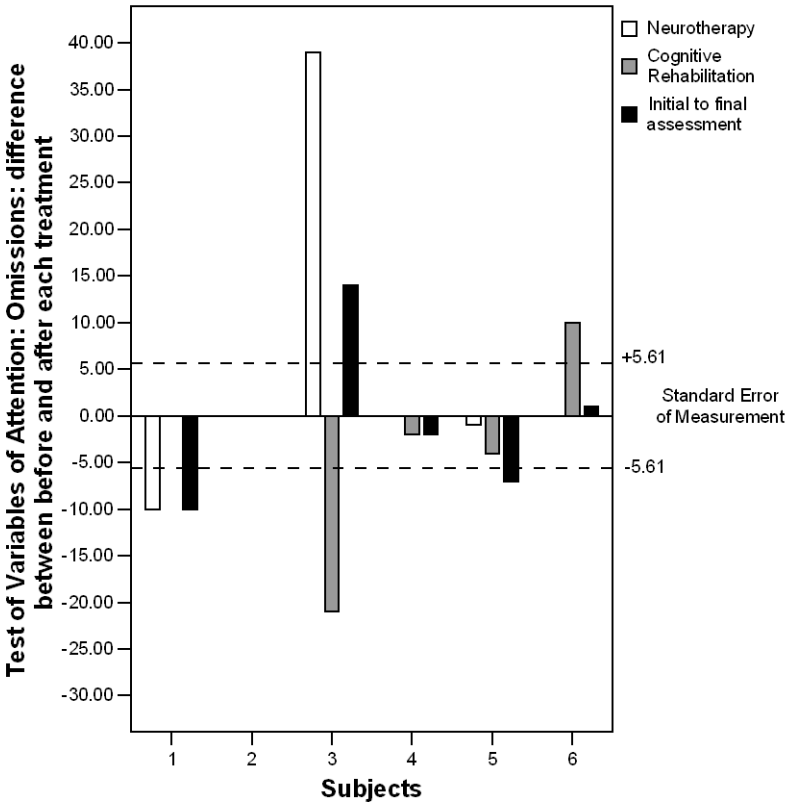


PARTICIPANT SIX

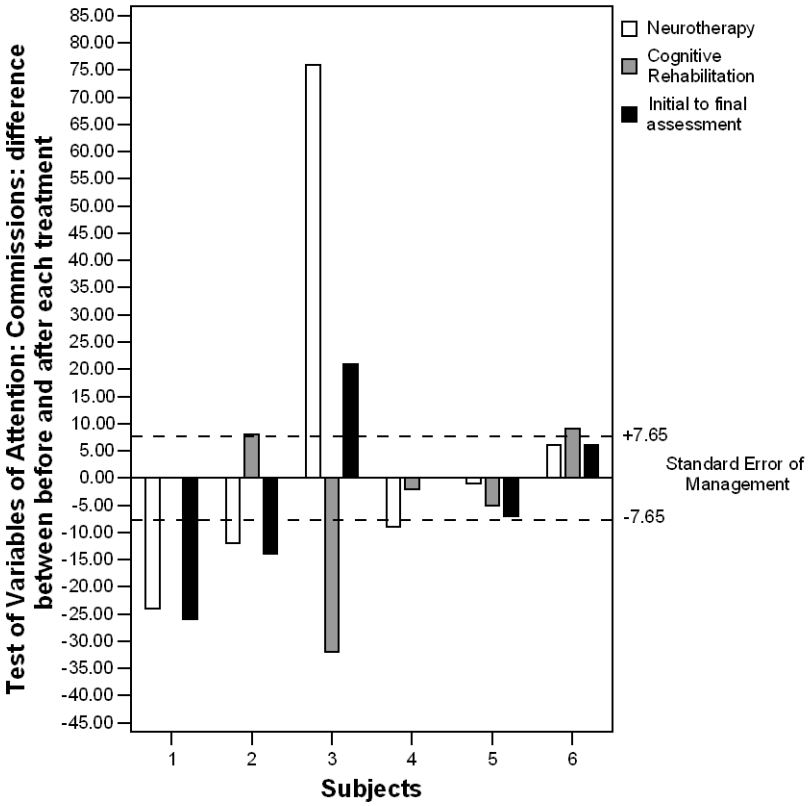
APPENDIX 10:

Bar Graphs for Individual Participants

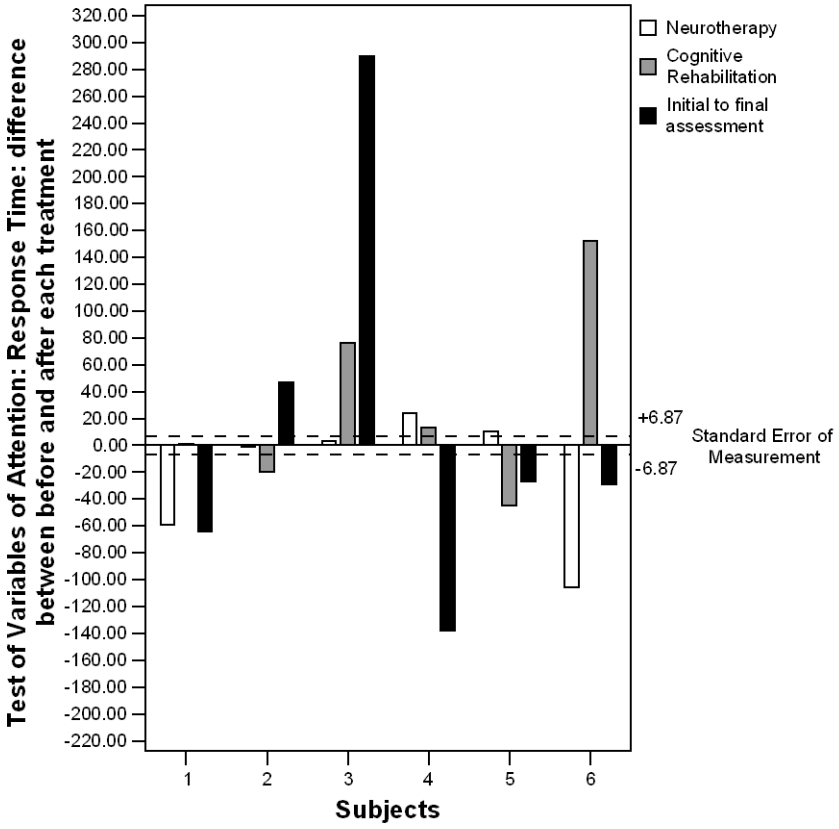
1) TEST OF VARIABLES OF ATTENTION (T.O.V.A.): OMISSIONS



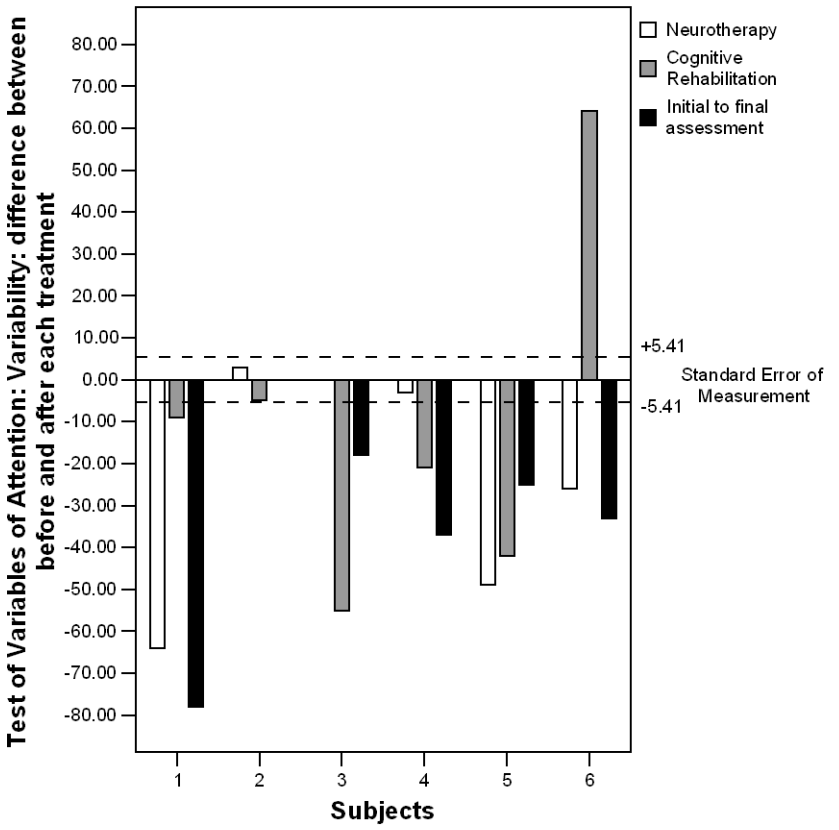
2) TEST OF VARIABLES OF ATTENTION (T.O.V.A.): COMMISSIONS



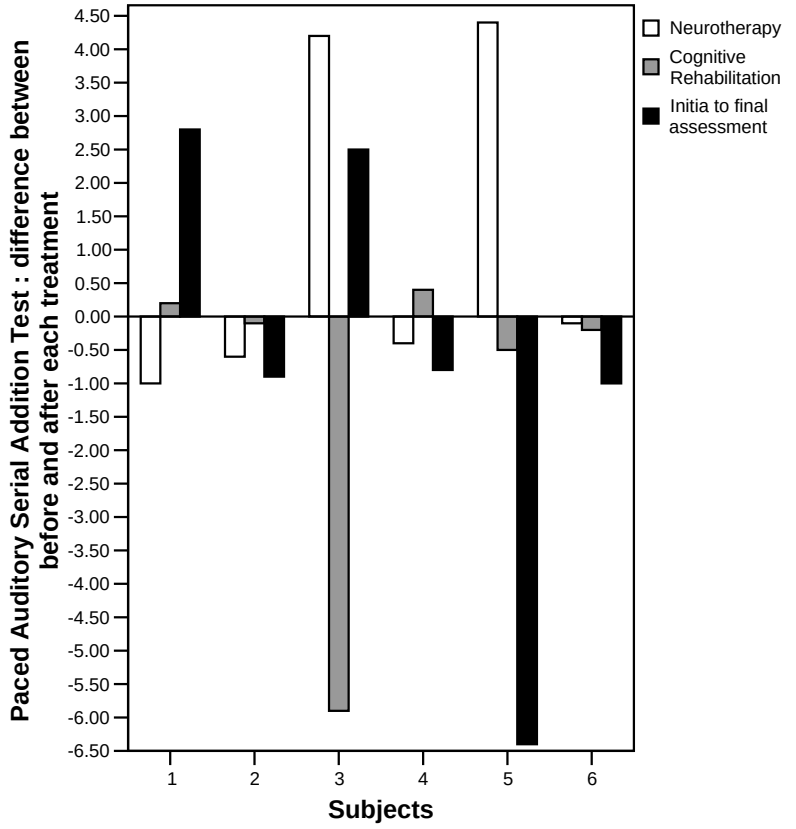
3) TEST OF VARIABLES OF ATTENTION (T.O.V.A.): RESPONSE TIME



