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Cognitive Change and Assessment during Electroconvulsive Therapy

A thesis presented in partial fulfillment of the requirements for the Degree of Doctor of
Clinical Psychology at Massey University, Wellington, New Zealand.

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Abstract

Electroconvulsive therapy (ECT) is the most effective treatment available for depression; however, cognitive side effects limit its use. If detected early in treatment, cognitive decline can be reduced by modifying the way the treatment is administered. Currently, no gold standard measures exist to assess ECT related cognitive change. The studies within this thesis aimed to improve the standard in which cognition is assessed during a course of ECT.

Study One surveyed health professionals across New Zealand on their current practice of cognitive assessment during ECT. Study Two collected normative data for a neuropsychological measure of motor speed which had potential to be included in a cognitive screening measure for use with people receiving ECT. Study Three prospectively investigated objective cognitive changes in 13 people receiving electroconvulsive therapy for a mood disorder. Study Four investigated the subjective cognitive changes by qualitatively analysing patients' reports of cognitive function throughout treatment. Finally, a brief but comprehensive cognitive screening measure was proposed for repeated use with patients receiving ECT. This measure was largely informed by findings from the aforementioned studies.

Time, resources and a lack of sensitive tests restricted professionals from conducting more frequent and thorough cognitive assessments. ECT resulted in both cognitive decline and improvement across a range of cognitive domains. Alleviation in depression was associated with improvement in cognitive function from baseline. Six weeks post ECT, cognitive decline most often resolved back to or was superior to the baseline functioning. Domains sensitive to decline during a course of treatment included retrograde memory, anterograde memory, verbal and visual learning, attention, verbal fluency and information processing speed. Retrograde amnesia was more likely for memories formed closer in time to ECT treatment. Subjective reports of cognitive change were broad, and varied as a function of treatment phase and severity of depression. It is proposed that future research determine the psychometric properties of the suggested screening measure.

Preface

As I was entering the Doctor of Clinical Psychology programme at Massey University, a predecessor in the programme, Dr Kiri Luther, also working under the supervision of Professor Janet Leathem, had just completed her doctoral research in the field of cognitive change associated with electroconvulsive therapy (ECT). Kiri conducted cognitive assessments with 19 people who had received ECT at least two years previously and found that some areas of cognitive deficit due to ECT endured into the long term. This finding highlighted the importance of minimising cognitive dysfunction early on in treatment by frequently monitoring and assessing cognitive function and thereby preventing long term cognitive deficits. Kiri and Janet's passion for the topic was contagious, and I was soon convinced that further research in this area was warranted.

A discussion with the ECT administering psychiatrist at Capital and Coast District Health Board (C&C DHB) Dr Nisar Contractor revealed longstanding frustration with the paucity of assessment measures available to sensitively detect cognitive change during ECT. Current measures were reportedly insensitive to detecting cognitive change reported by patients, and lengthier neuropsychological assessments, though more sensitive, were impractical due to time constraints. It was decided that further research into the neuropsychological assessment of patients receiving ECT, and the subsequent development of a brief screening measure tailored to detect cognitive change during ECT would be beneficial to both patients receiving ECT and health professionals responsible for their care.

At the outset of planning the research, it was hoped that a large enough number of participants would be recruited in order to analyse the results collectively using parametric statistics. After conversations with Professors Richard Porter and Bob Knight, two New Zealand researchers who have both contributed extensively to research in this field, the issue of recruitment was raised. It soon became apparent the greatest challenge in conducting this type of research would be in recruiting participants. This concern was also expressed by Susan Vella, ECT nurse at C&C DHB.

Recruitment challenges would be due to a number of factors: the small number of people who are prescribed ECT, the large physical distances between District Health Boards (DHBs) and thus the travel involved to recruit participants from across DHBs, and due to the characteristics of the population under investigation. For example, as most people who receive ECT suffer from severe depression, many would not be well enough to undergo cognitive assessments, to consent to taking part in research or be motivated to partake in research. In addition, there was a strict timeframe for completion of the research; the entirety of the research needed to be designed, receive ethics approval, be conducted, analysed, written up and submitted within 24 months.

Although early on in planning the research it was understood and acknowledged that recruiting participants would be difficult, it was agreed that a meaningful contribution to the existing pool of knowledge on cognitive effects of ECT could still be made even with a small participant sample. The implication, however, was that the way in which the results would be analysed would need to differ. Presenting the results as a series of case studies would abolish the need for a large participant pool required for hypothesis testing, and instead would represent what cognitive changes occurred for each individual as a function of how many treatments they had had, and how their mood changed over their course of treatment. The method of analysis was appropriate as each individual was variable in terms of ECT administration, seizure duration, threshold, number of treatments, psychiatric illness, co-morbidities, socio economic status, educational history, age, sex, and ethnicity to name a few. As analysing the results as a series of case studies would still provide rich and meaningful data, it was decided to go ahead with conducting the research. Despite receiving

support and referrals from four DHBs in the Lower North Island¹, the number of participants recruited was, as anticipated, low. However, meticulous efforts were made to ensure that baseline assessments, follow-up assessments and reassessments after every three treatments of ECT were made, which resulted in a total of 87 cognitive assessments conducted.

This thesis presents and discusses current research into the short term cognitive effects of electroconvulsive therapy and the assessment thereof. The research was conducted in order to better understand the nature and severity of the cognitive effects endured by 13 patients receiving ECT and to determine precisely, what cognitive changes occurred during their courses of ECT. In addition to adding to the existing literature on the short term cognitive effects of ECT, the current study also endeavoured to determine which measures were sensitive and insensitive to detecting the effects of ECT, and which cognitive changes people were reporting that were not being detected by the objective measures of assessment. The findings of this study later informed the suggestion of a screening measure, to assess and monitor cognitive function during a course of ECT.

¹ Capital and Coast DHB, Hutt Valley DHB, Mid Central DHB and Hawke's Bay DHB

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Conducting such research involves the help, support and collaboration of many individuals, and could never be achieved in isolation. First and foremost, I would like to acknowledge the people undergoing ECT who took part in the research, and the whānau of many of these people who supported their participation. It was a privilege to get to know each and every one of you and to work with you during this particularly difficult time in your lives. It takes great motivation and altruism to take part in voluntary research, particularly when suffering such debilitating illness. I have been touched by your courage and persistence, and I hope you have also benefitted from being a part of this research.

Thank you to the ongoing support and encouragement from Dr Nisar Contractor and Susan Vella psychiatrist and ECT nurse from C&C DHB who played a large part in the initiation of the research, provided ongoing support and encouragement throughout the duration of the research, and always kept me up to date with new referrals and treatment progress of participants.

Thank you to Erwin Sonnendecker, Clinical Psychologist at Hawke's Bay DHB for showing an avid interest in the research, and sharing a passion for improving the way in which cognition is assessed during a course of ECT; a passion which resulted in Erwin contributing some assessment data to the research.

Thank you to my parents for encouraging me in my first year of university to pursue my interests and to take papers which I enjoy, and not worry about whether or not they would someday lead to a career. Advice which has serendipitously led to me writing this doctorate and pursuing a career in clinical psychology. Thank you to my partner, Ben, whose endless support and numerous cups of teas and coffees (and at times dinner brought to my office) has helped me complete this thesis. I would also like to acknowledge my office peers in T4 who have now all become close friends.

Last but by no means least; I would like to extend my sincere gratitude to my wonderful supervisors, Professor Janet Leathem and Dr Ross Flett. Janet's clinical and research expertise has been indispensable throughout the duration of the research and I have been fortunate to have learnt from her. No problem was ever too big or too complicated for her to solve. If she didn't have the answers, she always knew the right person to contact. To my second supervisor Ross, thank you for your guidance, sense of humour and encouragement you generously provided.

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THESIS OVERVIEW

This thesis entitled, “Cognitive Change and Assessment during Electroconvulsive Therapy”, is written by publication. It contains six studies presented as manuscripts. These studies and their inter-relationships are shown in Figure 1 below. Chapters are imbedded between the manuscripts to connect the studies and bring cohesion. As ECT has an interesting and controversial history, the first chapter was devoted to outlining how this treatment came to be and how it has been used over the past eight decades.

Due to the thesis being written by publication, there is some unavoidable repetition throughout the thesis to ensure the manuscripts are complete when read in isolation from the thesis. The following overview details how the studies originated, the purpose each study serves, and how all studies, though some different in orientation and methodology, tie into the common theme of cognitive change and cognitive assessment during electroconvulsive therapy (ECT).

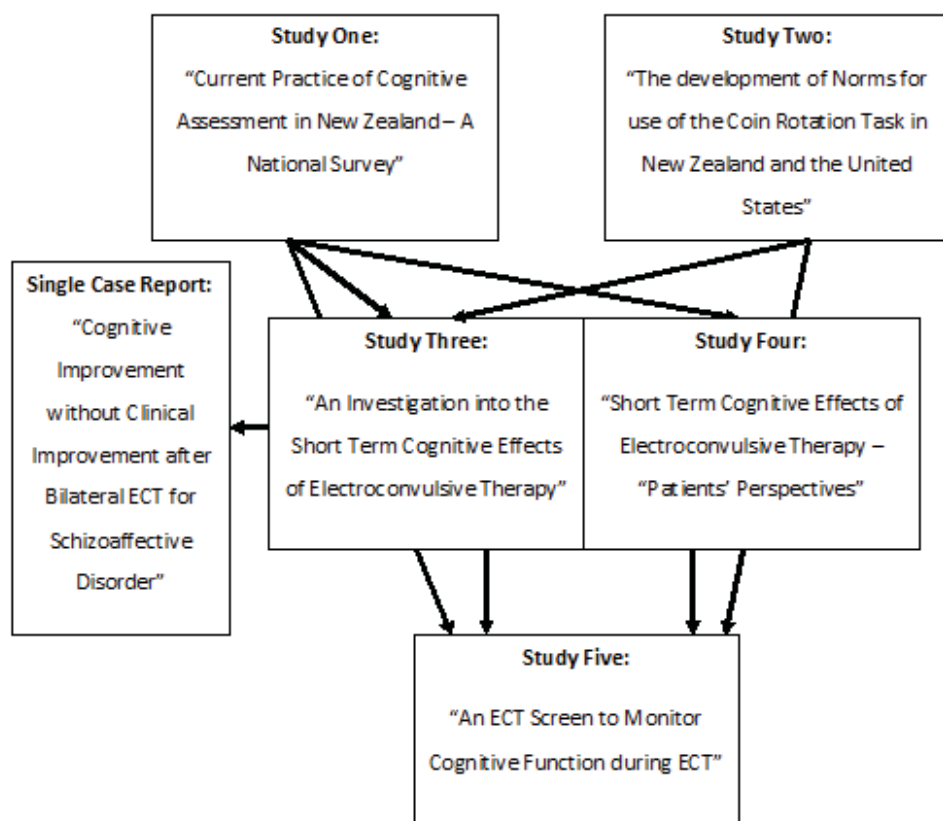


Figure 1. Flow diagram of thesis layout. This figure depicts how Study One informed study Three, Four and Five; how Study Two informed Study Three and Five; and how studies Three and Four also informed study Five. The figure also depicts how the single case report was an artifact from Study Three.

The Impetuses for the Studies

Reports of frustration with the dearth of sensitive measures available to assess ECT related cognitive change by a Wellington ECT administering psychiatrist provided one of the impetuses for conducting this research, as it appeared there was a need for the generation of a new screening measure tailored to detect the cognitive effects of ECT. Although it was assumed that this dissatisfaction was shared by other ECT administering and prescribing psychiatrists across New Zealand, it was decided that a more formal means of determining current practice of cognitive assessment during ECT would be beneficial to determine whether the need for a cognitive screen was widespread. This led to the first study “Cognitive Assessment during a Course of Electroconvulsive Therapy – A National Questionnaire Survey of Current Practice in Aotearoa, New Zealand” (see Study One, Figure 1). As well as determining current practice, the questionnaire acted as a tool to determine practicalities around conducting assessments, such as: how much time could professionals spend administering the screen, who would be the people likely to be conducting the assessments and what should the screen cover. This information also informed the suggested measures for the cognitive screen (Study Five, Figure 1).

To determine exactly what cognitive changes occur during ECT and therefore which cognitive constructs the proposed assessment tool would need to measure, the peer-reviewed literature was consulted. A literature search revealed a plethora of cognitive domains to be affected by electroconvulsive treatment, but whether or not these cognitive effects persist into the long term remained debated among researchers. A common and congruous finding was, however, that objective performance in cognitive tests seldom correlate with subjective reports of cognitive

functioning. In other words, patients often complain of cognitive impairment but this is not always detected by objective assessments. In addition, very few recommendations existed around which cognitive assessment tools are sensitive to detecting cognitive change during ECT. Thus it became apparent that more research was needed to address what difficulties patients were reporting which weren't being detected by objective assessment, which measures are sensitive or insensitive to detecting these complaints and what cognitive difficulties persist into the long term (if any). This led to the core and the largest components of the research, studies Three and Four (see Figure 1). Participants who were receiving ECT for a mood disorder were invited to take part in the studies; however, one exception was made for a man who was receiving ECT to treat schizoaffective disorder, as the ECT administering psychiatrist had concerns about his cognition and asked for him to be included in the study in order to receive more thorough cognitive assessments. As his psychiatric presentation differed from the other individuals' in the study, the results of these assessments were not included within the analysis found in Chapter Seven and are instead included in Appendix E.

The core component of the research involved prospectively carrying out cognitive assessments with patients receiving ECT through four DHBs across the lower North Island. To determine which cognitive changes were occurring, a carefully selected battery of tests covering a broad range of cognitive domains were included in the assessments, as well as a subjective reports from the patients to ensure that any cognitive deficits or improvements which were not being detected in the assessments were noted. Subsequently, the cognitive changes which occurred and the nature of these changes were examined. The results of studies One, Three and Four, supplemented by findings in the published peer-reviewed literature informed the suggestions for what should be included in a cognitive screen for people receiving ECT. Conducting the assessments which took part for the main study also gave the primary researcher an understanding of how these assessments pose different challenges and assessment requirements than working with other populations which was important to address when proposing the screen.

During the beginning stages of planning the research, a great deal of consideration went into deciding and adapting measures to include within the cognitive assessment which would be conducted with the patients participating in the research. As the selected measures could potentially end up as part of the screen which was to be developed, they needed to be simple to administer and score, have good psychometric properties and be relatively short to administer. One challenge was finding a measure of motor-speed which matched the aforementioned criteria. Eventually, the 'Coin Rotation Task' was discovered in the literature. This short, simple assessment measure appeared to be an ideal task to include the assessments. There were only two problems with the task: it was normed with the nickel in the United States; and the norms were limited. No normative data existed for the use of the task with females, or people below the age of 40 or above the age of 79. These factors led to the second study "Coin Rotation Task – The Development of Norms for New Zealand and the United States". In this study, normative data was collected with 215 New Zealanders and a comparison study was conducted which compared performance of the coin rotation task using the United States Nickel and the New Zealand 20 cent coin.

In conclusion, within this thesis six studies are presented based on three different participant samples: a questionnaire of health professionals in New Zealand investigating current practice of cognitive assessment during ECT; a study collecting norms for the use of a psychomotor task in New Zealand; a series of case studies examining the cognitive and clinical changes which occur during a course of ECT and at a six week follow-up; a qualitative content analysis of patients' reports of cognitive and clinical change during ECT; and finally, a paper offering suggestions of a cognitive screening measure for use with patients during a course of ECT. These studies are imbedded among chapters of literature reviews on the history of ECT, theories of ECT mechanism, variables affecting cognitive side effects of ECT, discussions and reflections of the studies. Each study, although variable in magnitude, design and purpose, collectively compliment the theme of the current thesis "The Assessment of Cognitive Change during ECT".

CHAPTER ONE

The History and Evolution of Electroconvulsive Therapy

Electroconvulsive therapy (ECT) also known as “Shock Therapy” is a widely used efficacious treatment for a variety of psychiatric conditions resistant to psychotherapy, medication and other forms of treatment (Chamberlin & Tsai, 1998). ECT is fast acting, has an efficacy as high as 85-90% compared to 60-65% for antidepressant medication (Nobler & Sackeim, 2001), and is considered to be the most effective acute treatment for treatment resistant major depressive disorder (Kellner et al., 2010; Sackeim et al., 2009). Electroconvulsive therapy works by passing a carefully controlled electric current through the brain via electrodes placed on the scalp to induce a grand mal seizure (National Institute of Clinical Excellence, 2003). The seizure induced by this current alleviates symptoms of psychiatric illness via a mechanism which remains unknown (Mankad, Beyer, Weiner, & Krystal, 2010). Since its origin in the 1930s, the way in which ECT is administered has been subjected to many modifications to increase the clinical efficacy and decrease the adverse side effects. This chapter will briefly outline how this controversial, yet life saving treatment has originated and evolved over the past 75 years. Modifications in administration and practice which have occurred over the past eight decades will be described as well as the factors contributing to the waxing and waning of the treatment’s popularity and the current theories of ECT mechanism.

The Origin of Electroconvulsive Therapy

Up until the 1930s, treatment options for psychiatric patients were limited. The principal treatment for psychiatric disorders was psychotherapy; predominantly psychoanalysis. For the very severely mentally ill, little could be offered in the way of treatment besides custodial care and prevention of harm to themselves and others (Mankad et al., 2010). The prognosis for severe mental illness was at this time, hopeless. A diagnosis of schizophrenia was seen as a death sentence and all a patient had

to look forward to was a lifetime placed in institutional care or a life at home locked in a bedroom and cared for by a distraught parent (Shorter & Healy, 2007). Prior to the 1930s, clinicians felt helpless, families despairing and patients were submitted to so much suffering and misery that suicide was a common escape (Shorter & Healy, 2007). Somatic treatments such as hydrotherapy, prolonged sleep therapy and insulin coma therapy were all attempts to treat the mentally unwell; however, all proved to be ineffective and unpleasant (Mankad et al., 2010).

Convulsive Therapies.

Prior to the 1930s, it had been established that epilepsy was uncommon in patients who had schizophrenia. It had also been observed that in the few patients who had schizophrenia and subsequently developed epilepsy, their symptoms of schizophrenia appeared to diminish subsequent to a seizure (Mankad et al., 2010; Shorter & Healy, 2007). Hungarian neuropsychiatrist Ladislav Joseph von Meduna hypothesised that seizures in epilepsy must therefore be protective against psychiatric illness. In 1933 Meduna concluded these two illnesses were in some way incompatible and supporting this hypothesis was an observation he made of the excessive growth of glia cells in epileptic brains and the apparent torpor of glia cells in schizophrenic brains (Shorter & Healy, 2007).

Meduna's speculation that epilepsy and schizophrenia were incompatible illnesses inspired a series of experiments to test this hypothesis. Meduna experimented with a number of different chemicals including caffeine, brucin, coramin, thebaine, alkaloids and camphor to induce seizures in guinea pigs. Camphor dissolved in oil proved to be the most reliable and effective chemical to induce a seizure (Gazdag, Bitter, Ungvari, Baran, & Fink, 2009). Two months later, in January of 1934, Meduna successfully treated his first human patient, Zoltan, who had been diagnosed with Schizophrenia and had been in a catatonic stupor for four years (Shorter & Healy, 2007). He was unable to move, was incontinent and required tube feeding. Zoltan's prognosis was considered bleak and his mental condition incurable (Abrams, 2002). After the fifth camphor induced seizure,

Zoltan was able to dress himself, he began to talk for the first time in four years and even requested breakfast (Shorter & Healy, 2007). After a few more treatments of camphor induced seizures, Zoltan decided he was well enough to see his wife whom he had not seen since he had become ill. Zoltan escaped from the institution where he was receiving his treatment and went home to see his wife. Upon returning home, he discovered his wife had a new lover. He beat up his wife's lover and threw him out of the house. From then on, Zoltan was considered cured (Shorter & Healy, 2007). He was later reported to have achieved a full recovery and gained employment (Fink, 2001).

Zoltan's successful treatment was the start of many successful treatments of convulsive therapy. In the first year, Meduna treated 26 patients. Ten recovered completely, three improved and 13 did not change (Fink, 1984). Although camphor successfully and reliably induced seizures, it was painful, had varying results and a seizure would develop only after a number of anxiously endured minutes had passed (Fink, 2001). Meduna then switched to a different chemical, pentylenetrazol, otherwise known as metrazol, which had rapid onset of action and produced fewer side effects (Abrams, 2002).

When metrazol started to take effect, patients' thoughts would race, heart rates increased, all consciousness was lost and a convulsion would occur. Post seizure the patient would wake having no recollection of any recent events, they would have muscle, back and headaches from the grand mal seizure and the patients' lips and tongues would often be bleeding. Some patients endured fractures of the spine, long bones and jaw (Fink, 2001). Others sustained microscopic lesions in the brain due to a lack of oxygen as the patients stopped breathing for several seconds to minutes after the seizure (Abrams, 1997).

The unpleasant terrors caused by metrazol led researchers to search for an alternative method to induce convulsions in patients. Experiments using electrically induced convulsions were first undertaken in Rome in 1937 by neuropsychiatrists Ugo Cerletti and Lucio Bini (Shorter & Healy, 2007). Cerletti and Bini came across the idea of using an electrical current to induce a seizure after

observations were made of pigs in slaughter houses which were electrically stunned into unconsciousness before being dispatched. If the pigs were not immediately dispatched, they clearly exhibited symptoms of a grand mal seizure (Shorter & Healy, 2007). The pigs appeared unharmed by the electrical current and the apparent safety of using electricity was encouraging to Bini and Cerletti. The neuropsychiatrists commenced trialing the use of electricity to induce seizures in animals. Bini was the first to demonstrate the safety and efficacy of applying electrodes to the temples of dogs to safely induce a seizure.

The first human patient to receive electrically induced seizures - electroconvulsive therapy - was a 38 year old man Enrico, in 1938 (Fink, 2001). Enrico was found wondering around a train station in Milan without a train ticket. He was delusional, hallucinating, gesticulating and altered between periods of mutism and neologistic speech (Fink, 2001). Enrico was diagnosed with schizophrenia and subsequently treated with ECT. His successful recovery after 11 treatments demonstrated that convulsions could indeed be induced safely, effectively, reliably and inexpensively with electricity (Fink, 2001). To this day, there has been no better convulsive agent than electricity (Shorter & Healy, 2007). The success of convulsive and electroconvulsive therapies changed attitudes towards psychiatric illness and instilled hope into the lives of families and patients suffering. The perception of mental illness being a hopeless condition changed through the invention of ECT to optimism and relief that successful treatment of mental illness was now possible (Fink, 2001).

Patterns of Use.

Throughout its 75 years of existence, ECT has waxed and waned in popularity. At its origin, ECT was considered the gold standard treatment for psychosis and depression and this remained so until the late 1940s (American Psychiatric Association, 2001). The mid 1950s saw a decline in the use of ECT and this decline continued for a number of years (Shorter & Healy, 2007) with ECT being a last resort treatment for major depression, suicidal ideation, anhedonia, catatonia, and those nonresponsive to medication. This is comparable to how it is used today (McCall, 2001). ECT's

decline in popularity was influenced by two main factors: the introduction of antipsychotic medications in the 1950s and later the portrayal of ECT in the media as a method of behaviour control and as a cruel and inhumane treatment (McDonald & Walter, 2001). Such images also evoked fears of punishment by the inherent association with the electric chair (Mankad et al., 2010).

In the meantime, continuous efforts were being made to improve the safety of ECT, and in 1978 the first Task Force Report was produced by the American Psychiatric Association where new standards for consent were published. The report encouraged unilateral over bilateral application to lessen cognitive side effects of the treatment (further explained in Chapter Three) (Rudorfter et al., 2003, cited in Zeman, Kapur, & Jones-Gotman, 2012). Additionally, the use of brief pulse electrical stimulation over sine wave was urged to further protect against cognitive decline from the treatment (Zeman et al., 2012).

In the 1980s and 1990s the use of ECT increased again, and it is still experiencing a modest resurgence (Munk-Olsen, Laursen, Videbech, Rosenberg, & Mortensen, 2006). This is due to its fast acting efficacy, modifications made to the treatment to improve clinical efficacy and reduce cognitive impairment, increase in safety and the growing acceptance by practitioners and patients (Mankad et al., 2010). ECT is being recognised as a proven and effective lifesaving intervention for patients with depression, mania and catatonia where other treatments have had little or no effect. In addition, ECT is benefitting from well informed medical journalism that portrays the procedure objectively as opposed to the Hollywood portrayal. However, many people still hold a stigmatised view of ECT despite recent media depictions tending to be more realistic, positive and supportive (Mankad et al., 2010).

Modern-day Electroconvulsive Therapy

ECT is widely used today across many different continents (Leiknes, Schweder, & Høie, 2012). It has been estimated that worldwide approximately 1,000,000 people receive ECT annually (Prudic,

Olfson, & Sackeim, 2001). Administration of electroconvulsive therapy still involves the application of electrodes onto the scalp to allow an electrical current to pass through the brain and induce a grand mal seizure (Ministry of Health, 2004). However, the way in which ECT is administered shows little resemblance to the historical application of the treatment (Mankad et al., 2010). Ongoing and extensive research has informed the way in which electroconvulsive treatment is currently practiced, and modifications have been put in place which increase clinical efficacy of the treatment and decrease cognitive adversities associated with ECT (the impact of these modifications on cognitive outcome are discussed in greater depth in Chapter Two). Despite a large amount of research dedicated to the field of ECT, the way in which the treatment works to relieve psychiatric illness remains unknown (Grover, Mattoo, & Gupta, 2005). The most prevalent and accepted hypothesis of ECT mechanism is that the seizure induces an alteration in the central nervous system neurotransmitter receptor activity and density (Lerer, 1984; National Institute of Clinical Excellence, 2003). However, other hypotheses of mechanism exist as outlined below.

Current Theories of ECT Mechanism

Since the origin of convulsive therapies, over 100 hypotheses have been proposed in attempt to explain the perplexing way in which ECT works (Grover et al., 2005). Psychological, neuro-physiological, neurochemical, neuro-endocrine and neuro-peptide theories have all been proposed, yet no single theory can stand alone to explain how it is that this treatment can so effectively relieve psychiatric illnesses (Grover et al., 2005). It has been suggested that the effects of ECT are likely to be acting in a way to normalise abnormal neuronal changes causing the psychopathology; however, it remains unclear which changes caused by ECT are responsible for improvement in clinical state, and which changes may be causing the adverse side effects (Anderson & Fergusson, 2013).

Reaching a better understanding of ECT mechanism will enable administration parameters to be more finely tuned to result in an even greater treatment efficacy with fewer adverse side-effects and less variability in outcome amongst patients (Peterchev, Rosa, Deng, Prudic, & Lisanby, 2010). In addition, a greater understanding of the influence the treatment has on brain function could lead to

the development of similar but less invasive treatments which would mirror the mechanism and clinical benefits of ECT but have fewer side effects (Perrin, 2012). Some of the most popular theories of ECT mechanism will be briefly outlined:

Generalised Seizure Theory.

The *Generalised Seizure Theory* suggests that it is the seizure that causes the therapeutic effect (Ottooson, 1960). This theory has been well supported by numerous studies in the literature.

Evidence for this theory, as reported by Bolwig (2011) is as follows:

- Placebo trials have demonstrated that sham ECT with the absence of a seizure produces no clinical effect.
- The longer the duration of the seizure, the better the clinical outcome for the patient (Ottooson, 1960).
- Bilateral electrode placement induces greater seizure generalisation than unilateral ECT and bilateral ECT has a superior clinical efficacy than that of unilateral ECT.
- Non-convulsive brain stimulation, such as Transcranial Magnetic Stimulation, has weak therapeutic effects.
- Treatment which is sub-convulsive has little or no antidepressant effect.

However, if the seizure is the critical component in inducing the therapeutic effect, what effect *specifically* is the seizure having on brain function to cause these effects? The following hypotheses offer some explanations as to how it is that the seizure can cause such therapeutic effects.

Neurotransmitter Hypothesis.

The first ever theory to explain the observed clinical effects of chemically inducing seizures was proposed by Ladislav Joseph von Meduna, the founder of convulsive therapy who hypothesised that clinical improvement was due to the changing of chemical compositions in the brain brought on by the convulsion (Meduna, 1936). This hypothesis suggested almost 80 years ago broadly resembles

the *Neurotransmitter Hypothesis*, one of the most widely accepted theories of ECT mechanism today. According to this hypothesis, the seizure produced during ECT releases neurotransmitters which act to reduce depression (Grover et al., 2005). Recurrent convulsive treatments have been shown to exert effects on several different neurotransmitter systems in the central nervous system (Ishihara & Sasa, 1999). For example, two subtypes of the neurotransmitter receptor for serotonin have been shown to become sensitised during ECT treatment. In addition, ECT decreases functioning of norepinephrine and dopamine inhibiting auto-receptors in the ceruleus and substantia nigra, consequently causing more of each neurotransmitter to be released and available within the synapse (Ishihara & Sasa, 1999). It is these neurotransmitters which have been postulated to play a role in the clinical improvement of depression.

The Neuroendocrine-Diencephalic Theory.

The *Neuroendocrine-Diencephalic Theory* proposed by Fink and Ottosson (1980) stipulates that ECT restores dysfunction associated with the neuroendocrine system during melancholic depression. Melancholic depressive symptoms such as: disturbed sleep, changes in appetite and decreased sexual drive, all show an improvement with ECT treatment (Fink & Ottosson, 1980). Supporting this theory is the evidence that ECT induces the release of endocrines and hormones related to the Hypothalamic-Pituitary-Adrenal (HPA) axis (Taylor & Fink, 2006). In addition, blood flow to the basal ganglia and brain stem increases during ECT, demonstrating that diencephalic limbic structures are stimulated by the treatment (Takano, Motohashi, & Uema, 2007). Currently, the neuroendocrine theory has been suggested to have the strongest foundation to explain the working mechanism of electroconvulsive therapy (Bolwig, 2011).

Increased Neurogenesis.

On-going neurogenesis occurs in the hippocampal formation during adult life in animals and in humans (Eriksson et al., 1998; Gould, Beylin, Tanapat, Reeves, & Shors, 1999). Studies have shown that after long periods of depression, a decrease in hippocampal volume occurs (Shah et al,

1998; Sheline et al., 1999 as cited in Grover et al., 2005). This decrease in neuronal proliferation is suggested to be caused by stress – and stress is also a precipitating factor of depression. Animal models have demonstrated that extended seizures can increase hippocampal neurogenesis (Grover et al., 2005). More recently, researchers have shown that ECT caused a significant increase in both the volume of both hippocampi in twelve patients who received a course of ECT (Nordanskog, Dahlstrand, Larsson, Knuttson, & Johanson, 2010). According to the increased neurogenesis theory, ECT reverses this decrease in hippocampal formation which occurs due to chronic stress and depression and stimulates neuronal proliferation, which in turn, works to relieve clinical depression. The effect ECT has on the hippocampi structures has been suggested to underlie the effects on cognition, particularly memory (Gregory-Roberts, Naismith, Cullen, & Hickie, 2010).

Anticonvulsant Hypothesis.

The Anticonvulsant Hypothesis, (Sackeim, 1999) another popular theory of ECT mechanism, suggests that anticonvulsant properties build up in the brain over the duration of ECT and these anticonvulsant changes are responsible for producing the therapeutic effect of ECT. During the first few treatments, a patient's seizure threshold increases and a greater dosage of electricity is required to produce the seizure. In addition, the seizure duration often decreases. Some physical anticonvulsant changes which occur include: a decrease of blood flow in the cerebral cortex lasting up to several months, changes in brain waves and the release of neurotransmitters and neuropeptides which have anticonvulsant properties (Sackeim, 1999). The anticonvulsant changes occur as the brain's natural response to prevent future seizures from occurring. It is suggested that one or more of these anticonvulsant properties is fundamental in causing the antidepressant effects of ECT (Sackeim, 1999).

Anatomical Theory.

While the aforementioned hypotheses describe the ECT mechanism as an *indirect* result of the treatment, the *Anatomical Theory* proposes that the anti-depressant effect of ECT is due to the direct

electrical stimulation of the frontal lobes – an area of the brain considered crucial for the integration of cognition, emotion and mood regulation (Michael, 2009). Supporting evidence for this theory is the finding that bilateral electrode placement has a greater clinical efficacy than unilateral electrode placement (see Chapter Two) and according to the *Anatomical Theory*, this is due to a greater area of the frontal lobes being directly stimulated. Therefore, via direct stimulation of the frontal lobes ECT helps to effectively regulate and moderate emotion thereby treating depressive symptoms.

In summary, although ECT is a longstanding, frequently used treatment for which the efficacy and safety have been well established, the mechanism of its action remains unknown (Grover et al., 2005). Many hypotheses have been formulated in attempt to better understand the mechanism of this effective treatment; some with more empirical evidence than others. Nevertheless, there is still no single theory which can stand alone to explain how it is that ECT functions to produce such profound clinical improvement for so many of its consumers. Future research should continue to investigate the mechanism behind ECT. The discovery of which will lead to greater clinical efficacy of the treatment, less individual variability and perhaps even the development of an alternative method with a parallel mechanism to provide relief of psychiatric disorders.

Modifications in the use of ECT

Historically, ECT was administered without anesthesia or muscle relaxants (= termed unmodified ECT). Modified ECT (ECT given under anesthesia and with muscle relaxants to reduce trauma from the convulsions) has replaced the unmodified mode of administration in most parts of Europe, Australasia and the United States; however, unmodified ECT is still used in parts of Asia, Africa, Latin America, Russia and Turkey (Leiknes et al., 2012). As well as the introduction of general anesthetics and muscle relaxants to prevent body spasms, further modifications to treatment include oxygenation during the procedure, careful monitoring of seizure activity, bite guards to prevent mouth trauma during the seizure and careful cognitive assessment to monitor cognitive effects of the treatment some time after the procedure. Up until the 1970s most ECT devices delivered a sine-

wave oscillating current. A high dosage of this electricity was used to induce the seizure. Standard practice has since changed to delivering a low dosage of brief fixed-pulse electrical stimulation. The change was made to allow achievement of a seizure with the least possible electrical energy (Ministry of Health, 2004). Modern ECT devices allow for the electrical dosage to be adjusted and tailored specifically to an individual's seizure threshold (Ministry of Health, 2004).

Randomised Control Trials (RCTs) have shown that the use of brief pulse electricity is associated with less severe short-term memory deficits compared to sine wave stimulation (Weiner, Rogers, Davidson, & Squire, 1986), and unilateral ECT induces fewer cognitive side effects than bilateral ECT (Dunne & McLoughlin, 2012). In New Zealand, bilateral high dose ECT has long been replaced with moderate dose unilateral ECT as the initial standard treatment to achieve good clinical effects while minimising cognitive side effects (Ministry of Health, 2004). The above innovations and modifications have increased the efficacy and popularity of electroconvulsive treatment in the western world (Lisanby, 2007).

Prescription of Electroconvulsive Therapy in New Zealand

In New Zealand, ECT is currently used as a last resort treatment under strict conditions. In order to be prescribed ECT the patient must meet at least one of the following criteria: a) patient has trialed at least two different types of antidepressant medication and been found to be "treatment resistant"; b) has experienced side effects from the antidepressant that make it an unsuitable treatment; c) the patient's life is in danger because they are suicidal; d) their life is in danger because their depression is chronic; e) or they have stopped eating or drinking; f) the patient is unable to move due to catatonia; g) or, if the patient is manic and mood stabilisers have been unable to treat this (Ministry of Health, 2009). A critical reason for these strict guidelines being put in place is due to ongoing reports that ECT can cause cognitive impairment, particularly in the form of memory loss (Lerer et al., 1995; Sackeim et al., 2007; Sienaert, Vansteelandt, Demyttenaere, Peuskens, 2010; Sobin et al., 1995; Squire, 1986b; Squire, Wetzel, & Slater, 1979; Weiner et al., 1986). Once a number of

medications have proven ineffective, as well as psychotherapy, a person may be considered an eligible candidate for ECT and prescribed a set number of treatments by their psychiatrist. If the patient is under the Mental Health Act and is unable to consent to treatment, a second opinion by an independent psychiatrist is sought.

ECT is prescribed as a course of treatments, rather than just one. One course can involve anything from six to 12 individual treatments of ECT (MOH; Ministry of Health, 2009). For patients under the mental health act, their ability to comprehend the risks and benefits of the treatment is reassessed after each treatment (MOH, 2009). The number of treatments a person may have will depend on the severity of illness and degree of treatment resistance, degree of complicating medical factors, the person's age (elderly patients may require longer courses) and technical parameters such as whether the ECT is administered bilaterally or unilaterally (Ministry of Health, 2012).

In New Zealand ECT is administered by trained, highly qualified and experienced personnel. At a minimum, the ECT team will comprise of a psychiatrist, anesthetist, ECT nurse and a recovery nurse (Ministry of Health, 2004). ECT is administered under general anesthetic in an operating theatre and a single treatment tends to take around half an hour (Ministry of Health, 2004). The electrical dose administered is individually tailored according to the patient's seizure threshold. A higher dose is given for unilateral ECT than for bilateral ECT, usually ranging somewhere between 50-150% above seizure threshold for bilateral ECT, and 150-600% for unilateral ECT. In New Zealand and the UK, ECT is administered twice a week which is less frequent than in the United States where ECT is typically administered three times weekly (Lerer et al., 1995). ECT is most commonly administered unilaterally (to one hemisphere of the brain) and this is most generally to the right side as it causes fewer cognitive effects than bilateral ECT and right unilateral ECT spares verbal functioning (Sackeim, Prudic, et al., 2000; Squire & Slater, 1978b). Treatment is discontinued either: after the course of ECT is completed, when ECT has been proven to be

ineffective for the individual, or after clinical efficacy has reached a plateau. Though in recent years maintenance ECT after the initial course seems to be common (McCall, 2001).

The latest public statistics on the number of patients who receive ECT in New Zealand are from 2011, and during this year 286 patients received ECT (Ministry of Health, 2012). The national rate of people receiving ECT was 6.5 per 100,000. Of these, 64% were women and 36% men. This gender difference reflects the greater number of women who present to mental health services with mood disorders. This ratio is similar to that reported in other countries (Ministry of Health, 2012). Europeans constituted the bulk of patients receiving ECT (84%), followed by Māori (7%), other (5%) and Asian (3%). The age range of patients who received ECT in 2011, ranged from 15-19 to 90-95, and patients were typically in the older age brackets. Figure 2 shows how the ages were dispersed among this group.

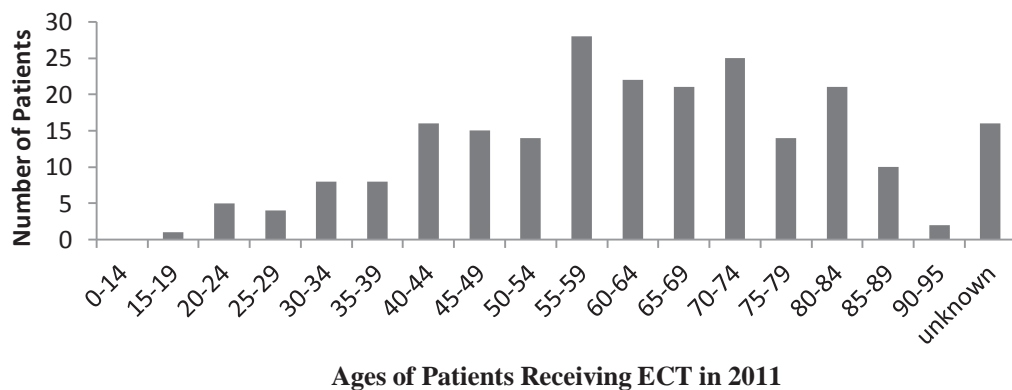


Figure 2. Age distribution of individuals receiving ECT from 1st of January to 31st of December 2011. Data abstracted from the Office of the Director of Mental Health Annual Report (2012).

Seventy five years after its conception, ECT remains the most effective treatment for major depressive disorder (UK ECT Review Group, 2003). The popularity of ECT has waxed and waned over the past eight decades; however, due to the superior clinical efficacy of the treatment and extensive research conducted into lessening the adverse cognitive effects, a resurgence in popularity

has occurred. Curiously, despite the considerable amount of research conducted into ECT, the way in which this remarkable treatment works remains undetermined (Fink, 2001). The following chapter provides an overview of some of the many variables which can moderate the cognitive and clinical side effects of the treatment.

CHAPTER TWO

Variables Affecting the Cognitive and Clinical Outcomes during Electroconvulsive Therapy

As with most medications and treatments ECT comes with side-effects; the most serious and unwelcome of these is cognitive impairment (Ingram, Saling, & Schweitzer, 2008). The cognitive changes which commonly occur during ECT are reported and discussed in the next chapter (Chapter Three); however, prior to understanding *how* ECT affects cognition, the numerous variables which act to moderate cognitive outcome should first be acknowledged. An appreciation of how these variables can impact cognitive function is necessary to understand the nature of cognitive changes during a course of ECT. Some of the moderating variables are related to the *person* receiving the treatment and other variables are related to the *treatment* itself. Such person related variables include: the effect of co-morbid illness (such as depression) on cognitive function, level of premorbid cognitive functioning, degree of cognitive reserve, concomitant medications and the individual's age. These will be discussed in Part I of this chapter. Treatment related variables that moderate cognitive outcome include: the number of given treatments, the frequency of treatments, the dosage of electricity used per treatment, the type of electricity used, the placement of the electrodes and location of electrical stimulation on the brain, the anesthetic agent used and duration of postictal confusion and disorientation. These will be discussed in Part-II of this chapter. The current chapter aims to raise awareness of the extensive number of variables which have been shown to affect degree and direction of cognitive change and to shed some light on how these can affect cognitive outcome.