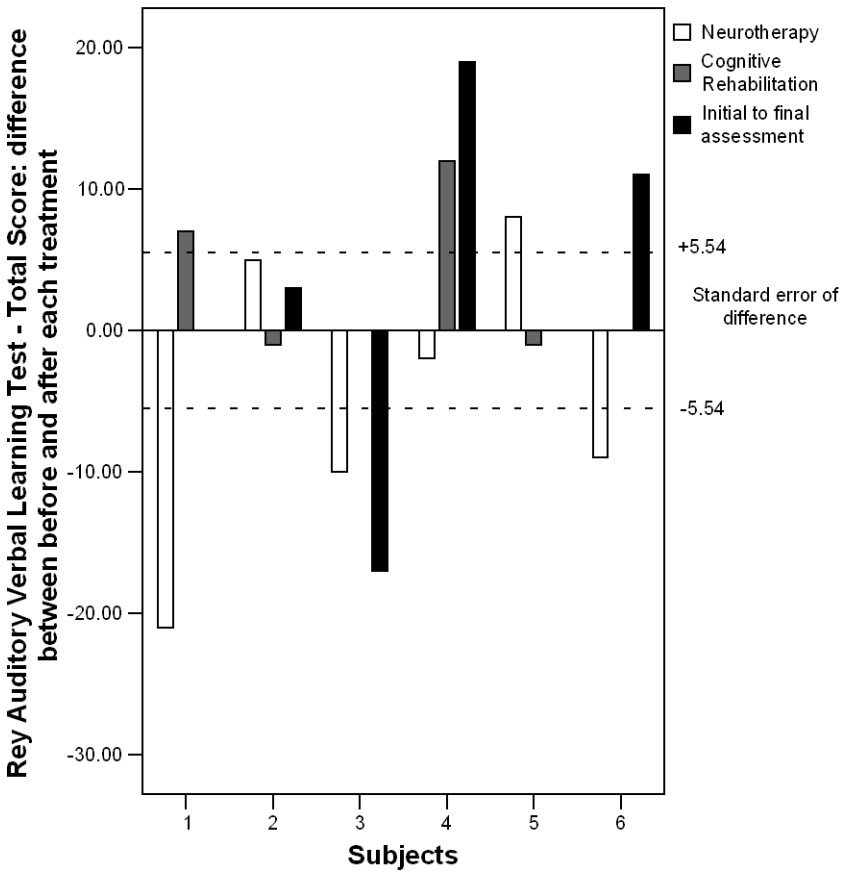
4) TEST OF VARIABLES OF ATTENTION (T.O.V.A.): VARIABILITY



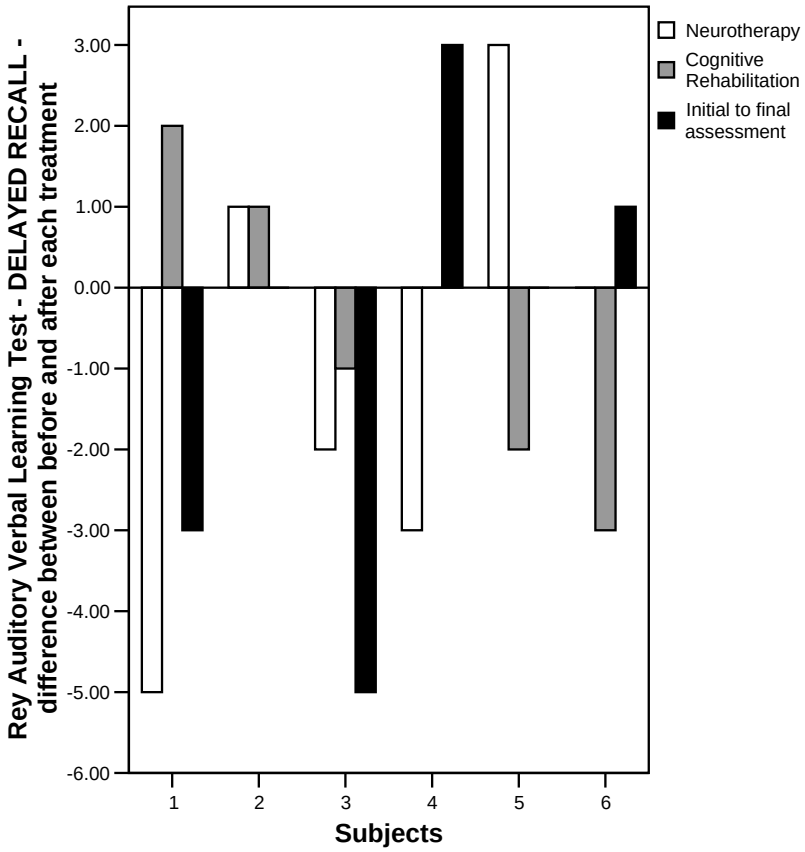
5) PACED AUDITORY SERIAL ADDITION TEST (PASAT)



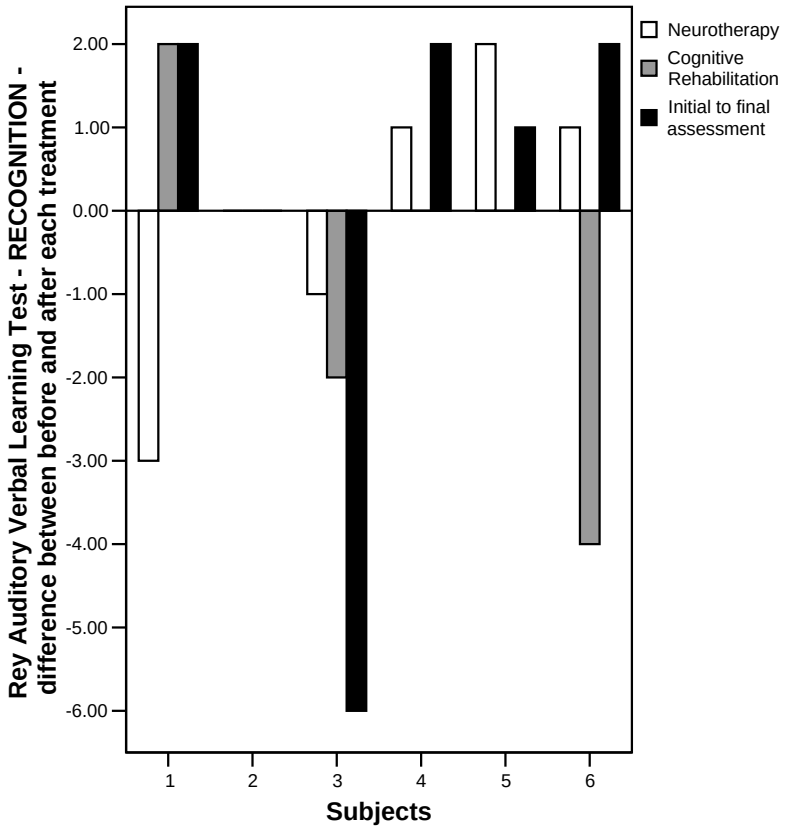
6) REY AUDITORY VERBAL LEARNING TEST (RAVLT): TOTAL RECALL



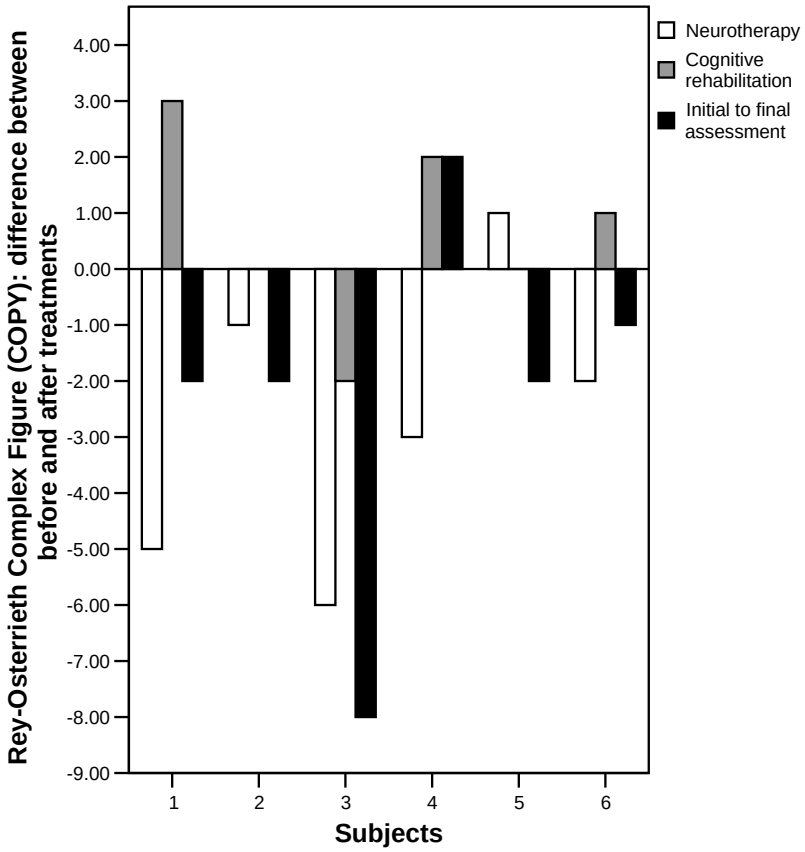
7) REY AUDITORY VERBAL LEARNING TEST (RAVLT): DELAYED RECALL



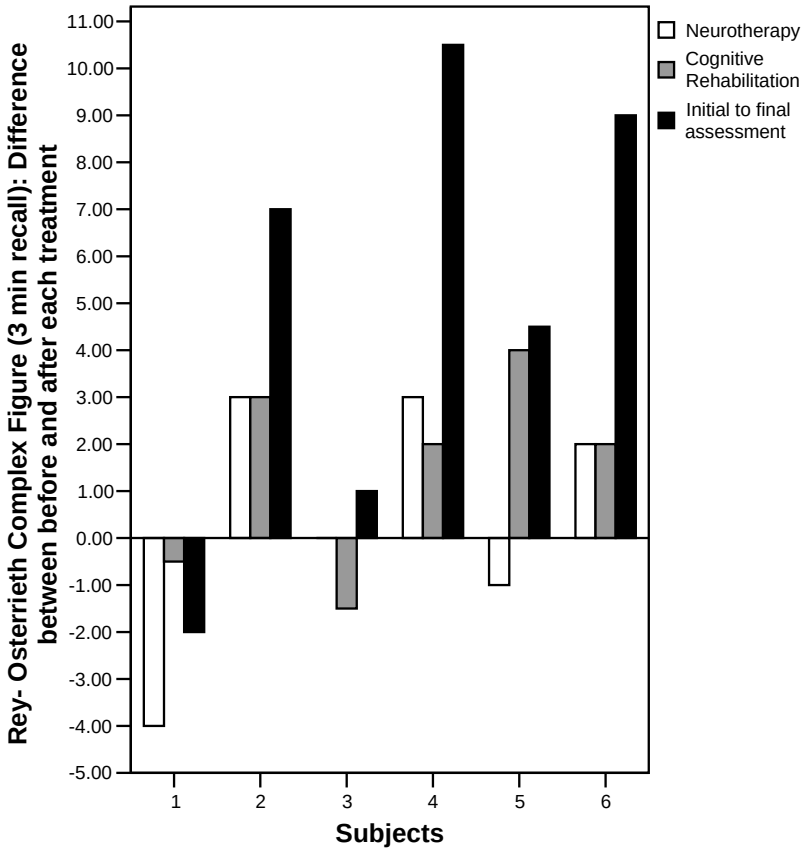
8) REY AUDITORY VERBAL LEARNING TEST (RAVLT): RECOGNITION



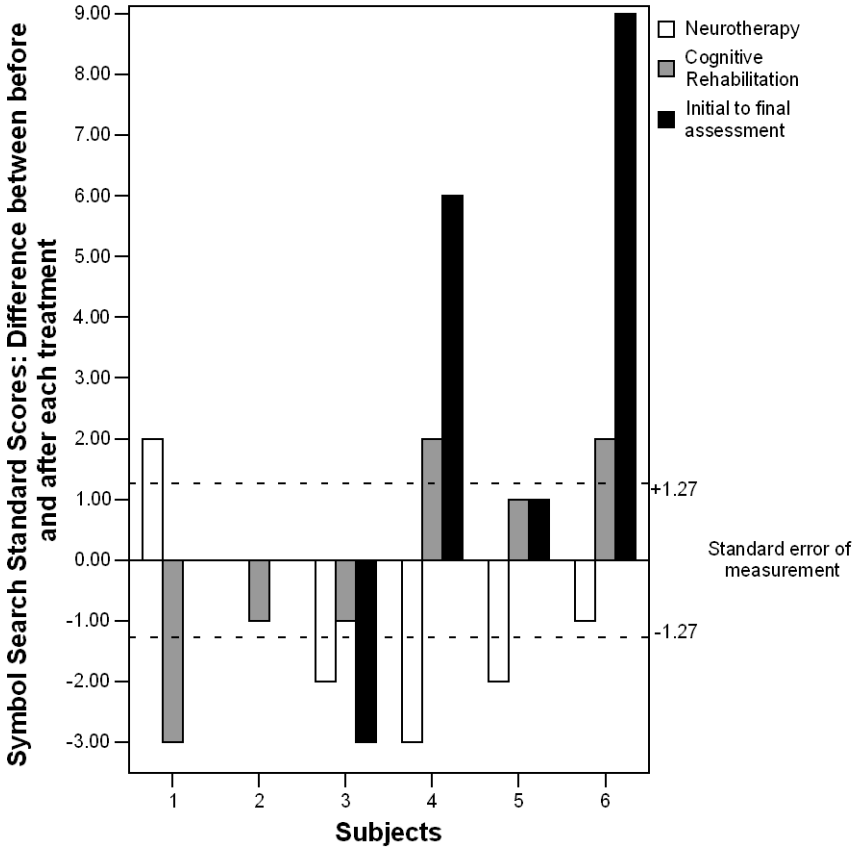
9) REY-OSTERRIETH COMPLEX FIGURE: COPY



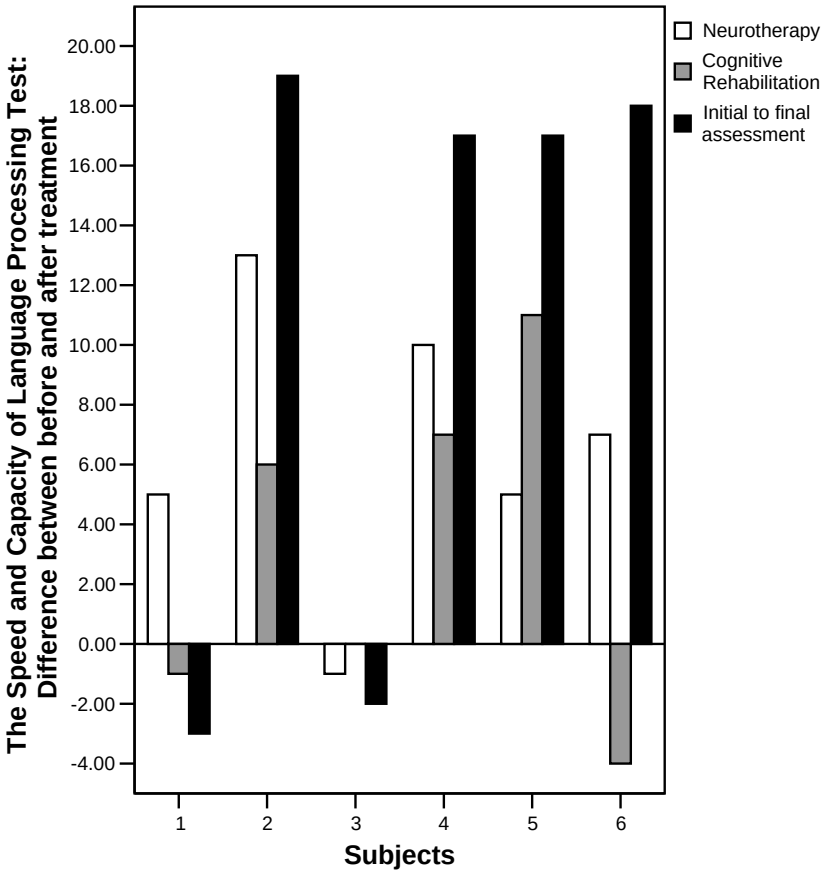
10) REY-OSTERRIETH COMPLEX FIGURE: RECALL



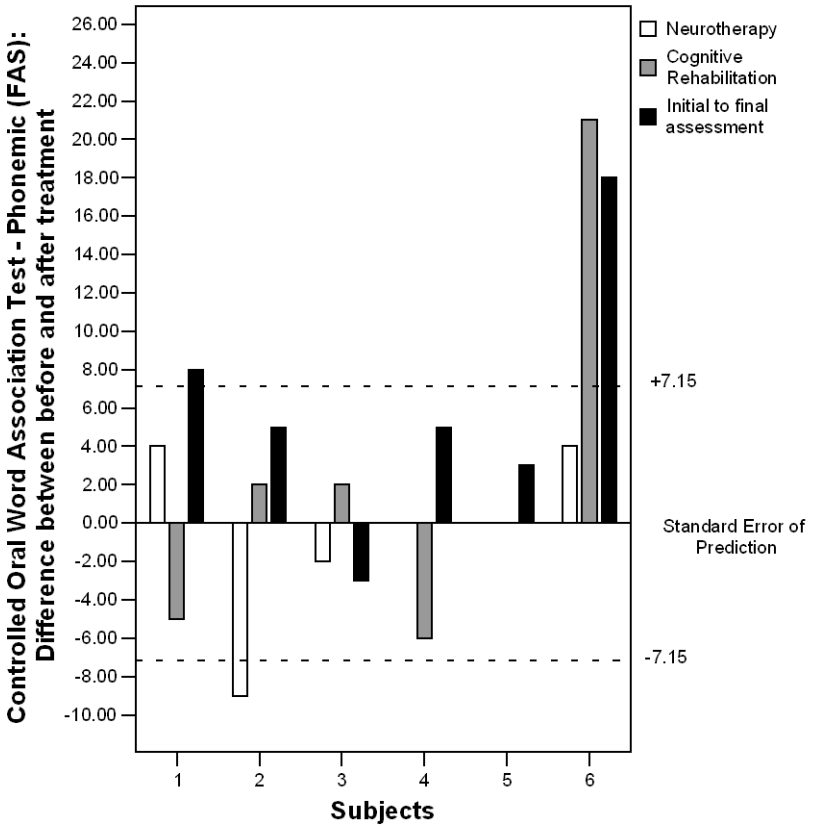
11) SYMBOL SEARCH



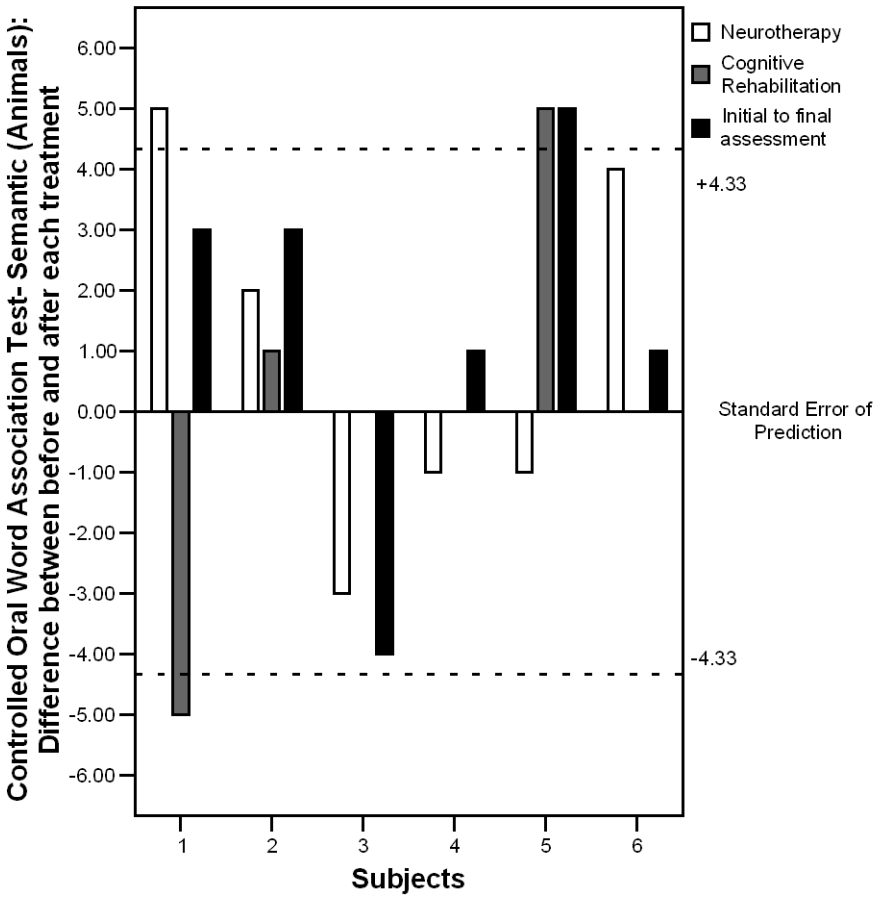
12) SPEED AND CAPACITY OF LANGUAGE PROCESSING TEST (SCOLP)



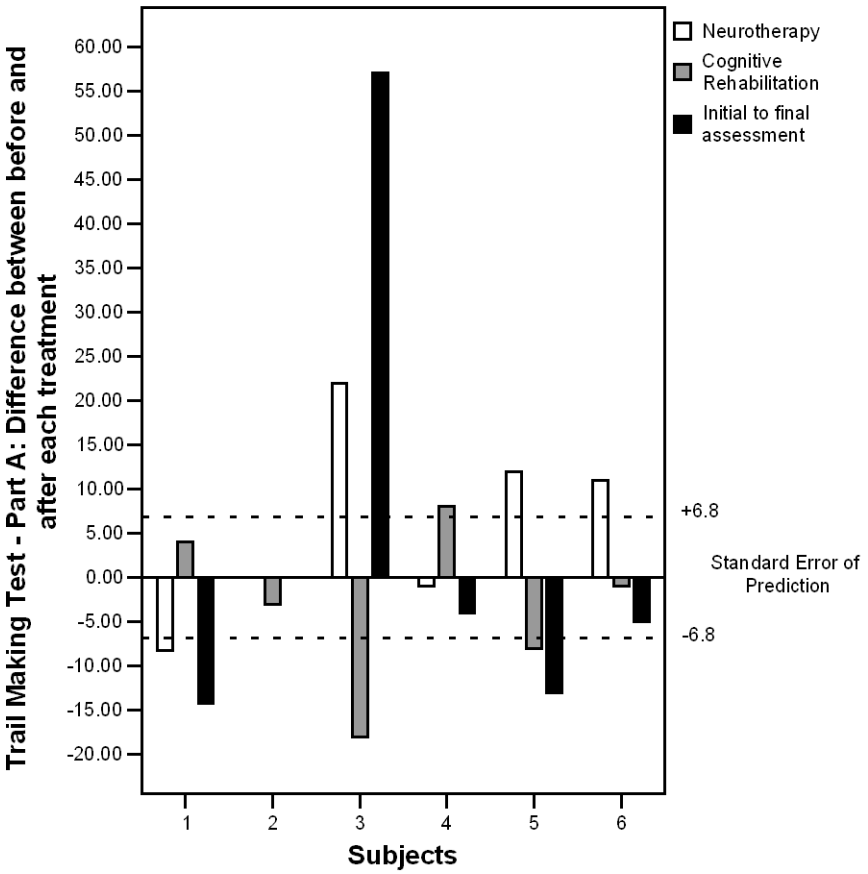
13) CONTROLLED ORAL WORD ASSOCIATION TEST: PHONEMIC (FAS)