As there are many ethical and practical challenges when conducting research in this population, the available literature on the variables affecting cognitive and clinical outcomes of ECT have limitations which are worthy of discussion. One of the most important limitations is attrition in

research, particularly when conducting follow-up assessments with patients. Often the individuals who are not included in the follow up assessments are the individuals who are the most impaired or most unwell, therefore this data may not be captured and therefore the research not entirely representative. Another limitation is the high variability among individuals included in this research, variables that are not often possible to control for. A final issue worthy of mention is the small sample sizes and the resultantly low power and weighting these studies have behind the conclusions and thus affect the generalisability of the results.

Part I: Person Related Variables

Depression

Cognitive impairment is a core component of depressive illness (Douglas & Porter, 2009). As ECT is a treatment for depression, patients are likely to present with cognitive complaints prior to commencing ECT. From a review of 30 studies published up until May 2009, Douglas and Porter (2009) found that the neuropsychological domains of verbal learning and memory, non-verbal learning and memory, sustained attention, executive functioning and psychomotor speed were most sensitive to improvement in clinical state in patients suffering from major depression. They concluded that whether mild or severe, depression is associated with broad cognitive impairment across many domains of cognitive functioning.

Cognitive Effects of Depression Vs. Cognitive Effects of ECT.

As the cognitive profiles of ECT and depression are similar, the cognitive side effects caused by ECT treatment can be challenging to tease apart from the cognitive side effects of ECT (Porter, Douglas, et al., 2008). Similarities include difficulty with verbal and non-verbal learning, memory functions, concentration and attention deficits and general dysfunction across a broad range of cognitive abilities (Douglas & Porter, 2009) (see Chapter Three for further discussion).

As Figure 3 portrays, prior to starting a course of ECT a patient may suffer cognitive dysfunction due to depression and during ECT treatment compound cognitive effects can occur from both depression and ECT– as demonstrated by the crossover section. If ECT is successful in alleviating depressive symptomatology, the cognitive effects of depression will subside but some residual effects from ECT may remain, as demonstrated by the red horizontal section. In some patients, residual cognitive impairment from depression remains even once the patient has recovered from the illness (Porter, et al. 2008).

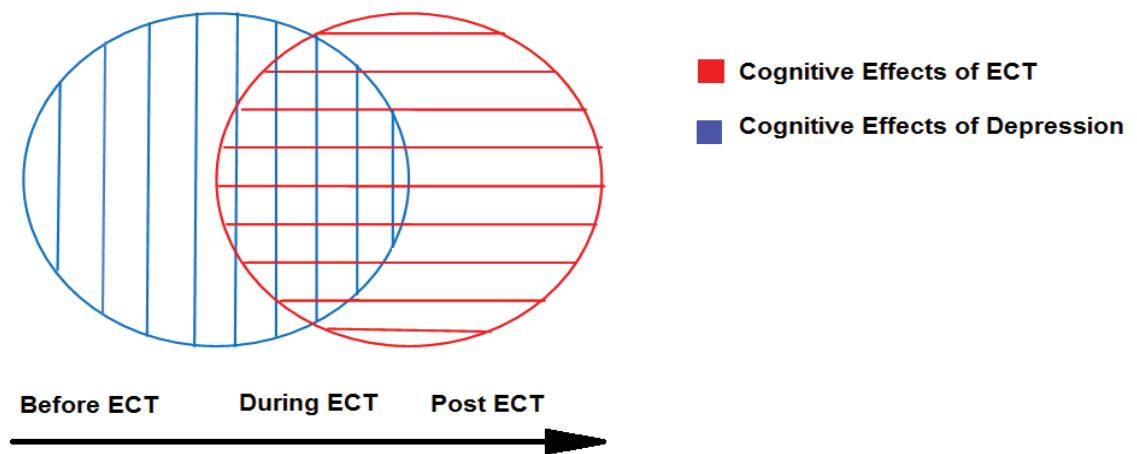


Figure 3. Depiction of how the depression and ECT related cognitive effects can overlap across time. Vertical lines represent the cognitive effects of depression and the horizontal lines represent the cognitive effects of ECT.

Although the cognitive profiles of depression and electroconvulsive therapy are similar, some differences in cognitive profiles exist. One key difference as noted by Sackeim and colleagues (1986) is that depression is associated with deficits of *acquiring* new information, whereas electroconvulsive therapy is more greatly associated with transient disruption in *retaining* new information. This difficulty in acquiring new information during depression may be due to attentional deficits associated with depression, whereas the disruption in retaining new information may be due to a disruption in the process of consolidating short term memories into long term

memories. During ECT, memory loss is more profound around the time of the treatment, whereas retrograde amnesia due to depression is more widespread. Detecting these subtle differences is another way of clarifying whether the cognitive deficit can be attributed to ECT or depression. In addition, it has been reported that with modern techniques of ECT administration (brief pulse stimulation at a low or moderate dose) the cognitive effects of ECT are less pronounced and usually do not exceed the effects of depression (Calev, Gaudino, Squires, Zervas, & Fink, 1995).

Global Cognitive Impairment prior to ECT

Patients who manifest global cognitive impairment prior to treatment with ECT have been found to be more vulnerable to persistent retrograde amnesia for autobiographical memories (loss of memories pertaining to the individual's life) (Sobin et al., 1995). In a study of 71 patients receiving ECT, those with lower baseline global cognitive functioning scores as measured by the Mini-Mental Status Examination (Folstein, Folstein, & McHugh, 1975) suffered a greater degree of retrograde amnesia two weeks after cessation of ECT (Sobin et al., 1995). According to the results of this study, premorbid cognitive functioning appeared to negatively predict cognitive impairment.

Cognitive Reserve

Cognitive Reserve (CR) theory stipulates that a greater level of education and occupational attainment is protective against cognitive dysfunction and a lower CR serves as a vulnerability factor for the effects of neurologic problems (Stern, 2002). A study by Legendere, Stern, Solomon and Furman (2003) tested the cognitive reserve (CR) theory with 50 patients receiving ECT. Cognitive reserve was calculated based on number of years of formal education and level of occupational attainment. These scores were transformed into standardised scores and patients were classified as either having high or low CR. The patients were assessed prior to starting the course of ECT and again after three treatments. Patients classified as having high CR remembered significantly more information after a 30 minute delay compared to the patients in the low CR

group. The authors proposed that high cognitive reserve is protective against functional impairment secondary to brain insult (Legendere et al., 2003).

Concomitant Medications

Medications have also been shown to affect degree of cognitive change during ECT. Sackheim and colleagues (2009) conducted a prospective, randomised, placebo controlled study to examine the effects of pharmacology on electroconvulsive therapy outcomes, including cognitive outcome. The authors found that some medications worsened cognitive adverse effects (for example, venlafaxine), whereas other medications actually enhanced ECT efficacy and reduced adverse cognitive side effects (for example, nortriptyline). In addition, the use of an antidepressant medication seemed to increase clinical efficacy and assisted in maintaining the clinical benefits received from ECT treatment (Sackheim et al., 2009). These authors concluded that reduced or increased cognitive side effects may differ as a function of concomitant medication used (Sackheim et al., 2009).

Age

ECT is used to treat patients across a wide range of ages, generally from adolescence until late adult life. The effect of a patient's age on cognitive outcome has been well researched; however, outcomes have been inconsistent. The results of some studies suggest that older adults may be more susceptible to cognitive deficits from ECT (Zervas, Calev, Jandorf, Schwartz, & et al., 1993), conversely, some researchers have found that cognition improves after ECT for all patients regardless of age (Brodaty, Hickie, Mason, & Prenter, 2000; Rey & Walter, 1997; Tielkes, Comijs, Verwijk, & Stek, 2008), and other researchers have found that the cognitive *improvement* occurs after ECT and the improvement is *greater* in older patients (Bosboom & Deijen, 2006; Wilkinson, Anderson, & Peters, 1993). These variable outcomes are explored in greater detail below.

It has been suggested that older adults may be more susceptible to cognitive difficulties post ECT as age in itself can induce cognitive frailty, and older adults are more likely to suffer comorbid

conditions which impact on cognitive function such as dementing illnesses and cerebro-vascular diseases (Tielkes et al., 2008). A study conducted in 1993 examined the relationship between age and cognitive impairment in 42 older adults receiving ECT for severe depression, found that older patients were more vulnerable to short term cognitive impairment in the domain of memory than younger patients. The memory difficulties suffered were more severe and longer lasting in the older patients. This pattern resolved at around six months when no differences in memory scores were observed (Zervas et al., 1993). The authors concluded that older patients may be more vulnerable to the short term cognitive side effects of electroconvulsive therapy but not the long term effects.

A study conducted by Brodaty, Hickie, Mason and Prenter (2000) found that regardless of age, cognition improved across a broad range of cognitive domains, and improvement in cognition was comparable between younger and older adults receiving ECT. These findings were replicated in a more recent review of cognitive outcome during ECT in older adults (Tielkes et al., 2008). In most studies reviewed, global cognitive functioning actually improved, and this improvement was most often maintained at follow-up assessments (Tielkes et al., 2008). Another review which compared the cognitive outcome of youth and adults receiving ECT also showed that type and frequency of cognitive impairment due to ECT was comparable for youth and adults (Rey & Walter, 1997). As a result of these studies, ECT was concluded to be as effective and safe for elderly patients suffering from depression as for younger patients (Brodaty et al., 2000).

Some studies have found that older patients experience cognitive improvement after ECT, and that this improvement is *greater* for older patients (Bosboom & Deijen, 2006; Wilkinson et al., 1993). This observation is supported by the cognitive reserve hypothesis discussed earlier in this chapter. The conclusions around what effects age has on degree of cognitive change during ECT remain mixed. However, most reviews suggest that age is protective against cognitive impairment.

The first section of this chapter has outlined how person related variables such as depression, premorbid cognitive functioning, cognitive reserve, concomitant medications and the patient's age can affect degree of cognitive change during a course of electroconvulsive therapy. The second section of this chapter will outline some of the treatment related variables which can affect cognitive outcome.

Part II - Treatment Related Variables

Frequency of ECT Treatment

The extent and existence of cognitive impairment has been shown to be dependent upon techniques used when administering the ECT treatment (Fraser, O'Carroll, & Ebmeier, 2008). One of the variations in ECT administration is the frequency at which the treatment is given. There are two commonly used frequency schedules of ECT administration: ECT administered twice, and three times per week (Lerer et al., 1995). As noted earlier, Australasian, British and most European clinicians most typically administer ECT at a frequency of twice per week whereas in the United States, three sessions of ECT per week tends to be standard procedure (Lerer et al., 1995). These schedules differ somewhat in their clinical and cognitive outcomes.

Twice weekly ECT is associated with less cognitive impairment than thrice weekly ECT. Lerer and colleagues conducted a double-blind study to compare clinical efficacy and cognitive outcomes for twice versus thrice weekly ECT. Both frequencies of ECT improved clinical outcome, and there was no difference in clinical outcome at one week and one month following cessation of treatment. While patients receiving more frequent ECT responded more quickly to the treatment, cognitive impairment was more profound. These findings have been replicated by Shapira and colleagues (1998). More recently Charlson, Siskind, Doi, McCallum, Broome and Lie (2012) conducted a systematic review and meta-analysis of twice weekly and thrice weekly ECT schedules. In accordance with previous studies, they too found no significant difference between twice and thrice

weekly ECT in terms of antidepressant effect, and patients receiving twice weekly ECT required fewer treatments. Remission rates did not differ between frequencies; however, cognitive side effects were more profound with more frequent treatment. It is recommended that twice weekly ECT be used over thrice weekly ECT, as clinical outcome is comparable and cognitive deficits are fewer; however, thrice weekly ECT should be used when fast clinical improvement is of primary importance (Lerer et al., 1995) such as in the case of patients who are suicidal.

Number of ECT Treatments

The number of treatments a patient receives also has an effect on the degree of cognitive impairment suffered. Many studies have shown that there is a cumulative effect on cognitive impairment temporally across treatments, with greater number of treatments being associated with a greater degree of amnesia suffered (Sackeim et al., 2007; Zinkin & Birtchnell, 1968). Conversely, Sackeim and colleagues (1986) showed that a cumulative cognitive *improvement* was observed when *low dosage* ECT was used. These authors suggested that low dosage titration may lessen some of the adverse cognitive side effects of ECT (Sackeim et al., 1986).

Dosage of Electricity

Adverse cognitive side effects have been associated with the magnitude of *excess* electrical stimulation above a patient's seizure threshold (Sackeim, Devanand, & Prudic, 1991). Seizure threshold is defined as "the smallest amount of electrical energy needed to induce an adequate seizure" (Arrufat, 1997). A patient's seizure threshold varies depending on electrical resistance of the skull (Prudic, 2008) and the patient's age and sex (Ministry of Health, 2004). To investigate the effects of the application of different electrical dosages, a prospective randomised double blind pilot study was conducted using ultra-brief electricity and right unilateral ECT with patients suffering from major depression. The electrical dosages were four, seven and ten times above seizure threshold. Higher electrical dosages were associated with greater cognitive impairment than lower doses (Quante et al., 2011).

Using excess electricity (supra-threshold dosing) is still commonly used as it has clinically superior results compared to threshold doses of electricity. Supra-threshold dosing is associated with a greater clinical response in right unilateral ECT, and a faster response in both right unilateral and bilateral ECT (Lisanby, Maddox, Prudic, Devanand, & Sackeim, 2000; Sackeim et al., 1993). Right unilateral ECT at a high dose is considerably more effective than low dosage right unilateral ECT and produces less severe cognitive effects than either form of bilateral ECT (Sackeim et al., 1993). As described, the cognitive and clinical outcome of electroconvulsive therapy is dependent on the dosage of electricity used and whether this dosage is above or at the patient's seizure threshold.

Type of Electricity

The type of electricity used in electroconvulsive therapy can also affect cognitive outcome. As noted earlier, sine wave electrical stimulation was the first type of electricity used in ECT but is now seldom used as it has been associated with short and long term cognitive deficits (Sackeim et al., 2007) and is inefficient at inducing seizures (Weiner, 1980). The use of oscillating sine-wave electricity has been replaced with square-wave brief-pulse or ultra-brief pulse electrical stimulation. See Figure 4 below for a depiction of the different types of electrical wave forms used for ECT treatment. Sine wave electricity has a pulse width of around 0.8 ms, which is relatively long compared to brief-pulse (0.5 ms) and ultra-brief pulse (0.2-0.3 ms) (Verwijk et al., 2012). Since the introduction of using brief and ultra-brief pulse electricity in ECT, the degree of cognitive impairment associated with the treatment has declined (American Psychiatric Association, 2001).

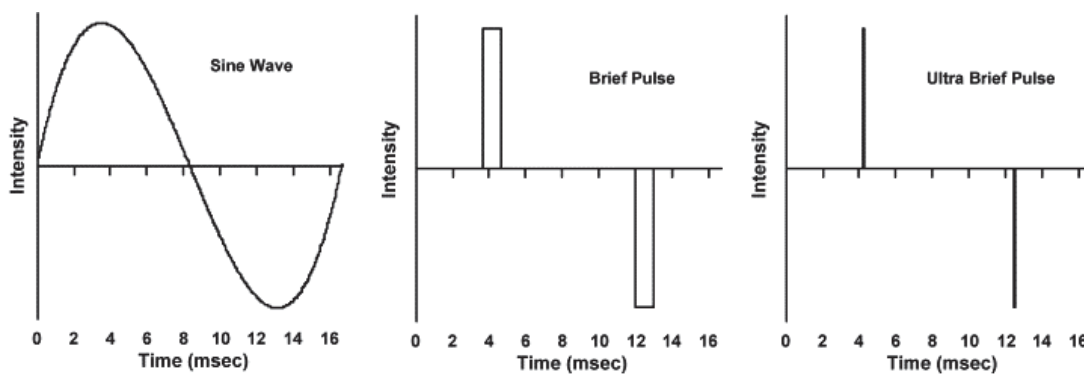


Figure 4. Demonstration of physical differences between sine-wave, brief-pulse and ultrabrief-pulse waves. This figure has been reproduced with modifications from Sackeim (2004).

Randomised Control Trials (RCTs) have demonstrated that more severe short-term memory deficits occur from using sine wave compared to brief pulse stimulation (Weiner, Rogers, Davidson, & Squire, 1986) due to excess energy associated with sine wave (Prudic, 2008). Yet even more effective at reducing cognitive side effects is the use of ultra-brief pulse electrical stimulation (Loo, 2007; Sienaert, Vansteelandt, Demyttenaere, Peuskens., 2006; Verwijk et al., 2012). This is because brief pulse waveform still delivers energy in excess of what is required to induce a seizure and it is this excess energy which is hypothesised to cause the cognitive impairment (Prudic, 2008). Sackeim and colleagues conducted a double-blind study where 90 patients receiving ECT for depression were randomly assigned to ECT using traditional brief pulse electricity to induce the seizure or ultra-brief pulse ECT. Cognitive functioning was assessed at baseline, during treatment, and at various time intervals after the therapy (Sackeim et al., 2008). Outcome cognitive assessments revealed that ultra-brief pulse stimulation reduced both the acute and long term cognitive effects associated with brief-pulse ECT. At a six month follow-up, ultra-brief pulse ECT was associated with less retrograde memory impairment than brief pulse ECT (Sackeim et al., 2008).

Electrode Placement

The locations at which electrodes should be placed remain a highly researched and hotly debated topic within the field of ECT practice (Abrams, 2002; Kellner et al., 2010). The debate is around where to best place electrodes in order to optimise clinical efficacy of the treatment while minimising the adverse effects of ECT on cognition (Prudic, 2008). The two most commonly used placements are: bilateral (also known as bitemporal) and unilateral. A third, less commonly used and less researched placement is bifrontal electrode positioning. These placement variations are depicted in Figure 5 below.

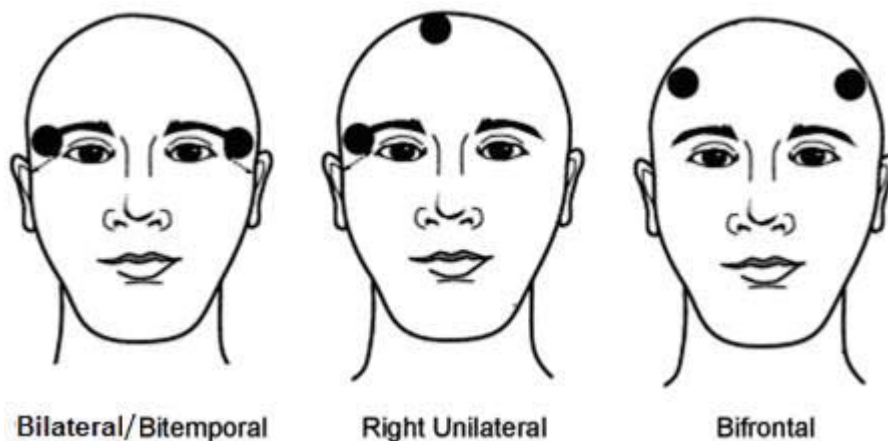


Figure 5. These diagrams depict bilateral/bitemporal, right unilateral and bifrontal electrode placements. This figure has been reproduced with modifications from Kellner, Pritchett, Beale and Coffey (1997).

Bilateral electrode placement has the longest history in ECT practice and remains a commonly used placement. Bilateral positioning requires both electrodes to be placed on either side of the head, four centimeters perpendicular to the middle of a line connecting the external ear canal with the angle of the eye (Sobin, Prudic, Devanand, Nobler, & Sackeim, 1996). Unilateral placement is currently the most highly recommended placement for ECT treatment (Mankad, Beyer, Weiner, &

Krystal, 2010). This position involves placing electrodes, and consequently administering the electrical stimulation, to one side of the patient's scalp. This is usually the right side, as the left hemisphere is largely responsible for speech functions in the majority of people. Unilateral positioning requires one electrode to be placed in the temporal position, and the second electrode to be placed one inch ipsilateral to the vertex of the scalp (Weiner et al., 1990). For patients who are left motor dominant, it is less clear which hemisphere speech functions will be primarily based (Pratt, Warrington, & Halliday, 1971). In these cases, cognitive testing shortly after treatment can clarify whether language functioning was temporarily disrupted by the treatment. Bi-frontal placement requires both electrodes to be placed 2.5cm anterior to bi-temporal sites symmetrically on either side of the forehead (Swartz & Nelson, 2005). Bilateral, unilateral and bifrontal electrode placements all stimulate different areas of the brain and are therefore associated with different degrees of cognitive disturbance.

Bilateral electrode placement has been repeatedly associated with greater short and long term cognitive deficits than unilateral ECT (Sackeim et al., 2007). In a large scale study of clinical and cognitive outcomes in 347 patients receiving ECT for depression, patients who had been treated with bilateral ECT suffered greater retrograde amnesia for autobiographical memory compared to patients receiving unilateral ECT (Sackeim et al., 2007). In addition, bilateral ECT was associated with greater deficits in cognitive functioning than any dosage of unilateral ECT for anterograde and retrograde memory. Two months post cessation of treatment, patients who received bilateral ECT generally had greater retrograde amnesia than patients who received right unilateral ECT (Sackeim, Prudic, et al., 2000). Performance on measures of global cognitive functioning is also more impaired after bilateral ECT than unilateral ECT (Sobin et al., 1995). Anterograde and retrograde memory for verbal material are more disrupted by bilateral than right unilateral ECT (Daniel & Crovitz, 1983; Prudic, 2008; Squire & Slater, 1978). After termination of a course of ECT performance on anterograde and retrograde memory measures for both verbal and spatial tasks has

been shown to be more impaired with bilateral ECT compared to unilateral ECT (Daniel & Crovitz, 1986; McElhiney et al., 1995; Sackeim et al., 1993; Sackeim, Prudic, et al., 2000; Steif, Sackeim, Portnoy, Decina, & Malitz, 1986; Weiner et al., 1986). At two months (Sackeim et al., 1993) and six months (Weiner et al., 1986) post treatment bilateral ECT is associated with greater retrograde amnesia. Retrograde memory deficits for autobiographical and public memories are also worse for bilateral than unilateral placement (Sackeim et al., 1993; Sackeim et al., 2007; Weiner et al., 1986). The literature is in agreement that right unilateral ECT is associated with fewer cognitive side effects than bilateral electrode placement.

The motivation behind using bi-frontal electrode placement is to decrease cognitive impairment by focusing the source of electrical stimulation directly onto the frontal lobes – the region of the brain thought to be most salient to the pathophysiology of depression - and away from the temporal lobes - regions thought to be associated with memory, to spare cognitive function (Prudic, 2008).

Bifrontal electrode placement is a relatively new electrode placement and fewer studies have assessed clinical and cognitive outcome of this placement. However, this placement appears to be an emerging topic of interest and the research which has been conducted on this placement shows promising results for the field of ECT.

Most studies comparing bifrontal electrode placement to bilateral and unilateral placement have demonstrated superiority of bifrontal placement in terms of cognitive and clinical outcome (Abrams & Taylor, 1973; Bailine, 2000; Bakewell, Russo, Tanner, Avery, & Neumaier, 2004; Lawson et al., 1990; Letemendia, 1993; Ranjkesh, 2005) with some exceptions of studies which have found no significant differences in cognitive outcomes. However, a major limitation to these studies is the often small sample sizes and large variability among the patients. When Kellner and colleagues (2010) randomly allocated a larger number of patients (N=230) into one of three electrode placement conditions: bifrontal at one and a half times seizure threshold, bilateral at one and a half times seizure threshold and right unilateral and six times seizure threshold, they found few

differences in cognitive outcome across the three electrode placements (Kellner et al., 2010).

Sienaert, Vansteelandt, Demyttenaere and Peuskens (2010) compared the cognitive side effects of bi-frontal and right unilateral ultra-brief pulse ECT and also found that both treatment placements resulted in cognitive and clinical improvement (Sienaert, Vansteelandt, Demyttenaere, Peuskens, 2010).

In summary, comparisons of cognitive outcome across electrode placements have been the focus of a number of studies. There are many issues related to electrode placement; these include individual characteristics of the patient receiving the treatment as well as issues with research comparing the clinical efficacy of the different placements. It is generally agreed that acute and short term adverse cognitive side effects are greater with bilateral ECT than with unilateral non-dominant ECT, and non dominant ECT spares cognitive functions dependent on the dominant hemisphere (Dunne & McLoughlin, 2012; Sackeim, 1992; Sackeim, Prudic, et al., 2000; Sackeim et al., 2008; Sobin et al., 1995; Squire & Slater, 1978). Bifrontal electrode placement is a relatively new placement in the field of ECT practice, and requires more research to better understand this placement's effect on cognitive and clinical outcome. The literature thus far, demonstrates that it is at the least as effective as bifrontal at one and a half times seizure threshold, bilateral at one and a half times seizure threshold and right unilateral and six times seizure threshold.

Anesthetic Agent

Some anesthetic agents may also affect cognitive impairment from ECT. There are two ways in which anesthetic agents can influence cognitive outcome: directly or indirectly. Anesthetic agents can directly act upon neuronal systems in the brain inducing cognitive change, or indirectly affect cognition by increasing seizure threshold, requiring higher dosages of electricity to induce the seizure, and the increase in electricity can affect cognitive outcome (MacPherson & Loo, 2008).

Recent studies have shown that cognitive outcome can actually be improved after ECT as a function of anesthetic agent used. For example, ketamine, a drug used both for pain and as an anesthetic, has

been suggested to be “neuro-protective” when used as the anesthetic agent in ECT (see MacPherson & Loo, 2008). Ketamine can also be effective for the treatment of depression as it exerts rapid beneficial effects on suicidal ideation (Price, Nock, Charney, & Mathew, 2009). A retrospective study investigating the use of ketamine as the anaesthetic agent in ECT found that ketamine acts synergistically with the clinical benefits on mood evoked by ECT. The study compared 16 patients who received ECT with ketamine, and 26 who received the barbiturate thiopental. Patients who received ketamine required significantly fewer treatments of ECT, had significantly lower depression scores on the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) and significantly higher Mini Mental State Examination (MMSE; Folstein et al., 1975) scores.

Postictal Confusion

Patients with longer acute disorientation after ECT treatment have been found to suffer from greater retrograde amnesia (Sobin et al., 1995). Disorientation refers to the length of time it takes for a patient to correctly reorient to time place and person. Recovery time during the postictal phase has been shown to predict short and long term retrograde amnesia for autobiographical memories.

Conclusion

There are numerous person and treatment related variables which affect clinical and cognitive outcome during treatment with electroconvulsive therapy. The current chapter, though not exhaustive, lists many of these variables. Understanding which person and treatment related variables may be affecting an individual’s cognitive outcome enhances treatment safety. In addition, the degree of cognitive impairment can better be predicted for each patient prior to beginning a course of ECT and changes can be made to reduce the impact of ECT on cognition and decrease the likelihood of the patient suffering long term cognitive effects (Prudic, 2008). The nature of the cognitive side effects and cognitive domains shown to be affected during a course of ECT are discussed in the following chapter.

CHAPTER THREE

Short Term Cognitive Side Effects of Electroconvulsive Therapy

“The effects of ECT on memory and cognition contribute to public fears. ECT does affect the mind – that unique and delicate essence of our individuality that distinguishes one human being from another...” (Fink, 1997).

Cognitive functioning is a collective psychological term referring to an individual’s ability to conduct a number of mental processes, including: processing information, forming memories and remembering, problem solving, decision making, producing and comprehending language, perceiving information and attending or concentrating (Goldstein, 2008). An individual’s cognitive abilities are inferred from their behaviour (Abikoff et al., 1987, as cited in Lezak, Howieson, & Loring, 2004) often via measuring performance on formal tests. Numerous studies have shown that individuals’ performances on these tests commonly decline throughout a course of ECT, and despite advancements in the way the treatment is administered, cognitive decline still remains a widely accepted and common side effect of ECT (Ingram, Saling, & Schweitzer, 2008).

A large proportion of research into the cognitive side effects of ECT since the change from using sine-wave electricity to brief-pulse indicates that the cognitive side effects of the treatment are short term, and *should* resolve within the first few months following cessation of ECT (Cohen & Squire, 1981; Quante et al., 2011; Semkovska & McLoughlin, 2010; Stoudemire, Hill, Morris, Martino-Saltzman, & Lewison, 1993; Williams, Iacono, Remick, & Greenwood, 1990) with no long term effects on memory (Abrams, 2002; Fink, 2004; Semkovska & McLoughlin, 2010). However, the findings are mixed, and some patients report permanent retrograde amnesia extending back several months or years after treatment (McElhiney et al., 1995; Rose, Fleischmann, Wykes, Leese, &

Bindman, 2003; Sackeim, 2000; Sobin et al., 1995). Although the degree of persistence of cognitive impairment from ECT remains under debate, short term cognitive impairment is consensually agreed upon as a real and undesirable side effect for many patients receiving the treatment. The current chapter offers a review of the common short term cognitive side effects associated with the treatment, the nature of these side effects, and some recommendations as to how these changes can be assessed.

Short Term Cognitive Side Effects of ECT

Extensive research has shown that cognitive impairment from electroconvulsive therapy is often global across a wide range of – but not all - cognitive functions (Semkovska & McLoughlin, 2010). Patients may experience difficulties with their memory, with the speed in which they are able to process information, their ability to concentrate and hold attention, their ability to plan and organise ideas, to make decisions, to mentally manipulate information and their general intellect may temporarily become impaired (Ingram et al., 2008). Luther (2012) reviewed 42 studies and literature reviews published between 1978 and the end of 2011 on the outcomes of ECT on cognition. Table 1 presents the domains of functioning she found most adversely affected by ECT.

Table 1
Domains of Functioning Most Commonly Adversely Affected during a Course of ECT and the Percentage of Studies Showing Impairment in this Domain

Domain	% of studies showing impairment
Encoding*	100%
Public Events Memory*	100%
Everyday Memory*	100%
Visual Processing Speed*	100%
Psychomotor Speed*	100%
Retrograde Amnesia/Memory	92%
Subjective Complaints	86%
Information Processing Speed*	86%
Autobiographical Memory and Retrieval	83%
Anterograde Amnesia/Memory	73%
Verbal Learning and Memory	68%
Semantic Memory*	67%
Global Cognitive Functioning	67%
Cognitive Flexibility*	67%
Visual Learning and Memory	50%
Executive Functioning	50%
Attention	46%
Working Memory	44%
Language*	33%

Note. * Indicates the domain was less commonly assessed; with fewer than seven studies included in the review having assessed these particular domains.

Luther (2012) concluded that the domains of functioning most commonly impaired by electroconvulsive therapy are: retrograde amnesia (for declarative memory including semantic, episodic and autobiographical memory), anterograde amnesia (impairment in learning new information and memory of events since ECT), executive functioning, psychomotor speed, visual processing speed, attention, working memory and information processing speed. Language was less commonly affected. Memory remained the most commonly impaired and assessed domain during and post ECT treatment (Luther, 2012). A limitation of this review is that it does not discern between the short term and long term effects of ECT.

The main short term cognitive changes associated with ECT can be categorised into the following categories: postictal confusion, retrograde amnesia, anterograde amnesia, subjective memory complaints and non-memory cognitive side effects (Nehra, Chakrabarti, Sharma, & Painuly, 2007). Each will be described briefly in the following sections.

Post Ictal Confusion

Postictal confusion is a transient secondary effect of treatment whereby the patient awakes from the anesthetic disorientated to time and place, agitated, unable to follow commands and may make repetitive stereotyped movements (Mankad, Beyer, Weiner, & Krystal, 2010). Post-ictal confusion is common and is estimated to occur in approximately 10% of patients receiving ECT (Mankad et al., 2010). It is a transient effect of ECT and within an hour the patient typically recovers from this state (Ministry of Health, 2012).

Retrograde Amnesia

The most concerning cognitive side effect of ECT is the *persistence* of retrograde amnesia (Goodman, 2011). Retrograde amnesia in ECT refers to the inability to consciously recall events that occurred or information that was learned prior to the course of ECT. At least one third of patients receiving ECT will experience some degree of retrograde amnesia post treatment (Rose et al., 2003). Shortly after ECT most patients will experience gaps in their memory for events that occurred close to the time of treatment (Cohen & Squire, 1981). Delayed recall of declarative or explicit memory is the type of memory identified to be most susceptible to the effects of ECT; non-declarative or implicit memory seems to be relatively preserved (Rami-Gonzalez et al., 2001; Squire, 1986; Squire & Chace, 1975). There are two main subtypes of declarative memory associated with dysfunction from ECT; namely autobiographical memory and impersonal semantic memory.

Autobiographical Memory

Autobiographical memory refers to the ability to recall events and facts that are specific to a person's past. This type of memory involves both *episodic* and *semantic* components. Semantic autobiographical memory contains knowledge and facts of one's past whereas episodic autobiographical memory is the conscious recollection of temporally and spatially specific events from one's past (Levine, 2004). These types of memory are complex as they consist of spatial,

emotional and sensory components (Goldstein, 2008). An example of an autobiographical memory would be remembering going on a picnic. The semantic component would be remembering the fact that one went on a picnic, and the episodic component would be remembering details about the episodes that occurred during the picnic. Levine (2004) refers to this episodic recollection as a conscious state of “mental time travel”. Over time, some autobiographical memories take on the quality of fact, in which the episodic details are lost (Levine, 2004). In keeping with the previous example, the person may remember going on a picnic at a particular time but not remember anything about the picnic. ECT has been shown to affect both these aspects of autobiographical memory (Semkowska & McLoughlin, 2010).

Autobiographical memory loss has been widely and repeatedly reported as an adverse secondary effect of ECT (see for example: Calev et al., 1993; Fraser, O'Carroll, & Ebmeier, 2008; Peretti, Danion, Grange, & Mobarek, 1996; Sackeim et al., 2000; Sackeim et al., 2007; Shapira et al., 1998; Sobin et al., 1995; Squire & Chace, 1975; Squire & Zouzounis, 1986) and has been deemed the most distressing side effect of ECT (Sackeim et al., 2008; Scott, 2010). A review of 15 studies published between 1980 and 2007 suggested that autobiographical memory impairment does occur as a result of ECT; however, the persistence of the memory loss is generally short term, resolving within 6 months post treatment (Fraser et al., 2008). Memories of personal events occurring close in time to treatment, generally within six months prior to starting the course of ECT, are more susceptible to memory loss than memories formed further back in a person's life (Fraser et al., 2008). Some researchers have found that autobiographical memory loss persists until one week after completion of a course of ECT (Peretti et al., 1996), while others have reported the effects to be long lasting (Lisanby, Maddox, Prudic, Devanand, & Sackeim, 2000) or permanent (Rose et al., 2003).

Impersonal Semantic Memory

Although autobiographical amnesia has been reported to be the most distressing cognitive side effect resulting from ECT, non-autobiographical semantic memories are more susceptible to forgetting due to ECT. Lisanby and colleagues (2000) found that ECT results in greater and more persistent cognitive impairment for knowledge about the world and public events than for personal knowledge, and this impairment was persistent at a two month follow-up. These authors proposed this was due to deeper encoding of personal information, and that less personal significance is attributed to impersonal world events and are therefore more susceptible to being forgotten. A reason why autobiographical amnesia has received greater attention in the literature than impersonal semantic amnesia may be due to the magnitude of personal distress caused by this impairment relative to other types of retrograde amnesia.

Anterograde Amnesia

Anterograde Amnesia refers to the inability to retain new information acquired after ECT. Most people who receive ECT will experience some degree of anterograde amnesia (Firth et al., 1983; Hasse-Sander, Muller, Schuring, & Möller, 1998). Both recall and recognition memory of newly learnt facts and information are adversely affected post ECT (Ghaziuddin et al., 1996; Squire, Slater, & Chace, 1976; Steif, Sackeim, Portnoy, Decina, & Malitz, 1986). However, the ability to learn and retain new information recovers typically within a few days of the last ECT treatment (Calev, Nigal, Shapira, & Tubi, 1991; Goodman, 2011; Sackeim et al., 1993).

Verbal and Visual Memory

A systematic review and meta-analysis conducted by Semkovska and McLoughlin (2010) revealed that in the short term, verbal and visual memory become acutely impaired during ECT treatment relative to baseline functioning, but improved in the long term. Immediate and short term memory is also impaired after ECT treatment (Steif et al., 1986). Porter, Heenan and Reeves (2008) found that after three treatments of ECT patients scores significantly reduced from baseline on the Rey

Auditory Verbal Learning Task. Delayed recall was more adversely affected than immediate recall (Semkovska & McLoughlin, 2010).

Non-Memory Cognitive Impairment

Non-memory cognitive impairment includes all cognitive domains of function besides memory, such as: concentration and attention, working memory and executive functions. Non-memory cognitive changes during ECT are often neglected within the ECT literature (Ingram et al., 2008). Amongst the few studies which have investigated the non-memory cognitive side effects of ECT mixed results have been reported (Ingram et al., 2008). These authors urged that more research is needed to characterise what the nature of impairment and recovery is for these non-memory cognitive domains during and after a course of ECT. Since some non-memory cognitive abilities such as attention and encoding are required for the formation of memories, this adds further importance to understanding these non-memory changes which occur (Robertson & Pryor, 2006).

Processing Speed

Research has shown that ECT can induce acute impairment in the speed at which an individual is able to process information (Tsourtos, Spong, & Stough, 2007); however, this tends to improve two weeks following cessation of ECT treatment (Semkovska & McLoughlin, 2010).

Executive functioning

Executive functioning, which includes mental shifting, inhibition, mental flexibility, semantic fluency and organisation in thinking is often affected by ECT. Semkovska and McLoughlin's (2010) meta analysis found medium to large impairments in patients' executive functioning ability shortly after ECT treatment; however, patients recovered back to their baseline level of functioning during short term follow-up and showed improvement at long term reassessment. Working memory tends to improve somewhat two weeks post ECT compared to patients' baseline assessment. Spatial problem solving has been shown to become impaired during a course of ECT relative to baseline;

however, after cessation of ECT treatment, patients generally recover to pre-treatment levels at long term follow-up (Semkovska & McLoughlin, 2010).

Attention/Concentration

An individual's ability to attend and concentrate has shown improvement relative to baseline at short and long term follow-ups post treatment with ECT. These changes are often associated with the magnitude of clinical improvement (Sackeim, 1992; Steif et al., 1986).

Subjective Reports of Changes in Cognition

Subjective Memory refers to the patient's *perception* of their memory function. It is an integral component of cognition to assess and document, as subjective reports of memory loss or dysfunction are not often detected by objective assessment (Nehra et al., 2007). Commonly, when objective and subjective cognitive outcome are compared, there is often a weak or no association between the two (Broadbent, Cooper, Fitzgerald, & Parkes, 1982; Nehra et al., 2007). Likewise, in healthy and neurological samples subjective reports of memory generally show weak or no association with objective neuropsychological assessment (Broadbent et al., 1982). Prior to 1975 when sine wave stimulation was used, subjective memory scores were significantly worse after ECT than they were at baseline (Nehra et al., 2007). Since the change in electrical pulse to brief pulse stimulation, subjective memory now significantly improves following right unilateral or bilateral ECT (Coleman et al., 1996; Pettinati & Rosenberg, 1984).

Although subjective and objective cognitive outcome are poorly correlated, subjective reports of cognitive function appear to correlate with patients' clinical states (Nehra et al., 2007). The better a patient's mood, the more positive a patient tends to rate his or her memory. It has been previously reported that a patient's subjective report of memory appears to be a greater reflection of their current mood state rather than their objective memory function (Coleman et al., 1996).

The Nature and Duration of ECT related Cognitive Change

Retrograde amnesia experienced from ECT is usually temporally graded, with events and information learnt closer to the ECT treatment more likely to be affected than memories formed earlier in the patient's life (Calev et al., 1993; Cohen & Squire, 1981; Fraser et al., 2008; Peretti et al., 1996; Rami-Gonzalez et al., 2001; Sackeim et al., 1993). Events occurring one year or less prior to ECT are more susceptible to memory loss than episodes occurring further back in time (Weiner, Rogers, Davidson, & Squire, 1986). Anterograde amnesia is usually confined to the time of the treatment course or shortly afterward (Rami-Gonzalez et al., 2001).

Current research on the persistence and severity of ECT related cognitive impairment remains inconsistent (Semkovska & McLoughlin, 2010); however, most studies conclude that memory loss is not permanent and the deficit disappears within a month after cessation of ECT treatment (Meeter, Murre, Janssen, Birkenhager, & van den Broek, 2011; Williams et al., 1990). Semkovska and McLoughlin (2010) found that ECT caused significant cognitive impairment which was evident within days of finishing a course of ECT. These deficits were shown to resolve during the two weeks following cessation of treatment and improved thereafter. Three days after ECT, medium to large deficits were found for memory and executive functioning. No persisting cognitive effects were found 15 days after the last ECT treatment. Deficits in memory have been reported to typically remain a few days after termination of ECT (Firth et al., 1983; Ghaziuddin et al., 1996; Mackenzie, Price, Tucker, & Culver, 1985, Semkovska & McLoughlin, 2010).