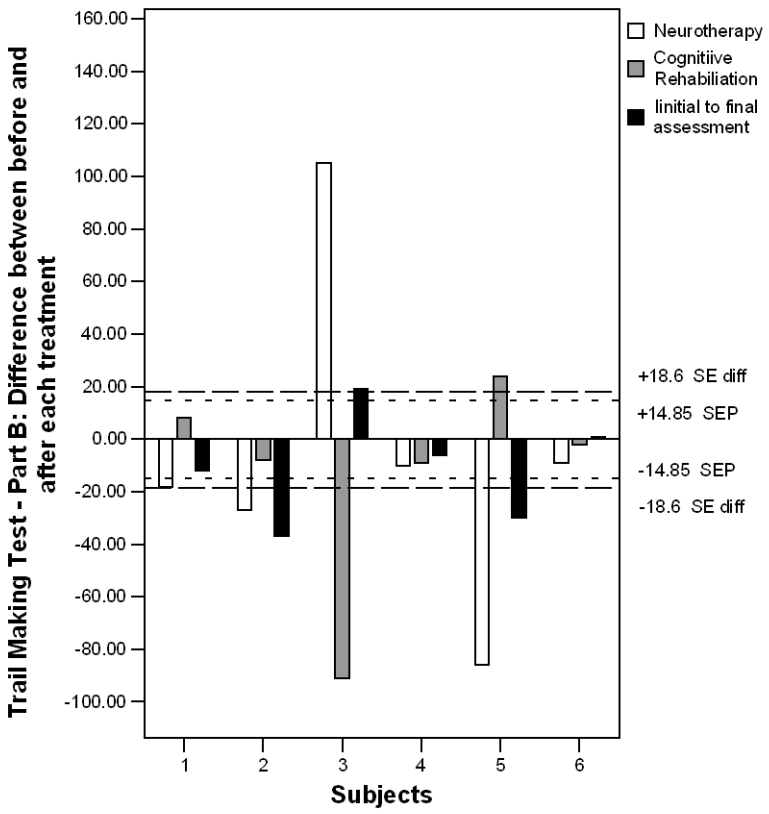
14) CONTROLLED ORAL WORD ASSOCIATION TEST: SEMANTIC (ANIMALS)



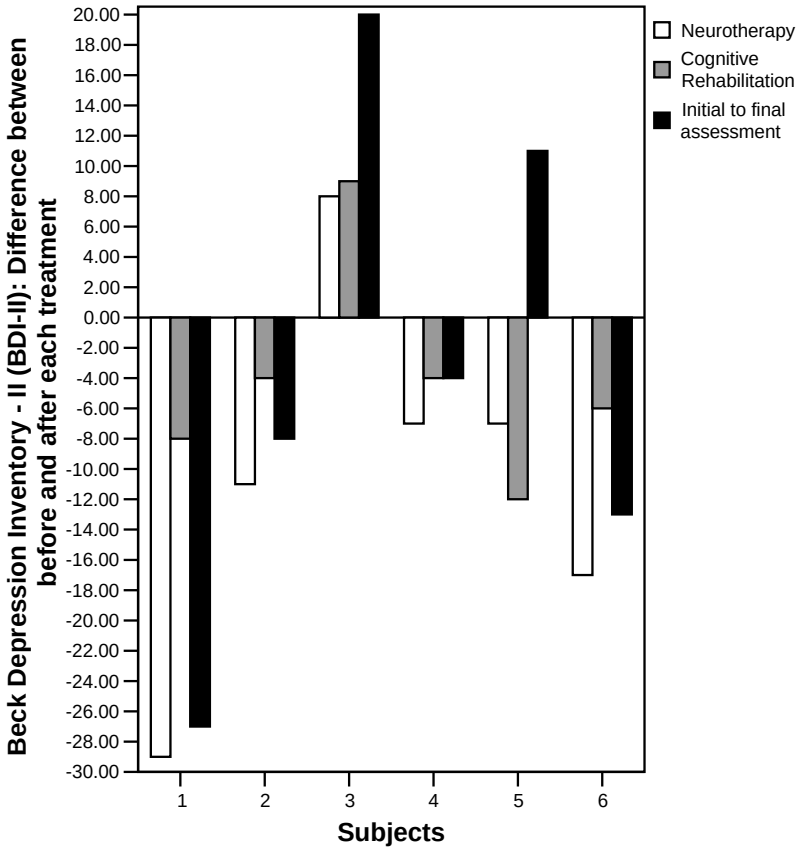
15) TRAIL MAKING TEST – PART A



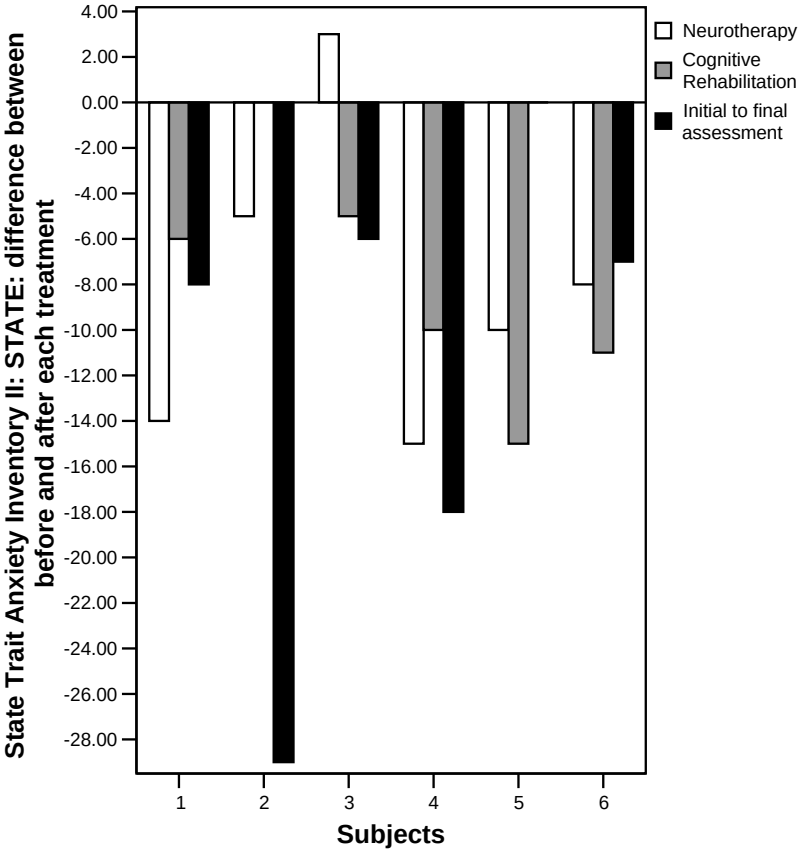
16) TRAIL MAKING TEST – PART B



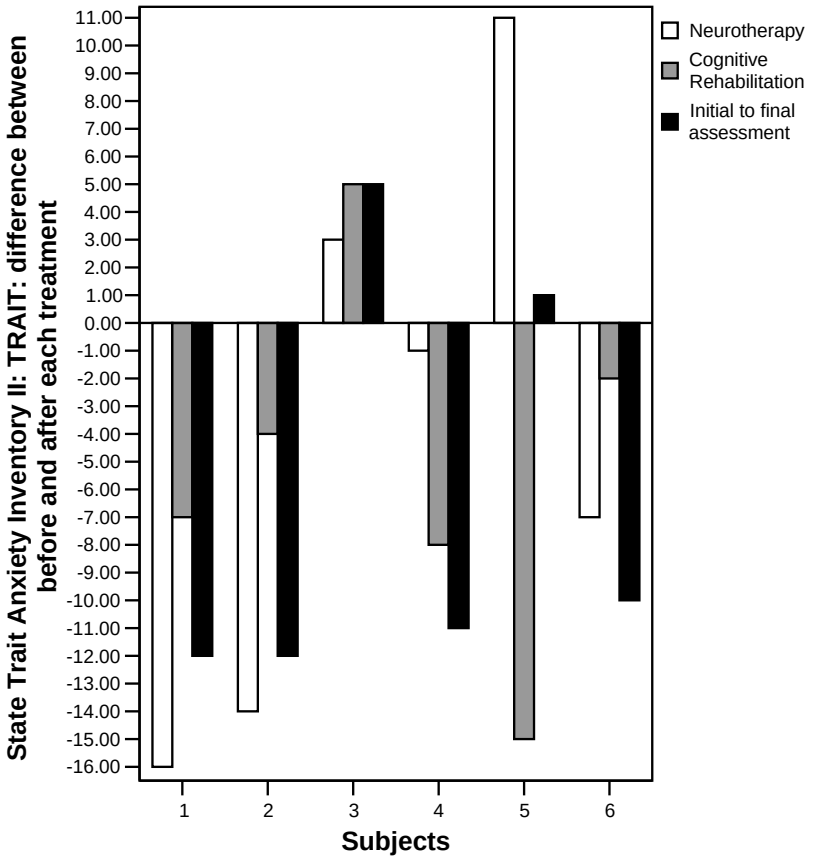
17) BECK DEPRESSION INVENTORY – II (BDI-II)



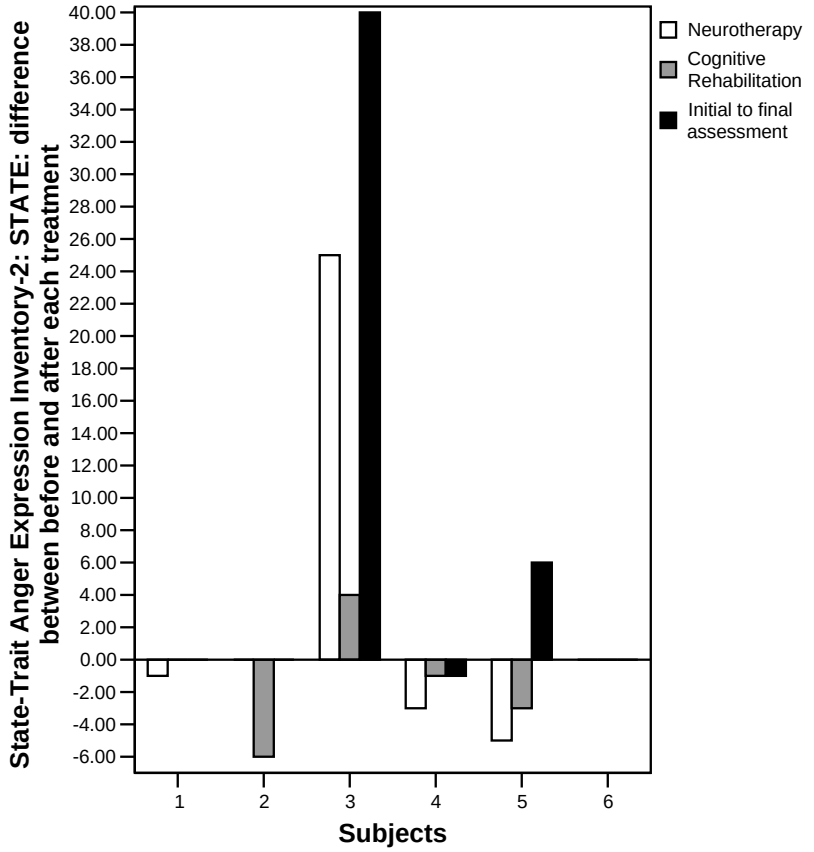
18) STATE TRAIT ANXIETY INVENTORY (STAI): STATE