The Positive effects of ECT on Cognition

Although cognitive dysfunction during treatment is common, cognitive improvement during ECT can also be observed. Global cognitive function improves as depression alleviates, therefore if ECT is successful in alleviating depressive symptomatology, cognitive improvement is often observed during or after ECT (Calev, Gaudino, Squires, Zervas, & Fink, 1995). Since the change from sine-wave electrical stimulation, most studies on memory following ECT indicate that patients report

equal or enhanced memory functioning within a few days following a course of ECT (Calev et al., 1991; Coleman et al., 1996; Greenberg & Kellner, 2005; Pettinati & Rosenberg, 1984; Weiner et al., 1986) and this improvement has been speculated to be due to the alleviation of depressive symptomatology (Coleman et al., 1996; Steif et al., 1986). Fifteen days post ECT treatment, processing speed, working memory and some aspects of executive function improve beyond baseline levels of functioning (Semkovska & McLoughlin, 2010).

In summary, cognitive impairment still remains a common and unwanted side effect of the treatment despite continual efforts to refine ECT practice (Abrams, 2002). Most research concludes that the effects of ECT on cognition are short term and temporary, and that no longstanding effects should remain. However, this is not the case for some individuals. Although no conclusive research exists, it has been hypothesised that marked cognitive decline early in treatment poses a risk factor for continual cognitive impairment as the treatment course progresses (Porter, Douglas, & Knight, 2008), therefore the earlier on in treatment cognitive decline is detected, the sooner modifications to treatment can be made and the better the prognosis for cognitive functioning. Thus, conducting frequent and thorough assessments of a patient's cognitive function is recommended as it facilitates detection of serious cognitive impairment early on in treatment (Nehra, 2008). The remainder of this chapter will describe some of the recommendations around assessment of cognition during the course of ECT and argues the benefits for assessing cognitive function during the course of treatment.

Recommendations and Guidelines for the Assessment of Cognitive Change during ECT

Various national and international organisations have recommended a patient's cognitive functioning is monitored intermittently throughout a course of ECT. The ECT Accreditation Service, the National Institute of Clinical Excellence, the American Psychiatric Association, the Royal Australian and New Zealand College of Psychiatrists, and the Department of Health in

Western Australia guidelines are some examples of which and will be briefly outlined within this chapter in addition to suggestions for assessment schedules and batteries which exist within the peer-reviewed literature. Some recommendations are brief, others more comprehensive, but all suggest that cognitive functioning *should* be assessed, and at best, regularly.

In the United Kingdom, for an ECT clinic to become accredited by the ECT Accreditation Service (ECTAS) the following standards around cognitive assessment must be met: patients' orientation and memory must be assessed before and after the first treatment of ECT, and reassessed at intervals throughout the treatment course. An 'excellent' clinic would also assess memory and cognitive functioning three and six months post cessation of treatment. At this time, it is recommended that both subjective and objective side effects are recorded (ECTAS, 2011). ECTAS suggests that patients' cognitive side effects and memory are assessed using the Mini Mental Status Examination (Folstein, Folstein, & McHugh, 1975), that subjective questioning occur within a clinical interview by the referring psychiatrist three to four working days after the end of the treatment course, and again one or two months post treatment. ECTAS encourages the use of a memory log to assess a patient's subjective memory between sessions.

The National Institute of Clinical Excellence (NICE, 2003) recommends that cognitive function be monitored on an ongoing basis and at a minimum at the beginning and end of each course of treatment. No recommendations for timing of assessments are given. Conversely, the American Psychiatric Association Task Force Report urges that objective assessments as well as patients' subjective reports of cognitive functioning be assessed at least weekly during a course of ECT and that orientation, anterograde and retrograde amnesia also be assessed (American Psychiatric Association, 2001).

The Royal Australian and New Zealand College of Psychiatrists' (RANZCP) guidelines for the administration of ECT state that "the patient must be reviewed after each ECT treatment by a

medical officer, who should assess the efficacy of treatment and adverse events, especially delirium ... standardised rating scales for longitudinal assessment of mental state (such as the Hamilton or Beck rating scales for depression) and of cognition (such as the Folstein Mini Mental State Examination) may be useful in assessing clinical progress” (RANZCP, 1999, p. 4).

Finally, the Chief Psychiatrist’s Guidelines for the use of ECT in Western Australia (2006) provided by the Western Australian Department of Health is more comprehensive. Within these guidelines medical professionals are urged to monitor the presence and severity of memory changes on an ongoing basis, at least 24 hours post treatment. Subjective reports of cognitive functioning as well as objective assessments are strongly encouraged. The practitioner is recommended to ask about patients’ distant memories, recent memories and memories which should have been laid down following commencement of ECT. Orientation to person, place and time should also be assessed as well as an informal assessment of anterograde and retrograde amnesia, with ECT as the time reference. Practitioners are encouraged to talk to the patient and other informants about the patient’s memory. Any changes in cognitive functioning should be documented and the assessment process should be repeated until memory is back to normal. If cognitive impairment persists after the course of ECT, a plan should be made for post ECT follow-up assessment and management. The guidelines recommend against formal neuropsychological assessment as it is impractical and has the potential to be confounded by practice effects. Instead they propose that the MMSE is used, but acknowledge that it has inadequate reliability to detect memory changes even when memory impairment is significant enough to cause functional impairment.

Recommendations in the published literature around ways in which cognitive changes should be measured are few. Based on a review of the current literature, Porter and colleagues (2008) offer the most comprehensive assessment schedule defining which cognitive domains to assess and recommendations as to how these domains can be assessed with various measures. Their assessment timing recommendations include: a baseline assessment and reassessment early in treatment, and

again after the sixth treatment. Porter and colleagues suggest that assessments are carried out at a standard time after treatment which should be at least 48 hours after treatment to allow for any transient treatment effects to resolve. They also recommended the same battery be repeated 2-3 months post treatment. A further recommendation is to include a mood measure alongside the cognitive assessment to allow for interpretation of cognitive change within the context of the patient's clinical state, as mood significantly affects cognitive performance.

Porter and colleagues reviewed the sensitivity of a number of cognitive tests to the effects of ECT treatment. From this review, a battery of tests was proposed for the use with patients receiving ECT. This 55 minute battery includes the MMSE or Modified Mini Mental State Examination (3MSE; Teng, 1987), Hopkins Verbal Learning Test (HVLT; Brandt, 1991), Autobiographical Memory Questionnaire- Short Form (AMI-SF; Kopelman, Wilson, & Baddely, 1989) and the Digit-symbol Substitution Task (DSST; Wechsler, 1997). These authors also urged that reorientation be assessed after each individual treatment (Porter, Douglas, et al., 2008).

A recent article originating from India argued that in developing countries Porter et al.'s proposed battery is unsuitable, due to the high caseload (where typically 10-15 patients receive ECT per day) and low resources available to clinicians (Viswanath et al., 2013). Thus they argued that the development of separate assessment measure was required to accommodate the cultural variation which exists globally. Viswanath et al. proposed a short (20-30 minute) sensitive battery for ECT Related Cognitive Deficits (B4ECT-ReCoDe) for the rapid assessment of cognitive deficits associated with ECT in the Indian setting. Their battery comprised of both subjective and objective components, and included: a shortened version of the Rey Auditory Verbal Learning Test (Spreen & Strauss, 1998), a set of questions assessing subjective memory, the orientation section of the Hindi Mental Status Examination (Pandav, Fillenbaum, Ratcliff, Dodge, & Ganguli, 2002), a culturally modified version of Squire's Autobiographic Memory Questionnaire (Squire, Slater, &

Miller, 1981), the Digit Symbol Substitution Test (Wechsler, 1981), Visual Memory Test (Strub & Black, 2000) and Letter Number Sequencing (Wechsler, 1997).

Another recently published article examined the utility of a screening battery to predict ECT cognitive change which happens later in a course of treatment (Martin et al., 2013). The battery comprised of MMSE, the first and second recall items of the Modified Mental State Examination (3MSE; Teng, 1987), and several mental control items such as counting backwards from 20, saying the alphabet (forwards), counting forwards in 3s starting with 1 ending after 14 responses, saying the days of the week backwards, starting with 'Sunday', and saying the months of the year backwards starting with 'December'. The items found to be most predictive of later changes in anterograde memory were: counting backwards from 20, and reciting the months of the year backwards (Martin et al., 2013).

Some common themes emerge from the national, international and academic guidelines outlined above. These include the recommendation of frequent and ongoing monitoring of a patient's cognitive functioning; a baseline assessment of cognitive functioning prior to commencing electroconvulsive treatment is beneficial to obtain a benchmark for cognitive change; the MMSE is the most commonly recommended cognitive screen; in addition to objective assessment of memory a report of subjective memory function should also be obtained; and a patient's clinical state should be assessed alongside their cognitive function. An apparent dearth within these recommendations is the allocation of responsibility for ensuring the cognitive assessments are conducted, except for ECTAS who explicitly state that it is the onus is on the referring psychiatrist.

Which Measures are used to assess Cognition during ECT?

A recent literature review of 28 studies assessing cognitive and clinical outcomes of ECT published between 1995 and 2011 revealed a large variation of measures were used to assess these outcomes (Luther, 2012). The review revealed that mood was most commonly assessed using the Hamilton

Depression Rating Scale (HDRS; Hamilton, 1960), followed by the Beck Depression Inventories (BDI; Beck & Steer, 1987; BDI II; Beck, Steer, & Brown, 1996) and the Montgomery Asberg Depression Rating Scale (MADRS; Montgomery & Asberg, 1979). The Mini Mental State Examination (MMSE; Folstein et al., 1975) was the most commonly used measure of global cognitive functioning followed by the Modified Mini Mental State Examination (3MSE; Teng, 1987). To assess verbal learning and memory word lists were popular and complex figures were often used to assess visual learning and memory. This review was based on the published peer-reviewed literature, thus the measures used within these studies do not necessarily reflect what measures are used in everyday practice (see Chapter Four for a review of measures used in hospital settings in New Zealand). The review revealed that no Gold Standard measure of assessment exists for use during ECT.

The MMSE has received considerable attention in the ECT literature with some few authors reporting on the measure favourably (e.g. Robertson & Pryor, 2006), and others reporting that the measure is inadequate for the assessment of ECT related cognitive change (e.g., Robertson & Pryor, 2006). Changes in global cognitive state measured by the MMSE do not to correlate well with the degree of anterograde or retrograde amnesia subsequent to ECT, with the potential for patients to show unchanged MMSE scores despite having considerable amnesia (Robertson & Pryor, 2006). A further limitation of the MMSE is that it has a ceiling effect and is therefore insensitive to early or subtle evidence of cognitive impairment, especially for high functioning individuals (de Jager, Milwain, & Budge, 2002). As the MMSE is insensitive for detecting memory difficulties, more sensitive measures of both anterograde and retrograde memory should be adopted when assessing memory impairment from ECT (Fox, 2001; Sobin et al., 1995).

In summary, the benefits of regularly assessing cognitive function are evident, and frequent assessment is recommended during a course of ECT. The MMSE is commonly used to assess cognitive change during ECT despite many studies demonstrating the unsuitability of the measure

in detecting ECT related cognitive decline. Guidelines around how and when to assess cognition during the course of ECT do exist, but must be searched for rigorously within handbooks for ECT practitioners, clinical guidelines for the administration of ECT and within the published literature. Despite the existence of these recommendations, thorough and frequent assessments are said to be rare and unsystematic (Porter, Douglas, et al., 2008). The extent to which this is factual in New Zealand is unclear as current practice around cognitive assessment during ECT to the best of our knowledge has not yet been evaluated in Aotearoa, New Zealand. The following chapter details a study into what practitioners and health professionals around New Zealand are doing to assess and monitor cognition for patients undergoing a course of ECT in New Zealand. The study is an attempt to address the dearth in the literature, as there appears to be an absence of studies investigating current practice of cognitive assessment during ECT, and also explores variables such as; who conduct the cognitive assessments, the frequency and timing of assessments and what factors prevent more frequent and thorough assessments.

CHAPTER FOUR

Cognitive Assessment during a Course of Electroconvulsive Therapy - A National Questionnaire Survey of Current Practice in Aotearoa, New Zealand

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Abstract

The objective of the current study was to shed light on current practice regarding cognitive assessment during electroconvulsive therapy (ECT) across Aotearoa. Twenty four medical professionals representing all ECT administering district health boards took part in the study by responding to questionnaire. The findings revealed that cognitive function was assessed at least once during a course of ECT by 73.7% of respondents. 27.3% conducted an assessment at baseline, at least once during the course and again post-treatment. Assessments were primarily conducted by nurses (38.8%), psychiatrists (22.2%) and psychologists (22.2%). It was reported by 66% of respondents that cognitive assessments were not conducted frequently or thoroughly enough in their workplace due to a lack of time, resources and sensitive tests. In conclusion, respondents recognised assessing cognitive change during a course of ECT was important, though large variations in the nature, frequency and length of assessments existed. Future research should focus on the development of a sensitive screening measure tailored for use with patients receiving ECT to help overcome the current restrictions to cognitive assessment.

Keywords: cognitive assessment, current practice, Electroconvulsive therapy,

Cognitive Assessment during a Course of Electroconvulsive Therapy - A National Questionnaire Survey of Current Practice in Aotearoa, New Zealand

Electroconvulsive therapy (ECT) is an effective treatment for a variety of psychiatric disorders (Mankad et al., 2010). It is fast acting and often effective when all other treatments have failed. Up to 50-60% of people who are non-responsive to medication will show clinical improvement from ECT (Prudic et al., 1996). Despite high treatment efficacy, ECT is only prescribed in New Zealand, as in other parts of the western world, under strict conditions (Ministry of Health, 2004). Central to these restrictions are ongoing reports that ECT may cause cognitive impairment (Ingram et al., 2008; Nehra et al., 2007). In addition, cognitive side effects limit the use of ECT by diminishing patient satisfaction and contributing to the stigma associated with the treatment (Prudic, 2008). Cognitive assessment during the treatment course is recommended in order to detect and monitor cognitive change (Nehra et al., 2007).

The most recent statistics on the number of patients receiving ECT in New Zealand are from 2011, and reveal 286 patients received ECT during this year, 6.5 people per 100,000 (Ministry of Health, 2012). In New Zealand no regulations exist which oblige treating professionals to monitor or assess cognitive functioning. As there are no enforced guidelines put in place to assess cognitive function it is unclear what practitioners are doing to assess cognitive function and whether or not practitioners have sufficient resources to do so. The aim of the current study is to shed some light onto what practitioners around the nation are doing at assess cognitive change during a course of ECT.

Recommendations and Guidelines for ECT Cognitive Assessment

Although there are no strict guidelines around how cognition should be assessed in New Zealand, various national and international organisations have recommended a patient's cognitive

functioning is monitored intermittently throughout ECT. See for example, The ECT Accreditation Service (The Royal College of Psychiatrists Centre for Quality Improvement, 2011), the National Institute of Clinical Excellence (National Institute of Clinical Excellence, 2003), the American Psychiatric Association (American Psychiatric Association, 2001), and the Royal Australian and New Zealand College of Psychiatrists (Royal Australian and New Zealand College of Psychiatrists, 1999).

Suggestions for assessment schedules and batteries also exist within the peer-reviewed literature. Porter and colleagues (2008), offer number of useful recommendations: a) to conduct a baseline assessment, reassess early in treatment, and again after the sixth treatment; b) to carry out assessments at a standard time after treatment which should be at least 48 hours post treatment to allow for any transient treatment effects to resolve; c) repeat the same battery 2-3 months post treatment; d) and to include a mood measure alongside the cognitive assessment as mood affects cognitive performance. Porter and colleagues proposed a 55 minute test battery including the Mini Mental Status Examination MMSE (MMSE; Folstein et al., 1975) or the Modified Mini Mental State Examination (3MSE; Teng, 1987), Hopkins Verbal Learning Test (HVLT; Brandt, 1991), Autobiographical Memory Questionnaire- Short Form (AMI-SF; Kopelman et al., 1989) and the Digit-symbol Substitution Task (DSST; Wechsler, 1997). A brief cognitive battery has recently been suggested by Viswanath et al., (2013) which offers an ECT battery appropriate for use in developing countries where the number of patients receiving ECT per day is high (10-15) and resourcing is low. The battery is short (20-30 minutes) and is culturally adapted for use with Eastern cultures.

Within the national, international and academic guidelines on assessment, some common themes emerge: a) the need for frequent and ongoing monitoring of a patient's cognitive functioning; b) the importance of a baseline assessment of cognitive functioning prior to commencing ECT to obtain a benchmark for cognitive change; c) a report of subjective memory function should also be obtained,

and d) a patient's clinical state should be assessed alongside their cognitive function. The recommendations are not clear regarding where the responsibility for doing the cognitive assessments lies (except for ECTAS who explicitly state that the onus is on the referring psychiatrist). Another common trend is the inclusion of the MMSE in guidelines, recommendations and suggested batteries, despite research suggesting that short cognitive screening measures such as the MMSE are problematic as they are insensitive in detecting ECT related cognitive change (Robertson & Pryor, 2006).

Benefits of Cognitive Assessment during Electroconvulsive Therapy

Since the introduction of ECT in 1938, efforts have been made to refine the ECT administration technique to increase clinical efficacy and reduce the cognitive side effects of the treatment (Abrams, 2002). Despite these efforts, cognitive impairment remains a common and undesirable side effect (Ingram et al., 2008). Patients can experience difficulties with the speed in which they are able to process information, their ability to sustain attention, to plan, organise and mentally shift between tasks, their visuospatial skills can become impaired as can general intellect (Ingram et al., 2008). The most severe, well researched and distressing cognitive side effect of ECT, however, is its negative impact on memory (Sackeim et al., 2007; Sienaert, Vansteelandt, Demyttenaere, Peuskens, 2010; Sobin et al., 1995).

Switching from sine-wave to brief-pulse electrical stimulation was one refinement to ECT which relieved the severity of cognitive impairment (Weiner et al., 1990). Since this change, research concludes that cognitive dysfunction is less severe and mostly limited to the first three days post treatment. After 15 days most dysfunction should have resolved (Semkowska & McLoughlin, 2010). Descriptive reviews agree that six months post treatment, all ECT related cognitive dysfunction should have resolved (Calev, 1994; Ingram et al., 2008). If this is the case, then why should medical professionals bother spending valuable time and resources assessing cognition? The motivation to do so derives from the fact that some patients report significant gaps in their memory

years after treatment (Rose et al., 2003). Monitoring a patient's cognitive functioning throughout their course of ECT allows for the detection of impairment early on in treatment, and cognitive impairment early on in treatment may pose as a risk factor for continual cognitive impairment as the treatment course progresses (Porter, Douglas, et al., 2008).

If impairment can be identified, parameters of ECT administration can be altered to reduce impairment, or if necessary, the treatment course can be suspended (Scott, 2010) or terminated (Porter, Douglas, et al., 2008). Modifications which are well documented to reduce cognitive impairment include: changing from bilateral to unilateral ECT, decreasing intensity of electrical stimulation, spacing of treatments from more to less frequent and altering dosages of medications and anaesthetics where possible (Scott, 2004). Treatment planning should aim to maximise clinical efficacy while minimising adverse cognitive side effects.

In addition to these practical and ethical arguments for regularly assessing cognition, documented evidence of careful and frequent cognitive monitoring will provide objective evidence that changes in cognition were taken seriously in the case of litigation (Porter, Douglas, et al., 2008). Although conducting regular cognitive assessments is time consuming and potentially costly, monitoring cognitive change is beneficial for the patient and can assist in guiding optimal treatment schedules and ultimately result in fewer memory and cognitive complaints.

In summary, the benefits of regularly assessing cognitive function are evident. Guidelines around how and when to assess cognition during the course of ECT do exist, but thorough and frequent assessments are said to be rare (Porter, Douglas, et al., 2008). Current practice around cognitive assessment during ECT has not yet been evaluated in Aotearoa, New Zealand. The aim of this study is to investigate what medical professionals are doing to assess cognition for patients undergoing a course of ECT in New Zealand. The frequency and length of assessments, domains of cognition assessed and measures used will be described. We consider who is conducting the assessments, and

what barriers if any, limit more frequent or thorough assessments from occurring in New Zealand hospitals.

Method

Procedure and Questionnaire Design

Participants were recruited via an email sent out to a National ECT treatment staff email list which included a link to the electronic questionnaire and information introducing the questionnaire (refer to Appendix A for the questionnaire). Participants were also contacted via a national annual ECT meeting. Forty-five individuals were contacted on this mail list, and 18 completed the questionnaire. The questionnaire was then sent out to a further 8 people to ensure coverage across all ECT administering DHBs was achieved. The questionnaire took approximately 10 minutes to complete and explored the following areas: measures in place for assessing cognition, whether a measure of clinical state is included within the assessment, who is responsible for conducting the assessments, timing of assessments, frequency of assessments, and whether, in the opinion of the respondent, patients' cognitive functioning was assessed frequently enough, and if not, what restricted the occurrence of more frequent or thorough cognitive assessments. The results of the questionnaire remained anonymous. The responses to the survey were collected from October 2012 until June 2013. The survey was generated using Qualtrics.TM

Participants

Respondents were psychiatrists, nurses, and psychologists across ECT administering district health boards (DHBs) throughout New Zealand. Of the 20 DHBs in New Zealand, ECT was administered at 15 at the time this survey was conducted and at least one response was received from each ECT administering DHB in New Zealand. A total of 24 completed questionnaires were received.

Results

The data were analysed using Statistical Package for Social Sciences (SPSS) Version 19.0. One DHB has a data analyst responsible for conducting all cognitive assessments; the three responses received from this DHB were treated as one response as they all reported answers based on a common system of assessment.

How frequently is Cognition Assessed?

Most respondents (73.7%) reported that some form of cognitive assessment is conducted during a course of ECT. Of these 27.3% conduct an assessment prior to ECT, at least once during the course and again after the course. Baseline cognitive assessment is routinely conducted by 45.5% and half conduct an assessment post treatment. A small proportion will conduct an assessment only if the patient complains of memory impairment post ECT (4.5%). Two thirds of respondents stated that assessment of cognitive functioning is currently not being carried out frequently *enough* due to: lack of time (100%), resources (50%), and a lack of suitable screening measures sensitive to ECT related cognitive impairment (41.6%).

Which Assessment Measures are being utilised?

As illustrated in Figure 1 the most frequently used cognitive assessment measure is the MMSE (81.25%). Also popular is the Montreal Cognitive Examination (37.5%) (MoCA; Nasreddine et al., 2005) and the Addenbrooke's Cognitive Examination-Revised (31.25) (ACE-R; Mioshi, Dawson, & Mitchell, 2006). Some respondents reported using the measures suggested by Porter et al. (2008) which includes the HVLT, AMI-SF, DSST in addition to the MMSE or the 3MSE (12.5%). Over a third of respondents (37.5%) use more than one measure to assess cognitive functioning.

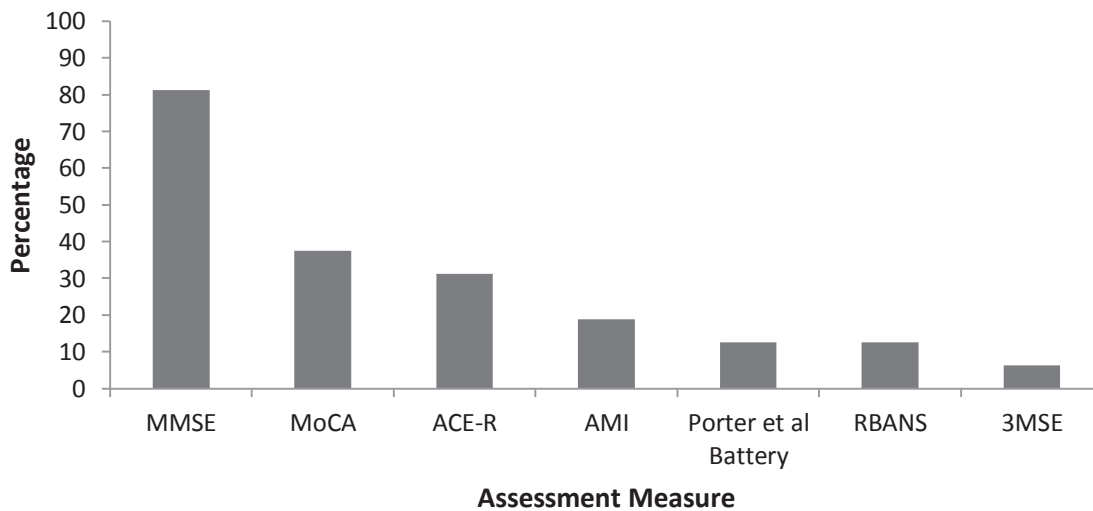


Figure 1. Frequency of assessment tools used to assess cognitive functioning.

Assessing Clinical State during ECT

Most practitioners conduct a mood assessment alongside the cognitive assessment (80.95%). The most commonly utilised assessment measure is the Montgomery Asberg Depression Rating Scale (MADRS; Montgomery & Asberg, 1979) (52.38%), less commonly utilised is the Beck Depression Inventory (BDI-II; Beck et al., 1996) (9.52%) and the Geriatric Depression Scale (GDS; Yesavage et al., 1982) (9.52%). Even less commonly utilised are the Hamilton Depression Rating Scales (HDRS; Hamilton, 1960) and the Hospital Anxiety and Depression Scales (HADS; Zigmond & Snaith, 1983). Many practitioners (38.10%) also assess anxiety and psychosis as well as mood alongside the cognitive assessment.

Who Conducts the Assessment?

As shown in Figure 2, most (38.88%) of the cognitive assessments are conducted by nurses, though, many are conducted by psychiatrists (22.22%) and clinical psychologists (22.22%). A minority of assessments are conducted by junior doctors/ registered medical doctors (11.11) and data analysts (5.55%).

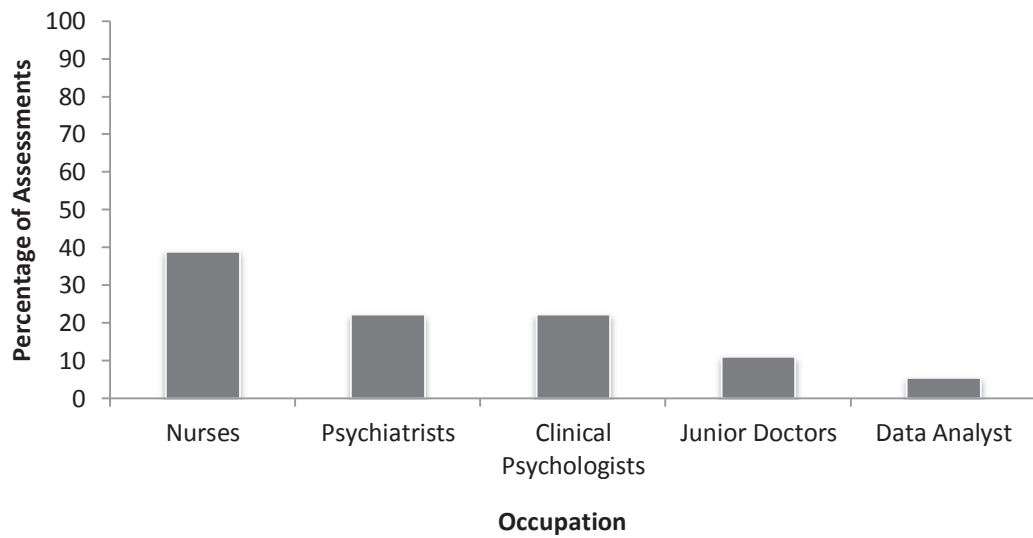


Figure 2. Professional groups involved in assessing cognitive functioning during ECT.

How Long is Spent Conducting the Cognitive Assessment?

The average reported time spent conducting cognitive assessments with patients was 23 minutes, with large variations between respondents (SD=16.8 minutes). One third of respondents reported spending 30 minutes to one hour conducting the assessment. On average respondents reported that 17 minutes (SD=8.26) would be feasible for an ideal cognitive screen to take.

Cognitive assessments are generally being conducted 24 hours post treatment (42.86%), although some conducted assessments one to five hours (21.43%), 48 hours (28.57%) and a few days to one week post treatment (7.14%). The delays reported between ECT and assessments are depicted in Figure 3.

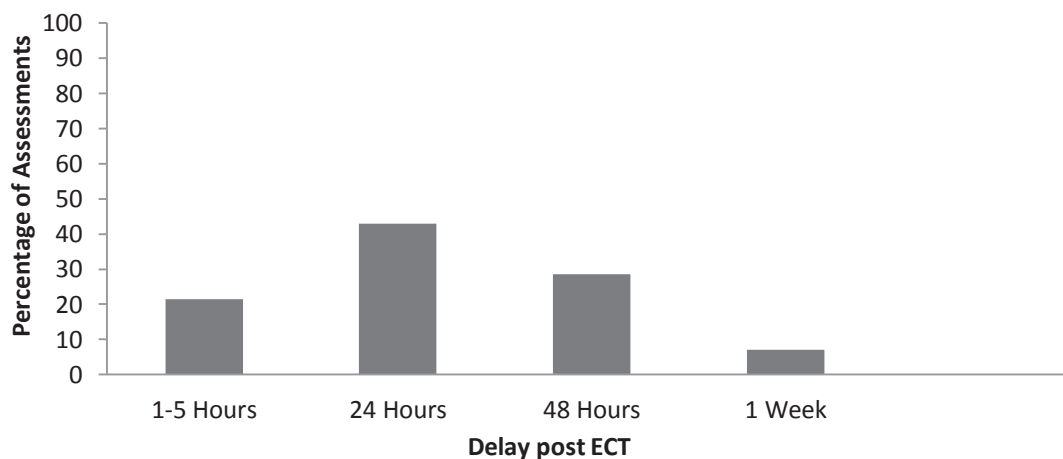


Figure 3. Reported time delays between ECT and cognitive assessment.

Discussion

Medical professionals in this sample recognise that cognitive assessment is an integral component of treatment with ECT. Most respondents reported that a cognitive assessment is conducted at least once during their course of ECT. Almost one third of the respondents conducted some form of cognitive assessment pre and post treatment and at least once during the course. Most of the cognitive assessments are augmented with a mood assessment; the MADRS is the most commonly used tool for this. Timing of the assessments varied; however, most are conducted at least 24 hours post treatment. The time spent conducting the assessment is often brief, around 10-20 minutes. In New Zealand, cognitive assessments are being conducted by nurses, psychologists, psychiatrists, doctors and data analysts. Monitoring of cognition is hampered by lack of time, resources and appropriate sensitive measures of cognitive change. Some respondents have adopted Porter et al.'s (2008) recommended battery of tests, but a lack of time and resources restrict many from carrying out this 55 minute long assessment. The MMSE was the most commonly utilised measure of cognitive functioning. The MMSE is often recommended within the ECT guidelines around

cognitive assessment and is a popular brief cognitive screening tool in New Zealand (Strauss, Leatham, Humphries, & Podd, 2012).

Due to the insensitivity of current measures being used to assess cognitive function during ECT, we argue that there is a need for the development of a new cognitive screening measure. The cognitive profile of ECT is different to that of dementia or mild cognitive decline, therefore the use of screening measures such as the MMSE designed to detect dementia related cognitive impairment are inadequate in detecting ECT related memory change. The assessment of ECT related cognitive change warrants its own specialised sensitive screen.

The results of the current study suggest that an ECT cognitive screen would need to take fewer than 20 minutes to administer, as time was the largest factor preventing cognitive assessment. The measure would need to be inexpensive and be sensitive to detecting ECT related cognitive change and have sound psychometric properties. As it is optimal that cognition is reassessed throughout a course of ECT, a screening measure with alternate forms would prevent practice effects. As assessments are being carried out by a wide range of professions, the assessment instrument would need to be easy to administer and score and require minimal training.

As a screening measure will take time to develop and validate, in the interim the Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005) could be utilised as an alternative to the MMSE. The MoCA may be more sensitive than the MMSE when assessing the long term cognitive effects of ECT (Luther, 2012). The MoCa is a one page 30 point test which takes approximately 10 minutes to administer. It assesses short term memory, visuospatial abilities, executive functioning, attention, abstraction, orientation, concentration, working memory, language, short term memory recall and delayed recall after approximately five minutes. The MoCA has superior sensitivity (90%) and specificity (87%) for detecting MCI, compared with 18% and 100% respectively for the

MMSE (Nasreddine et al., 2005). In addition, it is freely available in the public domain and has three alternate forms which make it suitable for reassessment.

Although the MoCA has been shown to be more sensitive to cognitive change during ECT than the MMSE (Luther, 2012), like the MMSE, it was designed to detect dementia related mild cognitive impairment, not ECT related cognitive change. A screening measure should not be used as a direct proxy to more sophisticated assessments such as Porter et al.'s (2008) suggested battery; however, when only a short time frame is permitted, the use of the MoCA has been shown to be superior to the MMSE and certainly to an absence of cognitive assessment (Luther, 2012).

There are some limitations to the current study. As completion of the questionnaire was voluntary there may have been a response bias such that the reported frequency of cognitive assessments may be inflated and the numbers of people not conducting cognitive assessments may be higher than reported. In addition, the response rate was low. Although at least one response was received from all of the district health boards which perform ECT, this does not capture likely intra-workplace variability. Despite the low response rate, the main objective of the current study was to provide insight into what some professionals are doing to monitor cognitive function, which previously remained largely unknown for New Zealand.

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STATEMENT OF CONTRIBUTION
TO DOCTORAL THESIS CONTAINING PUBLICATIONS

(To appear at the end of each thesis chapter/section/appendix submitted as an article/paper or collected as an appendix at the end of the thesis)

We, the candidate and the candidate's Principal Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of Candidate: Anneke Thornton

Name/Title of Principal Supervisor: Janet Leatham

Name of Published Research Output and full reference:

Cognitive Assessment during a Course of Electroconvulsive Therapy - A National Questionnaire Survey of Current Practice In Aotearoa, New Zealand

In which Chapter is the Published Work: Four

Please indicate either:

- The percentage of the Published Work that was contributed by the candidate:
and / or
- Describe the contribution that the candidate has made to the Published Work:

The candidate had full responsibility for the design, data collection, analysis and write up of the research. Supervisors provided guidance and were involved in decisions made around research processes and provided input with data analysis and formatting of the thesis. For these reasons Professor Janet Leatham and Dr Ross Flett were included as co-authors for the manuscripts which comprise this thesis.

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CHAPTER FIVE

Selecting and Refining the Assessment Measures for the Investigation into the Short Term Cognitive Effects of Electroconvulsive Therapy

To assess the cognitive changes which occurred during a course of ECT in Study Three (Chapter Seven), a carefully selected battery of tests was first assembled. The current chapter outlines the steps taken to develop this battery and considerations made when designing the assessment. Information about the tests and the rationale for the tests chosen are also offered within this chapter.

Prior to designing the assessment battery a number of considerations were made. These considerations arose from reviewing the current guidelines around cognitive assessment during ECT and current recommendations for testing within the peer reviewed literature (see Chapter Three) and consultation with a clinical psychologist who had experience conducting cognitive assessments with this population.

Considerations

To maximise sensitivity of the assessment, it was essential to assess the domains of functioning most commonly impaired during ECT, with an emphasis on the assessment of memory. Also important was how sensitive a measure would be at indicating who is likely to get cognitive disturbance long term, i.e. how well a measure would differentiate an individual who was at risk of developing cognitive impairment. A list of these domains can be found in Table 1 in Chapter Two.

- A. As the population under investigation would be unwell, it was aimed that the testing time would not exceed 50 minutes. This was to prevent fatigue, concentration, motivation loss and undue discomfort for the participant. Thus time taken to administer each measure was also taken into consideration.

- B. As the assessments were to be repeated over the duration of the course of ECT, tests with alternate forms were given precedence over tests without alternate forms.
- C. Depending on the reception of the tests from the participants and the sensitivity of the tests to detect cognitive change, the tests administered in the current study could potentially form the foundation of the screening measure to be suggested in Chapter Nine.
- D. As the tests could potentially form the basis of a yet to be developed screening measure, tests which were simple to score and straightforward to administer were considered for the assessment.
- E. Selected tests needed to be psychometrically sound, valid, and reliable.

To determine which cognitive constructs to assess, the domains of cognition most commonly associated with impairment or change during ECT were determined. These domains were based on the literature review of the cognitive side effects of ECT described in Chapter Three, which included a recent review of cognitive outcomes due to ECT conducted by Luther (2012). This review suggested that the domains of functioning most commonly impaired by electroconvulsive therapy are: retrograde amnesia (for declarative memory including semantic, episodic and autobiographical memory), anterograde amnesia (impairment in learning new information and memory of events since ECT), executive functioning, psychomotor speed, visual processing speed, attention, working memory and information processing speed. Memory remained the most commonly impaired and assessed domain during and post ECT treatment (Luther, 2012). This review's findings are congruent with a recent meta-analysis of cognitive outcomes of ECT (Semkovska & McLoughlin, 2010).

Selecting Measures

A number of cognitive tests and self rating scales were reviewed for suitability for inclusion in the battery for cognitive assessment with ECT patients. Below is an overview of some of the tests commonly used in ECT research and were considered for the inclusion for use in the current study.

Tests are divided into domains of functioning, starting with assessments of global cognitive impairment, and following with assessments of retrograde amnesia, anterograde amnesia, psychomotor speed and mood.

Tests of Cognitive Function

Global Cognitive Function

There is a lack of consensus as to which global cognitive screening measure is best suited to assess cognition during a course of ECT. Commonly used global cognitive screens include the Mini Mental Status Examination (MMSE: Folstein et al., 1975), the Modified Mini Mental Status Examination (3MS: Teng, 1987), Addenbrooke's Cognitive Examination – Revised (ACE-R: Mathuranath, Nestor, Berrios, Rakowicz, & Hodges, 2000) and the Montreal Cognitive Assessment (MoCa: Nasreddine et al., 2005). However, a brief global cognitive screen alone is insufficient to detect changes and monitor cognition during ECT (Robertson & Pryor, 2006).

Mini Mental Status Examination (MMSE).

The MMSE (Folstein et al., 1975) is a brief 30 point questionnaire that is used to test for cognitive impairment. Its most common use is to screen for dementia; however, it has been frequently utilised in ECT research due to its simplicity, validity and ease of administration (Nehra et al., 2007). The MMSE takes five to 10 minutes to administer and assesses for orientation to time, orientation to place, registration, attention and calculation, recall, language, and spatial ability.

The MMSE has been subjected to criticism due to its insensitivity in detecting memory impairments during ECT (Nehra et al., 2007). A further criticism is that it is based almost entirely on verbal assessment of memory and attention (Bak & Mioshi, 2007). It is insensitive to frontal executive dysfunction and visuospatial deficits. Furthermore the assessment of memory and language is superficial and it does not provide any qualitative information (Bak & Mioshi, 2007). The MMSE does not measure semantic memory and changes in global cognitive status measured by the MMSE

do not correlate well with the degree of anterograde or retrograde amnesia subsequent to ECT, with the potential of patients showing unchanged MMSE scores despite having considerable amnesia (Robertson & Pryor, 2006). Robertson and Pryor conclude that the MMSE lacks the sensitivity needed to detect memory dysfunction caused by ECT. A further limitation of the MMSE is it has a ceiling effect and is therefore insensitive to early or subtle evidence of cognitive impairment, especially for high functioning individuals (de Jager et al., 2002).

Conversely, a study by Nehra et al. (2007) found that MMSE scores correlated highly with memory impairment prior to, during and up to one month after ECT. These authors concluded that the MMSE may in fact reliably detect memory impairment during treatment and that pre-ECT scores could be used to screen for patients who are liable to develop more severe memory impairment during treatment (Nehra et al., 2007).

The Modified Mental Status Examination (3MS).

The 3MS was developed as a response to criticisms of the MMSE. Ten & Chui (1987) took the original MMSE and added four additional subtests to improve the scope of the MMSE assessment. These additions are: date and place of birth (semantic memory), word fluency (executive functioning), similarities (language) and delayed recall of words (long term memory). The maximum score was increased from 30 points to 100 points and a modified scoring procedure permitted partial credit on some items (Tombaugh, McDowell, Kristjansson, & Hubley, 1996). These modifications enable the test to cover a wider range of cognitive functions and enhanced the reliability and validity of scores (Tombaugh et al., 1996). The 3MS takes slightly longer than the MMSE to administer (Cullen, O'Neil, Evans, Coen, & Lawlor, 2007). Sobin et al. (1995) used the 3MS before treatment to assess pre-ECT global cognitive impairment. Using the 3MSE the authors successfully predicted the magnitude of retrograde amnesia for autobiographical information in the week after the course of ECT and at a two month follow-up.

Addenbrooke's Cognitive Examination – Revised (ACE-R).

The Addenbrooke's Cognitive Examination-Revised (ACE-R; Mioshi et al., 2006) is another test of global cognitive function and takes around 15 minutes to administer. The ACE-R consists of 26 tasks, divided into five domains: attention and orientation, memory, verbal fluency, language and visuospatial skills (Bak & Mioshi, 2007). The attention section assesses for orientation in time and space, registration of three words and serial subtraction or backwards spelling. The memory section assesses encoding information recalled after a 10 minute delay, making it more sensitive to the MMSE and the 3MS to mild cognitive impairment (MCI) (Bak & Mioshi, 2007). It assesses for recall and recognition memory. It also includes four general knowledge questions to test semantic memory. The verbal fluency component requires the patient to produce as many different words as possible in a minute according to specified rules. Language is assessed by requiring the patient to repeat single words and phrases and reading five irregular words. Finally, visuospatial ability is assessed in two parts: drawing overlapping pentagons (derived from the MMSE) and drawing a cube and a clock. The second part consists of counting dots and naming four incomplete letters. The ACE-R provides age and education norms for the total score and for scores on the individual subtests (Mioshi et al., 2006). The ACE-R has been less commonly used than the MMSE in ECT research.

Montreal Cognitive Assessment (MoCA).

Finally, the Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005) was developed as a screening tool to detect MCI. The MoCa is a widely used global cognitive screening tool which resembles the MMSE and has commonly been used in ECT literature. The MoCa is a one page 30 point test which takes approximately 10 minutes to administer. The MoCa assesses short term memory, visuospatial abilities, executive functioning, attention, abstraction, orientation,

concentration, working memory, language, short term memory recall and delayed recall after approximately five minutes.

Gill, Freshman, Blender and Ravina (2008) compared the MMSE and the MoCa as screening tools to assess cognitive dysfunction in patients with Parkinson's Disease. These authors found the MoCa to be more sensitive in detecting cognitive dysfunction than the MMSE in discriminating early and mild cognitive dysfunction. The MMSE exhibited a ceiling effect, whereas the MoCA did not. This is possibly due to the fact the MoCA's memory testing involves more words and a longer delay than the MMSE, and the MoCA assesses domains affected early in Parkinson's disease such as executive function and visuospatial function which the MMSE does not (Gill et al., 2008). Two further studies comparing the MoCA and the MMSE have been conducted with patients with Parkinson's disease, the MoCA identified cognitive deficits in these patients; however, the MMSE did not detect any cognitive dysfunction (Mamikonyan, Moberg, Siderowf, Duda, & Have, 2009; Nazem, Siderowf, Duda, Have, & Colcha, 2009). Furthermore, studies within other populations comparing the MoCa and the MMSE have found the MoCA to be more sensitive to cognitive dysfunction, for example in patients after a transient ischemic attack and stroke (Pendlebury, Cuthbertson, Welch, Mehta, & Rothwell, 2010). More recently, the MoCa has also been found to be more sensitive than the MMSE when assessing the long term cognitive effects of ECT on cognition (Luther, 2012).

The MoCA has good sensitivity (90%) and specificity (87%) for detecting MCI, compared with 18% and 100% respectively for the MMSE (Nasreddine et al., 2005). In addition to excellent psychometric properties, the MoCa is available free in three alternate forms to combat the confound effect of practice.

On this basis, the MoCA was selected as to assess global cognitive assessment.

Subjective Memory

It has been commonly reported that patients' self-reports of memory impairment are incongruent with objective measures of memory impairment (National Institute for Clinical Excellence, 2003), hence the importance of obtaining a combination of subjective and objective assessments of memory. Assessing subjective memory is also a good way to establish the patients' perceptions of cognitive function post ECT (Coleman et al., 1996).

Squire Subjective Memory Questionnaire.

The Squire Subjective Memory Questionnaire (SSMQ; Squire, Wetzel, & Slater, 1979) is the most common measure used to assess subjective memory complaints from ECT (Coleman et al., 1996). The SSMQ takes around 20 minutes to administer and was originally developed to differentiate memory complaints related to depression before ECT from memory complaints associated with amnesia after ECT (Robertson & Pryor, 2006; van Bergen, Brands, Jelicic, & Merckelbach, 2010). The SSMQ includes 18 items that are rated on nine-point likert scales, with answer options ranging from worse than ever before (-4) to better than ever before (+4). Squire et al. (1979) suggested that nine of the items are sensitive to the amnesic effects of ECT; the other nine are sensitive to mood state. The SSMQ has been found to have adequate reliability and good construct validity (van Bergen et al., 2010).

Cognitive Failures Questionnaire.

The Cognitive Failures Questionnaire (CFQ; Broadbent et al., 1982) is a 25 item assessment measure of self reported failures in perception, memory and motor function (Broadbent et al., 1982). Although the CFQ has been used in ECT literature little attention is given to retrograde amnesia for events in the recent past which is typically the most persistent and severe deficit after ECT, (Berman, Prudic, Brakemeier, Olfson, & Sackeim, 2008).

Subjective Report.

An additional way to assess subjective memory difficulties is simply to enquire with the patient about their current cognitive status and any changes they have noticed (negative or positive) since receiving ECT.

On this basis the SSMQ and Subjective Report were elected for assessing subjective reports of global cognitive change:

Retrograde Amnesia

Autobiographical Memory.

Temporally graded retrograde amnesia for personally relevant autobiographical information is common post ECT (Squire et al., 1981; Weiner et al., 1986) and is distressing for individuals who receive ECT (Scott, 2010). Therefore, autobiographical memory should always be monitored in patients who undergo ECT, specifically for events that have occurred 6 weeks prior to ECT treatment.

Autobiographical Memory Interview.

The Autobiographical Memory Interview (AMI; Kopelman et al., 1989) is the most commonly used assessment measure in the ECT literature to assess autobiographical memory. There is a long and short form of this test. Both assess retrograde amnesia, semantic memory and episodic memory and a temporal gradient in retrograde amnesia. The AMI- Short Form asks 30 questions about 5 different life events and takes around 10 minutes to administer. The long form is more thorough in that it asks 281 items about 9 events and takes 20-30 minutes to complete. The AMI has shown strong reliability and validity as a measure of retrograde amnesia and it is sensitive to variation in ECT technique (Lisanby et al., 2000; Sobin et al., 1995). An advantage of the AMI is that it does not need frequent updating as it is based on the individuals' personal semantic and episodic memory rather than current affairs and news events.

A major concern with the AMI is that the test has been found to be insensitive to ECT-induced amnesia (Robertson & Pryor, 2006) for two main reasons: it measures old information when ECT amnesia is densest for more recent memory; and around 60% of the items assessed in the AMI involve over-learned and highly rehearsed facts that are spared by the effects of ECT (such as grandparents names, and telephone numbers) (Robertson & Pryor, 2006). Amnesia induced by ECT shows a temporal gradient with memories close in time to the ECT treatment showing the greatest degree of amnesia (Weiner et al., 1986). To address these shortcomings and bearing in mind the restricted timing allowed for the assessment, only the last section of the AMI (section C) was selected to be administered to the participants. This section assesses memory for recent events such as: the hospital where the patient currently is, the location, address, staff members treating the patient, recent incidents, and recent events such as details about where the patient spent Christmas. This section, when administered in isolation from the long form takes approximately 5 minutes to administer.

On this basis, Section C of the Autobiographical Memory Interview was selected to assess autobiographical memory.

Anterograde Amnesia

Visual Learning and Memory.

Complex figures are a common way to assess visual learning and memory during a course of ECT (Luther, 2012). Essentially, complex figures involve requiring the participant to copy a complex geometrical figure and to reproduce the figure at a later time.

Rey-Osterrieth Complex Figure Test.

The Rey-Osterrieth Complex Figure Test, also known as the Rey Complex Figure Test (ROCFT/RCFT; Rey, 1941) is a popular measure of visuospatial constructional ability and visual memory skills and visual memory (Lezak, 1995). The RCFT is a well validated, often used

neuropsychological test (Meyers & Vollbrecht, 1999). The figure was initially derived to assess visuospatial constructional ability and visual memory in patients with brain injuries; however, it is presently used more broadly in neuropsychological settings (Lezak, 1995). An additional recognition trial has been developed by Meyers and Meyers (1995) which extends from the RCFT. Administering the RCFT involves a copy trial of the complex figure, a 3-minute immediate recall trial, a 30 minute delayed recall trial and a recognition trial which is administered directly after the delayed recall trial. The RCFT takes around 10 minutes to administer. The copy phase of the RCFT is a measure of visuospatial constructional ability, while the recall phase is a test of visual learning and memory. The recall phase evaluates the individual's ability to encode complex visual information into longer term storage and recall this information at a later time (Fernando, Chard, Butcher, & McKay, 2003).

Taylor Complex Figure (TCF).

A disadvantage of the Rey Complex Figure is the absence of parallel forms which poses the risk of practice effects with repeated administration. An alternative form to the RCFT was developed by Taylor (2010) which could be administered to an individual once he or she has already been exposed to the RCFT. Historically, the Taylor Complex Figure (TCF) was considered comparable to the RCFT and the figures were used interchangeably (Hubley, 2010). Research has since demonstrated that these two figures are not comparable and that the TCF is easier to learn and remember than the RCFT (Strauss & Spreen, 2006). There has since been a Modified Taylor Complex Figure (MTCF) developed which provides comparable accuracy scores to the RCFT (Hubley & Jassal, 2006). Yet the problem remains when using the RCFT and the MTCF that only two assessments are possible before patients will display a practice effect, therefore these tests are not suitable for repeated administration as is the case with assessment of cognition during a course of ECT.

Medical College of Georgia Complex Figures (MCGCF).

The Medical College of Georgia (MCG) Complex Figures (Ingram, Soukup, & Ingram, 1997) are four forms which can be used for repeated assessments. The MCG complex figures have a 36 point scoring system comparable to the RCFT. The four figures show equivalent performances on the initial copy and recall tests (Loring & Meador, 2003; Yamashita & Yasigo, 2008) and no practice effect is observed across the four conditions (Yasugi & Yamashita, 2010). Due to the four parallel forms and the sound psychometric properties of this test these forms of complex figures were selected for the current investigation.

The MCG figures were selected for the cognitive screen.

Verbal Learning and Memory

Rey Auditory Verbal Learning Test .

The Rey Auditory Verbal Learning Test (RAVLT; Schmidt, 2004) evaluates short term auditory verbal memory, rate of learning, learning strategies, retroactive and proactive interference, retention of information and differences between learning and retrieval. Participants are given a list of 15 unrelated words repeated over five different trials and are asked to repeat the list. Another list of 15 unrelated words are given and the client must again repeat the original list of 15 words and then again after 30 minutes. The RAVLT takes around 10-15 minutes to administer.

Verbal Learning and Memory- MoCA

Within the MoCA is a verbal learning and memory component. This assesses verbal learning and memory for five unrelated items after a short delay.

The Montreal Cognitive Assessment was used to assess verbal memory and an additional delayed recall phase was added on to assess delayed verbal memory.

Psychomotor Speed and Motor Speed

Psychomotor speed is a less commonly assessed domain; however, a test of psychomotor speed should be included in a cognitive assessment of a patient undergoing ECT as impairment is commonly found in this domain (Luther, 2012). Timed tests are sensitive compared to changes in cognition as a result of brain injury or psychopathology (Tulsky, Zhu & Ledbetter, cited in Porter et al. 2008). The most common method used to assess processing speed is to examine the number of accurate responses made in a fixed period (Porter, et al. 2008). Alternative to psychomotor speed, reaction time can be assessed which rules out effects of motor function and assesses purely speed of cognitive processes. Porter and colleagues go on to say that there is little evidence that psychomotor measures are consistently sensitive to the effects of ECT; however, they still recommend assessing for psychomotor speed.

Digit Symbol Substitution Test.

The digit symbol substitution test (DSST) was recommended in Porter's et al (2008) suggested battery of tests to assess for psychomotor speed. The DSST is a neuropsychological test sensitive to brain damage, dementia, age and depression. It consists of nine digit-symbol pairs. The participant is given a sheet with rows of digits, and for each digit, the corresponding code should be written as fast as possible. With the digit-symbol coding test, a subtest of the WAIS IV (Wechsler, 2008) the participant is allowed 120 seconds to complete as many digit-symbol pairs as possible.

Coding Task.

The coding task, which is found within the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS; Randolph, 1998) is similar to the DSST in that it assesses an individual's psychomotor and processing speed by requiring the person to code symbols into numbers as quickly as possible within a 90 second time frame.

Coin Rotation Task.

The coin-rotation task (CRT; Mendoza, Apostolos, Humphries, Hannah-Pladdy, & O'Bryant, 2010) is a measure of motor dexterity. The task requires the participant to rotate a coin 180 degrees using the thumb, index and middle fingers as quickly as possible for 20 rotations (Minor, Jones, Stewart, Hill, & Kulesza, 2010). The participant first does it with his/her dominant hand, followed by the non-dominant hand. It is a convenient, easily administered bedside test of motor dexterity and has demonstrated good convergent validity when compared with other standardised motor measures (Hill et al., 2010; Mendoza et al., 2010). Administration of the task takes approximately one minute (Minor et al., 2010). Sensitivity and specificity of the CRT is comparable with or better than other standardised tests of motor dexterity (Mendoza et al., 2010).

The RBANS Coding Task was selected to assess psychomotor speed and the Coin Rotation Task was selected to assess motor speed.

Mood

A patient's clinical state influences their objective cognitive performance (Porter, Douglas, et al., 2008) and their subjective report of cognitive function (Fraser, O'Carroll, & Ebmeier, 2008). Thus gauging the effect of depression on cognitive function is vital when determining what the cognitive effects of ECT are. There are three main methods which can be used to gauge the cognitive influence of depression when measuring the cognitive effects of electroconvulsive treatment. One is to closely assess and measure (objectively and/or subjectively) the severity of depression during a course of ECT to better understand the impact the illness is having on an individual's cognitive functioning. This enables the results of cognitive testing to be understood within the context of the patient's clinical state (Porter, Douglas, et al., 2008).

A second way to gauge the cognitive influence of depression during ECT is to obtain the patient's premorbid level of cognitive functioning and compare this against a baseline level of cognitive functioning before the patient begins their ECT treatment; however, a premorbid cognitive

assessment is very rarely available. The third and most rigorous way to determine the cause of cognitive dysfunction in patients receiving ECT would be to have a matched control group of patients who are suffering from the same degree of depression and therefore experiencing similar depression-related cognitive deficits, but not be receiving ECT. Any changes in cognitive functioning observed in the group receiving ECT that are not observed in the matched control group could be inferred to be a side effect of the treatment. This research design was not adopted for the current study for two main reasons: firstly, it would be unethical for a person suffering from severe depression to not be offered ECT as a treatment option, and secondly, it would be very difficult to match patients on all variables which can moderate cognitive outcome (See Chapter Two). The first method for gauging the effects of ECT was deemed the most appropriate method of controlling for the cognitive effects of depression for the current research.

Depression

Many different psychometric measures exist for the assessment of mood. The Hamilton Depression Rating Scale (HDRS; Hamilton, 1960) is the mood measure most commonly used to assess depression in ECT research (Luther, 2012), followed by the Beck Depression Inventory (BDI/BDI II; (Beck & Steer, 1987; Beck, Steer & Brown, 1996). The third most common mood assessment measure used in ECT research is the Montgomery Asberg Depression Rating Scale (MADRS; (Montgomery & Asberg, 1979). These three scales were considered for inclusion into the current battery.

Hamilton Depression Rating Scale (HDRS).

The Hamilton Depression Rating Scale also known as the Hamilton Rating Scale for Depression (HDRS/HRSD; Hamilton, 1960) is a 29 item multi-choice questionnaire to rate the severity of depression, more specifically, low mood, insomnia, agitation, anxiety and weight loss (Hedlund & Viewig, 1979). The HDRS takes around 20-30 minutes to administer. The HDRS has been the gold standard for the assessment of depression for over 40 years, but more recently it has come under a

lot of criticism around the psychometric strength of the measure (Bagby, Ryder, Schuller, & Marshall, 2004). A recent review of the measure suggested that the items require revision to redress the psychometric problems and thereby improve the measure, or better still rejection of the HDRS and replacement with an alternative measure has advantages over revision (Bagby et al., 2004). These authors recommend the MADRS as an alternative measure.

Montgomery–Åsberg Depression Rating Scale (MADRS).

The Montgomery–Åsberg Depression Rating Scale (MADRS; Montgomery & Asberg, 1979) is a ten-item diagnostic questionnaire used to measure the severity of depression and is often used in conjunction with the HDRS. There is, however, a high degree of statistical correlation between scores on the two measures (Heo, Murphy, & Meyers, 2007). The MADRS has a combination of subjective and objective items to assess clinical state and was designed to address the limitations of the HDRS (Bagby et al., 2004).

Beck Depression Inventory-II (BDI-II).

The Beck Depression Inventory- Second Edition (BDI-II; Beck et al., 1996) is the most often used self-rating instrument for depressive symptoms (Svanborg & Åsberg, 2001). The original Beck Depression Inventory (BDI; Beck & Steer, 1987) was developed from clinical observations about attitudes and symptoms frequently displayed by psychiatric patients (Beck et al., 1961). Beck et al. systematically consolidated these observations into 21 symptoms and attitudes which could be rated from 0 to 3 in terms of intensity. The BDI was designed to be administered by trained interviewers but it is most often self administered. The instrument generally takes 5 -10 minutes to complete and is simply scored by summing the ratings given to the 21 items.

Selected measure to assess for depression: Beck Depression Inventory- II. In the case of referrals of patients who are receiving ECT for the treatment of bipolar, the *Young Mania Rating Scale* will be used (Young, Biggs, Ziegler, & Meyer, 1978).

Final Selection of Measures

Table 2 displays the selected tests and how long each test is estimated to take to administer. Table 3 displays the domains of cognitive function assessed by the selected tests.

Table 2

Battery Selected for Assessment of Cognition and Mood for this Current Study

Assessment Measure	Time to Administer
Autobiographical Memory Interview (AMI) – (Section C Recent Life)	5 Minutes
Montreal Cognitive assessment (MoCA)*	10 Minutes
Medical College of Georgia Complex Figure (MCGCF)*	5 Minutes
RBANS Coding Task *	2 Minutes
Coin Rotation Task (CRT)	1 Minute
Subjective Report	5 Minutes
Squire Subjective Memory Questionnaire (SSMQ)	5 Minutes
Beck Depression Inventory (BDI-II)	10 Minutes
Recall of five words from MoCA*	1 Minute
Recall of Medical College of Georgia Complex Figure	5 Minutes
Total Time	49 Minutes**

Note. * The following cognitive tests have alternate forms which will be used on alternate assessments. **The administration time may vary, depending on severity of depression and number of previous assessments conducted with this battery.

Table 3

Domains of Cognitive Function Assessed by each Test

Domains Tested	Assessment Measure
Retrograde Amnesia	AMI
- Semantic, Episodic and Autobiographical	
Abstraction	MoCA
Anterograde Amnesia	MCGFG, MoCA
Short Term Memory Recall	MoCA
Delayed Memory Recall	MoCA
Executive Functioning	MoCA, MCGCF
Psychomotor Speed	Coding Task
Motor Speed	CRT
Visuospatial Ability	MoCA MCGCF
Attention/Concentration and Working Memory	MoCA, ROCFT
Information Processing Speed	RBANS coding task
Orientation	MoCA
Short Term Memory	MoCA
Mood	BDI
Language (Production and retrieval)	MoCA

Refining the Selected Measures and Procedure for use with Patients receiving Electroconvulsive Therapy

Some of the measures selected for the assessment needed adapting and refining to suit the assessment needs for the people in the current research. The remainder of this chapter will outline any changes made to measures and the rationale for the order of testing and the timing of testing.

Adapting the Assessment Measures

Squire Subjective Memory Questionnaire.

The 18 item measure of subjective memory was developed in 1979 (Squire et al., 1979) and since its development there have been no updated versions of this questionnaire. A visual analysis of the questionnaire items and the layout of the items evoked doubt about whether the measure would be suitable in its current form or whether it required modification, due to the length of the items and the way in which the items were worded. It was concluded that the SSMQ in its original form would not be suitable to administer to patients having ECT (refer to Appendix 1) for the adapted and original version of the SSMQ).

The items in the original questionnaire are lengthy, verbose and repetitious. For example, items 1, 3, 4, 6, 7, 8, 9, 10, 11, 12, 13, 14 and 18 all start with the words “My ability”. To reduce this repetition, the items were grouped according to headings determined by the beginning words of how each item appeared in the original questionnaire. The headings included: “my ability to recall...”, “My ability to remember” and “other”. Item 8, “My ability to remember the names and faces of people I meet is...” is a double barreled question, which essentially requires the participant to give one answer to two questions reflecting functioning of two separate cognitive domains. The ability to recall names requires function of semantic memory, and the ability to recall faces requires

function of visual memory. Therefore this question was altered to only reflect one's ability to recall faces, as the ability to recall names is already covered in item 1. "My ability to recall names is...."

Item 13 "My ability to know when the things I am paying attention to are going to stick in my memory is..." is lengthy, confusing and requires a high cognitive demand to understand what the question was asking. Keeping this item posed the risk of having participants offer an arbitrary response, or overwhelming a participant who may have diminished cognitive capacity due to suffering severe depression. This item, therefore, was omitted completely.

As it was anticipated that many people receiving ECT would be older adults based on statistics provided by the ministry of health of previous years (Ministry of Health, 2011, 2012), both the original and the updated measure were sent to a 77 year old woman for feedback. She was blind to which measure was the original, and which the adapted version was. After reviewing the original SSMQ she reported being confronted with a solid phalanx of 18 questions to be rather off putting. She preferred the updated version of the SSMQ, as the items were grouped in a more meaningful way and it was easier to understand what the items were assessing. She also reported that, for both questionnaires it was unclear what period of memory the SSMQ was assessing, as no time frame is stated.

Considering this feedback and through further discussion, the anchors of the updated measure were adapted. The anchors used in the original SSMQ are "Worse than Before" and "Better than Before". As pointed out by the woman who piloted the questionnaire, these anchors aren't specific, and leave the respondent thinking, "than before what?" As this current investigation looks at the effects of cognition due to ECT, the anchors were modified to "This is a questionnaire about your memory. Since having ECT, how would you rate your"... items were then listed down the left hand column with anchors "Worse", "No Change" and "Better". The original item rating scale went from -

4(Worse than Before) through to +4 (Better than Before), this was changed to 0 (worse), 5 (No Change) and 9 (better) as these numerical values appeared more logical.

It is acknowledged that altering the order of the items of the SSMQ and altering some of the wording of the items could affect the validity and some of the psychometric properties of the questionnaire; however, it was decided that having a participant become frustrated with the questionnaire or giving arbitrary answers due to not understanding the item would be a greater threat to the validity of the results and could damage rapport between the participant and the primary investigator. After the research was conducted, a Cronbach's alpha revealed that the internal consistency of the adapted SSMQ was good ($\alpha = .88$).

Autobiographical Memory Interview.

As the AMI assesses autobiographical memory over a large period of time, and there is very little evidence which suggests ECT affects memories for early life periods (Robertson & Pryor, 2006), and strong evidence to suggest amnesia due to ECT is temporally graded, with memories closer in time to treatment more susceptible to amnesia than memories formed further back in time (Squire et al., 1981; Weiner et al., 1986), it was decided that only Section C of the AMI – the Recent Life section be administered. The remaining sections of the AMI which assess memories for early life events were excluded. As well as increasing the sensitivity of the assessment, a further advantage of administering only a component of the AMI was that it took significantly less time to administer than the full version.

Two questions were added to the AMI to further assess autobiographical memory. One question was added to Part 7: Present hospital or institution – Personal/Semantic. After asking the question “Where are you currently living” a further question “how long have you resided there for?” was added. This section of the AMI also asks “Can you tell me about an incident which has occurred at this hospital?” As some of the patients who receive ECT are outpatients, the question was replaced

with “Can you tell me about something you did last week?” This question could relate to something in or out of hospital. A further measure of personal semantic memory was asked “Can you tell me what you had for dinner the night before last?”

Alongside the AMI, three questions of Public Events knowledge were asked.

1. “Who is currently the prime minister of New Zealand?”
2. “Who is the president of the United States?”
3. “Can you tell me about something that has been in the news in the past two weeks”
OR “Can you tell me about something that has been happening in your community in the past two weeks” (if the individual does not watch the news or read the paper).

Medical College of Georgia Complex Figures.

Complex figures are typically administered with a copy trial, a three minute immediate recall trial to assess for short term visual memory and a 20-30 minute delayed recall trial. Due to the strict time frame of the assessment, the immediate recall phase was not assessed. As participants’ scores were compared against their own previous performance’s not against norms this adaptation was justified.

Montreal Cognitive Assessment.

The five words used to assess ‘delayed’ memory after a five minute delay period in the MoCA were reassessed near the end of the assessment. To lift the ceiling on the digit span forward (DSF) and digit span backwards (DSB) tasks incorporated within the MoCA, three extra DSF and DSB strings were added, which increased in difficulty by one digit for each subsequent addition.

Coin Rotation Task.

The CRT was used with a New Zealand 20 cent coin, in place of the US nickel or quarter. An equivalence study was conducted to ensure that equivalence between the US nickel and NZ 20 cent coin existed. The following chapter (Chapter Six) presents this study.

Assessment Procedure

Test Order.

As the people participating in the current research would be unwell enough to warrant a prescription of ECT and most likely be suffering from severe depression, it was anticipated that some individuals would not be able to withstand a full hour of cognitive testing. It was also made clear to participants that if at any time during the assessment they wished to withdraw from the assessment, they could do so without question. Given the possibility of early withdrawal, the order of the assessment was designed in such a way that cognitive domains most associated with impairment were prioritised earlier on in treatment. In addition, practicalities of some of the measures, such as the requirement of a 20-30 minute delay for the recall phase of the complex figure and verbal memory task also informed the order the measures were administered.

The Autobiographical Memory Interview (AMI) part C was prioritised and administered first, as autobiographical memory loss is the most distressing cognitive impairment associated with ECT (Scott, 2010). The AMI is administered in interview format and starts with simple questions that participants would be likely to answer correctly, for example, item number one asks: "Can you name the hospital that you are in". It was hoped that this would assist in developing rapport with the patients and aid in putting them at ease with the testing. As the Montreal Cognitive Assessment (MoCA) covers a broad range of cognitive functions, it was prioritised and administered subsequent to the AMI. Placing the MoCA early on in the battery also allowed for the added component of long term verbal memory to be assessed after a 25-30 minute delay. The Medical College of Georgia Complex Figure (MCGCF) was then administered, allowing a sufficient period of time to pass

before the recall phase was administered at the end of the assessment (approximately 25 minutes). The MCGCF was followed by the coding task and the Coin Rotation Task (CRT). The participant was then asked about any subjective cognitive changes he or she had noticed since starting the course of ECT. The subjective report was followed by the Squire Subjective Memory Questionnaire (SSMQ) as the SSMQ complimented the topic of subjective memory change. The BDI-II followed the SSMQ as it is also a self rated questionnaire and flowed on from the SSMQ. The recall verbal memory phase and the visual memory recall phase ended the assessment (see Table 2).

Timing of Testing.

The initial cognitive assessment took place before any ECT treatment had begun to obtain a baseline measure of cognitive functioning. The second session was conducted 48 hours after the patients' third session of ECT and a further assessment took place 48 hours after the patients' sixth treatment. The 48 hour time period allowed for recovery of orientation and transient effects of the ECT; however, is a short enough period to detect cognitive impairment resulting from ECT (Porter, Douglas, et al., 2008). As different cognitive domains recover at different paces, e.g. reorientation to time, person and place recovers at a faster speed than recovery of working memory, and the magnitude of cognitive effects varies as a function of how much time surpasses after the ECT treatment (Daniel & Crovitz, 1983), it was important to assess cognitive function at a standard time, each time after treatment. Therefore an effort was made to adhere to the 48 hour delay as closely as possible.

CHAPTER SIX

Coin Rotation Task – The Development of Norms for New Zealand and the United States

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Abstract

The Coin Rotation Task (CRT) is a validated test of motor dexterity. Restricting its use is the absence of normative data with any NZ coin and only incomplete preliminary normative data exists for the use of the CRT with the US nickel. There were two objectives for the current study: To collect norms for the use of the CRT within New Zealand and to compare the US nickel against the NZ 20 cent coin to establish the degree of equivalency between the two coins. Two hundred and fifteen participants aged 16-87 took part in this normative study. Participants consecutively rotated the 20 cent coin 180 degrees in their dominant and non-dominant hands, as quickly as possible for 20 seconds per hand. Ninety-four participants also participated in the equivalence study, and carried out the task with both the 20 cent coin and the nickel. Normative data was collected for number of rotations per 20 seconds with the NZ 20 cent coin. No significant difference in the number of coin rotations was found between the nickel and the 20c coin. There was no effect of gender and age was inversely correlated with coin rotation performance. The CRT is a quick, convenient and inexpensive task of psychomotor speed and motor function. The current study offers norms for use of this task with the 20 cent coin to facilitate utilisation of this measure within New Zealand and the United States.

Keywords: motor dexterity, psychomotor speed, clinical assessment, bedside motor assessment, norms

Coin Rotation Task – the Development of Norms for the use in New Zealand and the United States

The Coin-Rotation Task (CRT) is a convenient, easily administered bedside test of motor dexterity (Mendoza et al., 2010). The task requires an individual to rotate a coin 180 degrees as quickly as possible utilising only the thumb, index and middle fingers. The individual performs the task with both their dominant and non-dominant hand. The origins of this practical task can be traced back to the Louisiana State University Health Sciences System, where the test was developed by John Mendoza to assess fine manual dexterity and collect qualitative information about a patient's coordination and motor speed (Hill et al., 2010). The task has now been used for over 20 years, although the bulk of the peer-reviewed literature which has utilised the CRT has been published during the past five years.

The CRT has been used across a broad range of studies to assess motor dexterity. For example, the CRT was used to assess motor dexterity and limb kinetic apraxia in patients with Parkinson's disease (Quencer et al., 2007; Vanbellingen et al., 2011), to assess motor slowing with patients who are HIV positive (Minor et al., 2010), to assess asymmetry of ipsilateral motor activation during bimanual and unimanual motor tasks (Ghacibeh et al., 2007), to investigate which hemisphere of right handed subjects exerts bilateral compared with contralateral motor control when performing precise motor moments (Hanna-Pladdy, Mendoza, Apostolos, & Heilman, 2002), to assess dexterity and inter-hemispheric motor communication (McGregor et al., 2012), and to investigate how amputation of the dominant and non-dominant upper or lower extremity alters deftness in the intact limbs (Swanberg et al., 2011).

Reasons for the recent increase in popularity may be due to the task's many merits which make it a practical bedside test of psychomotor speed. The CRT is inexpensive, accessible, available, easily replaceable and does not lose calibration (Hill et al., 2010). In addition, the CRT is free from the

effects of education, literacy and gender (Minor et al., 2010). Many of the commonly used tests of motor function, such as the Finger Tapping test (Dodrill, 1978) and the Grooved Pegboard Test (Spreeen & Strauss, 1991) require the use of expensive and bulky equipment not always accessible or appropriate for bedside examinations (Mendoza et al., 2010). The CRT, however, is compact and can be carried in the clinician's pocket (Hill et al., 2010). Administration of the task provides qualitative information about an individual's technique and coordination (Mendoza et al., 2010). Comparisons in CRT performance between the dominant and non-dominant hand can detect subtle hemispheric differences in cognitive processing (Hanna-Pladdy et al., 2002; Mendoza et al., 2010). The task is simple, time efficient and can be performed and scored in under a minute for both hands (Hill et al., 2010). In addition, the CRT has no significant floor effects which make it sensitive to motor impairment, and the sensitivity and specificity of the CRT is comparable with, or better than other standardised tests of motor dexterity (Hill et al., 2010).

Luria refers to behaviour as the process of various complex functional systems of the brain, which depend on the coordination of groups of interrelated functional areas which make their own specific contribution to the entire functional system (Christensen & Jensen, 1989). Depending on which area of the brain is impaired, the disturbance of the function will manifest in different ways. The structure impaired can only be identified after detailed analysis of the system has been conducted (Christensen & Jensen, 1989). The act of rotating a coin would be the result of a number of neuronal processes each contributing to this task. The coin rotation task could thus be used not only to assess motor speed, but also to qualitatively assess motor function and assist in the investigation to determine area and structure of damage.

Previous research investigating whether limb-kinetic apraxia (LKA), loss of hand and finger dexterity, is associated with Parkinson's disease compared performance in the use of the finger tapping test and the coin rotation task with people diagnosed with Parkinson's disease and a control group. There was no difference in finger tapping performance between the two groups; however,

performance on the coin rotation task was impaired, indicating that the coin rotation task was sensitive in detecting LKA whereas the FTT was not (Quencer et al., 2007). This is some evidence to suggest that the CRT could potentially be used to dissociate between disorders of coordination and disorders of motor-speed.

Hill and colleagues (2010) validated the CRT against the Finger Tapping Test (FTT), the Grip Strength Test (GST) and the Grooved Pegboard Test (GPT) – the latter being considered the gold standard measure of psychomotor speed (Strauss & Spreen, 2006). The CRT was found to be significantly correlated with all these measures suggesting a high level of convergent validity (Hill et al., 2010). The CRT was most strongly related to the GPT. The CRT predicted fine motor and psychomotor processing speed impairment with 0.80 sensitivity and 0.62 specificity for dominant hand performance, and 0.83 sensitivity and 0.43 specificity for non-dominant hand performance. These authors concluded that the CRT was a convenient and inexpensive psychomotor processing speed screening tool for clinical settings.

Mendoza and colleagues (2010) evaluated the construct validity and diagnostic accuracy of motor impairment of the CRT. These authors used the CRT as a test of motor skill in 60 right handed men aged from 40-79 years. As with Hill and colleagues' (2010) validation study, Mendoza and colleagues also validated the CRT against the FTT, GST and the GPT. Again, the CRT demonstrated convergent validity with these tests and divergent validity with a measure of upper extremity strength (grip strength). The CRT was concluded to be a valid assessment of motor functioning.

As well as validating the task against a number of tasks of fine motor skill, dexterity and strength, Mendoza and colleagues (2009) also collected preliminary norms for use of the Coin Rotation Task with the US nickel; however, the limited sample used poses clear limitations for widespread use of these norms. Accordingly, the authors suggested future research should collect normative data for

women and younger adults stratified by age and sex in order to complete the standardisation of the task. The current study aims to fill this gap in the literature by employing a larger sample and including both left and right motor dominant males and females, across a wide range of ages.

Currently, no gold standard procedure exists for the administration of the CRT and there appears to be some discrepancies in the literature regarding how the task is administered. The essence of the task, however, is consistent, with all studies requiring the participant to rotate a US coin through serial 180 degree turns using the thumb, index and middle finger. The participant first does this with his/her dominant hand, followed by the non-dominant hand. Mendoza and colleagues (2010) required participants in their validation study to rotate a nickel through 180 degree turns using the thumb, index and middle fingers as rapidly as possible for 20 rotations. They then recorded the time taken to complete the rotations. Participants were allowed to rotate the coin towards or away from themselves according to their preference. If the coin was dropped, participants were given the coin back immediately and told to continue rotating the coin, unless the elapsed time was greater than two seconds. Hill and colleagues (2010) validated the CRT with the US quarter. They allowed 10 seconds for as many rotations as possible, and if the participant dropped the coin the task was started again. Minor and colleagues (2010) and Barkemeyer, Santa Maria, Browndyke, Callon and Dunn (1998) followed this same procedure, rotating a US quarter 180 degrees as many times as possible in 10 seconds, first with the dominant hand, followed by the non-dominant hand. Hill and colleagues (2010) noted a ceiling effect with the number of coin rotations possible within the short amount of time permitted in their study.

As variations in the way this task is administered exist, there is an obvious need for standardisation of this task. As this study aims to provide new normative information for the task, the current authors saw this as an ideal opportunity to optimise the administration procedure of the task, without deviating significantly from previous modes of administration. Preliminary trials of the task showed that acceleration of motor speed, and grasping coordination of the task generally took place

in the first few seconds, after which participants tended to become familiar with the task and maintained a steady, constant motor speed. Allowing the relatively short time limit of 10 seconds for the task could result in many false positives of people being classified as ‘motor impaired’ using the cut-off score proposed of 13 proposed by Hill and colleagues (2010). It was therefore decided to lengthen the allotted time in order to create a finer distinction between motor-impaired and non-motor impaired individuals and increase specificity of the task, as well as lift the ceiling effect noted by Hill and colleagues. Mendoza and colleagues (2010) permitted as much time as the patient required for 20 rotations, which combats this threat to specificity. However, capping the allotted time allowed for the task, as is required by Hill’s mode of administration, ensures the task is quick, efficient, and any patient who is severely motor impaired will not have to endure the task for a prolonged period of time. Therefore, for the current study, participants were required to rotate a New Zealand 20 cent coin and a US nickel as quickly as possible for duration of 20 seconds.

This article reports the procedure and results of two studies. The primary objective of the current study (Study 1) was to develop norms for the use of the CRT in New Zealand. The second study aimed to establish whether equivalency between the New Zealand 20 cent coin and the United States nickel exists, to determine whether the New Zealand norms could be utilised in the United States. Based on previous findings by Mendoza and colleagues (2010) it is expected that no significant gender or age effects will be found and that the number of coin rotations will be significantly higher in individuals’ dominant versus non-dominant hand. It is also expected that handedness will not have an effect on coin rotation performance. The impact of practice will also be explored in Study 2.

Method

Participants

Two hundred and fifteen community dwelling participants were recruited via verbal invitations

from the researchers for study 1. Participants included staff and students from a Wellington university, patrons of a cinema located in Wellington, New Zealand, and people were approached in public locations and invited to take part in the study. The ages of the participants ranged from 16-87 years ($M=41.20$, $SD=17.96$). The sample included both females (62%) and males (38%). The majority of the participants were right hand dominant (81%). Exclusion criterion was the existence of a motor condition which disabled or impaired fine motor skill. Examples of such conditions include Carpel Tunnel syndrome, Arthritis, and Occupational Overuse Syndrome. Ninety four of the above participants also participated in the equivalence study (Study 2).

Apparatus

A New Zealand 20 cent coin was used to norm this task. The coin has a weight of 4.0 grams, diameter of 21.75 mm, and thickness of 1.56mm and has 7 grooves on the edge. The United States nickel was used in the comparison study (Study 2). The nickel has a weight of 5.0 grams, diameter of 21.21 mm, thickness of 1.95 mm and a smooth edge. A stopwatch was used to time the task.

Procedure

Study 1

Participants were asked to rotate the coin as quickly as possible, 180 degree turns for 20 seconds, using their thumb, index and middle fingers (see Figure 1), first in their dominant hand, then in their non dominant hand. The participant could turn the coin towards or away from them according to their personal preference, but were asked remain consistent. The task was first demonstrated to the participant. If the coin was dropped (which seldom occurred), and picked up again within two seconds, the participant continued the task. If the coin was dropped and it took longer than two seconds to collect the coin, the participant was asked to start again. This procedure is described by Mendoza et al. (2010). All participants performed the task while seated. The number of rotations for each hand and coin were recorded, as was the individuals' ages and sex.

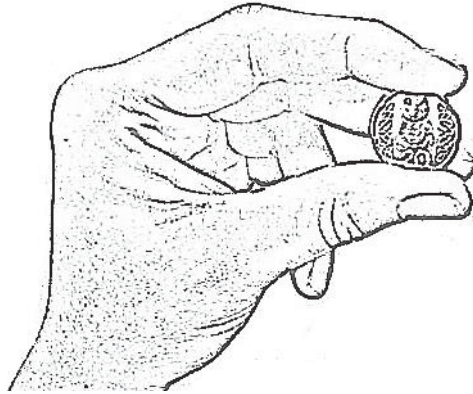



Figure 1. Finger positioning for the coin rotation task, based on demonstration provided by Hill et al., (2010).

Study 2

The comparison study adopted a within subjects design and compared both the NZ 20 cent coin and the US nickel. Participants completed the coin rotation task twice, once with each coin and following the same instructions as used in Study 1. Across subjects counterbalancing was used to control for order effects. A 2x2 Latin square was used to counterbalance the order of coin rotations with the nickel and the 20 cent coin, and participants were randomly allocated in condition A or B by flipping a coin prior to them completing the task. This process is presented in Figure 2.

Condition A	Nickel Dominant/ Non Dominant	20 cent Dominant/Non Dominant
Condition B	20 cent Dominant/Non Dominant	Nickel Dominant /Non Dominant



Time

Figure 2. Latin square showing conditions A and B used to control order effects in the equivalence study.

Results

Preliminary tests

Independent samples t-tests, paired samples t-tests, ANOVAs and regression analyses were conducted with SPSS Statistics (Version 19.0). The critical assumptions underlying these tests were checked and met. Six outliers were detected and subsequently removed prior to analysis. Levene's tests for homogeneity were non-significant for all dependent variables. An alpha value of $\alpha=0.05$ was set for all conducted statistical analyses, unless stated otherwise, and all tests were two-tailed.

Study 1

Normative data for gender, age, and number of rotations completed in participants' dominant and non-dominant hands are provided in Tables 1, 2 and 3 below.

Table 1
Mean Dominant Hand Performance for Males and Females for the Coin Rotation Task

Age	Male		Female		<i>P</i>	<i>d</i>	π
	Dominant Hand	<i>N</i>	Dominant Hand	<i>N</i>			
16-24	30.07(4.01)	15	28.90 (3.61)	30	.863	0.31	0.25
25-32	30.85 (5.06)	20	28.21 (6.13)	28	.431	0.47	0.47
33-40	30.17 (5.64)	12	27.91 (5.26)	11	.333	0.41	0.25
41-48	29.50 (8.26)	6	27.75 (5.45)	16	.567	0.25	0.13
49-56	26.72 (3.69)	11	28.56 (5.52)	18	.341	-0.39	0.26
57-64	28.67 (2.89)	6	26.50 (3.14)	16	.143	0.72	0.08
65+	23.25 (5.29)	12	25.42 (5.43)	14	.342	-.40	0.26
All ages	28.51(5.69)	82	27.75 (5.16)	133	.319	-0.83	0.99

No significant differences existed between male and female performance in the individuals' dominant hands for all age brackets. Mean dominant hand performance showed a gradual decline with age, with a slight peak in performance at ages 57-64 in males, and at ages 49-56 in females. Post-hoc analysis with a Bonferroni adjustment revealed that although there were significant differences among the norm groups for the males ($F(6,74)=3.58$, $p=.004$), the peak in performance at ages 57-64 was not statistically significantly different to the mean performance in the other age groups. There were no significant differences between the age brackets for females ($F(6,126)=1.02$, $p=.108$).