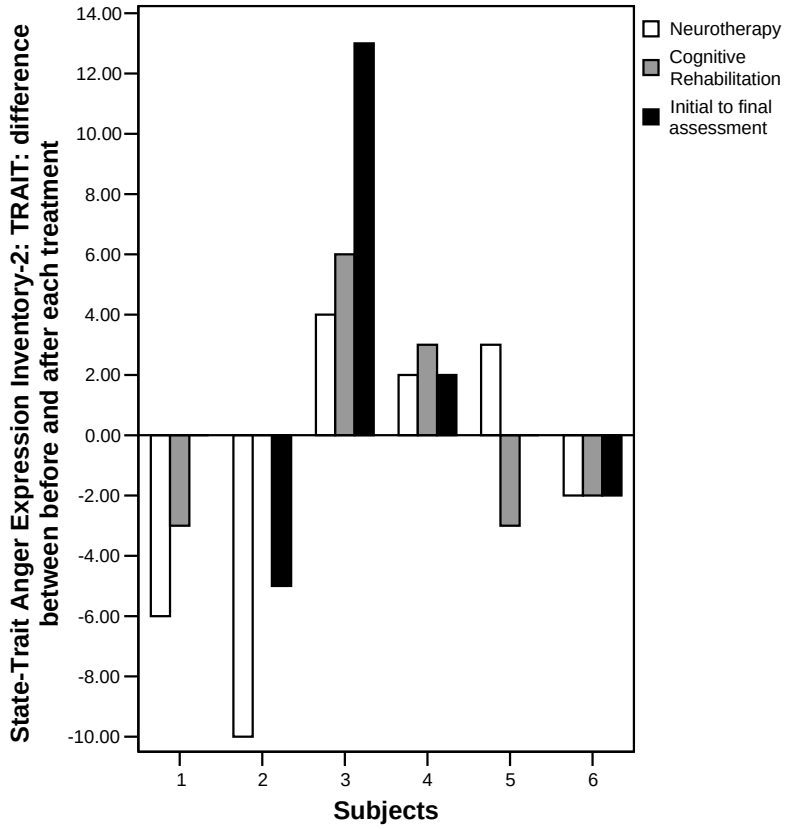
19) STATE TRAIT ANXIETY INVENTORY (STAI): TRAIT



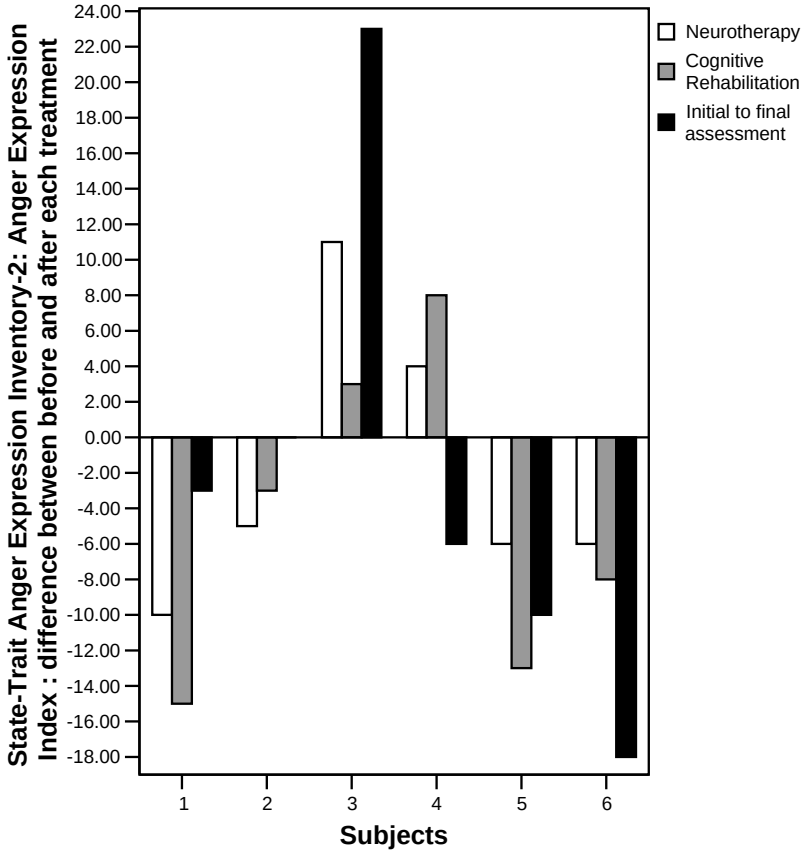
20) STATE TRAIT ANGER EXPRESSION INVENTORY-II (STAXI): STATE



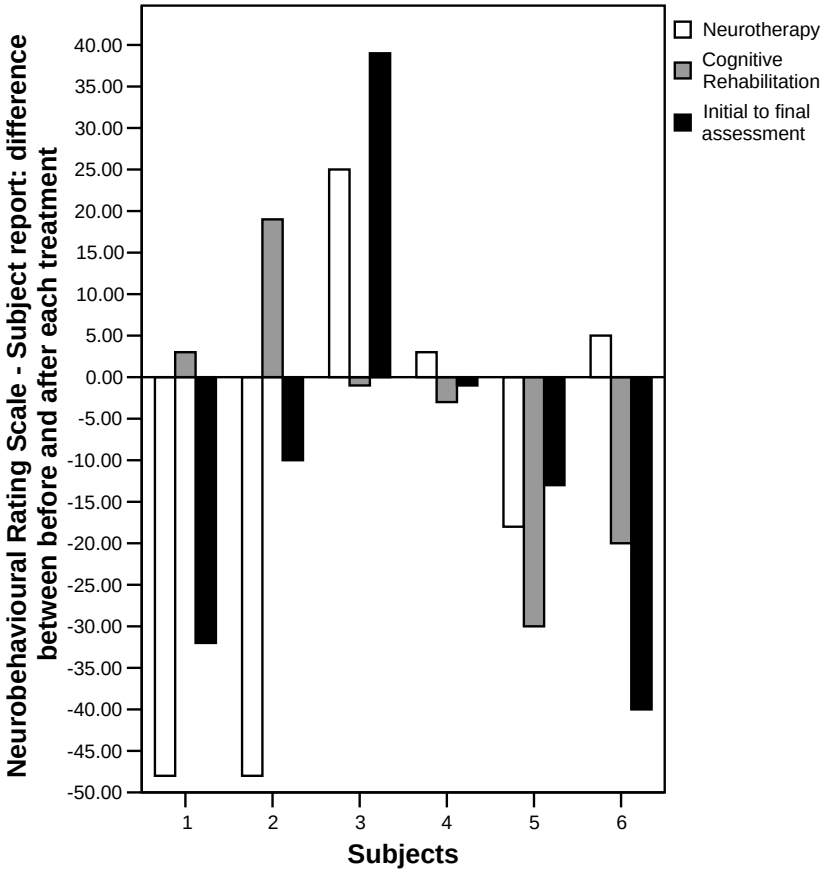
21) STATE TRAIT ANGER EXPRESSION INVENTORY-II (STAXI): TRAIT



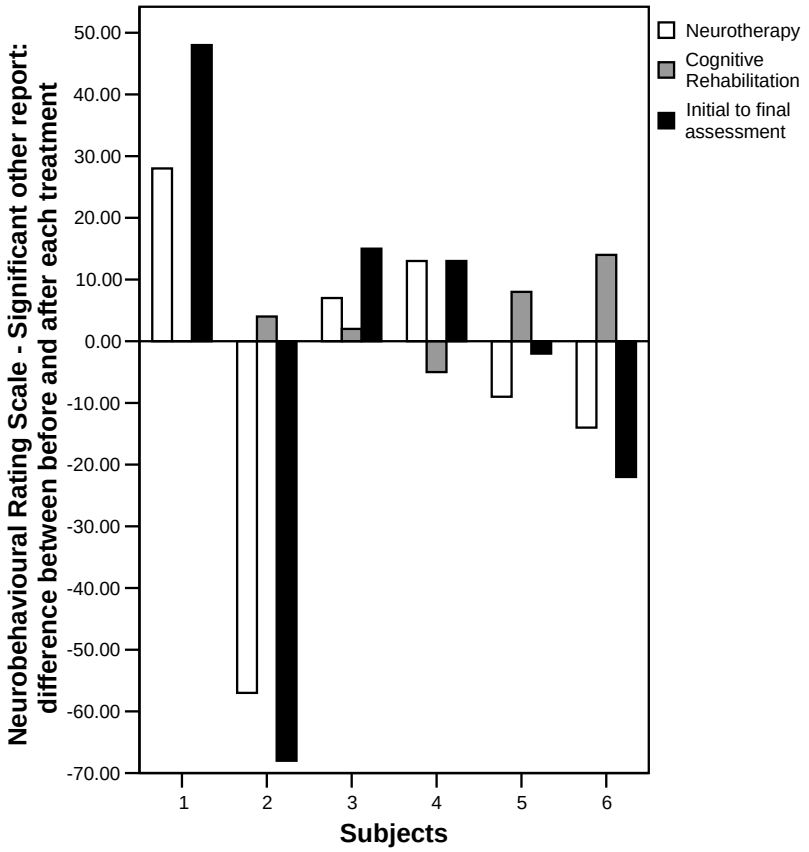
22) STATE TRAIT ANGER EXPRESSION INVENTORY-II (STAXI): ANGER EXPRESSION INDEX



23) NEUROBEHAVIOURAL RATING SCALE (NRS): SUBJECT REPORT



24) NEUROBEHAVIOURAL RATING SCALE (NRS): SIGNIFICANT OTHER



APPENDIX 11:

Examination of Treatment Order: Mann-Whitney U Results

Test of Variables of Attention - Omissions

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Test of Variables of Attention: Omissions: difference between before and after Neurotherapy	Neurotherapy first	2	2.50	5.00
	cognitive rehab first	3	3.33	10.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Test of Variables of Attention: Omissions: difference between before and after Cognitive Rehab	Neurotherapy first	2	3.50	7.00
	cognitive rehab first	3	2.67	8.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Test of Variables of Attention: Omissions: difference between initial and final assessment	Neurotherapy first	2	2.50	5.00
	cognitive rehab first	3	3.33	10.00
	Total	5		

Test Statistics(b)

	Test of Variables of Attention: Omissions: difference between before and after Neurotherapy
Mann-Whitney U	2.000
Wilcoxon W	5.000
Z	-.645
Asymp. Sig. (2-tailed)	.519
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Test of Variables of Attention: Omissions: difference between before and after Cognitive Rehab
Mann-Whitney U	2.000
Wilcoxon W	8.000

Z	-.592
Asymp. Sig. (2-tailed)	.554
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Test of Variables of Attention: Omissions: difference between initial and final assessment
Mann-Whitney U	2.000
Wilcoxon W	5.000
Z	-.577
Asymp. Sig. (2-tailed)	.564
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test of Variables of Attention – Commissions

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Test of Variables of Attention: Comissions: difference between before and after Neurotherapy	Neurotherapy first	2	1.50	3.00
	cognitive rehab first	3	4.00	12.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Test of Variables of Attention: Comissions: difference between before and after Cognitive Rehab	Neurotherapy first	2	3.50	7.00
	cognitive rehab first	3	2.67	8.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Test of Variables of Attention: Comissions: difference between initial and final assessment	Neurotherapy first	2	1.50	3.00
	cognitive rehab first	3	4.00	12.00
	Total	5		

Test Statistics(b)

	Test of Variables of Attention: Comissions: difference between before and after Neurotherapy
Mann-Whitney U	.000
Wilcoxon W	3.000
Z	-1.732
Asymp. Sig. (2-tailed)	.083
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Test of Variables of Attention: Comissions: difference between before and after Cognitive Rehab
Mann-Whitney U	2.000
Wilcoxon W	8.000
Z	-.577
Asymp. Sig. (2-tailed)	.564
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Test of Variables of Attention: Comissions: difference between initial and final assessment
Mann-Whitney U	.000
Wilcoxon W	3.000
Z	-1.732
Asymp. Sig. (2-tailed)	.083
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test of Variables of Attention - Response Time

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Test of Variables of Attention: Response Time: difference between before and after Neurotherapy	Neurotherapy first	2	2.50	5.00
	cognitive rehab first	3	3.33	10.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Test of Variables of Attention: Response Time: difference between before and after Cognitive Rehab	Neurotherapy first	2	2.50	5.00
	cognitive rehab first	3	3.33	10.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Test of Variables of Attention: Response Time: difference between initial and final assessment	Neurotherapy first	2	3.50	7.00
	cognitive rehab first	3	2.67	8.00
	Total	5		

Test Statistics(b)