Table 2

Mean Non-Dominant Hand Performance for Males and Females for the Coin Rotation Task

Age	Male		Female		<i>P</i>	<i>d</i>	π
	Non-Dominant	<i>N</i>	Non-Dominant	<i>N</i>			
16-24	27.20(5.65)	15	25.10 (3.52)	29	.138	0.44	0.39
25-32	27.79(5.07)	19	25.61 (5.72)	28	.590	0.40	0.67
33-40	27.91 (4.14)	12	24.27 (4.54)	11	.057	0.84	0.61
41-48	25.50 (3.72)	6	24.07 (4.21)	15	.161	0.36	0.35
49-56	25.36 (4.27)	11	27.22 (5.11)	18	.295	-0.39	0.26
57-64	24.83 (5.42)	6	24.81 (3.69)	16	.992	0.00	0.05
65+	22.75 (5.08)	12	23.61 (4.94)	13	.603	-0.17	0.11
All ages	26.39(5.23)	81	25.18(4.71)	130	.084	0.24	0.52

There were no significant differences between males and females. A post hoc power calculation for all ages showed that no significant difference was calculated with 0.93 power. Thus males and females were combined for analyses.

As with the dominant hand, no difference in mean rotations existed between males and females, and non-dominant performance declined slowly with age; however, differences between age groups were not statistically significant for both males ($F(6,74)=1.82, p = .107$) and females ($F(6,123)=1.17, p = .326$).

As no significant differences between males and females were detected for any of the age brackets in both the dominant and non-dominant hands, combined norms for males and females are offered in Table 3. In addition, paired-samples t-tests were conducted to determine whether a significant difference between average number of coin rotations in an individual's dominant and non-dominant hand exists. The probability values are also offered in Table 3.

Table 3

Mean Performance for the Coin Rotation Task in Dominant and Non-Dominant hands for Males and Females combined

Age	Dominant Hand	Non-Dominant Hand	N	P	<i>d</i>	π
16-24	29.27 (3.73)	25.82 (4.42)	44	.000	0.84	0.99
25-32	29.31 (5.80)	26.49 (5.51)	47	.000	0.49	0.91
33-40	29.08 (5.45)	26.17 (4.62)	23	.002	0.58	0.76
41-48	28.22 (6.17)	24.48 (4.04)	21	.005	0.61	0.76
49-56	27.86 (4.91)	26.51 (4.24)	29	.068	0.27	0.29
57-64	27.09 (3.16)	24.81 (4.08)	22	.079	0.72	0.89
65+	24.42 (5.71)	23.20 (4.92)	25	.456	0.23	0.20
All ages	28.15 (5.20)	25.55 (4.74)	215	.000	0.50	1.00

Age and Motor Speed

One-way ANOVAs revealed a significant main effect of age for number of rotations in the dominant hand ($F(6,207)=3.415$, $p=0.003$), but not in the non-dominant hand ($F(6,204)=1.94$, $p=0.077$). A Bonferroni post-hoc analysis with an adjusted alpha level of 0.007 ($\alpha=0.05/7$) revealed that the average number of coin rotations in the dominant hand for the 65+ group was significantly fewer than those in the 16-24 year group ($p=.003$) and the 25-32 year group ($p=.002$). This difference also approached significance in comparison to the 33-40 year group ($p=.033$). All other pair-wise comparisons were not statistically significant.

As demonstrated in Table 3, motor dexterity appeared to decline slowly with age. A Pearson's correlation coefficient calculated using participants' average motor speed revealed that this decline is moderate but statistically significant ($r(210)=-.266$, $p=0.000$). However, further analyses revealed that motor dexterity declines at a faster rate in the individual's dominant hand ($r(214)= -.319$, $p<.001$) than in their non-dominant hand ($r(210)= -.19$, $p=.006$). A Fisher Z-transformation was calculated and showed a non-significant difference in the relationships between decline in the dominant versus the non dominant hand ($Z=-1.41$, $p=0.159$).

Hand Dominance, Age and Motor Speed

As predicted, individuals achieved greater mean scores with their dominant hand than their non-dominant hand. Interestingly, this difference became less statistically significant with increasing age (see p -values in Table 3), indicating that with age hand dominance has less of an effect on motor speed.

There was a strong significant correlation between number of rotations made in individuals' dominant and non-dominant hands ($r(211)=.71, p<.001$). A linear regression determined that the difference in scores between the dominant and non-dominant hand was a positive predictor of age ($R^2=0.04, (F(1,208)=8.41, p=0.004)$). The regression equation was used to calculate at what age there would be no difference between number of coin rotations in the dominant hand and those in non-dominant hand. The results of the linear regression predicted that this would occur at 100 years of age ($\beta=-0.42, t(208)=6.47, p=.000$).

Handedness and Motor Speed

Independent samples t -tests were run to establish whether handedness had an effect on coin rotation performance. As predicted, there was no significant difference in motor speed for people who were left hand dominant and right hand dominant for both the dominant ($t(212)=.57, p=.572$) and the non dominant hands ($t(209)=-3.8, p=.698$).

Study 2

To determine whether a significant difference between the number of coin rotations made with the New Zealand coin and the U.S coin in a 20 second period exists, two paired samples t -tests were conducted. One compared the number of coin rotations with the non-dominant hand using the 20 cent coin and the nickel and the other compared dominant hand rotations with the 20 cent coin and the nickel.

There was no statistically significant difference in number of coin rotations between the US nickel and the 20 cent coin with either the non-dominant hand ($t(93)=-.106, p=0.916$) or the dominant hand ($t(93)=-1.352, p=0.180$). Number of rotations made with both coins correlated strongly for both dominant ($r(93)=.85, p = 0.000$) and non-dominant hands ($r(93) = .756, p= 0.000$). Descriptive data for number of coin rotations in each had for each coin are presented below in Table 4.

Table 4
Number of Coin Rotations for Non-Dominant (ND) and Dominant (D) Hands for the US Nickel and the NZ 20 Cent Coin (N=94)

Hand	Coin	Minimum	Maximum	Mean	SD
Dominant	US Nickel	17	39	29.11	(5.11)
	NZ 20 Cent	17	40	28.61	(4.93)
Non Dominant	US Nickel	14	37	25.44	(5.18)
	NZ 20 Cent	14	39	25.46	(5.40)

To test the final hypothesis that the number of coin rotations would increase with practice, paired samples t-tests were conducted to compare the first coin rotation against the second with the dominant hand and then with the non-dominant hand. Both the dominant and non-dominant hands showed an increase in number of coin rotations over time. The increase in rotations with the dominant hand at time one ($M=28.37, SD=5.27$) was significantly lower than coin rotations at time two ($M=29.22, SD=4.66$) indicating a practice effect in the dominant hand ($t(92)=-2.48, p=0.015$). A significant increase from time one ($M=24.92, SD=5.25$) to time two ($M=26.06, SD=5.24, t(92)=-4.20, p=0.000$) was also found in the non-dominant hand.

Discussion

To date, the only normative data available against which to compare coin rotation performance has been for right handed men aged 40-79 (Mendoza et al., 2010). The current study provides a new set of normative data for the coin rotation task based on a sample of 215 participants, male and female aged 16-87. A significant but moderate decline in motor speed with age was found in the current

study, which is consistent with finding from Minor, Jones, Stewart, Hill and Kulesza (2010) and studies which have evaluated the effect of age with other measures of motor speed (Ruff, 1993). A significant age effect was not found in Mendoza and colleagues' study. Although a significant effect of age was found for the current study, a Fisher's Z analysis revealed that these two correlations were not significantly different $Z = -0.62, p = 0.53$. No significant effect of gender was found which is also consistent with findings from Minor and colleagues (2010).

Individuals completed more coin rotations in their dominant versus their non-dominant hands which has also been noted in the literature as a characteristic of this test (Hill et al., 2010; Mendoza et al., 2010). Over time, the difference in performance between the dominant and non-dominant hand became smaller. This effect may be due to performance in the dominant hand being superior to begin with, and therefore greater scope exists for decline in the dominant hand with age, which is not the case with the non-dominant hand. However, the differences between the correlations of motor decline in the dominant and non-dominant hand were not statistically significant.

The comparison study found no significant difference between coin rotation performance with the US nickel and the NZ 20 cent coin, and this was true for both the dominant and non-dominant hands. This implies that the nickel would be a valid substitute for the 20 cent coin when conducting the coin rotation task in the United States, and conversely, the validation studies conducted with the nickel should generalise to the 20 cent coin. A small, practice effect was found after the second consecutive trial of the task for both hands. The second trial was performed immediately subsequent to the initial trial. As the CRT has been proposed as a convenient way to monitor motor dexterity during the course of an illness (Hill et al., 2010) the nature and longevity of the practice effect of this task should be further researched. If the practice effect remains over time, no change in CRT score after a second administration could actually imply motor slowing.

A limitation of the current study is the relatively low sample in the higher age brackets. Nonetheless, the data yielded significant results when the genders were combined, thereby increasing statistical power. In addition, the nature of the data followed the trends found in previous studies of motor speed, such as the gradual decline in motor dexterity with age and the superiority of dominant hand performance over non-dominant performance. A larger sample with greater statistical power may have detected a possible gender effect. Although this has been reported for other measures of motor speed (Ruff, 1993), it has not yet been detected in the CRT. The CRT has been validated against a number of tests of motor speed and psychomotor function (Hill et al., 2010). As the way in which the task was administered for the current study differed somewhat in an attempt to optimise the mode of administration the validity of the task should be re-evaluated as this may have changed as a result.

As well as a test of motor speed and dexterity, the CRT requires a degree of cognitive involvement. In order to carry out the task, the patient must carefully monitor the location of the coin in their fingers, sequence the movements and carefully coordinate the thumb, index and middle fingers to successfully rotate the coin (Hill et al., 2010). Future studies should evaluate the construct validity of the CRT comparing it with tests of psychomotor speed involving a greater cognitive component, such as the coding subtest of the WAIS-IV (Wechsler, 2008) as past research has shown that the CRT correlated more highly with the Grooved Pegboard Test than the Finger Tapping Test (Hill et al., 2010), the former having a higher cognitive component than the latter. The CRT could be used in addition to the FTT to dissociate between pure motor impairment and fine motor coordination impairment. In addition, by qualitatively observing the patient perform this task, the clinician will be able to observe whether the person has difficulty with the maneuvering and coordination of turning the coin.

In addition, future research should assess the predictive validity of the coin rotation task with patients suffering conditions where psychomotor slowing is common, such as with patients

suffering from depression. Finally, throughout the limited literature available on this task, there have been some variations in the way the task is administered and the coin which is used. It is recommended that one universal method of administering this task be adopted, so that comparisons across studies can be made more easily. It is hoped that the results of this study will facilitate widespread use of this convenient, inexpensive, validated measure of motor dexterity and psychomotor function.

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STATEMENT OF CONTRIBUTION
TO DOCTORAL THESIS CONTAINING PUBLICATIONS

(To appear at the end of each thesis chapter/section/appendix submitted as an article/paper or collected as an appendix at the end of the thesis)

We, the candidate and the candidate's Principal Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of Candidate: Anneke Thornton

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Name of Published Research Output and full reference:

Coin Rotation Task – The Development of Norms for New Zealand and the United States

In which Chapter is the Published Work: Six

Please indicate either:

- The percentage of the Published Work that was contributed by the candidate:
and / or
- Describe the contribution that the candidate has made to the Published Work:

The candidate had full responsibility for the design, data collection, analysis and write up of the research. Supervisors provided guidance and were involved in decisions made around research processes and provided input with data analysis and formatting of the thesis. For these reasons Professor Janet Leatham and Dr Ross Flett were included as co-authors for the manuscripts which comprise this thesis.

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CHAPTER SEVEN

Short Term Cognitive Change during Electroconvulsive Therapy for Unipolar and Bipolar Depression: A Prospective Study

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Abstract

Although Electroconvulsive therapy (ECT) is the most effective treatment for major depressive disorder; its use is limited due to ongoing reports of cognitive impairment secondary to the treatment. Patients commonly report cognitive dysfunction post ECT which is often not detected in objective cognitive assessments. To effectively assess ECT related cognitive change, further research needs to be conducted into increasing the sensitivity of the cognitive assessment. The current study aimed to determine: 1) what cognitive changes occurred during the course of ECT; 2) the nature of impairment and recovery of these changes over time; and 3) which assessment measures were sensitive to detecting change. Thirteen patients receiving ECT for treatment resistant major depressive disorder or bipolar disorder aged between 18 to 78 were recruited into the study. A comprehensive cognitive assessment was conducted prior to starting the course of ECT, again after every three treatments of ECT and at a 6 week follow-up. The data are presented as a series of case studies and are also analysed collectively. The nature of cognitive impairment varied across individuals and was dependent upon mood and number of treatments. Cognitive impairment typically occurred during the course of treatment; however, by the follow-up assessment most domains of cognition had returned to at least the baseline level of function. In conclusion, ECT caused cognitive dysfunction across a broad range of functions in the short term for the current sample; however, this dysfunction typically resolved by six weeks post ECT. The assessment measures sensitive to detecting the cognitive changes are reported.

Keywords: Cognitive Assessment, Electroconvulsive Therapy, Short Term Cognitive Effects

An Investigation into the Short Term Cognitive Changes during Electroconvulsive Therapy for Uni and Bipolar Depression: A Prospective Study

Electroconvulsive therapy (ECT) is currently the most effective treatment for depression and a variety of psychiatric disorders; however, its use is restricted due to ongoing reports that ECT can cause cognitive impairment (Ingram et al., 2008). As cognitive change is a common side effect of the treatment, frequent and thorough cognitive assessments are recommended throughout the course of ECT (Nehra et al., 2007; Porter, Douglas, et al., 2008). Early detection of emerging cognitive dysfunction allows for modifications in treatment administration to be implemented, and subsequently reduce the risk of longstanding cognitive impairment (Martin et al., 2013). Although frequent cognitive assessment is recommended, the way in which these changes should best be assessed remains under debate, as does the nature and duration of the cognitive side effects (National Institute of Clinical Excellence, 2003; Verwijk et al., 2012). To date, no gold standard measure is available to monitor and assess cognition during a course of ECT (Gangadhar & Thirthalli, 2010). Thus, cognitive functioning is rarely monitored in a systematic way (Porter et al., 2008; see Chapter Four).

The assessment of cognitive function during ECT is complicated by incongruence between patients' subjective reports of cognitive function and their performance on objective cognitive tests (Berman et al., 2008; Vamos, 2008). One suggested hypothesis for this incongruence is that the cognitive tests selected are not sensitive enough to detect the changes which occur (NICE, 2003; Porter, et al., 2008). The widespread use of screening measures such as the Mini Mental State Examination (MMSE; Folstein et al., 1975) to assess ECT related cognitive function despite their insensitivity to detecting the cognitive effects of ECT perpetuates this finding of incongruence (Robertson & Pryor, 2006).

Assessing cognitive change during a course of ECT with insensitive measures could result in cognitive impairment remaining undetected, and failure to modify treatment. In addition, as most empirical research into the cognitive outcomes of ECT has relied on the use of objective neuropsychological assessment, it is possible that memory impairment following ECT may be more persistent than what is reported in the literature (Rose et al., 2003). Recent research into ECT related cognitive change which has relied on objective outcome measures of cognitive function suggests that most cognitive impairment is short term and should resolve within 15 days post treatment (Semkowska & McLoughlin, 2010; Verwijk et al., 2012); however, the subjective effects of ECT appear to last much longer than this (Fraser et al., 2008). If the degree of cognitive impairment reported in the literature is underestimated due to the inadequacy of objective measures to detect the entire magnitude of impairment, this would have implications in the way informed consent to treatment should be gained from patients.

Further research into the cognitive side effects of ECT using sensitive measures is required to address the ongoing debate surrounding the nature and duration of the cognitive effects of ECT, which will in turn enable patients to make a fully informed decision when consenting to treatment. Further research will also assist in determining which measures are sensitive to detecting the cognitive effects of ECT. Determining which measures are sensitive to detecting ECT related cognitive change will aid in preventing further use of insensitive or redundant measures in practical and research settings and will ultimately narrow the incongruence which exists between subjective and objective cognitive outcomes. Addressing these research questions will ultimately improve the quality and sensitivity of cognitive assessment with patients receiving ECT and aid in enabling cognitive impairment to be detected earlier on in a treatment course to decrease the risk of longer term cognitive impairment.

Cognitive Effects of ECT

Although there is extensive research into the short term cognitive effects of ECT, most has focused on memory related outcomes (Ingram et al., 2008), particularly anterograde and

retrograde amnesia (Ingram et al., 2008; Rami-Gonzalez et al., 2001). Very few studies have investigated in depth the cognitive changes associated with non-memory cognitive domains, such as attention and concentration, executive functions, processing speed and visuospatial abilities (Bayless et al., 2010; Verwijk et al., 2012). Whether these non-memory cognitive impairments persist into the long term remains inconclusive (Ingram et al., 2008; Rami-Gonzalez et al., 2001). Investigating non-memory cognitive change is important, as the function of memory is dependent on non-memory cognitive functions such as attention and concentration (Robertson & Pryor, 2006).

A recent meta-analysis analysed 84 studies (2981 patients in total) that had investigated ECT related cognitive outcome and included the few studies that had investigated the non-memory cognitive side effects (Semkovska & McLoughlin, 2010). The meta-analysis revealed that ECT affects a broad range of cognitive functions, including processing speed, attention, working memory, verbal and visual memory, spatial problem solving and executive function, but not intellectual ability or vocabulary (Semkovska & McLoughlin, 2010). Effect sizes were calculated to compare changes from pretreatment, to three and 15 days post-treatment. Medium to large deficits were found for episodic memory and all areas of executive functioning three days post treatment. Delayed recall was significantly more affected than immediate recall for both verbal and visual memory; although more for verbal memory. Learning and retrieval of unstructured information showed greater impairment than the ability to retrieve organised and contextualised information (for example, performance on word lists showed greater impairment than story recall). Tests of attention and working memory showed no change from baseline. Fifteen days post treatment, only a small deficit in unstructured delayed verbal memory remained. Furthermore, for the majority of variables there was a small to medium improvement beyond baseline of cognitive functions after 15 days.

Depression and Cognitive Impairment

It has long been established that depression is also associated with cognitive impairment (Douglas & Porter, 2009). As treatment resistant depression is the main indication for ECT, most patients suffer some degree of cognitive impairment prior to starting a course of ECT (Porter, Douglas, et al., 2008). This depression related cognitive dysfunction improves as a result of depression being ameliorated by ECT (Bayless et al., 2010; Calev et al., 1995; Sobin et al., 1995; Stoudemire, Hill, Morris, & Dalton, 1995). The extent of cognitive improvement has been found to co-vary with the extent of clinical improvement (Sobin et al., 1995). As depression and ECT are both associated with cognitive side effects, it is important to administer a measure of mood alongside cognitive measures in order to understand the cognitive changes which occur within the context of the patients' clinical state (Porter, Douglas, et al., 2008).

Research Aims

The main objective of the current research was to determine the nature, extent and duration of cognitive changes occurring at multiple stages during a course of ECT. In addition to memory, assessment would include a range of cognitive domains and be able to detect the cognitive effects of ECT as they occurred. A final aim was to determine which available neuropsychological measures were sensitive to detecting changes.

The following predictions were made based on previous findings in the literature:

- ECT will be effective in relieving the subjective reports of depression and mania.
- Impairment in cognitive function will occur as a result of ECT.
- The nature of cognitive impairment will manifest as a decrease in cognitive performance from baseline which will continue over the course of treatment.
- Recovery or improvement in cognitive function from the baseline level of functioning should occur by the six week follow-up cognitive assessment.

- Subjective and objective reports of cognitive function will be incongruent.

Method

Participants

Thirteen people (5 male, 9 female) about to receive ECT for treatment resistant depression (76.92%, N=10) or bipolar disorder (23.08%, N=3) were recruited from four ECT clinics in the lower North Island of New Zealand. Participant age range was 18-78, with the mean age of 47 years, (SD=13.5). Ethnicities included: NZ European (N=7, 53.85%), Maori (N=3, 23.08%), South African, Dutch and Chinese (N=1, 7.69% respectively). All participants were fluent in English and all assessments were conducted in English. During the course of treatment, 38.46% (N=5) of participants were outpatients, 30.77% (N=4) inpatients, and the remainder 30.77% (N=4) spent part of the time during their treatment in hospital, or respite care. All participants were prescribed ECT as medications had been ineffective or inadequate at relieving psychiatric symptomatology.

See Table 1 on the following page for a summary of patient characteristics.

Table 1

Summary of Patient Characteristics

Participant	Primary Diagnosis	Specifier	Age	Sex	Ethnicity	Courses of ECT	Total ECT treatments	Electrode Placement	Inpatient/Out-patient	Medication
P1	Major Depressive Episode,	Recurrent, Severe. Without psychosis.	78	Male	NZ/European	1	8	Right Unilateral	Inpatient	Unknown
P2	Major Depressive Disorder	Recurrent, Severe. Without psychosis.	56	Male	NZ/Dutch	3	25	Bilateral	Inpatient	Lithium carbonate 230mg 3x daily Mirtazapine 30mg 2x daily
P3	Major Depressive Disorder	Recurrent, Severe. Without psychosis.	36	Female	NZ/ Māori	1	12	Right Unilateral	Outpatient	Zopiclone Parnate
P4	Bipolar I	Most recent episode hypomanic	39	Male	NZ/Chinese	1	15	Right Unilateral	Outpatient	Fluoxetine 40mg, Carbamazepine 300mg
P5	Major Depressive Disorder	Recurrent, Severe. Without psychosis.		Male	Māori	1	12	Right Unilateral	Inpatient	Nortriptyline
P6	Major Depressive Disorder	Recurrent, Severe. Without psychosis.	51	Male	NZ/European	1	11	Right Unilateral	Outpatient	Nortriptyline 200mg and Quetiapine 50mg
P7	Major Depressive Disorder	Recurrent, Severe. Without psychosis.	18	Female	NZ/European	1	7	Right Unilateral	Inpatient	None
P8	Major Depressive Disorder	Recurrent, Severe. Without psychosis.		Female	Māori	3	47	Right Unilateral	Inpatient and Outpatient	Zopiclone 7.5mg
P9	Bipolar-I disorder		44	Female	NZ European	1	9	Right Unilateral	Outpatient	Unknown
P10	Major Depressive Disorder	Recurrent, Severe. Without psychosis.		Female	British	1	12	Right Unilateral	Outpatient	Venlafaxine 300mg Metazapine 30mg
P11	Major Depressive Disorder	Recurrent, Severe. Without psychosis.	56	Female	NZ/European	2	10	Left Unilateral	Outpatient	Unknown
P12	Bipolar-I	Most recent episode manic	45	Female	NZ/European	1	12	Right Unilateral	Outpatient	Lithium, 600mg Tipramate, 400mg
P13	Major Depressive Disorder	Recurrent, Severe. Without psychosis.		Female	South African	1	10	Right Unilateral	Inpatient	Prazosin 2mg nocte Lorazepam 1mg, promethazine 25mg, riperidone 0.5mg, Mirtazapine 15mg, Venlafaxine 187.5mg

Procedure

Recruitment.

Recruitment occurred through four District Health Boards (DHBs) in the Lower North Island region of New Zealand from August 2012 through to October 2013. After being prescribed a course of ECT, the patient was informed about the study by the ECT nurse or ECT administering psychiatrist. Any patient who expressed interest in participation met the primary researcher who explained the study in greater detail. Patients were given an information pack² containing further detail about the study and contact information for the researchers and written consent was obtained. Participation was voluntary and the patient was able to withdraw from the study at any time. Exclusion criteria included patients deemed by the ECT nurse or psychiatrist as too unwell to consent to taking part in a study or to undergo neuropsychological assessment, and patients receiving ECT for a condition other than a mood disorder.³ Ethical approval for conducting this research was obtained from the Health and Disability Ethics Committee, New Zealand.

ECT Treatment.

Twice weekly Right Uni Lateral (RUL) ECT using an ultra-brief 0.3ms pulse width was first choice of treatment. The MECTA SpECTrum Q5000 machine was used to administer the ECT and dose-titration determined using the limb isolation technique to establish seizure threshold. This technique involves isolating a limb from the effects of the muscle relaxant, by inflation of a blood pressure cuff to above systolic pressure. As the muscle relaxant does not affect that limb,

² Information pack can be found in Appendix D.

³ One exception was made for a man receiving ECT for schizoaffective disorder. This case is reported in Appendix E.

the seizure can be observed in that limb while the rest of the body is paralysed. Once the individual's seizure threshold was determined, the treatment dose was delivered at 6 times the seizure threshold. This dose remained constant for the entirety of the course, unless inadequate seizure duration occurred, and only then would the dosage be reviewed. Bi-lateral ECT (BL ECT) was used only in exceptional circumstances. The treatment dose for BL ECT was 1.5 to 2.0 times seizure threshold. The MECTA SpECTrum has the capability of varying the pulse width, the frequency (Hertz), the duration of the stimulus charge and the flow of current (Amps), allowing psychiatrists to tailor the treatment to best suit the individual patient. The average number of ECT treatments received was 11.25 (SD=6.86).

Measures.

Participants underwent neuropsychological and clinical assessments as outlined in the assessment procedures. Measures were chosen on the basis that they a.) had alternate forms (since tests would be repeated throughout the course of ECT), b.) had sound psychometric properties or had been designed for the use in this population c.) covered the cognitive constructs previously shown to be affected by ECT, d.) together would take no more an hour to administer. A measure of mood was added to determine the cognitive effect of depression on test performance.

The final assessment battery comprised of the following: Section C of the Autobiographical Memory Inventory (AMI; Kopelman et al., 1989) which assesses retrograde autobiographical memory for recent life events. The five words used to assess short term memory the Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005) in the MoCA were reassessed after a 30 minute delay to assess for long term verbal memory. To lift the ceiling on the digit span forward (DSF) and digit span backwards (DSB) tasks in the MoCA, three extra DSF and DSB strings were added which increased in difficulty by one extra digit for each subsequent addition. The copy and delayed recall phase of the Medical College of Georgia Complex Figures (MCGCF; Loring & Meador, 2003) were used, as was the Coding Task from the Repeatable Battery for

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the Assessment of Neuropsychological Status (RBANS; Randolph, 1998). The Coin Rotation Task (CRT; Mendoza et al., 2010) was used to assess motor speed. Finally, the Beck Depression Inventory – Second edition (BDI-II; Beck et al., 1996) and the Young Mania Rating Scale (YMRS; Young et al., 1978) were used as a measure of mood (See Chapter Five for considerations for choice of tests). To obtain a subjective measure of memory, the Squire Subjective Memory Questionnaire (Squire et al., 1979) was administered at each assessment. Table 2 depicts how the selected assessment measures map onto the cognitive domains to be tested.

Table 2

Cognitive Domains Assessed by the Selected Measures.

Domains Tested	Assessment Measure
Retrograde Amnesia - Semantic, Episodic and Autobiographical	AMI
Abstraction	MoCA
Anterograde Amnesia	MCGFG, MoCA
Short Term Memory Recall	MoCA
Delayed Memory Recall	MoCA
Executive Functioning	MoCA, MCGCF
Psychomotor Speed	Coding Task
Motor Speed	CRT
Visuospatial Ability	MoCA, MCGCF
Attention/Concentration and Working Memory	MoCA, MCGCF
Information Processing Speed	RBANS coding task
Orientation	MoCA
Short Term Memory	MoCA
Mood	BDI
Language (Production and retrieval)	MoCA
Subjective Memory	SSMQ

Assessment Procedure.

To maximise the validity of the assessment, each patient was asked at the beginning of each assessment to give their best effort when completing the measures, and to let the researcher know if fatigue or concentration loss occurred, or if they needed to have a break. The initial assessments took approximately one hour as this session involved providing an explanation for the research and obtaining informed consent. Subsequent assessments took approximately 45

minutes. All measures were administered in the same standardised order at each assessment, as outlined below:

1. Autobiographical Memory Interview (AMI) –Recent Life section (Section C)
2. Montreal Cognitive assessment (MoCA)
3. Medical College of Georgia Complex Figure- copy phase (MCGCF)
4. RBANS Coding Task
5. Coin Rotation Task (CRT)
6. Squire Subjective Memory Questionnaire (SSMQ)
7. Beck Depression Inventory (BDI-II)
8. Recall of five words from MoCA
9. Recall phase of MCGCF

Assessments were conducted in a one to one format and took place either in the patients' homes, at Massey University's psychology clinic or in an interview room in the inpatient unit at the hospital.

Assessment Schedule.

A formal cognitive assessment was undertaken with patients prior to their first treatment of ECT, and again after every three treatments during their course. A post-treatment assessment was conducted at cessation of the course and finally a six week follow-up assessment was conducted. All assessments were conducted 48 hours post ECT. The 48 hour time period allowed for recovery of orientation and transient effects of the ECT to resolve (Porter, Douglas, et al., 2008).

Design

A naturalistic prospective case-series design was adopted for the current study.

Results

Analysis

A case series design was deemed the most appropriate way to interpret the results of the study as the sample was small (N=13) and the patients were variable in the number of ECT treatments they received, the severity of depression, the way in which their treatment was administered, and the medications they were taking. However, group comparisons were also made across findings to evaluate general trends in cognitive changes. Each case study is presented visually and descriptively and Standard Mean Difference All effect sizes (SMDall) were used to evaluate changes which occurred across the group. The combination of single subject visual analysis and group SMDall analysis is recommended to ensure that important individual patient characteristics are not masked by the group analysis (Olive & Smith, 2005). Due to the high variability among the individuals included in this sample, there were many variables (such as age, comorbidity, severity of illness, and number of treatments) that were not able to be controlled for. This resulted in the variables not being normally distributed at baseline.

While some researchers advocate logarithmic and square root transforms for the sorts of variables of interest in this study given the inherent non-normality of their distributions, in this case we reported the simple descriptive statistics for the untransformed variables. This gave a context within which other researchers might interpret the relevant scores reported here. The simple correlational analyses that were reported here allowed us to paint a general picture of the nature of the relationships between variables. We may have considered data transformation if we had sought to pursue more sophisticated inferential analysis which would have been warranted with a larger available N.

Group Analysis

Standardised Mean Difference all (SMDall) effect sizes were calculated to demonstrate how each measure, overall, changed as a function of treatment phase. SMDall is computed utilising

all data points from the phases being compared (Olive & Smith, 2005). Variations in calculating standard mean differences exist; however, the logarithm utilised to analyse the data in the current study was as follows: $SMD_{all} = (\text{Baseline Score} - \text{Average of Treatment Scores}) / \text{Standard Deviation of the Baseline Score}$. Effect sizes were interpreted using Cohen's (1988) guidelines. Changes were assessed across three phases: changes from the baseline assessment to during the course of ECT; changes from during the course of ECT to the follow-up assessment; and changes from baseline to the follow-up assessment. When comparisons were made for during the course of treatment, the average performance was used as a more conservative approach rather than simply taking the post-treatment assessment score as often variability in cognitive performance existed in cognitive performance across assessments throughout treatment.

Table 3 displays the changes in cognitive function which occurred from baseline to the participants' average performance on the assessment measures achieved during their course of ECT. As predicted, a large decline occurred in degree of depressive symptomatology from baseline to during the ECT course, and a medium decline for mania. A large decline in autobiographical retrograde amnesia occurred during the course of ECT. Medium declines were observed in short term verbal memory and delayed visual and verbal memory. Working memory as measured by digit span backwards appeared to be a more sensitive measure of working memory than serial sevens; though the effect sizes were small for both. A small decline in information processing speed and verbal fluency occurred and small improvements in attention and motor speed occurred. Language function appeared insensitive to change from ECT, as did orientation to month, day of the week, place and city. A medium improvement in visuo-constructional ability as measured by the shape copy component of the MoCA occurred.

Table 3

Effect sizes of Cognitive Change which occurred from Baseline to Average Performance during the Course of ECT

<i>Variable</i>	<i>Assessment Measure</i>	<i>Effect Size</i>	<i>95% CI Lower-Upper</i>	<i>Qualitative Size</i>	<i>Direction</i>
Highly sensitive to change					
Depression	Beck Depression Inventory II	1.64	1.00-2.25	Large	Decline ¹
Retrograde Amnesia	Autobiographical Memory Interview	1.07	0.47-1.65	Large	Decline
Sensitive to Change					
Mania	Young Mania Rating Scale	0.76	-0.62-0.02	Medium	Decline
Short term Verbal Memory	Delayed recall phase – MoCa	0.62	0.04-1.19	Medium	Decline
Long term Visual Memory	Delayed Recall – (30min) MCGCF	0.57	-0.01-0.74	Medium	Decline
Long term Verbal Memory	Delayed Recall (30 min)- MoCA	0.49	-0.08-0.76	Medium	Decline
Visuo-constructional ability	Shape Copy – MoCA	-0.45	-1.02-0.13	Medium	Improvement
Moderately Sensitive to Change					
Verbal Fluency	MoCA	0.39	-0.19-0.95	Small	Decline
Orientation – Year	MoCA	-0.38	-0.95-0.20	Small	Improvement
Motor Speed – Non-dominant	Coin Rotation Task	-0.35	-0.94-0.25	Small	Improvement
Visuo-constructional ability	Clock drawing task – MoCA	-0.35	-0.87-0.27	Small	Improvement
Attention	Vigilance - MoCA	-0.28	-0.85-0.29	Small	Improvement
Visuo-constructional Ability	Copy phase of the MCGCF	-0.26	-0.83- -0.31	Small	Improvement
Attention	Digit Span Forwards=MoCA	-0.23	-0.80-0.34	Small	Improvement
Information Processing Speed	Coding Task	0.20	-0.39-0.76	Small	Decline
No or Very Small Change					
Orientation – Date	MoCA	-0.18	-0.40-0.74	Very Small	Improvement
Working Memory	Digit Span Backwards-MoCA	0.17	-0.40-0.74	Very Small	Decline
Motor Speed – Dominant Hand	Coin Rotation Task	-0.16	-0.76-0.43	Very Small	Improvement
Global Cognitive Function	Montreal Cognitive Assessment	0.13	-0.44-0.70	Very Small	Decline

Abstraction	Similarities-MoCA	-0.13	-0.70-0.44	Very Small	Improvement
Working Memory	Serial Sevens	-0.08	-0.64-0.49	Very Small	Improvement
Language	Naming – MoCA	0.01	-0.55-0.58	Very Small	Decline
Language	Sentence repetition – MoCA	-0.01	-0.58-0.56	Very Small	Improvement
Orientation	Month, day of the week, place and city	0	0	No Change	No Change

1. Decline on BDI-II indication higher mood.

As the AMI appeared to be sensitive to change from baseline to during the course of treatment, further analysis was conducted to determine exactly which items in the AMI were most sensitive. Table 4 displays the items from the AMI and the additional items of retrograde memory from most sensitive to least.

Table 4

Change in items Assessing Autobiographical and Semantic Memory from Baseline to During the Course of ECT

<i>Question</i>	<i>Effect Size</i>	<i>Confidence Interval</i>	<i>Qualitative Size</i>	<i>Direction</i>
Sensitive to Change				
What date did you start your treatment?	0.68	0.15-1.31	Medium	Decline
Can you tell me about something that has happened in the news over the past couple of weeks?	0.65	0.12-1.28	Medium	Decline
How long have you resided at your current address?	0.61	-0.31-0.83	Medium	Decline
What did you have for dinner the night before last?	0.61	0.05-1.20	Medium	Decline
The last holiday you went on – When was this? (Date?)	0.48	-0.27-0.87	Medium	Decline
Where did you spend last Christmas?	0.44	-0.09-1.06	Medium	Decline
Can you tell me about an incident involving a relative that has happened in the past year?	0.44	-0.10-1.06	Medium	Decline
Moderately Sensitive to Change				
Can you tell me the names of three staff members of the previous hospitalisation?	0.39	-0.16-0.98	Small	Decline
Can you tell me the name of our current prime minister?	0.34	-0.20-0.95	Small	Decline
Can you tell me who you spent last Christmas with?	0.34	-0.09-1.06	Small	Decline

Can you tell me the location of the previous hospital you were admitted to?	0.31	-0.23-0.91	Small	Decline
Can you tell me your previous address?	0.28	-0.27-0.98	Small	Decline
Can you tell me about a holiday you have been on?	0.28	-0.27-0.87	Small	Decline
Can you tell me the name of the previous hospital you were admitted to?	0.27	-0.27-0.87	Small	Decline
Can you tell me the name of the US president?	0.25	-0.31-0.83	Small	Decline
Can you tell me the date you arrived at the previous hospital?	0.25	-0.32-0.82	Small	Decline
Can you tell me about an incident that occurred on your previous holiday?	0.25	-0.32-0.82	Small	Decline
Can you tell me your current address?	0.23	-0.31-0.83	Small	Decline
Tell me about something that you did last week?	0.22	-0.77-0.37	Small	Decline
No or Very Small Change				
Who were you with on this previous holiday?	0.15	-0.42-0.72	Very Small	Decline
Can you tell me the location of this previous hospital?	0.13	-0.42-0.72	Very Small	Decline
Can you tell me the names of three staff members from this hospital?	0.02	-0.59-0.55	No Change	No Change
Can you tell me the name of the hospital you are in?	0.00	-	No Change	No Change

Table 5 displays changes in cognitive function which occurred from during the course of ECT to the six week follow-up. It was predicted that all cognitive decline that occurred during the course of ECT should resolve back to the baseline level of cognitive functioning or improvement from baseline was expected. The results largely supported this prediction as the table is manifested by improvements in performances across the majority of cognitive domains assessed. This table mainly represents recovery of cognitive function which occurred from the treatment up until six weeks following cessation of the course of treatment.

All large to medium effects occurred in a positive direction, indicating a significant improvement in functioning for visual and verbal memory (short term and delayed), working memory, verbal fluency, abstraction and information processing speed since ceasing ECT

treatment. Depressive and manic symptomatology increased indicating a relapse in symptoms; however, this effect was small. Small declines occurred in performance in motor speed and working memory. Measures of attention, language and orientation appeared to be insensitive to change during this treatment phase.

Table 5

Effect Sizes of Cognitive Change which occurred from During the Course to Follow-up

<i>Variable</i>	<i>Assessment Measure</i>	<i>Effect Size</i>	<i>95% CI Lower-Upper</i>	<i>Qualitative Size</i>	<i>Direction</i>
Highly Sensitive to Change					
Visual Memory	Delayed Recall – (30min) MCGCF	-1.07	-1.69 --0.44	Large	Improvement
Short term Verbal Memory	MoCa – delayed recall phase (5 min delay)	-0.84	-1.44 --0.21	Large	Improvement
Working Memory	Digit Span Backwards-MoCA	-0.70	-1.31--0.08	Large	Improvement
Sensitive to Change					
Long term Verbal Memory	Delayed Recall (30 min)- MoCA	-0.67	-1.28 --0.05	Medium	Improvement
Mania	Young Mania Rating Scale	-0.49	-1.09 -0.13	Medium	Improvement
Retrograde Amnesia	Autobiographical Memory Interview	-0.48	-1.08-0.13	Medium	Improvement
Verbal Fluency	MoCA	-0.48	-1.08 -0.13	Medium	Improvement
Abstraction	Similarities - MoCa	-0.84	-1.44 --0.21	Medium	Improvement
Information Processing Speed	Coding Task	-0.41	-1.02 -0.21	Medium	Improvement
Moderately Sensitive to Change					
Global Cognitive Function	Montreal Cognitive Assessment	-0.37	-0.97 -0.24	Small	Improvement
Depression	Beck Depression Inventory II	-0.30	-0.90 -0.30	Small	Improvement
Orientation – Month	MoCA	-0.23	-0.83 -0.37	Small	Improvement
Motor Speed – non dominant hand	Coin Rotation Task	0.22	-0.42 -0.85	Small	Decline
Working Memory	Serial Sevens	0.20	-0.40 -0.80	Small	Decline
No or Very Small Change					

Orientation – Day of week	MoCa	-0.19	-0.79 -0.42	Very Small	Improvement
Visuo-constructional Ability	Clock drawing task – MoCA	-0.19	-0.79 -0.42	Very Small	Improvement
Language	Sentence repetition - MoCA	-0.18	-0.78 -0.43	Very Small	Improvement
Attention	Digit Span Forwards-MoCA	-0.17	-0.77 -0.44	Very Small	Improvement
Attention	Vigilance – MoCA	0.16	-0.44 -0.76	Very Small	Decline
Visuo-constructional Ability	Shape Copy – MoCA	0.15	-0.45 -0.75	Very Small	Decline
Visuo-constructional ability	Copy phase of the MCGCF	-0.12	-0.72 -0.48	Very Small	Improvement
Orientation – Date	MoCA	0.08	-0.52 -0.68	Very Small	Decline
Motor Speed – Dominant Hand		0.08	-0.55-0.71	Very Small	Decline
Language	Naming - MoCA	0.05	-0.56-0.65	Very Small	Decline
Orientation Year, Place, City	. MoCA	0	0-0	No Change	No Change

Table 6 displays the changes in cognitive function which occurred from baseline to follow-up. The only large effect size computed from baseline to follow-up, was for depression, indicating superior treatment efficacy for the treatment of depression; an improvement which was maintained at follow-up. Medium improvements occurred for working memory, visuo-constructional ability and attention.

For individuals receiving ECT for bipolar disorder, a medium decline in manic symptomatology remained from baseline to follow-up. Small improvements from baseline occurred in patients' delayed visual memories, autobiographical retrograde amnesia, visuoconstructional ability, sentence repetition global cognitive functioning as measured by the MoCA, and delayed verbal memory. Short term verbal memory, attention as measured by vigilance component of the MoCA, orientation to time and place, motor speed verbal fluency, language and orientation endured only very small change from baseline to follow-up and were insensitive to change.

Again, working memory was more sensitive to change as measured by digit span backwards, than by serial sevens at this assessment phase.

Table 6

Effect sizes of Cognitive Change which occurred from Baseline to Follow-up

<i>Variable</i>	<i>Assessment Measure</i>	<i>Effect Size</i>	<i>95% CI Lower-Upper</i>	<i>Qualitative Size</i>	<i>Direction</i>
Highly Sensitive to Change					
Depression	Beck Depression Inventory	1.26	0.41-2.03	Large	Decline
Sensitive to Change					
Working Memory	Digit Span Backwards-MoCA	-0.69	-1.43-0.10	Medium	Improvement
Mania	Young Mania Rating Scale	0.51	-1.21-2.02	Medium	Decline
Abstraction	Similarities-MoCA	-0.45	-1.19-0.32	Medium	Improvement
Visuo-constructional Ability	Shape Copy – MoCA	-0.45	-1.91-1.06	Medium	Improvement
Visuo-constructional Ability	Clock drawing task – MoCA	-0.42	-1.16-0.34	Medium	Improvement
Attention	Digit Span Forwards	-0.41	-1.14-0.36	Medium	Improvement
Moderately Sensitive to Change					
Visual Memory	Delayed Recall – (30min) MCGCF	-0.39	-1.13-0.37	Small	Improvement
Retrograde Amnesia	Autobiographical Memory Interview	-0.33	-1.07-0.42	Small	Improvement
Visuo-constructional ability	Copy phase of the MCGCF	-0.30	-1.04-0.45	Small	Improvement
Orientation – Date	MoCA	0.30	-0.46-1.04	Small	Decline
Information Processing Speed	Coding Task	0.26	-0.50-1.00	Small	Decline
Language	Sentence repetition – MoCA	-0.21	-0.95-0.54	Small	Improvement
Global Cognitive Function	Montreal Cognitive Assessment	-0.20	-0.94-0.55	Small	Improvement
Long term Verbal Memory	Delayed Recall (30 min)- MoCA	-0.20	-0.94-0.55	Small	Improvement
No or Very Small Change					
Short term Verbal Memory (5 min delay)	MoCa – delayed recall phase	-0.19	-0.93-0.56	Very Small	Improvement

Attention	Vigilance – MoCA	-0.16	-0.90-0.59	Very Small	Improvement
Orientation – Year	MoCA	-0.16	-0.90-0.59	Very Small	Improvement
Working Memory	Serial Sevens- MoCA	0.11	-0.63-0.85	Very Small	Decline
Motor Speed – Dominant Hand	Coin Rotation Task	-0.10	-0.86-0.68	Very Small	Improvement
Motor Speed – Non-Dominant Hand	Coin Rotation Task	-0.07	-0.84-0.70	Very Small	Improvement
Verbal Fluency	MoCA	0.06	-0.68-0.80	Very Small	Decline
Language	Naming – MoCA	0.06	-0.69-0.80	Very Small	Decline
Orientation to month, day of the week, place, city	MoCA	0.00	-0.95-0.54	No Change	No Change

Depression

The average Beck Depression Inventory at baseline was: 39.80 ($SD=9.33$) indicating severe depression, during the assessments 24.32 ($SD=12.00$) indicating moderate depression, and at follow-up, the average score was 28.07 ($SD=13.47$) also indicating moderate depression.

Subjective Cognitive Function

Patients reported increasing subjective cognitive improvement throughout the course of treatment as measured by the SSMQ. SSMQ scores improved from baseline ($M=67.53$, $SD=16.91$) to during the treatment ($M=72.42$, $SD=20.83$, $d=-0.26$), and again from during the course to the six week follow-up ($M=81.08$, $SD=15.82$, $d=-0.47$). This improvement in subjective memory may have been the result of the alleviation of subjective depression.

The Relationship between Subjective and Objective Cognitive Function

To test whether there was a relationship between patients' objective performances on cognitive tests and their subjective reports of cognition, Pearson's correlation coefficients were conducted between SSMQ scores and scores on various cognitive tests. However, it must be noted that due to the large number of analyses conducted, it would be predicted that just by chance alone that some of the correlations produced would be statistically significant.

Table 7

Pearson's Correlation Coefficients between Subjective Memory Scores (SSMQ) and Objective Cognitive Outcome at Baseline, During the Course and at a Six Week Follow-Up

Cognitive Domain	Baseline	During	Follow-Up
Global Cognitive Function (MoCA)	.26	-.14	.14
Information Processing Speed (Coding Task)	.44	-.17	-.41
Retrograde Memory (AMI)	.55*	.09	.41
Short Term Verbal Memory (MoCA)	.45	-.06	.16
Long Term Verbal Memory (MoCA)	.42	-.06	-.06
Long Term Visual Memory (Complex Figure Recall)	.50	.10	-.10
Verbal Fluency (Letter Fluency)	.27	-.36**	.08
Attention (DSF)	.23	-.28	-.25
Working Memory (DSB)	.36	-.25	-.29
Motor Speed Non-Dominant (CRT)	.59*	-.44**	-.38
Motor Speed Dominant (CRT)	.33	-.47**	-.32
Orientation (Date)	.32	.31*	.03
Visuoconstructional Ability (Clock Drawing Task)	.04	-.15	.28
Divided Attention (trails MoCA)	.84*	-.08	.31
Language (Naming MoCA)	-.22	.11	.13

Note: * Correlation is significant at the 0.05 level (2 tailed).

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

Observations of the correlations across the different treatment phases suggest that at baseline, the correlations (although most did not reach the $\alpha 0.05$ level of statistical significance), are positive and stronger than the correlations observed during the other two phases. The positive correlations at baseline suggest that patients' subjective reports of cognitive function are congruent with their objective cognitive performances; patients may rate their memory as poor and achieve low scores on tests of memory. Interestingly, during the course of treatment, the majority of the correlations are negative, indicating that patients' reports of subjective function are incongruent with their objective performances; patients rated their subjective cognitive function as high despite their objective performances on tests being low. At the follow-up assessment, correlations between objective and subjective cognitive function remained mainly weak; however, a mixture of negative and positive correlations were found.

Single Case Analysis

To visually present how each domain of cognitive function changed for an individual over the course of their treatment as a function of changes in their mood, all scores for each domain of function were standardised based on the individual's average cognitive performance across all of their assessments. This allowed for the presentation and comparison of data on a single

graph. Participants' baseline assessments were not selected as point to compare subsequent assessments as only one baseline assessment could be conducted prior to starting the course of ECT and therefore using an average was seen as a more conservative approach. In addition, standardising the baseline scores as $M=0$, $SD=1$ would give the false impression that the baseline score was the individual's 'normal' performance, when in fact, prior to receiving ECT cognition was commonly affected by depression, and an individual's performance on measures of cognitive function were often well below their average performance. Therefore, all changes presented on the graphs should be interpreted as change relative to the individual's average performance rather than their change from baseline.

Changes in mood and cognitive function are presented for each case report. The cognitive domains represented on the graphs for individual analysis are: Global Cognitive Function (GCF) as measured by the MoCA; visual memory, as measured by the delayed recall phase of the Medical College of Georgia Complex Figure; Verbal Memory; as measured by a delayed recall phase of the words presented in the MoCA; Processing Speed, as measured by the RBANS Coding Task; Verbal Fluency, as measured by the MoCA; and Working Memory, as measured by the extended Digit Span Backwards task found within the MoCA. The descriptive changes reported in the tables are based on percentage changes of the individual's raw test scores from the first cognitive assessment prior to ECT (baseline) to the average performance during the course, and from the first assessment to follow-up. Raw BDI-II scores are also included in parentheses above the data points.

Case Study One

Participant One (P1), a 78 year old male received one course (consisting of 8 treatments) of ECT as an inpatient to treat major depressive disorder. ECT was administered biweekly and the electrode placement was right unilateral. P1's cognitive and clinical changes occurring over the course of treatment are presented visually in Figure 1 and descriptively in Table 8.

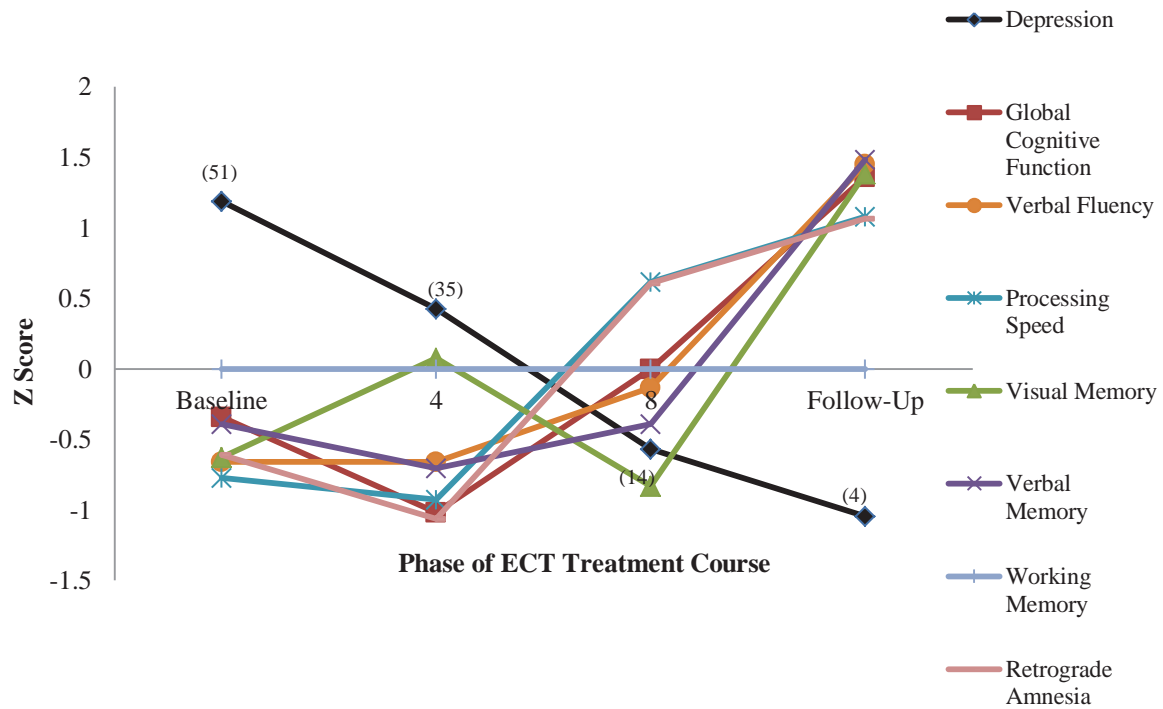


Figure 1. Standardised changes in scores of depression and performance on a test of global cognitive functioning, verbal fluency, processing speed, visual and verbal memory, working memory and retrograde amnesia prior to ECT treatment, over the course of 8 treatments of ECT, and at a six week follow-up.

Table 8

PI's Cognitive and Clinical changes over a course of ECT and at a Six Week Follow-Up

Modality Assessed	Change baseline to during the course*	Direction	Change Baseline to Follow-Up	Direction
Depression	51.96%	Decline	92.5%	Decline
Global Cognitive Function	2.27%	Decline	22.72%	Improvement

Verbal Fluency	4.17%	Improvement	33.33%	Improvement
Processing Speed	25%	Improvement	75%	Improvement
Visual Memory	10.42%	Improvement	83.33%	Improvement
Working Memory	No Change	No Change	No Change	No Change
Verbal Memory	12.5%	Decline	150%	Improvement
Retrograde Amnesia	10.86%	Improvement	47.82%	Improvement

Summary of P1

ECT was effective in treating depression severity progressively over the course of treatment, and this improvement in mood remained at the six week follow-up. At the baseline assessment, all domains of cognition assessed except working memory were below P1's average cognitive function, this impairment was most likely due to the impact of depression as depression steadily decreased throughout the course, and cognition improved across all domains. After the final treatment of ECT, some cognitive impairment existed for visual and verbal memory, and verbal fluency; however, at the six week follow-up, marked cognitive improvement had occurred as demonstrated by the steep inclines presented on Figure 1 from treatment 8 to follow-up. At the follow-up assessment, all domains of cognitive function assessed showed an improvement from the baseline level of cognitive function (see Table 8).

Case Study Two

Participant Two (P2), a 56 year old male received two courses of ECT as an inpatient to treat major depressive disorder. The first course consisted of 22 treatments, the second of 10. No baseline cognitive assessment could be conducted prior to commencing the first course of ECT due to the urgency in starting the treatment. The first course of ECT was administered right unilaterally. A weak treatment response resulted in the shift of electrode placement to bilateral for the second course of treatment. P2's cognitive and clinical changes which occurred over the two courses of treatments are presented visually in Figure 2 and descriptively in Tables 9 and 10.

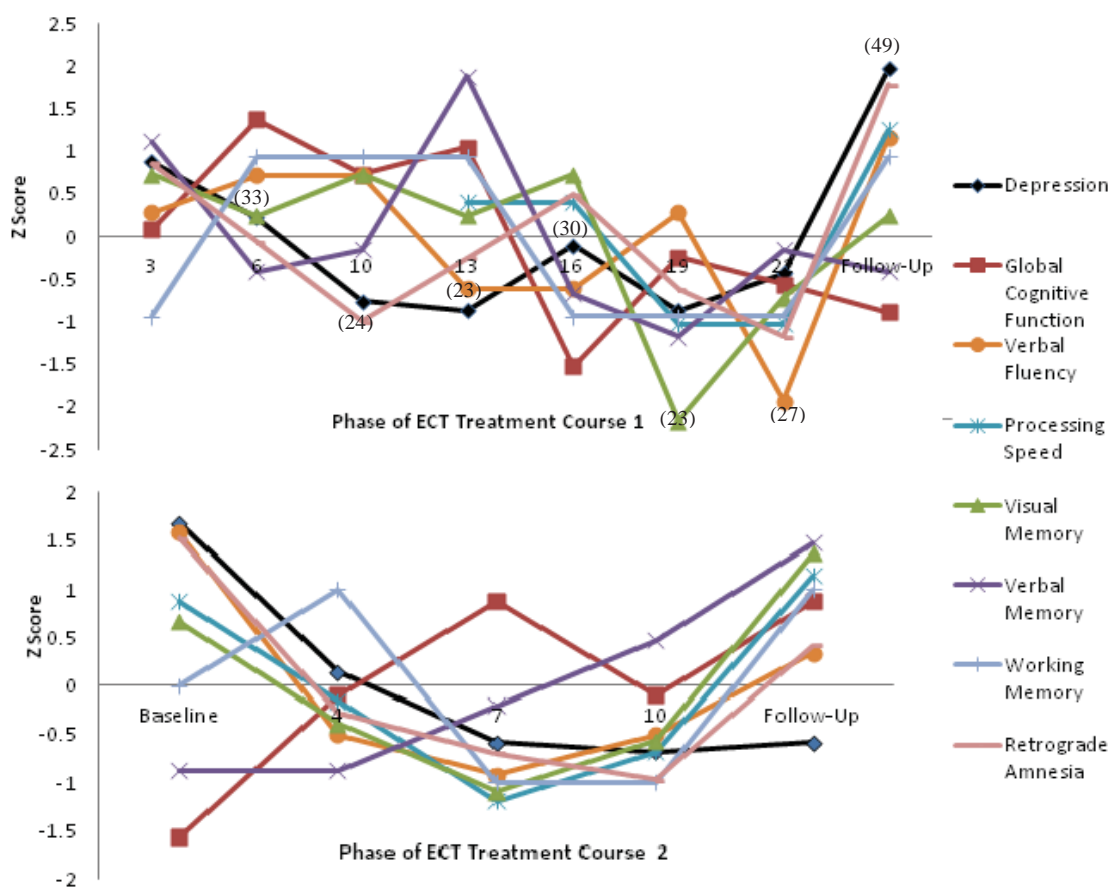


Figure 2. Standardised changes in scores of depression and performance on a test of global cognitive functioning, verbal fluency, processing speed, visual and verbal memory, working

memory and retrograde amnesia prior to ECT treatment, over two courses of ECT treatments, and at six week follow-ups.

Table 9

P2's Cognitive and Clinical changes over a the first course of ECT and at a Six Week Follow-Up

Modality Assessed	Change from third* ECT to during the course	Direction	Change third ECT to Follow-Up	Direction
Depression	31.62%	Decline	25%	Increase
Global Cognitive Function	0.79%	Improvement	14.26%	Improvement
Verbal Fluency	31.37%	Decline	7.14%	Decline
Processing Speed	13.16%	Decline	15.79%	Improvement
Visual Memory	17.09%	Decline	7.15%	Decline
Working Memory	50%	Improvement	100%	Improvement
Verbal Memory	48.33%	Decline	60%	Decline
Retrograde Amnesia	29.93%	Decline	18.52%	Improvement

Note: * As no baseline assessment was able to be conducted prior to the first course of ECT, the third treatment was used as a benchmark comparison for subsequent cognitive change. It is likely that after the third treatment, some cognitive impairment would already exist; therefore the percentages offered in Table 9 are likely to be a more conservative indication of the amount of cognitive change which would have occurred than if a baseline assessment would have been conducted.

Table 10

P2's Cognitive and Clinical changes over the second course of ECT and at a Six Week Follow-Up

Modality Assessed	Change from baseline ECT to during the course	Direction	Change Baseline to Follow-Up	Direction
Depression	87.08%	Decline	95.92%	Decline
Global Cognitive Function	20.37%	Improvement	37.78%	Improvement
Verbal Fluency	44.44%	Decline	25%	Decline
Processing Speed	27.27%	Decline	4.54%	Improvement
Visual Memory	29.49%	Decline	15.38%	Improvement
Working Memory	16.67%	Decline	50%	Improvement
Verbal Memory	50%	Improvement	175%	Improvement
Retrograde Amnesia	48.95%	Decline	25%	Decline

Summary of P2

As demonstrated by Figure 2 and Tables 9 and 10, depression decreased over both courses of ECT; however, there was a relapse of depressive symptoms following the first course of ECT, and a sharp spike in depression scores at the follow-up assessment. Despite the worsening of depression symptomatology, cognitive function did show an improvement for global cognitive function, processing speed, working memory and retrograde amnesia. Verbal fluency, visual memory and verbal memory did not revert back to baseline level of function, and it is unclear whether this is due to the level of depression P2 was experiencing at this assessment, residual effects from the ECT, or both. A visual comparison of course 1 and course 2 demonstrates that for P2, bilateral ECT was more effective in relieving depression which was maintained at the six week follow-up.

Case Study Three

Participant Three (P3), a 36 year old female received one course consisting of 12 treatments of ECT as an outpatient to treat major depressive disorder. ECT was administered biweekly and the electrode placement was right unilateral. P3's cognitive and clinical changes which occurred over the course of treatment are presented visually in Figure 3 and descriptively in Table 11.

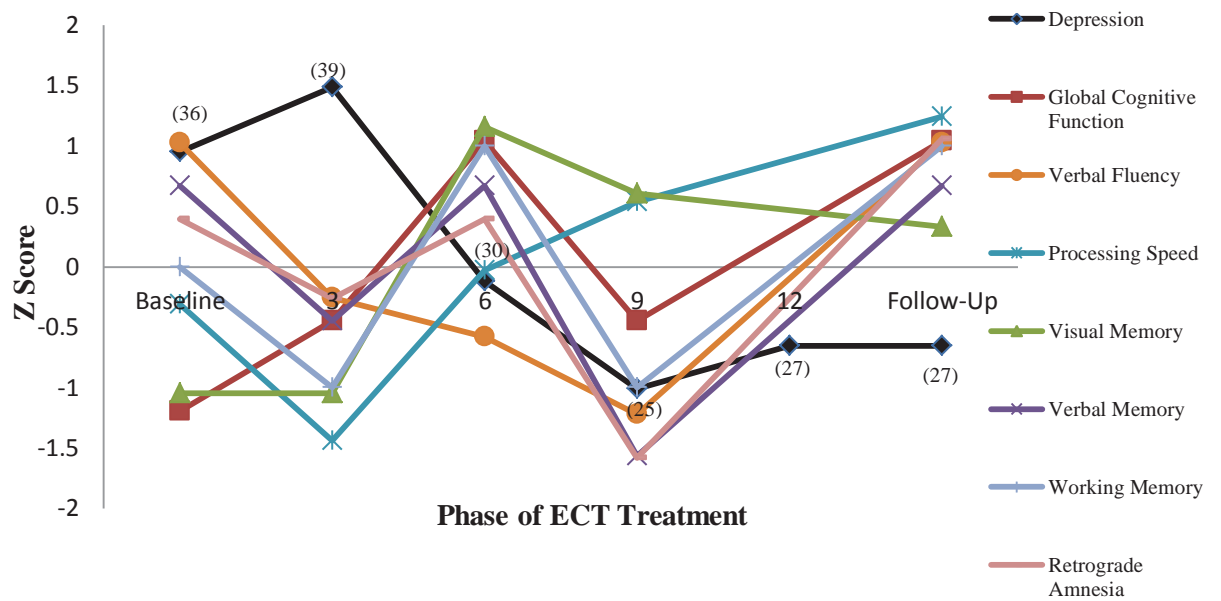


Figure 3. Standardised changes in scores of depression and performance on a test of global cognitive functioning, verbal fluency, processing speed, visual and verbal memory, working memory and retrograde amnesia prior to ECT treatment, over the course of 12 treatments of ECT, and at a six week follow-up.

Table 11

P3's Cognitive and Clinical changes over a course of ECT and at a Six Week Follow-Up

Modality Assessed	Change from baseline to during the course	Direction	Change Baseline to Follow-Up	Direction
Depression	15.97%	Decline	25%	Decline
Global Cognitive Function	6.41%	Improvement	11.5%	Improvement
Verbal Fluency	31.37%	Decline	No Change	No Change
Processing Speed	No Change	No Change	20%	Improvement
Visual Memory	17.28%	Improvement	18.5%	Improvement
Working Memory	11.13%	Decline	25%	Improvement
Verbal Memory	6.67%	Decline	No Change	No Change
Retrograde Amnesia	4.04%	Decline	3%	Improvement

Summary of P3

ECT was effective in treating depression severity progressively over the course of treatment for P3. From the cognitive assessment conducted prior to conducting the course of ECT, it is evident that some cognitive impairment existed prior to starting the course of ECT due to the effects of depression. Cognitive decline occurred after the sixth treatment of ECT; however, at the six week follow-up, all domains of cognitive function assessed had improved beyond the baseline level of cognitive function or had reverted back to the baseline scores. At this assessment point, clinical benefits of ECT on mood were maintained.

Case Study Four

Participant Four (P4), a 39 year old male received one course consisting of 15 treatments of ECT as an outpatient to treat bipolar-I disorder. ECT was administered biweekly and the electrode placement was right unilateral. Cognitive and clinical changes occurring over the courses of treatment are presented visually in Figure 4 and descriptively in Table 12. P4 was in a depressive state during the treatment and a hypomanic state at the baseline assessment phase and the six week follow-up.

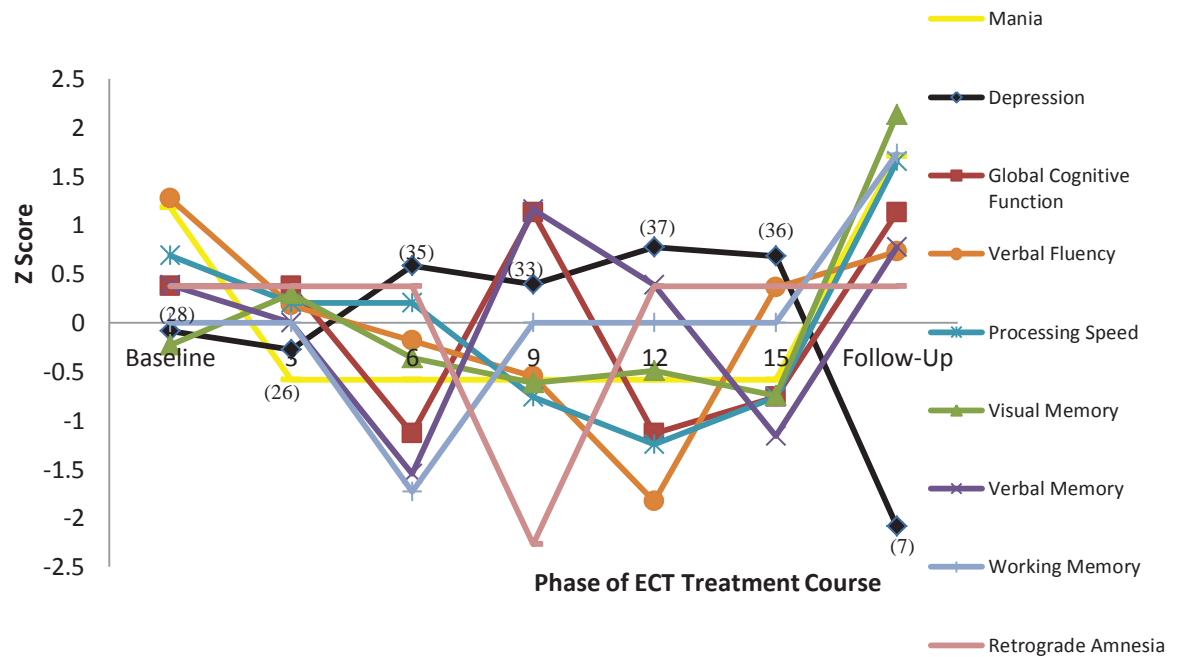


Figure 4. Standardised changes in scores of depression and performance on a test of global cognitive functioning, verbal fluency, processing speed, visual and verbal memory, working memory and retrograde amnesia prior to ECT treatment, over the course of 15 treatments of ECT, and at a six week follow-up.

Table 12

P4's Cognitive and Clinical changes over a course of ECT and at a Six Week Follow-Up

Modality Assessed	Change from baseline to during the course	Direction	Change Baseline to Follow-Up	Direction
Depression	19.28%	Increase	75%	Decline
Mania	100%	Decrease	30%	Increase
Global Cognitive Function	6.67%	Decrease	7.407%	Improvement
Verbal Fluency	40%	Decline	13.43%	Decline
Processing Speed	11.71%	Decline	9.76%	Improvement
Visual Memory	4%	Decline	60%	Improvement
Working Memory	10%	Decline	50%	Improvement
Verbal Memory	40%	Decline	13%	Decline
Retrograde Amnesia	0.58%	Decline	No Change	No Change

Summary of P4

Although ECT was not effective in decreasing depression symptomatology for P4 during the course of ECT treatment, with a slight increase in depressive symptoms over that time. At the six week follow-up assessment, depressive symptoms had been alleviated. The drastic decrease in depressive symptomatology is likely due to P4 being in a hypomanic phase of the bipolar disorder, rather than due to the clinical effects of ECT. On all domains of cognition assessed, cognitive impairment occurred during the course of treatment. At the six week follow-up, recovery in cognitive function had occurred across all domains except verbal fluency, which although was superior to P4's average performance during the course of treatment, did not revert completely back to baseline levels of cognitive function. An inverse relationship between scores of depression and mania can be seen on Figure 4, suggesting good discriminate validity of the mood measures.

Case Study Five

Participant Five (P5), a 49 year old male received one course consisting of 9 treatments of ECT as an inpatient to treat major depressive disorder. ECT was administered biweekly, and the electrode placement was right unilateral. P5's cognitive and clinical changes which occurred over the course of treatment are presented visually in Figure 5 and descriptively in Table 13.

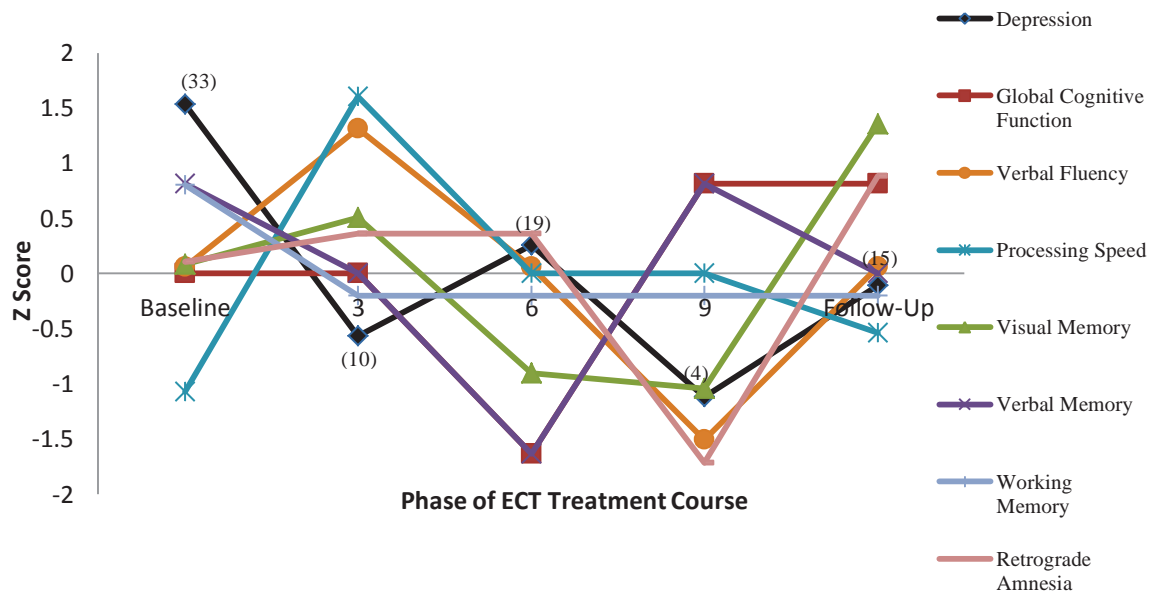


Figure 5. Standardised changes in scores of depression and performance on a test of global cognitive functioning, verbal fluency, processing speed, visual and verbal memory, working memory and retrograde amnesia prior to ECT treatment, over the course of 9 treatments of ECT, and at a six week follow-up.

Table 13

P5's Cognitive and Clinical changes over a course of ECT and at a Six Week Follow-Up

Modality Assessed	Change from baseline to during the course*	Direction	Change Baseline to Follow-Up	Direction
Depression	66.67%	Decline	54.54%	Decline
Global Cognitive Functioning	1.33%	Decline	4%	Improvement
Verbal Fluency	4.16%	Decline	No Change	No Change
Processing Speed	10%	Improvement	3.3%	Improvement
Visual Memory	21.05%	Decline	47.36%	Improvement
Working Memory	50%	Decline	50%	Decline
Verbal Memory	8.89%	Decline	6.66%	Decline
Retrograde Amnesia	5.37%	Decline	9.67%	Improvement

Summary of P5

ECT was effective in treating depression for P5, and the reduction in scores of depression remained at the six week follow-up. There was a decline in all domains of cognition assessed during the course of ECT, except for processing speed which improved slightly. At the six week follow-up, improvement beyond baseline occurred for scores of global cognitive function, processing speed visual memory and retrograde amnesia but not for working and verbal memory.

Case Study Six

Participant Six (P6), a 51 year old male received one course consisting of 11 treatments of ECT as an outpatient to treat major depressive disorder. ECT was administered biweekly and the electrode placement was right unilateral. P6's cognitive and clinical changes which occurred over the course of treatment are presented visually in Figure 6 and descriptively in Table 14. A follow-up assessment could not be conducted due to the follow-up assessment falling between Christmas and New Years day.

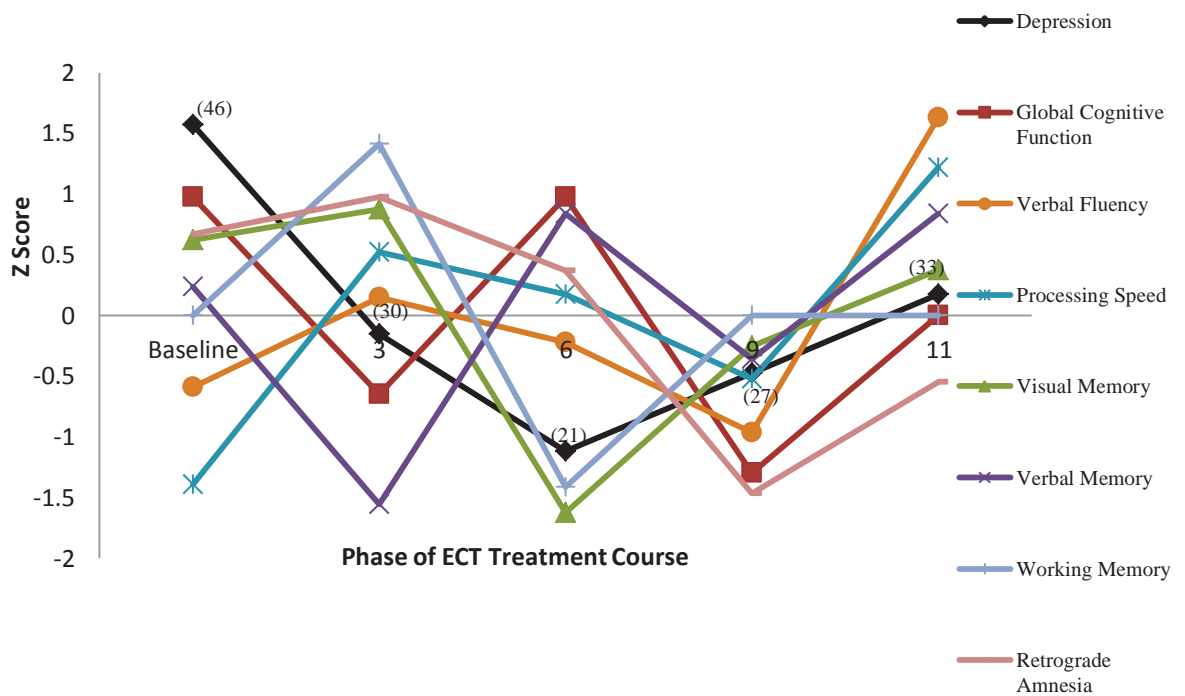


Figure 6. Standardised changes in scores of depression and performance on a test of global cognitive functioning, verbal fluency, processing speed, visual and verbal memory, working memory and retrograde amnesia prior to ECT treatment, over the course of 11 treatments of ECT.

Table 14

P6's Cognitive and Clinical changes over a course of ECT and at a Six Week Follow-Up

Modality Assessed	Change from baseline to during the course*	Direction	Change Baseline to Follow-Up	Direction
Depression	36.67%	Decline	-	-
Global Cognitive Functioning	13.39%	Decline	-	-
Verbal Fluency	16.67%	Improvement	-	-
Processing Speed	20.83%	Improvement	-	-
Visual Memory	18.38%	Decline	-	-
Working Memory	No Change	No Change	-	-
Verbal Memory	3.57%	Decline	-	-
Retrograde Amnesia	8.59%	Decline	-	-

Summary of P6

ECT was effective in relieving depression for P6; however, after the sixth treatment of ECT, scores of depression began to increase. Cognitive decline occurred during the course of treatment, with declines in global cognitive functioning, visual and verbal memory and retrograde amnesia occurring. Improvement in processing speed and verbal fluency occurred throughout the course of treatment.

Case Study Seven

Participant Seven (P7), an 18 year old female received one course consisting of 6 treatments of ECT as an inpatient to treat major depressive disorder. ECT was administered biweekly, and the electrode placement was right unilateral. P7's cognitive and clinical changes which occurred over the course of treatment are presented visually in Figure 7 and descriptively in Table 15. No follow-up cognitive assessment was conducted due to the participant relocating to the South Island of New Zealand.

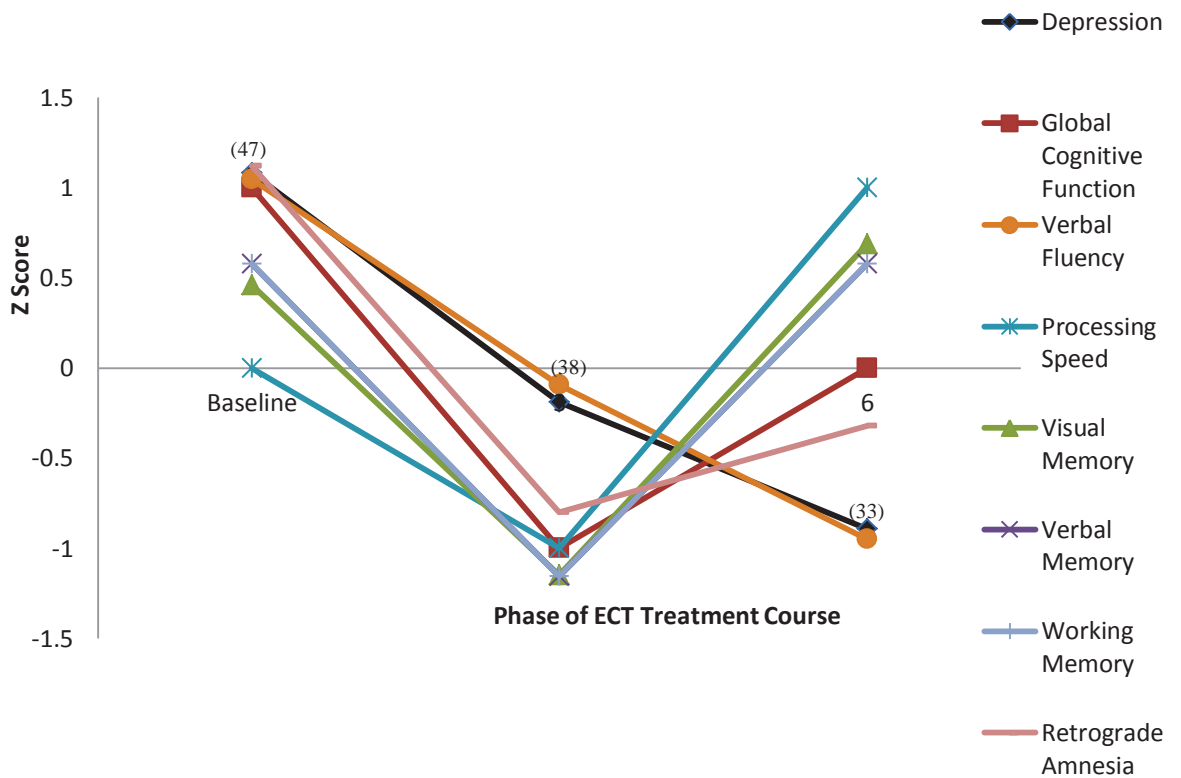


Figure 7. Standardised changes in scores of depression and performance on a test of global cognitive functioning, verbal fluency, processing speed, visual and verbal memory, working memory and retrograde amnesia prior to ECT treatment, over the course of 6 treatments of ECT.

Table 15

P7's Cognitive and Clinical changes over a course of ECT and at a Six Week Follow-Up

Modality Assessed	Change from baseline to during the course	Direction	Change Baseline to Follow-Up	Direction
Depression	32.39%	Decline	-	-
Global Cognitive Functioning	5.26%	Decline	-	-
Verbal Fluency	40.74%	Decline	-	-
Processing Speed	No Change	No Change	-	-
Visual Memory	9.36%	Decline	-	-
Working Memory	14.28%	Decline	-	-
Verbal Memory	3.45%	Decline	-	-
Retrograde Amnesia	12.28%	Decline	-	-

Summary of P7

ECT was effective in treating depression for P7, and depression scores dropped steadily after every three treatments. A decline in cognitive function was detected after the third treatment; however, the cognitive assessment conducted after the sixth treatment revealed cognitive recovery had occurred in all domains of cognitive function except verbal fluency. On average, the cognitive assessments conducted during the course of ECT showed a decline in function relative to the baseline assessment, except for processing speed which did not change.

Case Study Eight

Participant Eight (P8), a 52 year old female received ECT for major depressive disorder. P8 received three courses of ECT; the first two as an inpatient and the final course as an outpatient. The courses and treatments occurred over the course of 12 months, and each course was separated by 6 weeks or more without ECT treatment. ECT was administered right unilaterally, biweekly for the first two courses. The final course, ECT was delivered as a maintenance therapy once per week. The first course consisted of 14 treatments, the second of three and the final course, P8 received 30 treatments of maintenance ECT. No baseline cognitive assessment could be conducted prior to commencing the course of ECT, due to P8 starting her treatment prior to ethics approval for this study being obtained. P8’s cognitive and clinical changes which occurred over the courses of treatment are presented visually in Figure 8 and descriptively in Tables 16, 17 and 18.