	Test of Variables of Attention: Response Time: difference between before and after Neurotherapy
Mann-Whitney U	2.000
Wilcoxon W	5.000
Z	-.577
Asymp. Sig. (2-tailed)	.564
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Test of Variables of Attention: Response Time: difference between before and after Cognitive Rehab
Mann-Whitney U	2.000
Wilcoxon W	5.000
Z	-.577
Asymp. Sig. (2-tailed)	.564
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Test of Variables of Attention: Response Time: difference between initial and final assessment
Mann-Whitney U	2.000
Wilcoxon W	8.000
Z	-.577
Asymp. Sig. (2-tailed)	.564
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test of Variables of Attention – Variability

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Test of Variables of Attention: Variability: difference between before and after Neurotherapy	Neurotherapy first	2	3.00	6.00
	cognitive rehab first	3	3.00	9.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Test of Variables of Attention: Variability: difference between before and after Cognitive Rehab	Neurotherapy first	2	3.50	7.00
	cognitive rehab first	3	2.67	8.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Test of Variables of Attention: Variability: difference between initial and final assessment	Neurotherapy first	2	3.00	6.00
	cognitive rehab first	3	3.00	9.00
	Total	5		

Test Statistics(b)

	Test of Variables of Attention: Variability: difference between before and after Neurotherapy
Mann-Whitney U	3.000
Wilcoxon W	9.000
Z	.000
Asymp. Sig. (2-tailed)	1.000
Exact Sig. [2*(1-tailed Sig.)]	1.000(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Test of Variables of Attention: Variability: difference between before and after Cognitive Rehab
Mann-Whitney U	2.000
Wilcoxon W	8.000
Z	-.577
Asymp. Sig. (2-tailed)	.564
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Test of Variables of Attention: Variability: difference between initial and final assessment
Mann-Whitney U	3.000
Wilcoxon W	9.000
Z	.000

Asymp. Sig. (2-tailed)	1.000
Exact Sig. [2*(1-tailed Sig.)]	1.000(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Paced Auditory Serial Addition Test

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Paced Auditory Serial Addition Test : difference between before and after Neurotherapy	Neurotherapy first	2	1.50	3.00
	cognitive rehab first	3	4.00	12.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Paced Auditory Serial Addition Test : difference between before and after Cognitive Rehab	Neurotherapy first	2	3.50	7.00
	cognitive rehab first	3	2.67	8.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Paced Auditory Serial Addition Test : difference between initial and final assessment	Neurotherapy first	2	4.00	8.00
	cognitive rehab first	3	2.33	7.00
	Total	5		

Test Statistics(b)

	Paced Auditory Serial Addition Test : difference between before and after Neurotherapy
Mann-Whitney U	.000
Wilcoxon W	3.000
Z	-1.732
Asymp. Sig. (2-tailed)	.083
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Paced Auditory Serial Addition Test : difference between before and after Cognitive Rehab
Mann-Whitney U	2.000
Wilcoxon W	8.000
Z	-.577
Asymp. Sig. (2-tailed)	.564
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Paced Auditory Serial Addition Test : difference between initial and final assessment
Mann-Whitney U	1.000
Wilcoxon W	7.000
Z	-1.155
Asymp. Sig. (2-tailed)	.248
Exact Sig. [2*(1-tailed Sig.)]	.400(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Rey Auditory Verbal Learning Test - Total Recall

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Rey Auditory Verbal Learning Test - TOTAL RECALL - difference between before and after neurotherapy	Neurotherapy first	2	2.50	5.00
	cognitive rehab first	3	3.33	10.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Rey Auditory Verbal Learning Test - TOTAL RECALL - difference between before and after Cognitive Rehabilitation	Neurotherapy first	2	2.75	5.50
	cognitive rehab first	3	3.17	9.50
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Rey Auditory Verbal Learning Test - TOTAL RECALL - difference between initial and final assessment	Neurotherapy first	2	2.25	4.50
	cognitive rehab first	3	3.50	10.50
	Total	5		

Test Statistics(b)

	Rey Auditory Verbal Learning Test - TOTAL RECALL - difference between before and after neurotherapy
Mann-Whitney U	2.000
Wilcoxon W	5.000
Z	-.577
Asymp. Sig. (2-tailed)	.564
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Rey Auditory Verbal Learning Test - TOTAL RECALL - difference between before and after Cognitive Rehabilitation
Mann-Whitney U	2.500
Wilcoxon W	5.500
Z	-.296
Asymp. Sig. (2-tailed)	.767
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Rey Auditory Verbal Learning Test - TOTAL RECALL - difference between initial and final assessment

Mann-Whitney U	1.500
Wilcoxon W	4.500
Z	-.889
Asymp. Sig. (2-tailed)	.374
Exact Sig. [2*(1-tailed Sig.)]	.400(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Rey Auditory Verbal Learning Test - Delayed Recall

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Rey Auditory Verbal Learning Test - DELAYED RECALL - difference between before and after neurotherapy	Neurotherapy first	2	2.50	5.00
	cognitive rehab first	3	3.33	10.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Rey Auditory Verbal Learning Test - DELAYED RECALL - difference between before and after Cognitive Rehabilitation	Neurotherapy first	2	4.50	9.00
	cognitive rehab first	3	2.00	6.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Rey Auditory Verbal Learning Test - DELAYED RECALL - difference between initial and final assessment	Neurotherapy first	2	1.75	3.50
	cognitive rehab first	3	3.83	11.50
	Total	5		

Test Statistics(b)

	Rey Auditory Verbal Learning Test - DELAYED RECALL - difference between before and after neurotherapy
Mann-Whitney U	2.000
Wilcoxon W	5.000
Z	-.577
Asymp. Sig. (2-tailed)	.564
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Rey Auditory Verbal Learning Test - DELAYED RECALL - difference between before and after Cognitive Rehabilitation
Mann-Whitney U	.000
Wilcoxon W	6.000
Z	-1.732
Asymp. Sig. (2-tailed)	.083
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Rey Auditory Verbal Learning Test - DELAYED RECALL - difference between initial and final assessment
Mann-Whitney U	.500
Wilcoxon W	3.500
Z	-1.481
Asymp. Sig. (2-tailed)	.139
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Rey Auditory Verbal Learning Test - Recognition

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Rey Auditory Verbal Learning Test - RECOGNITION - difference between before and after neurotherapy	Neurotherapy first	2	1.50	3.00
	cognitive rehab first	3	4.00	12.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Rey Auditory Verbal Learning Test - RECOGNITION - difference between	Neurotherapy first	2	4.00	8.00
	cognitive rehab first	3	2.33	7.00

before and after Cognitive Rehabilitation	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Rey Auditory Verbal Learning Test - RECOGNITION - difference between initial and final assessment	Neurotherapy first	2	2.50	5.00
	cognitive rehab first	3	3.33	10.00
	Total	5		

Test Statistics(b)

	Rey Auditory Verbal Learning Test - RECOGNITION - difference between before and after neurotherapy
Mann-Whitney U	.000
Wilcoxon W	3.000
Z	-1.777
Asymp. Sig. (2-tailed)	.076
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Rey Auditory Verbal Learning Test - RECOGNITION - difference between before and after Cognitive Rehabilitation
Mann-Whitney U	1.000
Wilcoxon W	7.000
Z	-1.291
Asymp. Sig. (2-tailed)	.197
Exact Sig. [2*(1-tailed Sig.)]	.400(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Rey Auditory Verbal Learning Test - RECOGNITION - difference between initial and final assessment
Mann-Whitney U	2.000
Wilcoxon W	5.000
Z	-.645
Asymp. Sig. (2-tailed)	.519
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Rey Osterrieth Complex Figure - Copy

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Rey Complex Figure Copy - difference between before and after neurotherapy	Neurotherapy first	2	2.50	5.00
	cognitive rehab first	3	3.33	10.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Rey Complex Figure Copy - difference between before and after Cognitive rehabilitation	Neurotherapy first	2	3.25	6.50
	cognitive rehab first	3	2.83	8.50
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Rey Complex Figure Copy - difference between the initial and final assessment	Neurotherapy first	2	2.00	4.00
	cognitive rehab first	3	3.67	11.00
	Total	5		

Test Statistics(b)

	Rey Complex Figure Copy - difference between before and after neurotherapy
Mann-Whitney U	2.000
Wilcoxon W	5.000
Z	-.577
Asymp. Sig. (2-tailed)	.564

Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Rey Complex Figure Copy - difference between before and after Cognitive rehabilitation
Mann-Whitney U	2.500
Wilcoxon W	8.500
Z	-.296
Asymp. Sig. (2-tailed)	.767
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Rey Complex Figure Copy - difference between the initial and final assessment
Mann-Whitney U	1.000
Wilcoxon W	4.000
Z	-1.291
Asymp. Sig. (2-tailed)	.197
Exact Sig. [2*(1-tailed Sig.)]	.400(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Rey Osterrieth Complex Figure - Recall

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Rey Complex Figure 3 min Recall - difference between before and after neurotherapy	Neurotherapy first	2	2.75	5.50
	cognitive rehab first	3	3.17	9.50
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Rey Complex Figure 3 min Recall - difference between before and after Cognitive Rehabilitation	Neurotherapy first	2	2.50	5.00
	cognitive rehab first	3	3.33	10.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Rey Complex Figure 3 min Recall - difference between initial and final assessment	Neurotherapy first	2	2.00	4.00
	cognitive rehab first	3	3.67	11.00
	Total	5		

Test Statistics(b)

	Rey Complex Figure 3 min Recall - difference between before and after neurotherapy
Mann-Whitney U	2.500
Wilcoxon W	5.500
Z	-.296
Asymp. Sig. (2-tailed)	.767
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Rey Complex Figure 3 min Recall - difference between before and after Cognitive Rehabilitation
Mann-Whitney U	2.000
Wilcoxon W	5.000
Z	-.592
Asymp. Sig. (2-tailed)	.554
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Rey Complex Figure 3 min Recall - difference between initial and final assessment
Mann-Whitney U	1.000
Wilcoxon W	4.000
Z	-1.155
Asymp. Sig. (2-tailed)	.248
Exact Sig. [2*(1-tailed Sig.)]	.400(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Symbol Search

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Symboldiff21	Neurotherapy first	2	4.50	9.00
	cognitive rehab first	3	2.00	6.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Symboldiff43	Neurotherapy first	2	1.50	3.00
	cognitive rehab first	3	4.00	12.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Symboldiff50	Neurotherapy first	2	1.75	3.50
	cognitive rehab first	3	3.83	11.50
	Total	5		

Test Statistics(b)

	Symboldiff21
Mann-Whitney U	.000
Wilcoxon W	6.000
Z	-1.732
Asymp. Sig. (2-tailed)	.083
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Symboldiff43
Mann-Whitney U	.000
Wilcoxon W	3.000
Z	-1.777
Asymp. Sig. (2-tailed)	.076
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Symboldiff50
Mann-Whitney U	.500
Wilcoxon W	3.500

Z	-1.481
Asymp. Sig. (2-tailed)	.139
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Speed and Capacity of Language-Processing Test

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
The Speed and Capacity of Language-Processing Test - difference between before and after neurotherapy	Neurotherapy first	2	3.25	6.50
	cognitive rehab first	3	2.83	8.50
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
The Speed and Capacity of Language-Processing Test - difference between before and after cognitive rehabilitation	Neurotherapy first	2	2.50	5.00
	cognitive rehab first	3	3.33	10.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
The Speed and Capacity of Language-Processing Test - difference between Initial and final assessment	Neurotherapy first	2	3.00	6.00
	cognitive rehab first	3	3.00	9.00
	Total	5		

Test Statistics(b)

	The Speed and Capacity of Language-Processing Test - difference between before and after neurotherapy
Mann-Whitney U	2.500
Wilcoxon W	8.500
Z	-.296
Asymp. Sig. (2-tailed)	.767
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	The Speed and Capacity of Language-Processing Test - difference between before and after cognitive rehabilitation
Mann-Whitney U	2.000
Wilcoxon W	5.000
Z	-.577
Asymp. Sig. (2-tailed)	.564
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	The Speed and Capacity of Language-Processing Test - difference between Initial and final assessment
Mann-Whitney U	3.000
Wilcoxon W	9.000
Z	.000
Asymp. Sig. (2-tailed)	1.000
Exact Sig. [2*(1-tailed Sig.)]	1.000(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Controlled Oral Word Association - FAS

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Controlled Oral Word Association - Phonemic - difference between before and after neurotherapy	Neurotherapy first	2	2.75	5.50
	cognitive rehab first	3	3.17	9.50
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Controlled Oral Word Association - Phonemic - difference between	Neurotherapy first	2	3.00	6.00
	cognitive rehab first	3	3.00	9.00

before and after Cognitive Rehabilitation	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Controlled Oral Word Association - Phonemic - difference between initial and final assessment	Neurotherapy first	2	3.25	6.50
	cognitive rehab first	3	2.83	8.50
	Total	5		

Test Statistics(b)

	Controlled Oral Word Association - Phonemic - difference between before and after neurotherapy
Mann-Whitney U	2.500
Wilcoxon W	5.500
Z	-.304
Asymp. Sig. (2-tailed)	.761
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Controlled Oral Word Association - Phonemic - difference between before and after Cognitive Rehabilitation
Mann-Whitney U	3.000
Wilcoxon W	9.000
Z	.000
Asymp. Sig. (2-tailed)	1.000
Exact Sig. [2*(1-tailed Sig.)]	1.000(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Controlled Oral Word Association - Phonemic - difference between initial and final assessment
Mann-Whitney U	2.500
Wilcoxon W	8.500
Z	-.296
Asymp. Sig. (2-tailed)	.767
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Controlled Oral Word Association - Animals

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Controlled Oral Word Association - Semantic - difference between before and after neurotherapy	Neurotherapy first	2	4.00	8.00
	cognitive rehab first	3	2.33	7.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Controlled Oral Word Association - Semantic - difference between before and after Cognitive Rehabilitation	Neurotherapy first	2	2.50	5.00
	cognitive rehab first	3	3.33	10.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Controlled Oral Word Association - Semantic - difference between initial and final assessment	Neurotherapy first	2	3.50	7.00
	cognitive rehab first	3	2.67	8.00
	Total	5		

Test Statistics(b)

	Controlled Oral Word Association - Semantic - difference between before and after neurotherapy
Mann-Whitney U	1.000
Wilcoxon W	7.000
Z	-1.185

Asymp. Sig. (2-tailed)	.236
Exact Sig. [2*(1-tailed Sig.)]	.400(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Controlled Oral Word Association - Semantic - difference between before and after Cognitive Rehabilitation
Mann-Whitney U	2.000
Wilcoxon W	5.000
Z	-.592
Asymp. Sig. (2-tailed)	.554
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Controlled Oral Word Association - Semantic - difference between initial and final assessment
Mann-Whitney U	2.000
Wilcoxon W	8.000
Z	-.609
Asymp. Sig. (2-tailed)	.543
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Trail Making Test - Part A

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Trail Making Test - Part A - difference between before and after neurotherapy	Neurotherapy first	2	2.00	4.00
	cognitive rehab first	3	3.67	11.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Trail Making Test - Part	Neurotherapy first	2	3.00	6.00

A - difference between before and after Cognitive Rehabilitation	cognitive rehab first	3	3.00	9.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Trail Making Test - Part A - difference between initial and final assessment	Neurotherapy first	2	3.00	6.00
	cognitive rehab first	3	3.00	9.00
	Total	5		

Test Statistics(b)

	Trail Making Test - Part A - difference between before and after neurotherapy
Mann-Whitney U	1.000
Wilcoxon W	4.000
Z	-1.155
Asymp. Sig. (2-tailed)	.248
Exact Sig. [2*(1-tailed Sig.)]	.400(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Trail Making Test - Part A - difference between before and after Cognitive Rehabilitation
Mann-Whitney U	3.000
Wilcoxon W	9.000
Z	.000
Asymp. Sig. (2-tailed)	1.000
Exact Sig. [2*(1-tailed Sig.)]	1.000(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Trail Making Test - Part A - difference between initial and final assessment
Mann-Whitney U	3.000
Wilcoxon W	9.000
Z	.000
Asymp. Sig. (2-tailed)	1.000

Exact Sig. [2*(1-tailed Sig.)]	1.000(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Trail Making Test - Part B

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Trail Making Test - Part B - difference between before and after neurotherapy	Neurotherapy first	2	2.50	5.00
	cognitive rehab first	3	3.33	10.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Trail Making Test - Part B - difference between before and after Cognitiive Rehabiliation	Neurotherapy first	2	3.00	6.00
	cognitive rehab first	3	3.00	9.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Trail Making Test - Part B - difference between initial and final assessment	Neurotherapy first	2	2.00	4.00
	cognitive rehab first	3	3.67	11.00
	Total	5		

Test Statistics(b)

	Trail Making Test - Part B - difference between before and after neurotherapy
Mann-Whitney U	2.000
Wilcoxon W	5.000
Z	-.577
Asymp. Sig. (2-tailed)	.564
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Trail Making Test - Part B - difference between before and after Cognitiive Rehabiliation
Mann-Whitney U	3.000
Wilcoxon W	9.000
Z	.000
Asymp. Sig. (2-tailed)	1.000
Exact Sig. [2*(1-tailed Sig.)]	1.000(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Trail Making Test - Part B - difference between initial and final assessment
Mann-Whitney U	1.000
Wilcoxon W	4.000
Z	-1.155
Asymp. Sig. (2-tailed)	.248
Exact Sig. [2*(1-tailed Sig.)]	.400(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Beck Depression Inventory - II

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Beck Depression Inventory II : difference between before and after Neurotherapy	Neurotherapy first	2	2.00	4.00
	cognitive rehab first	3	3.67	11.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Beck Depression Inventory II : difference between before and after Cognitive Rehab	Neurotherapy first	2	3.25	6.50
	cognitive rehab first	3	2.83	8.50
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Beck Depression Inventory II : difference between	Neurotherapy first	2	2.00	4.00
	cognitive rehab first	3	3.67	11.00

initial and final assessment	Total	5		

Test Statistics(b)

	Beck Depression Inventory II : difference between before and after Neurotherapy
Mann-Whitney U	1.000
Wilcoxon W	4.000
Z	-1.185
Asymp. Sig. (2-tailed)	.236
Exact Sig. [2*(1-tailed Sig.)]	.400(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Beck Depression Inventory II : difference between before and after Cognitive Rehab
Mann-Whitney U	2.500
Wilcoxon W	8.500
Z	-.296
Asymp. Sig. (2-tailed)	.767
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Beck Depression Inventory II : difference between initial and final assessment
Mann-Whitney U	1.000
Wilcoxon W	4.000
Z	-1.155
Asymp. Sig. (2-tailed)	.248
Exact Sig. [2*(1-tailed Sig.)]	.400(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

State Trait Anxiety Inventory - STATE

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
State Trait Anxiety Inventory II: STATE: difference between before and after Neurotherapy	Neurotherapy first	2	3.50	7.00
	cognitive rehab first	3	2.67	8.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
State Trait Anxiety Inventory II: STATE: difference between before and after Cognitive Rehab	Neurotherapy first	2	4.50	9.00
	cognitive rehab first	3	2.00	6.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
State Trait Anxiety Inventory II: STATE:difference between initial and final assessment	Neurotherapy first	2	2.00	4.00
	cognitive rehab first	3	3.67	11.00
	Total	5		

Test Statistics(b)

	State Trait Anxiety Inventory II: STATE: difference between before and after Neurotherapy
Mann-Whitney U	2.000
Wilcoxon W	8.000
Z	-.577
Asymp. Sig. (2-tailed)	.564
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	State Trait Anxiety Inventory II: STATE: difference between before and after Cognitive Rehab
Mann-Whitney U	.000
Wilcoxon W	6.000

Z	-1.732
Asymp. Sig. (2-tailed)	.083
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	State Trait Anxiety Inventory II: STATE:difference between initial and final assessment
Mann-Whitney U	1.000
Wilcoxon W	4.000
Z	-1.155
Asymp. Sig. (2-tailed)	.248
Exact Sig. [2*(1-tailed Sig.)]	.400(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

State Trait Anxiety Inventory - TRAIT

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
State Trait Anxiety Inventory II: TRAIT: difference between before and after Neurotherapy	Neurotherapy first	2	1.50	3.00
	cognitive rehab first	3	4.00	12.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
State Trait Anxiety Inventory II: TRAIT: difference between before and after Cognitive Rehab	Neurotherapy first	2	3.50	7.00
	cognitive rehab first	3	2.67	8.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
State Trait Anxiety Inventory II: TRAIT: difference between initial and final assessment	Neurotherapy first	2	1.50	3.00
	cognitive rehab first	3	4.00	12.00
	Total	5		

Test Statistics(b)

	State Trait Anxiety Inventory II: TRAIT: difference between before and after Neurotherapy
Mann-Whitney U	.000
Wilcoxon W	3.000
Z	-1.732
Asymp. Sig. (2-tailed)	.083
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	State Trait Anxiety Inventory II: TRAIT: difference between before and after Cognitive Rehab
Mann-Whitney U	2.000
Wilcoxon W	8.000
Z	-.577
Asymp. Sig. (2-tailed)	.564
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	State Trait Anxiety Inventory II: TRAIT: difference between initial and final assessment
Mann-Whitney U	.000
Wilcoxon W	3.000
Z	-1.777
Asymp. Sig. (2-tailed)	.076
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

State-Trait Anger Expression Inventory - II - STATE

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
State-Trait Anger Expression Inventory-2: STATE: difference between before and after Neurotherapy	Neurotherapy first	2	3.75	7.50
	cognitive rehab first	3	2.50	7.50
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
State-Trait Anger Expression Inventory-2: STATE: difference between before and after Cognitive Rehab	Neurotherapy first	2	2.75	5.50
	cognitive rehab first	3	3.17	9.50
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
State-Trait Anger Expression Inventory-2: STATE: difference between initial and final assessment	Neurotherapy first	2	3.00	6.00
	cognitive rehab first	3	3.00	9.00
	Total	5		

Test Statistics(b)

	State-Trait Anger Expression Inventory-2: STATE: difference between before and after Neurotherapy
Mann-Whitney U	1.500
Wilcoxon W	7.500
Z	-.889
Asymp. Sig. (2-tailed)	.374
Exact Sig. [2*(1-tailed Sig.)]	.400(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	State-Trait Anger Expression Inventory-2: STATE: difference between before and after Cognitive Rehab
Mann-Whitney U	2.500

Wilcoxon W	5.500
Z	-.296
Asymp. Sig. (2-tailed)	.767
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	State-Trait Anger Expression Inventory-2: STATE: difference between initial and final assessment
Mann-Whitney U	3.000
Wilcoxon W	9.000
Z	.000
Asymp. Sig. (2-tailed)	1.000
Exact Sig. [2*(1-tailed Sig.)]	1.000(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

State-Trait Anger Expression Inventory - II - TRAIT

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
State-Trait Anger Expression Inventory-2: TRAIT: difference between before and after Neurotherapy	Neurotherapy first	2	1.50	3.00
	cognitive rehab first	3	4.00	12.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
State-Trait Anger Expression Inventory-2: TRAIT: difference between before and after Cognitive Rehab	Neurotherapy first	2	2.75	5.50
	cognitive rehab first	3	3.17	9.50
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
State-Trait Anger Expression Inventory-2: TRAIT: difference between initial and final assessment	Neurotherapy first	2	2.25	4.50
	cognitive rehab first	3	3.50	10.50
	Total	5		

Test Statistics(b)