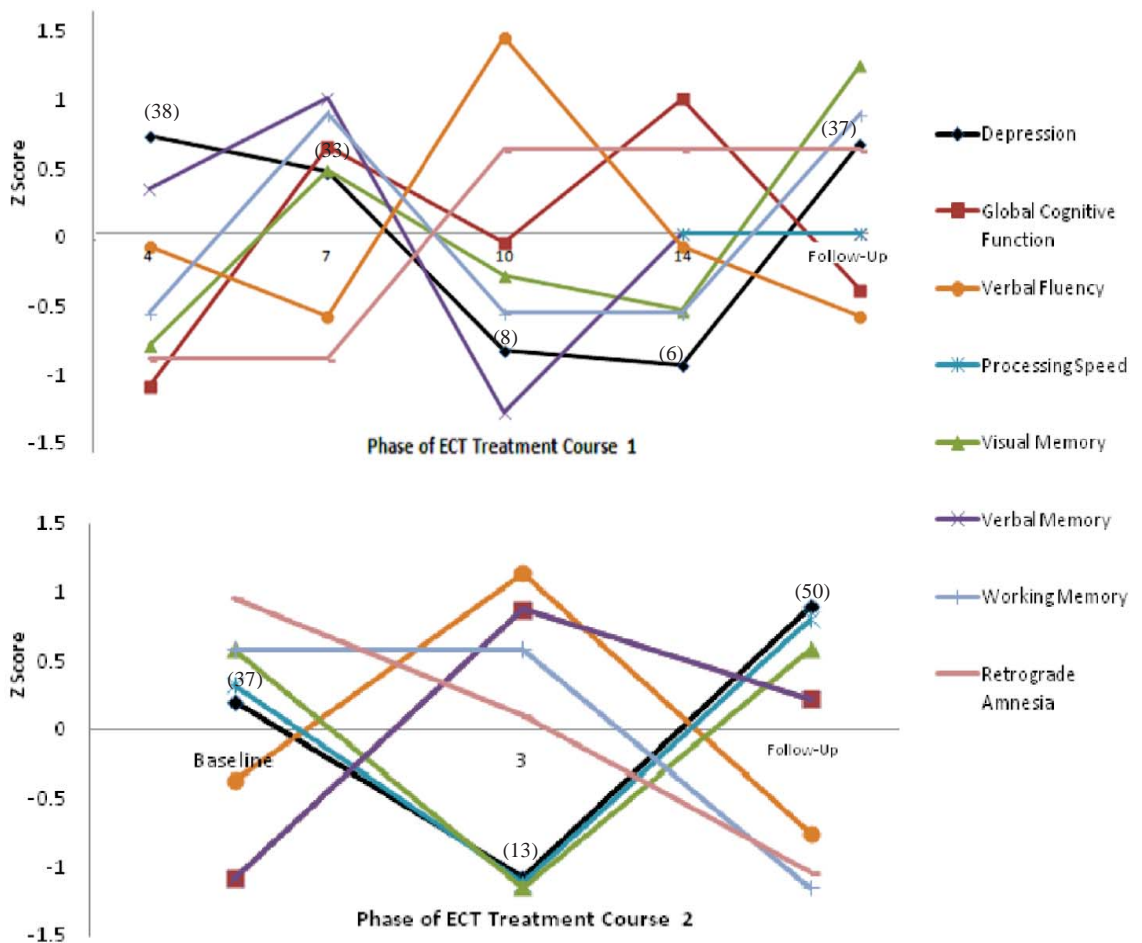


Figure 8. Standardised changes in scores of depression and performance on a test of global cognitive functioning, verbal fluency, processing speed, visual and verbal memory, working memory and retrograde amnesia prior to ECT treatment, over two courses of treatments and at six week follow-up assessments.

Table 16

P8's Cognitive and Clinical changes over the first course of ECT and at a Six Week Follow-Up

Modality Assessed	Change from fourth ECT* to during the course	Direction	Change Fourth ECT to Follow-Up	Direction
Depression	58.77%	Decline	2.63%	Decline
Global Cognitive Function	27.45%	Improvement	11.65%	Improvement
Verbal Fluency	6.67%	Improvement	10%	Decline
Processing Speed	No Change	No Change	No Change	No Change
Visual Memory	22.22%	Improvement	66.67%	Improvement
Working Memory	33.33%	Improvement	100%	Improvement
Verbal Memory	12.12%	Decline	9.09%	Decline
Retrograde Amnesia	9.87%	Improvement	14.86%	Improvement

Note: * As no baseline assessment was able to be conducted with P2 prior to commencing the first course of ECT, the fourth treatment was used to compare subsequent cognitive decline against to give an indication of cognitive change which occurred from early on in treatment to later on in treatment. It is likely that after the fourth treatment, some cognitive impairment would already exist; therefore the percentages offered in Table 16 are likely to be a more conservative indication of the amount of cognitive change which would have occurred than if a baseline assessment would have been conducted.

Table 17

P8's Cognitive and Clinical changes over the second course of ECT and at a Six Week Follow-Up

Modality Assessed	Change from Baseline to during the course	Direction	Change from Baseline to Follow-Up	Direction
Depression	64%	Decline	35.14%	Decline
Global Cognitive Function	31.57%	Improvement	21.05%	Improvement
Verbal Fluency	44.44%	Improvement	11.11%	Decline
Processing Speed	9.09%	Decline	3.03	Improvement
Visual Memory	40%	Decline	No Change	No Change
Working Memory	No Change	No Change	50%	Decline
Verbal Memory	30%	Improvement	20%	Improvement
Retrograde Amnesia	9.67%	Decline	22.58%	Decline

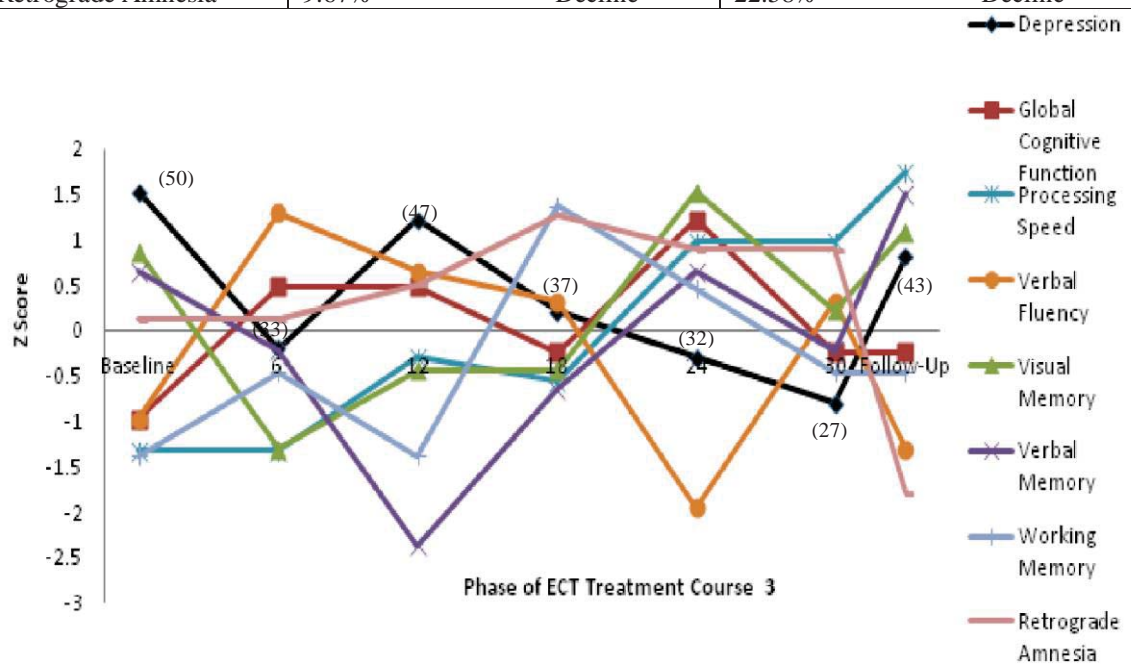


Figure 9. Standardised changes in scores of depression and performance on a test of global cognitive functioning, verbal fluency, processing speed, visual and verbal memory, working memory and retrograde amnesia prior to ECT treatment, after every six treatments of maintenance ECT, and at a six week follow-up.

Table 18

P8's Cognitive and Clinical changes over the course of maintenance ECT and at a Six Week Follow-Up

Modality Assessed	Change from Baseline to during the course	Direction	Change from Baseline to Follow-Up	Direction
Depression	34.8%	Decline	14%	Decline
Global Cognitive Function	6.52%	Improvement	4.34%	Improvement
Verbal Fluency	46.25%	Improvement	12.5%	Decline
Processing Speed	14.70%	Improvement	35.29%	Improvement
Visual Memory	24.5%	Decline	5%	Improvement
Working Memory	170%	Improvement	100%	Improvement
Verbal Memory	16.67%	Decline	16.67%	Improvement
Retrograde Amnesia	0.4%	Improvement	20.83%	Decline

Summary of P8

P8 had three courses of ECT; courses which were variable in number of treatments and frequency of administration. Figure 9 shows that in both the first and second course of treatment, ECT provided some symptom relief; however, depression scores soon increased subsequent to ceasing ECT treatment. Cognitive changes occurred during both these courses of ECT, and cognitive impairment did not revert back to the baseline level of performance for either of the follow-up assessments. This may have been due to the cognitive effects of depression, residual cognitive effects of ECT, or both. Figure 9 presents the cognitive changes which occurred during maintenance ECT. Improvements in working memory, processing speed and visual memory are apparent throughout the course. Verbal fluency and retrograde amnesia declined from during the course to follow-up which is in an unpredicted direction and may be due to the increase in depressive symptoms.

Case Study Nine

Participant Nine (P9), a 44 year old female received one course consisting of 6 treatments of ECT as an outpatient to treat bipolar I disorder. ECT was administered biweekly, and the electrode placement was right unilateral. P9's cognitive and clinical changes which occurred over the course of treatment are presented visually in Figure 10 and descriptively in Table 19.

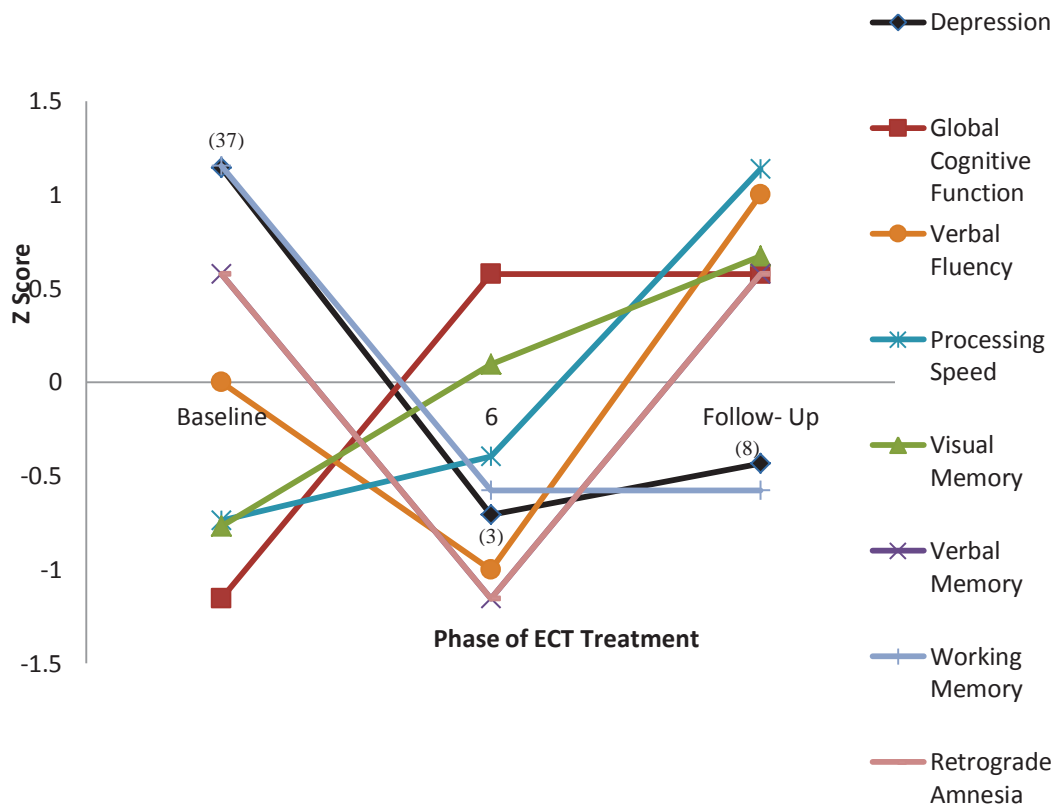


Figure 10. Standardised changes in scores of depression and performance on a test of global cognitive functioning, verbal fluency, processing speed, visual and verbal memory, working memory and retrograde amnesia prior to ECT treatment, over the course of 6 treatments of ECT and at a six week follow-up.

Table 19

P9's Cognitive and Clinical changes over a course of ECT and at a Six Week Follow-Up

Modality Assessed	Change from baseline to during the course	Direction	Change Baseline to Follow-Up	Direction
Depression	91.89%	Decline	78.37%	Decline
Global Cognitive Function	4.17%	Improvement	4.17%	Improvement
Verbal Fluency	25%	Decline	25%	Improvement
Processing Speed	9.52%	Improvement	52.38%	Improvement
Visual Memory	18.75%	Improvement	31.25%	Improvement
Working Memory	100%	Decline	100%	Decline
Verbal Memory	54.55%	Decline	No Change	No Change
Retrograde Amnesia	2.94%	Decline	No Change	No Change

Summary of P9

ECT was effective in relieving depressive symptomatology for P9 over the six treatments. The improvement in mood remained at the six week follow-up with only a slight relapse in symptoms. Cognitive function was impaired at baseline, likely due to the cognitive effects of depression. Cognitive function improved as symptoms of depression were alleviated, except for working memory which declined. Cognitive function was superior at the six week follow-up than it was at baseline, except for working memory which did not revert back to baseline level of functioning.

Case Study Ten

Participant Ten (P10), a 44 year old female received one course consisting of 9 treatments of ECT as an outpatient to treat major depressive disorder. ECT was administered biweekly, and the electrode placement was right unilateral. P10's cognitive and clinical changes which occurred over the course of treatment are presented visually in Figure 11 and descriptively in Table 20.

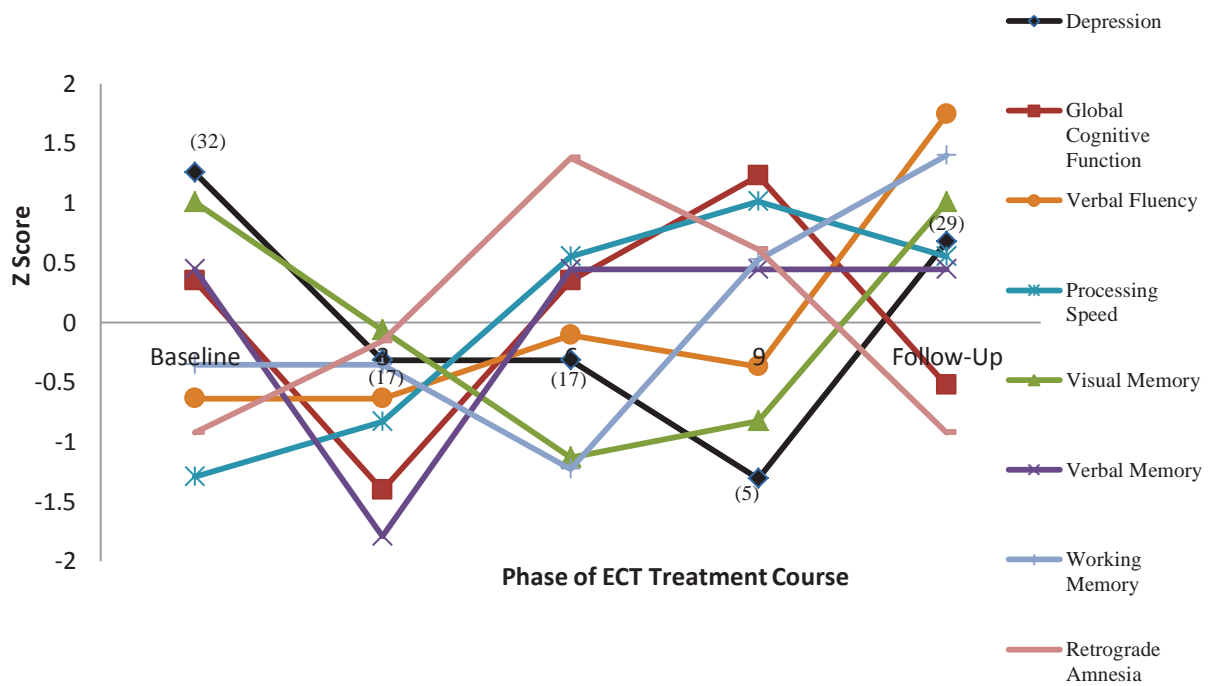


Figure 11. Standardised changes in scores of depression and performance on a test of global cognitive functioning, verbal fluency, processing speed, visual and verbal memory, working memory and retrograde amnesia prior to ECT treatment, over the course of 9 treatments of ECT and at a six week follow-up.

Table 20

P10's Cognitive and Clinical changes over a course of ECT and at a Six Week Follow-Up

Modality Assessed	Change from baseline to during the course*	Direction	Change Baseline to Follow-Up	Direction
Depression	68.89%	Decline	19.44%	Decline
Global Cognitive Functioning	1.19%	Improvement	3.57%	Improvement
Verbal Fluency	8.33%	Improvement	75%	Improvement
Processing Speed	7.092%	Improvement	8.51%	Improvement
Visual Memory	15.27%	Decline	No Change	No Change
Working Memory	No Change	No Change	100%	Improvement
Verbal Memory	13%	Decline	No Change	No Change
Retrograde Amnesia	6.89%	Improvement	No Change	No Change

Summary of P10

ECT was effective in relieving depression for P10; however, a relapse in depressive symptoms had occurred by the six week follow-up. Although a relapse had occurred, depression severity remained less than what it was at the baseline assessment. The baseline cognitive assessment revealed some cognitive decline, likely due to the effects of depression. During the course of ECT, some domains of cognition declined (delayed visual and verbal memory), and others improved (global cognitive function, verbal fluency, processing speed, retrograde amnesia). At the follow-up assessment, all cognitive domains had reverted back to the baseline level of functioning or were functioning superior to the baseline assessment.

Case Study Eleven

Participant Eleven (P11), a 51 year old female received ECT as an outpatient to treat major depressive disorder. P11 received two courses of ECT as an outpatient. The first course consisted of six treatments, the second of three. ECT was administered biweekly, and the electrode placement was right unilateral for the first few treatments of the first course and then a shift to left unilateral electrode placement was made. P11 was left hand dominant, and suffered expressive dysphasia after receiving right unilateral ECT, indicating that speech function may have been located in the right hemisphere for P11, thus a shift in electrode placement was made. This shift alleviated speech difficulties. P11's cognitive and clinical changes which occurred over the course of treatment are presented visually in Figure 12 and descriptively in Tables 21 and 22.

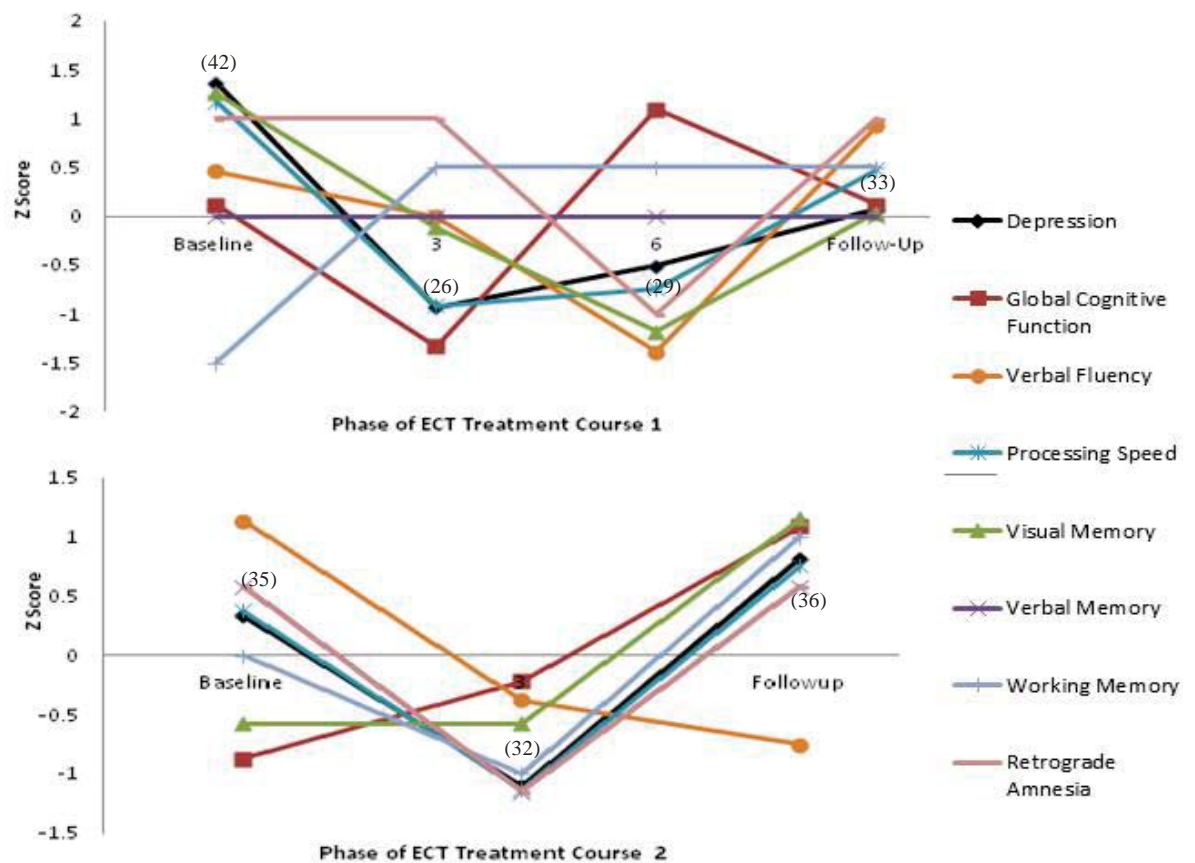


Figure 12 shows depression scores and performance on tests of global cognitive functioning,

visual and verbal memory, processing speed, verbal fluency and working memory prior to ECT treatment, over two courses of ECT treatment and at a six week follow-up.

Table 21

P11's Cognitive and Clinical changes over the first course of ECT and at a Six-Week Follow-Up

Modality Assessed	Change from baseline to during the course	Direction	Change Baseline to Follow-Up	Direction
Depression	34.52%	Decline	21.43%	Decline
Global Cognitive Function	1.92%	Decline	No Change	No Change
Verbal Fluency	15.62%	Decline	6.25%	Improvement
Processing Speed	19.49%	Decline	6.77%	Decline
Visual Memory	35.71%	Decline	22.85%	Decline
Working Memory	50 %	Improvement	50%	Improvement
Verbal Memory	No Change	No Change	No Change	No Change
Retrograde Amnesia	1.51%	Decrease	No Change	No Change

Table 22

P11's Cognitive and Clinical changes over the second course of ECT and at a Six-Week Follow-Up

Modality Assessed	Change from baseline to during the course*	Direction	Change Baseline to Follow-Up	Direction
Depression	8.5%	Decline	2.86%	Increase
Global Cognitive Functioning	3.84%	Improvement	11.54%	Improvement
Verbal Fluency	23.52%	Decline	29.41%	Decline
Processing Speed	14.54%	Decline	3.63%	Improvement
Visual Memory	No Change	No Change	29.63%	Improvement
Working Memory	33.33%	Decline	33.33%	Improvement
Verbal Memory	6.67%	Decline	No Change	No Change
Retrograde Amnesia	3.03%	Decline	No Change	No Change

Summary of P11

P11 had 9 treatments of ECT spread out over two courses. The cognitive profiles of both courses show a pattern of cognitive decline during the course of ECT, and cognitive improvement at the six week follow-up. P11 experienced a relapse in depression at the follow-up of the second course of ECT. Although depression was high, cognitive recovery still occurred, and cognitive function was at or above baseline levels of functioning for all domains except verbal fluency.

Case Study Twelve

Participant Twelve (P12), a 41 year old female received one course of ECT consisting of 12 treatments as an outpatient to treat rapid cycling bipolar-I disorder. ECT was administered biweekly and the electrode placement was right unilateral. P12's cognitive and clinical changes which occurred over the courses of treatment are presented visually in Figure 13 and descriptively in Table 23.

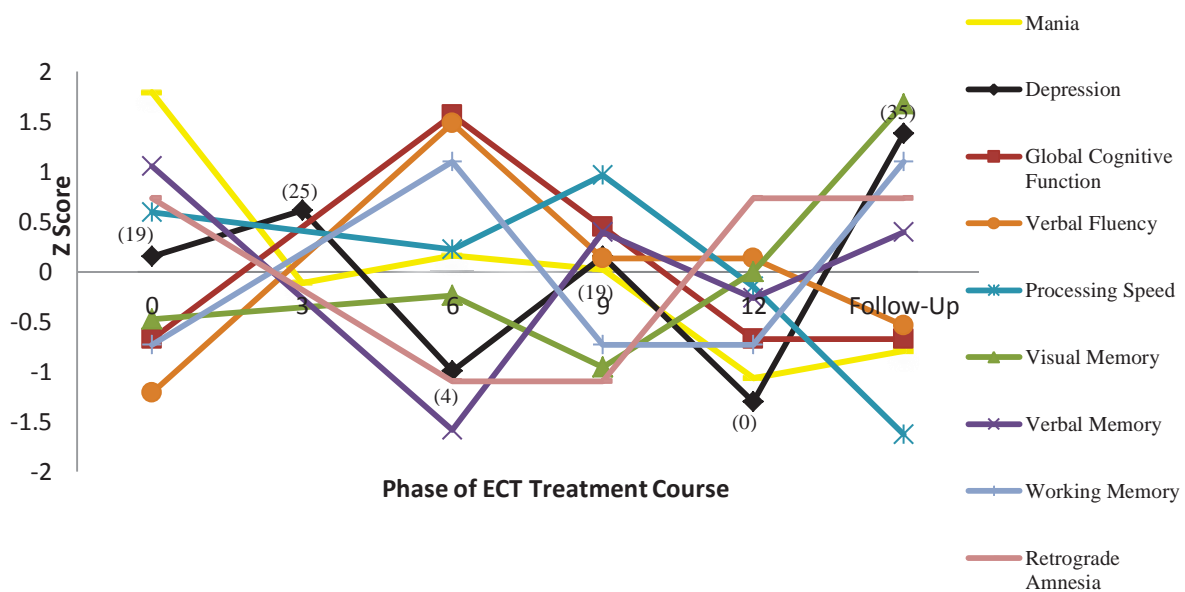


Figure 13 shows depression and mania scores and performance on tests of global cognitive functioning, visual and verbal memory, processing speed, verbal fluency and working memory prior to ECT treatment, over a course of ECT treatment and at a six week follow-up.

Table 23

P12's Cognitive and Clinical changes over the second course of ECT and at a Six-Week Follow-Up

Modality Assessed	Change from baseline to during the course*	Direction	Change Baseline to Follow-Up	Direction
Depression	36.84%	Decline	84.24%	Increase
Mania	71.49%	Decline	90.48%	Decline
Global Cognitive Function	8.67%	Improvement	No Change	No Change
Verbal Fluency	38.1%	Improvement	14.29%	Improvement
Processing Speed	1.86%	Decline	16.67%	Decline
Visual Memory	2.08%	Improvement	56.25%	Improvement
Working Memory	33.33%	Improvement	100%	Improvement
Verbal Memory	31.11%	Decline	13.33%	Decline
Retrograde Amnesia	1.96%	Decline	No Change	No Change

Summary of P12

ECT was effective in treating the manic symptoms of bipolar for P12. At baseline, P12 was in a manic phase, and her scores on the Young Mania Rating Scale decreased steadily over the course of 12 treatments. At the six week follow-up, P12 was in a depressive phase, and her symptoms of mania remained low; however, she now scored highly on the measure of depression. At the six week follow-up, processing speed and verbal memory showed a decline from baseline, all other domains of cognitive function demonstrated either no change from baseline or improvement.

Case Study Thirteen

Participant Thirteen (P13), a 44 year old female received one course consisting of 10 treatments of ECT as an outpatient to treat major depressive disorder. ECT was administered biweekly, and the electrode placement was right unilateral. P13's cognitive and clinical changes which occurred over the course of treatment are presented visually in Figure 14 and descriptively in Table 24.

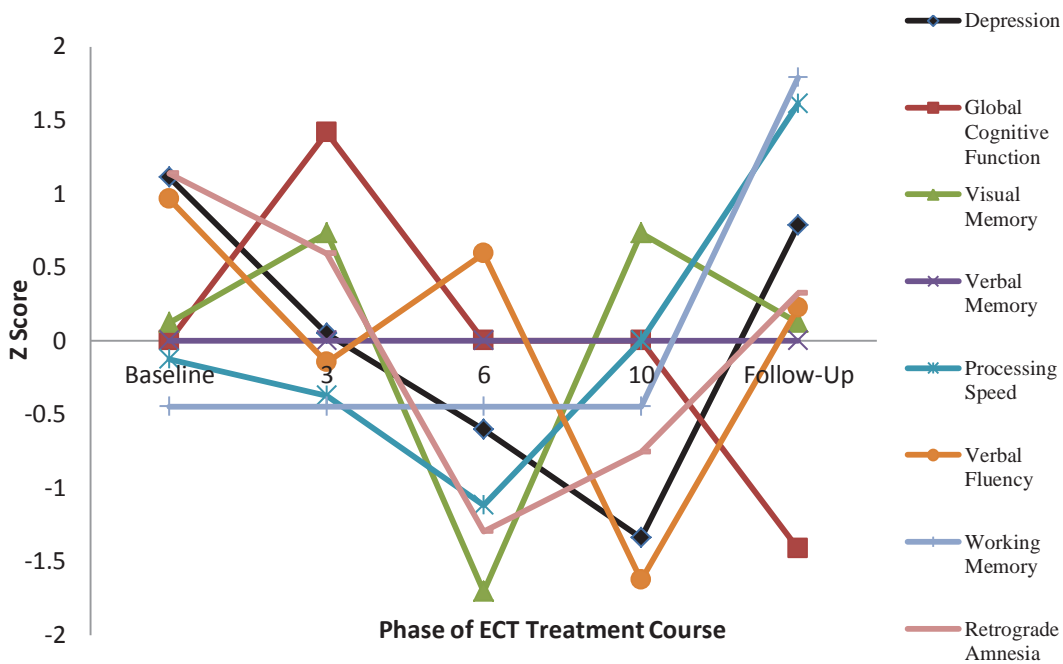


Figure 14. Standardised changes in scores of depression and performance on a test of global cognitive functioning, verbal fluency, processing speed, visual and verbal memory, working memory and retrograde amnesia prior to ECT treatment, over the course of 10 treatments of ECT and at a six week follow-up.

Table 24

Participant 13's Cognitive and Clinical changes over a course of ECT and at a Six Week Follow-Up

Modality Assessed	Change from baseline to during the course*	Direction	Change Baseline to Follow-Up	Direction
Depression	41.83%	Decline	7.84%	Decline
Global Cognitive Function	1.19%	Improvement	3.57%	Decline
Verbal Fluency	22.92%	Decline	12.5%	Decline
Processing Speed	6.52%	Decline	30.44%	Improvement
Visual Memory	4.167%	Decline	No Change	No Change
Working Memory	No Change	No Change	50%	Improvement
Verbal Memory	No Change	No Change	No Change	No Change
Retrograde Amnesia	17.64%	Decline	8.82%	Decline

Summary of P13

ECT was effective in relieving depression for P13; however, a relapse in depressive symptomatology occurred at the six week follow-up. Although a relapse in symptoms occurred, the degree of depression was not as severe as it was during the baseline cognitive assessment. Cognitive decline occurred during baseline on all cognitive domains assessed, except for global cognitive function which showed a very small improvement. At the six week follow-up, all domains of cognitive function returned to or above the baseline level of performance, except for retrograde amnesia and verbal fluency which showed a small decline in comparison to P13 baseline cognitive performance.

Discussion

The main objective of the current research was to determine which cognitive changes occurred for the current participant sample during their course/s of ECT, and to contribute to the existing literature on the nature and duration of ECT related cognitive side effects. The current

investigation also aimed to address the dearth of research into the non-memory cognitive changes which occur due to ECT by assessing a broad range of cognitive functions in addition to memory, as purely memory related changes have been a strong focus of previous research in this area. In addition to evaluating which cognitive domains were sensitive to change, the measures which detected these changes were also reported.

It was predicted that ECT would be effective at relieving mood symptoms associated with major depressive disorder and bipolar I disorder. It was also predicted that cognitive impairment from the baseline cognitive assessment would occur during the course of ECT, and that this cognitive impairment would manifest as a decline across a broad range of cognitive domains from baseline, and that a recovery in cognitive function should occur after the patients ceased their treatment with ECT. A final prediction was that objective and subjective cognitive outcome would be incongruent.

As predicted, ECT was effective in relieving depressive symptoms, and many of the patients included in the research benefited from acute decreases in depressive and manic symptomatology soon after starting the course. The group analysis showed large and medium effect sizes for the decrease in depressive and manic symptomatology respectively, and this change was reflected in most single case analyses. Comparisons of the 13 case studies and 17 courses of ECT analysed in this study revealed that although ECT was highly effective in alleviating symptoms of depression in the short term, at the six week follow-up the clinical benefits of the treatment did not remain for over half of the participants, and a significant relapse in depressive symptoms occurred for these people. This finding was masked by the group analysis which showed a small relapse in depressive symptoms at the six week follow-up assessment over all for the group.

Cognitive change due to ECT was evident, and all participants who took part in the study underwent a considerable degree of cognitive change. As seen by the single case analyses, it

was common for an individual's cognitive performance to fluctuate two to three standard deviations over the course of treatment, and across all domains of functioning represented in these analyses. This fluctuation included both cognitive decline and cognitive improvement; the improvement was likely to be due to the rapid alleviation of depression (Bayless et al., 2010) and recession of ECT related side effects. All cognitive profiles varied; however, some trends across participants were evident.

A common profile resembled a U shape with cognitive functioning being superior at the baseline assessment, decline occurring during the course and a remission back to baseline functioning at the six week follow-up assessment. This profile was roughly demonstrated by P2, P4, P5, P6, P7, P9, P10, P11, P12 and P13; although anomalies existed across these participants. When cognitive function had not resolved back to the baseline level of functioning it was still evident that some cognitive recovery had occurred, and a longer follow-up assessment may have revealed further or complete recovery. For example, P2 had a 48.95% decline in retrograde memory from baseline to during the treatment, and from baseline to follow-up a 25% decline. Although retrograde memory had not completely returned to the baseline level of functioning significant recovery had occurred.

A visual analysis of P1 demonstrates how cognitive function can be impaired by depression prior to starting a course of ECT and how the relief of depression can positively affect cognitive function. From baseline to the final treatment of ECT, cognitive function remained relatively stable for P1. Expected cognitive decline from ECT did not objectively occur for P1; however, any decline caused by the treatment may have been masked by the rapid alleviation of depression resulting in a alleviation of the cognitive effects of the illness. Thus two opposing parallel processes were likely occurring resulting in no objective cognitive change; cognitive decline due to ECT and cognitive improvement due to the lifting of depression. From the final treatment of ECT to the six week follow-up, a great improvement in cognitive function occurred, which was likely to be the result of two factors: recovery of any cognitive decline

over time since treatment, and the influence of depression on cognitive function being alleviated. P3 presented a similar cognitive profile manifested by cognitive impairment at baseline due to high scores of depression, and as depression was alleviated cognitive function improved. At the six week follow-up, depression scores remained low, and cognitive function improved beyond the baseline level of functioning. P8 received maintenance ECT after two courses of ECT administered as usual. During the period of maintenance ECT no clear trends could be observed with P8, and her cognitive function appeared to fluctuate across assessments, as did her mood. The larger spacing between treatments during maintenance ECT may have allowed for cognitive recovery to occur between the treatments, resulting in less cognitive impairment occurring from baseline.

The group analyses revealed a trend reflective of many of the single case analyses. Cognitive decline during the treatment was common, and a recovery of function returning to the baseline level or superior to baseline performance at the follow-up assessment was apparent for most cognitive domains. The improvements which occurred from baseline to follow-up as shown by Table 5 are likely to be due to both the alleviation of depression and recovery of cognitive decline which occurred during the ECT. This is further evidence to suggest that both ECT and depression are responsible for cognitive decline, and the alleviation of depression in addition to time lapsing from treatment promotes a recovery in cognitive functioning. A large decline occurred in retrograde memory from baseline to during the course of treatment, and this finding is consistent with previous studies investigating the cognitive effects of ECT (Ingram et al., 2008; Rami-Gonzalez et al., 2001; Sackeim et al., 2007).

Short term and delayed verbal memory and delayed visual memory were also sensitive to decline from baseline to during the course of treatment, as was the ability to copy a shape. Shape copy may be impaired during the course of ECT, as the electrodes were placed on the right side of the head, and visuospatial ability is most commonly situated in the right hemisphere (Squire & Slater, 1978). This deficit, however, did not persist into the long term.

Verbal fluency and information processing speed showed small declines. Some improvements occurred from baseline to during the course; however, these improvements are not likely to be due to the ECT treatment, but to the alleviation of depression. The aforementioned cognitive declines occurred despite the alleviation of depression. Hypothetically, if the research had a control group matched on all variables except for depression, it is likely that the non-depressed group would show even greater cognitive declines than the depressed group, as the depressed group benefits from cognitive improvement from the alleviation of depression.

The cognitive changes which occurred during the course of treatment to follow-up were largely in the positive direction indicating cognitive recovery across a broad range of cognitive domains in the six weeks following ECT treatment. All changes which elicited large and medium effects were in the positive direction. After finishing the course of ECT, participants in the current sample experienced a large or medium recovery across the following domains: visual memory, short term verbal memory, working memory, delayed verbal memory, retrograde amnesia, verbal fluency, abstraction and information processing speed. Previous research has found that ECT does not affect abstract reasoning (Tiller & Lyndon, 2003). Insensitive to change from during the course to follow-up was orientation to date, year, place or city, language tasks, tests of attention, visuoconstructional ability and motor speed.

Perhaps of highest clinical importance, are the changes which occurred from baseline to six week follow-up assessment, as it is these changes are considered the *long term* cognitive effects of the treatment. These changes are of great clinical importance as longstanding cognitive impairment is a concerning and distressing side effect of ECT and also restrict its use. However, the current research findings are reflective of most other outcomes which have been produced since the change from using sine wave electricity to brief pulse electricity. That is, the finding that the cognitive changes of ECT are short term, and no longstanding effects of the treatment occur (Bayless et al., 2010; Semkovska & McLoughlin, 2010). The group cognitive profile was manifested by cognitive decline during the course of treatment and improvements across

cognitive functions beyond baseline for most domains assessed. Medium improvements in cognitive performance occurred for working memory, abstraction, visuo-constructional ability and attention. Small improvements occurred in retrograde amnesia, language, global cognitive function and delayed verbal memory. These results were consistent with the meta-analysis conducted by Semkowska and McLoughlin (2010) who also found that no cognitive deficits persisted beyond 15 days after ECT, and a small-medium improvement occurred beyond baseline.

The following cognitive domains showed no change from the baseline assessment to follow-up: short term verbal memory, attention, orientation to year, month, day of the week, place and city, working memory as measured by serial sevens task, motor speed, verbal fluency and language. The only domain in cognitive function which showed a decline from baseline to the six week follow-up assessment was orientation to date and information processing speed, and the effect sizes for both these declines were small. It has been well established that information processing speed is sensitive to the effects of mild traumatic brain injury (Schoenberg & Scott, 2011) and the finding that information processing speed was still slightly impaired six weeks after the course of ECT may be due to that cognitive domain being especially sensitive to the effects of ECT. This finding differed to Semkowska and McLoughlin's study, as they found a small improvement in information processing speed 15 days post treatment.

The results as determined by the group analyses support previous research which suggest that no long lasting cognitive effects of ECT occur; however, when analysing the data on a case by case basis, it is evident that for some individuals, cognitive impairment did remain at the six week follow-up. It is possible that a longer term follow-up may have resulted in complete cognitive recovery; however, further research should assess the long term outcomes of ECT (National Institute of Clinical Excellence, 2003). An implication of this is that when patients are providing informed consent for treatment; they should be warned that although most people benefit from a full recovery of cognitive function, some people do have lasting impairment at

six week follow-up. Conducting the single case analyses in addition to group analysis was beneficial in that it allowed for the different cognitive profiles to be recognised, as they would have otherwise been undetected had only the group analysis been conducted. It also allowed for the detection of changes which occurred for individuals, which were at times lost by the pooling of the data as a group.

The final prediction was that subjective and objective cognitive outcome would be incongruent. Subjective cognitive function as measured by the SSMQ improved increasingly throughout a course of ECT, and objective cognitive function declined during a course of ECT and improved again at the six week follow-up assessment. At the baseline assessment, Pearson's correlation coefficients were mainly moderate in strength and positive in direction. Although most of these correlations did not reach statistical significance, this finding is suggestive that at baseline, subjective reports of cognitive function may be a good indicator of objective cognitive performance. During the course of ECT, most correlations between the SSMQ scores and cognitive outcomes scores were weak and negative, replicating the common finding of incongruence between the two (Berman et al., 2008; Nehra, Chakrabarti, Khehra, Sharma, & Painuly, 2008; Vamos, 2008). An explanation for this incongruence may be that objective assessment is insensitive during a course of ECT or that reports of cognitive function may be elevated due to the alleviation of depression, as subjective report of cognitive function has previously been suggested to be a better indicator of mood than objective cognitive function (Coleman et al., 1996). At the follow-up assessment, a mixture of negative and positive correlations existed, which may be reflecting the return in cognitive function back to baseline level of functioning.