	State-Trait Anger Expression Inventory-2: TRAIT: difference between before and after Neurotherapy
Mann-Whitney U	.000
Wilcoxon W	3.000
Z	-1.732
Asymp. Sig. (2-tailed)	.083
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	State-Trait Anger Expression Inventory-2: TRAIT: difference between before and after Cognitive Rehab
Mann-Whitney U	2.500
Wilcoxon W	5.500
Z	-.296
Asymp. Sig. (2-tailed)	.767
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	State-Trait Anger Expression Inventory-2: TRAIT: difference between initial and final assessment
Mann-Whitney U	1.500
Wilcoxon W	4.500
Z	-.889
Asymp. Sig. (2-tailed)	.374
Exact Sig. [2*(1-tailed Sig.)]	.400(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

State-Trait Anger Expression Inventory - II - Anger Expression Index

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
State-Trait Anger Expression Inventory-2: Anger Expression Index : difference between before and after Neurotherapy	Neurotherapy first	2	2.50	5.00
	cognitive rehab first	3	3.33	10.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
State-Trait Anger Expression Inventory-2: Anger Expression Index : difference between before and after Cognitive Rehab	Neurotherapy first	2	2.50	5.00
	cognitive rehab first	3	3.33	10.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
State-Trait Anger Expression Inventory-2: Anger Expression Index : difference between initial and final assessment	Neurotherapy first	2	4.50	9.00
	cognitive rehab first	3	2.00	6.00
	Total	5		

Test Statistics(b)

	State-Trait Anger Expression Inventory-2: Anger Expression Index : difference between before and after Neurotherapy
Mann-Whitney U	2.000
Wilcoxon W	5.000
Z	-.592
Asymp. Sig. (2-tailed)	.554
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	State-Trait Anger Expression Inventory-2: Anger Expression Index : difference between before and after Cognitive Rehab
Mann-Whitney U	2.000
Wilcoxon W	5.000
Z	-.577
Asymp. Sig. (2-tailed)	.564
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	State-Trait Anger Expression Inventory-2: Anger Expression Index : difference between initial and final assessment
Mann-Whitney U	.000
Wilcoxon W	6.000
Z	-1.732
Asymp. Sig. (2-tailed)	.083
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Neurobehavioural Rating Scale - Subject Report

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Neurobehavioural Rating Scale - Subject report: difference between before and after Neurotherapy	Neurotherapy first	2	1.50	3.00
	cognitive rehab first	3	4.00	12.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Neurobehavioural Rating Scale - Subject report: difference between before and after Cognitive Rehab	Neurotherapy first	2	4.50	9.00
	cognitive rehab first	3	2.00	6.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Neurobehavioural Rating Scale - Subject report: difference between initial and final assessment	Neurotherapy first	2	3.00	6.00
	cognitive rehab first	3	3.00	9.00
	Total	5		

Test Statistics(b)

	Neurobehavioural Rating Scale - Subject report: difference between before and after Neurotherapy
Mann-Whitney U	.000
Wilcoxon W	3.000
Z	-1.777
Asymp. Sig. (2-tailed)	.076
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Neurobehavioural Rating Scale - Subject report: difference between before and after Cognitive Rehab
Mann-Whitney U	.000
Wilcoxon W	6.000
Z	-1.732
Asymp. Sig. (2-tailed)	.083
Exact Sig. [2*(1-tailed Sig.)]	.200(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Neurobehavioural Rating Scale - Subject report: difference between initial and final assessment
Mann-Whitney U	3.000
Wilcoxon W	9.000
Z	.000
Asymp. Sig. (2-tailed)	1.000
Exact Sig. [2*(1-tailed Sig.)]	1.000(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Neurobehavioural Rating Scale - Significant Other Report

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Neurobehavioural Rating Scale - Significant other report: difference between before and after Neurotherapy	Neurotherapy first	2	3.00	6.00
	cognitive rehab first	3	3.00	9.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Neurobehavioural Rating Scale - Significant other report: difference between before and after Cognitive Rehab	Neurotherapy first	2	2.50	5.00
	cognitive rehab first	3	3.33	10.00
	Total	5		

Ranks

	Treatment Groups	N	Mean Rank	Sum of Ranks
Neurobehavioural Rating Scale - Significant other report: difference between initial and final assessment	Neurotherapy first	2	3.00	6.00
	cognitive rehab first	3	3.00	9.00
	Total	5		

Test Statistics(b)

	Neurobehavioural Rating Scale - Significant other report: difference between before and after Neurotherapy

Mann-Whitney U	3.000
Wilcoxon W	9.000
Z	.000
Asymp. Sig. (2-tailed)	1.000
Exact Sig. [2*(1-tailed Sig.)]	1.000(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Neurobehavioural Rating Scale - Significant other report: difference between before and after Cognitive Rehab
Mann-Whitney U	2.000
Wilcoxon W	5.000
Z	-.577
Asymp. Sig. (2-tailed)	.564
Exact Sig. [2*(1-tailed Sig.)]	.800(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

Test Statistics(b)

	Neurobehavioural Rating Scale - Significant other report: difference between initial and final assessment
Mann-Whitney U	3.000
Wilcoxon W	9.000
Z	.000
Asymp. Sig. (2-tailed)	1.000
Exact Sig. [2*(1-tailed Sig.)]	1.000(a)

a Not corrected for ties.
b Grouping Variable: Treatment Groups

APPENDIX 12:

Examination of Treatment Differences: Wilcoxon Test Results

Mean, Standard Deviation, Minimum, & Maximum

Descriptive Statistics

	N	Mean	Std. Deviation	Minimum	Maximum
Symbol Search: difference between before and after neurotherapy	5	-1.2000	5.54076	-6.00	8.00
The Speed and Capacity of Language-Processing Test - difference between before and after neurotherapy	5	8.0000	3.46410	5.00	13.00
Rey Complex Figure Copy - difference between before and after neurotherapy	5	-2.0000	2.23607	-5.00	1.00
Rey Complex Figure 3 min Recall - difference between before and after neurotherapy	5	.6000	3.04959	-4.00	3.00
Trail Making Test - Part A - difference between before and after neurotherapy	5	2.7480	8.60915	-8.26	12.00
Trail Making Test - Part B - difference between before and after neurotherapy	5	-30.0000	32.13254	-86.00	-9.00
Controlled Oral Word Association - Phonemic - difference between before and after neurotherapy	5	-.2000	5.31037	-9.00	4.00
Controlled Oral Word Association - Semantic - difference between before and after neurotherapy	5	1.8000	2.77489	-1.00	5.00
Rey Auditory Verbal Learning Test - TOTAL RECALL - difference between before and after neurotherapy	5	-3.8000	11.64903	-21.00	8.00
Rey Auditory Verbal Learning Test - DELAYED RECALL - difference between before and after neurotherapy	5	-.8000	3.19374	-5.00	3.00
Rey Auditory Verbal Learning Test - RECOGNITION - difference between before and after neurotherapy	5	.2000	1.92354	-3.00	2.00
Neurobehavioural Rating Scale - Subject report: difference between before and after Neurotherapy	5	-21.2000	26.07106	-48.00	5.00
Neurobehavioural Rating Scale - Significant other report: difference between before and after Neurotherapy	5	-7.8000	32.30635	-57.00	28.00

	N	Mean	Std. Deviation	Minimum	Maximum
Beck Depression Inventory II : difference between before and after Neurotherapy	5	-14.2000	9.23038	-29.00	-7.00
State Trait Anxiety Inventory II: STATE: difference between before and after Neurotherapy	5	-10.4000	4.15933	-15.00	-5.00
State Trait Anxiety Inventory II: TRAIT: difference between before and after Neurotherapy	5	-5.4000	10.92245	-16.00	11.00
State-Trait Anger Expression Inventory-2: STATE: difference between before and after Neurotherapy	5	-1.8000	2.16795	-5.00	.00
State-Trait Anger Expression Inventory-2: TRAIT: difference between before and after Neurotherapy	5	-2.6000	5.45894	-10.00	3.00
State-Trait Anger Expression Inventory-2: Anger Expression Index : difference between before and after Neurotherapy	5	-4.6000	5.17687	-10.00	4.00
Test of Variables of Attention: Omissions: difference between before and after Neurotherapy	5	-2.2000	4.38178	-10.00	.00
Test of Variables of Attention: Comissions: difference between before and after Neurotherapy	5	-8.0000	11.37981	-24.00	6.00
Test of Variables of Attention: Response Time: difference between before and after Neurotherapy	5	-26.4000	54.56464	-106.00	24.00
Test of Variables of Attention: Variability: difference between before and after Neurotherapy	5	-27.8000	28.83921	-64.00	3.00
Paced Auditory Serial Addition Test : difference between before and after Neurotherapy	5	.4600	2.22666	-1.00	4.40
Symbol Search: difference between before and after cognitive rehabilitation	5	.6000	5.22494	-6.00	5.00
The Speed and Capacity of Language-Processing Test - difference between before and after cognitive rehabilitation	5	3.8000	6.14003	-4.00	11.00

	N	Mean	Std. Deviation	Minimum	Maximum
Rey Complex Figure Copy - difference between before and after Cognitive rehabilitation	5	1.2000	1.30384	.00	3.00
Rey Complex Figure 3 min Recall - difference between before and after Cognitive Rehabilitation	5	2.1000	1.67332	-.50	4.00
Trail Making Test - Part A - difference between before and after Cognitive Rehabilitation	5	.0000	6.20484	-8.00	8.00
Trail Making Test - Part B - difference between before and after Cognitiive Rehabiliation	5	2.6000	13.74045	-9.00	24.00
Controlled Oral Word Association - Phonemic - difference between before and after Cognitive Rehabilitation	5	2.4000	10.92245	-6.00	21.00
Controlled Oral Word Association - Semantic - difference between before and after Cognitive Rehabilitation	5	.2000	3.56371	-5.00	5.00
Rey Auditory Verbal Learning Test - TOTAL RECALL - difference between before and after Cognitive Rehabilitation	5	3.4000	5.85662	-1.00	12.00
Rey Auditory Verbal Learning Test - DELAYED RECALL - difference between before and after Cognitive Rehabilitation	5	-.4000	2.07364	-3.00	2.00
Rey Auditory Verbal Learning Test - RECOGNITION - difference between before and after Cognitive Rehabilitation	5	-.4000	2.19089	-4.00	2.00
Neurobehavioural Rating Scale - Subject report: difference between before and after Cognitive Rehab	5	-6.2000	19.27952	-30.00	19.00
Neurobehavioural Rating Scale - Significant other report: difference between before and after Cognitive Rehab	5	4.2000	7.29383	-5.00	14.00
Beck Depression Inventory II : difference between before and after Cognitive Rehab	5	-6.8000	3.34664	-12.00	-4.00
State Trait Anxiety Inventory II: STATE: difference between before and after Cognitive Rehab	5	-8.4000	5.68331	-15.00	.00

	N	Mean	Std. Deviation	Minimum	Maximum
State Trait Anxiety Inventory II: TRAIT: difference between before and after Cognitive Rehab	5	-7.2000	4.96991	-15.00	-2.00
State-Trait Anger Expression Inventory-2: STATE: difference between before and after Cognitive Rehab	5	-2.0000	2.54951	-6.00	.00
State-Trait Anger Expression Inventory-2: TRAIT: difference between before and after Cognitive Rehab	5	-1.0000	2.54951	-3.00	3.00
State-Trait Anger Expression Inventory-2: Anger Expression Index : difference between before and after Cognitive Rehab	5	-6.2000	9.20326	-15.00	8.00
Test of Variables of Attention: Omissions: difference between before and after Cognitive Rehab	5	.8000	5.40370	-4.00	10.00
Test of Variables of Attention: Comissions: difference between before and after Cognitive Rehab	5	2.0000	6.20484	-5.00	9.00
Test of Variables of Attention: Response Time: difference between before and after Cognitive Rehab	5	20.2000	76.90709	-45.00	152.00
Test of Variables of Attention: Variability: difference between before and after Cognitive Rehab	5	-2.6000	39.91616	-42.00	64.00
Paced Auditory Serial Addition Test : difference between before and after Cognitive Rehab	5	-.0400	.35071	-.50	.40

APPENDIX 13:

Ethics Document of Approval