Limitations

There are limitations in the design of the study for both the series of case studies, and for the group analyses. A robust case study design should encompass multiple baseline assessments until a steady baseline is achieved. The longer and more stable the baseline, the more

confidence one can have in the prediction made from the data (Center & Leach, 1984). Multiple baseline assessments could not be conducted with the current population due to the limited time prior to which many people were prescribed and given the ECT. In addition, an excess of one cognitive assessment prior to starting a course of ECT could not be justified for the interest of the patient, i.e., it was deemed unethical to subject participants to cognitive assessments in excess of what was sufficient to detect cognitive change during their course of treatment. Thus conclusions reached via visual analysis of the series of case studies should be made with caution, as fluctuations at baseline may give an inaccurate representation of baseline function. Due to the small sample size, and the resultant low power, hypothesis testing could not be conducted thus limiting generalisations to the greater population; however, the findings of the current study reflected those found in studies with greater numbers of participants. Finally, without a matched control group including people not receiving ECT, it is difficult to determine how the changes in mood influenced cognitive change. Ethical and practical considerations restricted the recruitment of a matched control group, as it would have been unethical to recruit 13 participants who had the same level of depression and matched on all other variables, and not offer these people the most effective treatment for their disorder. Without having a comparable control group, it is not possible to determine whether the patients returned to their premorbid level of functioning prior to depression. Visual analysis of many of the case studies revealed that even when a person was depressed at the six week follow-up, cognitive recovery still occurred, indicating that the effects of ECT are more severe than those of depression (see for example participant 11).

There are some limitations in the assessment of autobiographical retrograde amnesia. The ability for a participant to recall whether they have forgotten something can only occur when prompted by others to remember it (Robertson & Pryor, 2006). Another challenge when assessing autobiographical memory is the inability to verify the accuracy of recall (Sobin et al., 1995). Inconsistencies in responses to the AMI from the participant's baseline reports were

taken to be instances of amnesia, although this assumes that the baseline report of autobiographical events were correct.

Implications

Previous research has found the MoCA to be more sensitive than the MMSE in detecting ECT related cognitive impairment (Luther, Leathem, & Humphries, 2012a). The current study found that the MoCa, although more sensitive than the MMSE, was only slightly sensitive to ECT related cognitive change, thus the MoCA should not be used in isolation to detect ECT related cognitive impairment. However, some components of the MoCA were more sensitive to cognitive change than others. For example, the language components did not change considerably during the course of treatment, nor did most components of orientation, abstraction and working memory as measured by the serial sevens task. The short term verbal memory component, visuconstructional ability as measured by the shape copy task and clock drawing task, orientation to year, and attention as measured by the vigilance component of the MoCA appeared to be more sensitive to detecting cognitive change which occurred from baseline to during the course of treatment. Sensitive measures of cognitive impairment from baseline to during the course included the Autobiographical Memory Interview – Section C, the delayed recall phase of the Complex Figure Task, the five minute delay for the verbal memory task within the MoCA, and the added 30 minute delay for the verbal memory assessment. Verbal fluency was moderately sensitive to change, as was information processing speed. These domains of cognitive function should be assessed during the course of ECT as they have been shown to be most sensitive to cognitive decline. Subjective report of cognitive function is incongruent with objective cognitive performance during a course of ECT but not at baseline. During a course of treatment patients may rate their cognitive function as high, despite performing low on objective measures of cognition. The high self ratings of cognitive function may be a better indication of the alleviation of depression rather than cognitive status; therefore, subjective report should not be relied on as a measure of cognitive function.

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STATEMENT OF CONTRIBUTION
TO DOCTORAL THESIS CONTAINING PUBLICATIONS

(To appear at the end of each thesis chapter/section/appendix submitted as an article/paper or collected as an appendix at the end of the thesis)

We, the candidate and the candidate's Principal Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of Candidate: Anneke Thornton

Name/Title of Principal Supervisor: Janet Leathem

Name of Published Research Output and full reference:

Short Term Cognitive Change during Electroconvulsive Therapy for Unipolar and Bipolar Depression: A Prospective Study

In which Chapter is the Published Work: Seven

Please indicate either:

- The percentage of the Published Work that was contributed by the candidate:
and / or
- Describe the contribution that the candidate has made to the Published Work:

The candidate had full responsibility for the design, data collection, analysis and write up of the research. Supervisors provided guidance and were involved in decisions made around research processes and provided input with data analysis and formatting of the thesis. For these reasons Professor Janet Leathem and Dr Ross Flett were included as co-authors for the manuscripts which comprise this thesis.

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CHAPTER EIGHT

Patients' Perspectives on the Cognitive Effects of Electroconvulsive Therapy

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Abstract

Despite extensive attempts to decrease the cognitive side effects of electroconvulsive therapy (ECT), cognitive impairment remains a common and undesirable side effect. Objective tests of cognitive assessment often fail to reflect the extent of cognitive impairment reported by patients, or fail to provide any evidence of reported cognitive decline. The aim of the current study was to investigate the nature and content of patients' subjective cognitive change throughout a course of ECT. Subjective reports of cognitive function were obtained from 13 participants prior to receiving a course of ECT, after every three treatments of ECT, and again six weeks following cessation of the course. Participants were asked about any cognitive change (negative or positive) they had experienced since having ECT, completed the Squire Subjective Memory Questionnaire and the Beck Depression Inventory-II. Subjective reports were analysed using qualitative content analysis. The types of cognitive complaints and improvements varied as a function of the assessment phase. Cognitive decline across a broad range of functions occurred during the course of ECT. Follow-up assessment revealed improvement across a broad range of cognitive functions (e.g. general cognitive function, short term memory, executive function, general memory, and semantic memory). Subjective depression decreased rapidly after starting the course of treatment and increased slightly after finishing ECT. In conclusion, enquiring about subjective cognitive change due to ECT is an integral component of an ECT cognitive assessment. Until the gap between subjective reports of cognitive function and objective assessment is narrowed, practitioners should continue to obtain the patient's report of cognitive change to increase sensitivity of the cognitive assessment.

Keywords: subjective report, electroconvulsive therapy, subjective cognitive impairment,
subjective cognitive change during ECT

Patients' Perspectives on the Short Term Cognitive Effects of Electroconvulsive Therapy

Electroconvulsive therapy (ECT) is the most effective treatment for a range of psychiatric disorders (American Psychiatric Association, 2001; UK ECT Review Group, 2003); however, its use is limited due to ongoing reports that the treatment causes cognitive impairment, particularly with memory (Ingram et al., 2008; Prudic, 2008). Cognitive assessment throughout an individual's course of ECT is recommended to detect the occurrence of cognitive dysfunction (Nehra et al., 2007). If cognitive impairment is detected, the way in which the treatment is administered can be altered to decrease the cognitive side effects (Porter, Douglas, et al., 2008). Challengingly, a patient's subjective report of cognitive impairment and their performance on objective tests of cognitive function are often incongruent (Berman et al., 2008; Nehra et al., 2008; Vamos, 2008); a patient may complain of significant cognitive impairment or memory loss, but this impairment may not be reflected on objective cognitive assessment. Furthermore, while objective assessment finds memory loss to be short term, resolving within six months post treatment, subjective memory loss is reported to persist over a much greater period of time (Fraser et al., 2008; Rose et al., 2003). This gap between anecdotal reports of cognitive dysfunction and the extent of memory loss and cognitive impairment reported in the literature is yet to be adequately explained (Donahue, 2000).

A number of hypotheses have been suggested as to why subjective and objective cognitive assessment outcomes are incongruent. Some researchers propose that the measures used to assess memory are not sensitive enough to detect the impairment patients are reporting (NICE; 2003; Porter, Douglas, et al., 2008; Rose et al., 2003). Meanwhile, others suggest subjective cognitive impairment may be a better indication of a patient's current mood state rather than their actual cognitive state (Coleman et al., 1996) as mood and subjective cognitive function are often highly correlated, whereas subjective and objective cognitive function are not (Minett, Da Silva, Ortiz, & Bertulocci, 2008). Other researchers suggest that psychiatrists and health professionals are under-assessing or failing to assess cognitive function during ECT and are

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therefore unaware that memory loss is occurring despite the patient reporting gaps in their memory (Sterling, 2000). The former two hypotheses are discussed in greater detail below.

Many studies investigating the relationship between subjective and objective cognitive function both in the ECT and the wider literature have used simple, brief measures of cognitive function such as the Mini-Mental State Examination (MMSE; Folstein et al., 1975). Among these studies, only weak or no correlation between objective memory impairment and subjective cognitive complaints were found (see for example: Bassett & Folstein, 1993; Jorm et al., 1994; Prudic, Peyser, & Sackeim, 2000). On the other hand, studies which used more sophisticated tests of cognition have found stronger associations between the two (Gagnon et al., 1994; Jonker, Launer, Hooijer, & Lindeboom, 1996; Riedel-Heller, 1999). This suggests that the lack of association between subjective and objective cognitive state may be due to the insensitivity of the tests used to detect memory impairment. If this hypothesis is correct, building on the existing knowledge and understanding of the nature and types cognitive change reported by a patient during ECT should increase the sensitivity of objective assessment. Objective neuropsychological tests which capture the domains of cognitive function reported to have changed by the patient could be added to the assessment to fill the gap between objective assessment and subjective report. Resultantly increasing the validity of the objective cognitive assessment to capture ECT related impairment or change.

Whether subjective memory complaints actually indicate cognitive impairment remains under debate (Riedel-Heller, 1999). Strong evidence suggests that cognitive complaints are exacerbated by psychiatric illness (Riedel-Heller, 1999) and that the relationship between subjective report of cognitive function and cognitive performance is low in psychiatric patients (Moritz, Ferahli, & Naber, 2004; Mowla et al., 2007). This does not suggest an absence of cognitive impairment in patients who report cognitive dysfunction post ECT, but rather that the extent of cognitive impairment reported could be exacerbated by comorbid depression.

Therefore clinicians in clinical and research settings working with patients receiving ECT

should determine whether subjective complaints of memory dysfunction result from ECT or impaired cognition from the underlying psychiatric condition (Nehra et al., 2008) or a combination of the two.

Irrespective of the cause of the subjective cognitive dysfunction, and whether or not it is seen on formal assessment, cognitive impairment post ECT can be a major source of patient distress (Nehra et al., 2008). A patient's subjective perception of their cognitive change during ECT is important information to gather not only as the complaints may not be detected by an objective assessment but also because it will impact on an individual's opinion of how effective ECT was for them (Rose et al., 2003). A number of organisations have already recommended that subjective reports of cognitive function be obtained alongside objective psychometric assessment, (e.g., the United Kingdom ECT Accreditation Service (ECTAS, 2011) the American Psychiatric Association Task Force Report (APA, 2001), and the Chief Psychiatrist's Guidelines for the use of Electroconvulsive Therapy in Western Australia (2006)).

Subjective reports of cognitive change can be obtained simply by enquiry with the patient, or via the use of psychometric instruments. The Squire Subjective Memory Questionnaire (SSMQ; Squire et al., 1979) is the most commonly used psychometric in ECT research to assess patients' subjective memory (Coleman et al., 1996; Prudic et al., 2000). The SSMQ is an 18 item measure of subjective memory designed to differentiate the cognitive effects of ECT from those of depression. Squire and colleagues suggested that 9 of the items were sensitive to the amnesic effects of ECT, while the remainder were sensitive to detecting cognitive effects of depression (Coleman et al., 1996). Although the SSMQ is a popular measure in the field of ECT research, it is limited in the dimensions of metacognition it assesses, and the instructions are said to be extraordinarily complex (Sackeim, 2000).

Previous research into ECT induced Subjective Cognitive Impairment

Numerous studies have used psychometric tools such as the SSMQ, or the Cognitive Failures Questionnaire (CFQ; Broadbent et al., 1982) to investigate the subjective cognitive effects of ECT (see for example: Calev et al., 1991; Coleman et al., 1996; Schulze-Rauschenbach et al., 2005; Sienaert, De Becker, Vansteelandt, Demyttenaere, & Peuskens, 2005). Coleman and colleagues (1996) used the SSMQ to assess subjective memory complaints prior to and following a course of ECT in 70 patients. They found that prior to ECT marked deficits in self-rated memory function existed which may be attributable to the effects of depression. The magnitude of subjective deficit experienced co-varied with the severity of clinical symptoms. Shortly following the course of ECT SSMQ scores improved, and the scores continued to improve at a two month follow-up assessment; however, did not reach those of matched controls. Patients rated their memory on the SSMQ as improved even when objective assessments suggested the existence anterograde or retrograde amnesia.

Qualitative studies into the subjective cognitive effects of ECT are fewer, and amongst these, most investigate an individual's experience of ECT as a whole, rather than specifically the experience of cognitive change. Vamos (2008) explored the subjective experiences of patients who were treated with ECT by using quotations derived from first person written accounts. Although cognitive change was not directly investigated, the issue of cognitive disruption arose under themes: importance of validation of experience, and, impact of daily disruptions. Koopowitz, Chur-Hansen, Reid and Blashki (2003) also used qualitative methods to investigate eight patients' experiences of ECT via the use of semi structured interviews. Eleven themes were derived from the analysis; two of which pertained to cognitive function. These were: side effects of ECT (including short term and current side effects and the distress caused by these) and cognitive functioning and memory (including global impressions of functioning pre and post ECT). Seven out of eight patients felt that their memory or cognition was affected and

reported some form of memory impairment endured from the treatment although it patients was difficult for patients to disentangle the effects of normal aging, depression and ECT.

Luther, Leathem and Humphries (2012b) used interpretative phenomenological analysis (IPA) to investigate the experiences of 19 patients who had undergone ECT two or more years previously. Three main themes emerged from the analysis, namely: reports of impairment in attention/concentration, retrieval, and executive functioning. They also found that of the 19 participants, 17 stated that they still experienced deficits from ECT, and that these deficits had not changed since the time of ECT (one had never experienced any memory or cognitive effects from the treatment; one had experienced only short term cognitive effects).

In addition to researchers reporting on the subjective experiences of ECT, some consumers of the treatment have also documented their experience of ECT, and many of whom have alluded to the cognitive effects endured during their treatment. Janet Frame, one of New Zealand's most distinguished literary figures, offered an account of her experience of ECT detailed in her autobiography:

“After having received over two hundred applications of unmodified ECT, each the equivalent, in degree of fear, to an execution, and in the process having my memory shredded and in some aspects weakened permanently or destroyed... I arrived home, outwardly smiling and calm, but inwardly with all confidence gone.” (Frame, 1989).

Personal experiences of ECT are also seen within the peer-reviewed literature. In a manuscript outlining the importance of informed consent which includes the possible long term cognitive effects of ECT, Anne Donohue gave an account of her experience of severe memory loss she endured after ECT.

“My long term memory deficits far exceed anything my doctors anticipated...hosting and driving Mother Teresa for a full day visit to Los Angeles in 1989; the dinner reception for my

National Jefferson Award in Washington, D.C., in 1990, where I sat beside my co-honoree, General Colin Powell; my brother's wedding in 1991 – the list goes on, and keeps growing as people bring up references to the past in casual conversations. Human memory seems to me to be one of the most precious aspects of our personality, since our memories are so critical to who we are and how we see ourselves and others. The memories of our past give us an understanding of where we fit in the world. I have experienced more than a 'cognitive deficit.' I have lost a part of myself." (Donahue, 2000).

One can infer from anecdotal accounts such as these, that memory loss and cognitive impairment is deeply distressing and can contribute to a person's self-esteem, sense of identity and self-worth. Autobiographical memory loss, such as that described by Donahue (2000) is an essential component of human memory as it holds the individual's past, which represents an important basis of one's identity and continuity of self (Conway, 2001). Accounts such as Frame and Donahue's further reiterate the importance of research in this area, and the importance of obtaining a patient's subjective perspective of cognitive change within the cognitive assessment or treatment review.

The current study aims to investigate the short term cognitive changes which occur during and after ECT using a qualitative approach. The results of this study provide information regarding what cognitive changes people report at different stages of treatment, which will inform which domains should be assessed in objective cognitive assessments. This would be a first step in narrowing the gap which exists between objective assessments and subjective reports of cognitive change. This study is unique in that it obtains accounts of subjective change from individuals while they are completing their course of ECT. This design allows for the interpretation of these changes as they occur and within the context of their clinical change.

The current research sought to answer the following lines of enquiry: What subjective cognitive changes occur during a course of ECT? What is the nature of these changes? What subjective

changes in mood occur? And finally, do the cognitive domains affected differ as a function of treatment phase? It is predicted that the subjective reports of cognitive function will vary as a function of severity of depression and number of ECT treatments. It is further predicted that there will be a negative correlation between scores of subjective cognitive function and depression, as detected by the SSMQ and BDI-II. It is predicted that subjective report of dysfunction will be present at baseline, and this will be reflective of depression related cognitive dysfunction, and that the severity of cognitive dysfunction will reduce at six week follow-up.

Method

Participants

13 people who received ECT for treatment resistant depression or bipolar disorder were recruited from the four ECT clinics in the lower North Island of New Zealand from August 2012 to October 2013. Five of the 13 were male. All participants were fluent in English and all interviews were conducted in English. The age range was 18-78 years, with the mean age of 47.04, (SD=13.49 years). Ethnicities included were: NZ European (N=7, 53.85%), Maori (N=3, 23.08%), South African (N=1, 7.69%), Dutch (N=1, 7.69%) and Chinese (N=1, 7.69%).

ECT Treatment

ECT was given at a frequency of twice weekly, and most typically Right-Unilateral ECT (RUL ECT) with an ultra-brief 0.3ms pulse width was used. The MECTA SpECTrum Q5000 machine was used to administer the ECT. Dose titration was determined using the limb isolation technique to establish seizure threshold. This technique involves isolating a limb from the effects of the muscle relaxation by inflation of a blood pressure cuff to above systolic pressure. As the muscle relaxant does not affect that limb, the seizure can be observed in that limb while the rest of the body is paralysed. Once the individual's seizure threshold was determined, the treatment dose delivered was six times the patients' seizure threshold. This dose remained constant for the whole course, unless inadequate seizure duration was occurring. Bi-Lateral ECT

(BL ECT) was done only in very exceptional circumstances. The treatment dose for BL ECT was 1.5 to 2.0 times seizure threshold. The MECTA SpECTrum has the capability of varying the pulse width, frequency (Hertz), duration of the stimulus charge and the flow of current (Amps), allowing psychiatrists to tailor the treatment to best suit the individual. The average number of ECT treatments received was 11.25 (SD=6.86).

Research Design

A naturalistic within-subjects prospective qualitative enquiry was adopted for the current research.

Measures

The SSMQ (Squire et al., 1979) was adapted for use in this study, Although it is acknowledged that the psychometric properties of the scale may be compromised by adapting the scale, the researchers believed that administering the original version of the SSMQ developed in 1979 would threaten the validity of the scale due to the complexity of the questions, and could potentially damage rapport with the participants. The main adaptations included removing item 13 “My ability to know when the things I am paying attention to are going to stick in my memory is...” from the questionnaire due to the complex nature of the item and high cognitive capacity required to accurately provide an answer. The remainder of the SSMQ items were shortened; however, the content of the items remained the same. The anchors were changed from “Worse than before” and “Better than ever before” to “Worse Since ECT” “No Change” and “Better Since ECT” to give the respondent clear time frames to compare cognitive change. The numbers within the likert scale were changed from -4 to +4 to 0-9. The Beck Depression Inventory – Second edition (BDI-II; Beck et al., 1996) is a 21-item self-report instrument which measures the severity of depression in adults and adolescents. Each item is rated on a 4-point scale ranging from 0 to 3 and possible total scores range from 0 to 63. Support for the validity and reliability of the BDI-II is well established with samples from various populations (Beck et al., 1996). Subjective reports of cognitive and clinical change were also obtained by asking the

patient about any positive or negative changes they had noticed since starting their course of ECT.

Procedure

After being prescribed a course of ECT, the patient was informed about the study by the ECT nurse or ECT administering psychiatrist. Patients who were deemed too unwell to consent to taking part in the study were excluded, as were patients receiving ECT for a psychotic disorder. Patients agreeing to take part in this study provided written consent. Participation was voluntary at each stage of the research and the patient was able to withdraw from the study at any time. This study received ethical approval from the Health and Disability Ethics Committee, New Zealand. The current study was part of a larger study which included a broader range of neuropsychological assessment.

Assessment Schedule

The first author (AT) met with the patient prior to their commencement of treatment to conduct a baseline assessment, and met again after every three treatments of ECT and at a 6 week follow-up. Assessments were conducted at least 48 hours after a treatment of ECT to allow for any residual effects of the ECT (such as disorientation) to resolve and patients were assessed at a standard time post treatment (Porter, Douglas, et al., 2008). This schedule was adhered to as closely as possible.

Assessment Procedure

Baseline: During the baseline assessment, the patient was asked to comment on any positive or negative effects they were experiencing with their cognition. The term “cognition” was avoided; instead the terms “thinking processes” and “brain functions” were used. The primary researcher recorded verbatim what the patient had said and the dialogue was repeated back to the patient to ensure accuracy of the recording. The baseline assessment phase was conducted to detect preexisting cognitive impairment due to depression. The BDI-II was administered, as was the

SSMQ. The SSMQ required the patient to rate how they perceived their memory to be right now, the anchors were changed to “Poor” “Ok” and “Excellent”.

During the Course: During the course of ECT, subjective memory was assessed by asking, “Have you noticed any *positive* changes since having ECT?” and subsequently “Have you noticed any *negative* changes since having ECT?” The SSMQ and the BDI-II were administered.

Follow-up: At the six week follow-up, the patient was asked, “Since finishing your course of ECT, have you noticed any *positive* changes?” and subsequently “Since finishing your course of ECT, have you noticed any *negative* changes?” the same procedure as above was followed and the SSMQ and the BDI-II were administered.

Results

Analysis

Qualitative content analysis was employed to analyse the patients’ subjective reports. This method enabled the transformation of the qualitative data for use in a *descriptive* and *operational* means. Descriptively, qualitative content analysis allowed for the transformation of dialogue into themes and categories, and operationally, this dialogue could then be presented numerically. Interview dialogues were analysed for the three treatment phases: *baseline*, *monitoring* and *follow-up*. The specific approach used was *directed qualitative content analysis*, which adopts both a deductive and inductive approach to coding the data (Hsieh & Shannon, 2005). With directed content analysis, initial coding starts with theories or findings based on previous research (deductive), then during data analysis additional themes or categories are derived from the data (inductive). By letting the categories emerge from the dialogue, important categories are not overlooked (Bailey, 1987). The steps of qualitative analysis described by Zhang and Wildemuth (2009) were used as a guideline to analyse the data sets. All subjective

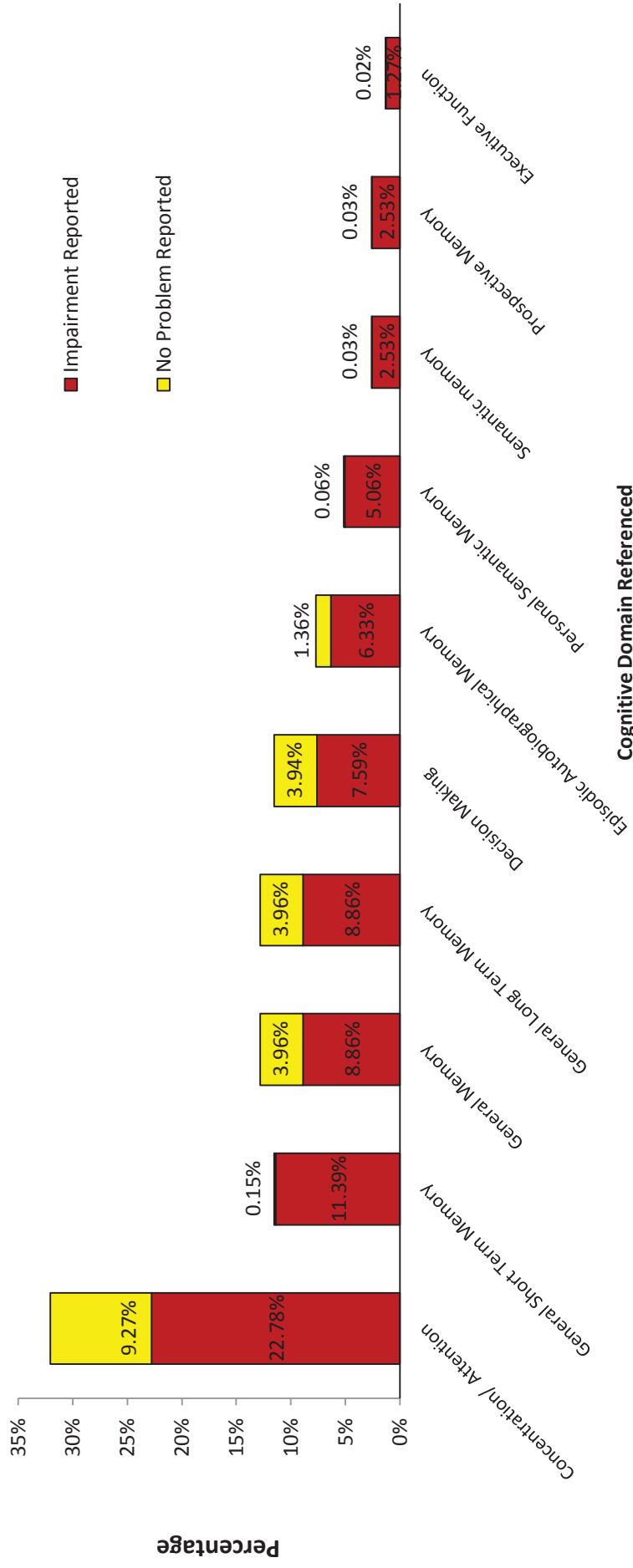
reports for each phase were amalgamated which resulted in three groups of dialogues; one for each treatment phase. All subjective reports of positive and negative changes during ECT were read and reread a number of times. Then, an initial list of coding categories was generated and new categories were added as the data were analysed inductively.

When deriving categories inductively, the constant comparative model was used (Glaser & Strauss, 1967). This involves comparing each text unit applied to a certain category with other texts already in that category and integrating categories and properties when needed. Individual themes were the unit for analysis; the theme may have been in the form of a single word, a phrase, a sentence or an entire paragraph. Although everything the patient said was recorded, only content referring to cognitive, somatic or emotional states were included in the analysis in order to answer the research questions. Information provided by the participants that did not fit into these categories were disregarded. An example of disregarded information was: "I have started a different medication."

Qualitative content analysis allows for the assignment of a unit of text to more than one category simultaneously (Tesch, 1990). This was appropriate for the current data set, due to some references of cognitive dysfunction involving more than one cognitive domain. For example, the complaint: "I go into a room and forget why I went in there" may be due to a combination of slowed processing speed, inattention and memory, and would therefore be categorised into "attention" and "processing speed" and "memory" categories. Once all data were categorised, an independent rater assessed the categorisation. Any differences in coding were discussed until an agreement was reached. Once coding was completed, the frequencies for each domain were converted into percentages to represent the portion of references to that cognitive domain out of the total number of references. The results of which are displayed in Figures 1, 2 and 3.

The baseline phase resulted in a total of 74 individual references pertaining to current cognitive function. Most of the cognitive reports during this assessment related to concentration or attention (32.1%) and the majority of these were complaints of dysfunction in this domain (22.78%). Other complaints included general memory, general long term memory, decision making, episodic autobiographical memory, semantic memory, prospective memory and executive functioning. Just over one fifth (22.78%) of the total reports were of cognitive functions being intact (e.g., “My memory is fine”).

The average BDI-II score at baseline was 39.80 ($SD=9.33$) indicating that patients were suffering from *severe* depression. Thus the cognitive complaints reflected in the baseline assessment most likely reflect the cognitive effects of depression and/or any cognitive effects of medications the patients may have been taking.



Cognitive Domain Referenced

Figure 1. Displays the subjective reports of cognitive function patients made prior to receiving any treatment of ECT. Reports for each cognitive domain are displayed as percentages of the total number of reports, thus all bars combined add up to 100%. Reports of cognitive impairment are displayed in red, and when patients referred to a cognitive domain which was functioning, this is displayed in yellow.

During the course of ECT, repeated assessments after every three treatments yielded a total of 225 references pertaining to cognitive change. Impairment occurred across a broad range of cognitive functions. Four new domains emerged which had not been reported in the baseline cognitive assessment. These included: orientation, information processing speed, general cognitive function and visual memory. Most commonly, general memory and concentration was reported to become impaired during the course. Personal semantic memory, general short term memory, decision making, executive function, orientation, information processing speed, general cognitive function, general long term memory, prospective memory, episodic autobiographical memory, visual memory and semantic memory were all reported to have worsened or declined during the course of treatment. Some cognitive improvement occurred for some individuals, and this is likely to be due to the alleviation of depression. Most improvement occurred in concentration/attention, (7.11% of all the reports) with some improvement, in general short term memory (5.78%), general long term memory (3.11%), decision making abilities (1.78%), general cognitive function (2.22%), general memory (0.89%) and semantic memory (0.89%).

The average BDI-II score was 24.32 (SD=12.00) during these assessments, indicating patients were suffering *moderate* depression. Thus cognitive complaints reflected during the course of treatment most likely reflect the cognitive effects of depression and those of ECT.

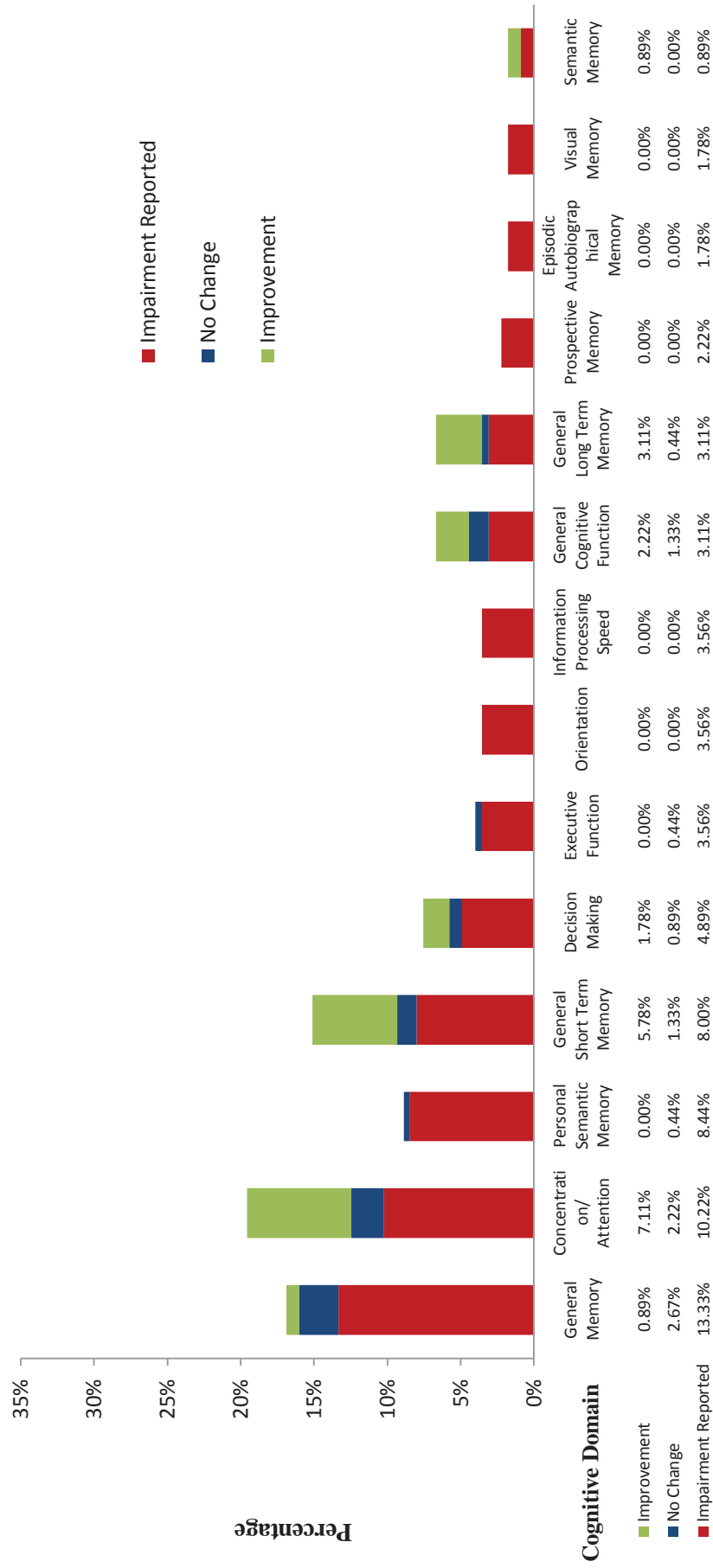


Figure 2. Displays the changes in cognitive function reported during a course of ECT. Reports for each cognitive domain are displayed as percentages of the total number of reports, thus all bars combined add up to 100%. Impairment in cognitive function is represented in red, no change is represented in blue and improvement in cognitive function is represented in green.

A common theme which emerged throughout this assessment phase was that cognitive dysfunction was reported to be most impaired shortly after treatment and the intensity of the impairment lessened as a function of time after treatment. Another theme which emerged was the insight patients had between their mood and their cognitive function. It was not uncommon for patients to report that their cognitive function had improved due to their mood improving, or was impaired due to their depression. Somatic complaints also emerged during this assessment phase, a total of 15 references were made, the majority of which pertained to headaches occurring after the treatment.

In addition to the cognitive and somatic changes due to ECT, changes in mood also occurred. During this assessment phase there were 128 references to mood state or changes in depressive symptoms. Of these 73.44% indicated an improvement in mood, 14.84% reported no change in mood, and 11.72% of the references made pertaining to mood, indicated a worsening in depressive symptoms.

Six weeks from finishing the course of ECT, a follow-up assessment was conducted. This assessment yielded a total of 52 reports pertaining to cognition. At the six week follow-up, it was evident some subjective cognitive impairment from the treatment had remained and subjective cognitive improvement had also occurred. Over half of the reports of cognitive function during this treatment were reports of improvement since finishing the course (59.6% of the reports). However, lasting cognitive impairment from the ECT was also commonly reported (40.4%). Figure 3 displays the subjective cognitive function at the follow-up assessment phase.

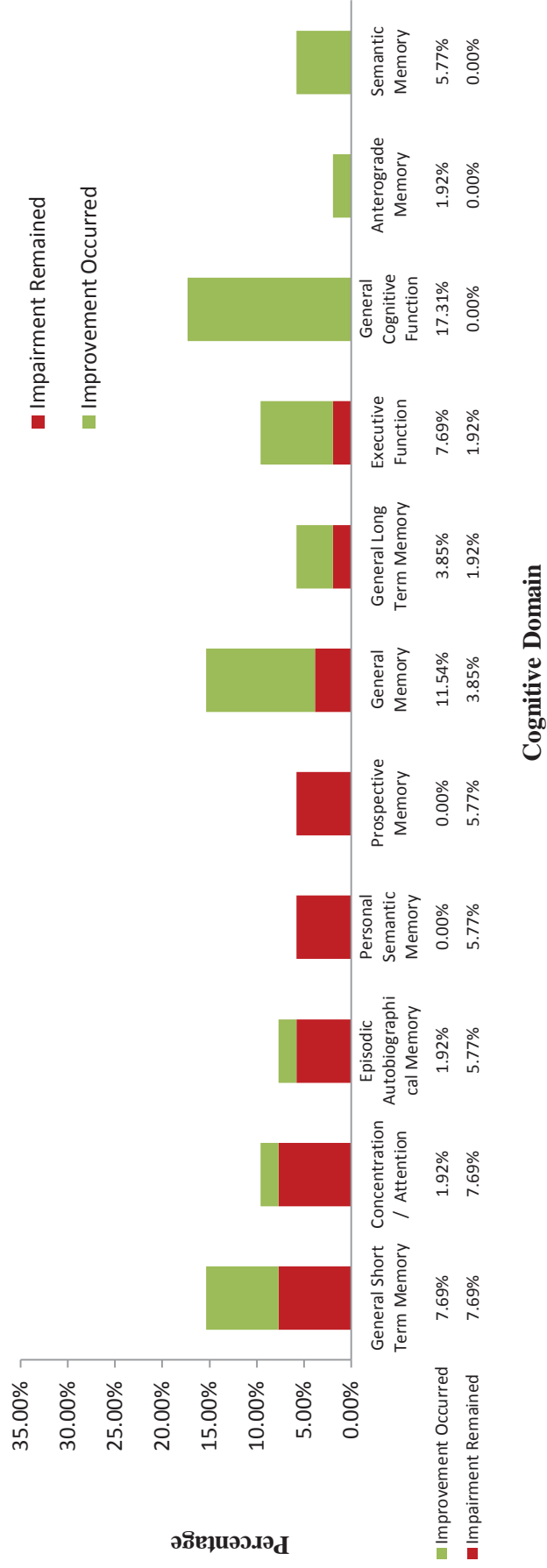


Figure 3. Displays the changes in cognitive function reported since finishing the course of ECT six weeks prior. Reports for each cognitive domain are displayed as percentages of the total number of reports, thus all bars combined add up to 100%. Impairment in cognitive function is presented in the red bars, and improvements in cognitive function are presented in the green bars.

Cognitive impairment remained for some individuals in the domain of general short term memory, concentration and attention, episodic autobiographical memory, personal semantic memory, prospective memory, general memory, general long term memory and executive functioning. The greatest improvement occurred for general cognitive function; many patients reported that overall their general cognitive function had improved since stopping the ECT. Improvements also occurred for general memory, general short term memory, executive function, semantic memory, anterograde amnesia, concentration and attention and episodic autobiographical memory. Anterograde memory was a new category which emerged during this assessment phase. Some categories which were referenced during the treatment phase were not referenced during the follow-up phase, indicating that impairments in those domains may have resolved. These included: orientation, decision making, visual memory and information processing speed.

A common theme which emerged during the follow-up assessment phase was the persistence of amnesia only for events occurring around the time of the course of treatment. Complaints of anterograde amnesia tended to pertain to the difficulty learning new information during the course of treatment. A new theme which occurred at this phase was the persistence of the clinical effects and the reemergence of depressive symptoms. There were 21 references to change in mood from ECT and whether these changes lasted. Of these references 38.10% pertained to the continual improvement in mood from treatment, and 61.9% pertained to deterioration in mood since stopping ECT. During the follow-up assessment phase, the average score on the BDI-II was 28.07 (SD=13.47) which indicates patients were suffering moderate depression during this time. Thus the cognitive complaints reflected in the follow-up assessment most likely reflect the cognitive effects of depression and/or any residual effects of the ECT.

Changes in Subjective Memory as measured by the SSMQ

In addition to obtaining qualitative subjective reports of cognitive change, the SSMQ was administered at each assessment. Subjective changes which occurred from baseline to during the course of treatment are presented in Table 1. Changes which occurred from during the course of treatment to the six week follow-up are presented in Table 2. Finally, changes which occurred from baseline to the follow-up assessment are presented in Table 3.

Table 1

<i>Effect sizes of change in SSMQ items occurring from Baseline to During the course of ECT</i>				
Item in SSMQ	Effect Size	Confidence Interval	Qualitative Size	Direction
Highly Sensitive to Change				
My ability to remember what I read and watch on television:	-1.18	-1.69- -0.50	Large	Improvement
Sensitive to Change				
My ability to make a memory that is on the tip of my tongue available:	-0.70	-1.30-0.015	Medium	Improvement
My ability to pay attention to what goes on around me:	-0.63	-1.18- -0.03	Medium	Improvement
My ability to follow what people are saying	-0.54	-1.10-0.04	Medium	Improvement
My ability to remember things when I really try:	-0.54	-1.12-0.02	Medium	Improvement
My ability to remember things that happened over a year ago	-0.52	-1.14-0.01	Medium	Improvement
My general alertness to things happening around me is:	-0.48	-1.06-0.08	Medium	Improvement
My ability to recall names:	-0.44	-0.394-0.20	Medium	Improvement
Moderately Sensitive to Change				
In one month's time my ability to remember this questionnaire is:	-0.36	-0.94-0.20	Small	Improvement
My ability to remember what happened a few minutes ago:	0.30	-0.22-0.92	Small	Decline
My ability to remember the faces of people I meet is:	-0.25	-0.83-0.30	Small	Improvement
My ability to make sense of what people explain to me is:	-0.22	-0.79-0.35	Small	Improvement
My relatives and acquaintances judge my memory to be:	-0.21	-0.79-0.34	Small	Improvement
No Change				

My ability to remember things that happened a long time ago is:	-0.19	-0.75-0.38	Very Small	Improvement
My ability to remember things that happened during my childhood is:	0.17	-0.39-0.74	Very Small	Decline
My ability to remember things I have learned is:	-0.08	-0.66-0.47	Very Small	Improvement
My ability to remember what I was doing after I have taken my mind off it is:	0.08	-0.49-0.64	Very Small	Decline

As shown on Table 1, most of the evaluated SSMQ items improved from the baseline ratings.

The item most sensitive to change was the item regarding the individual's ability to remember what they read and watch on television. This cognitive ability relies on concentration/attention, encoding and recall memory. This is consistent with the reported improvement in concentration and general memory which were the two most frequent reports of improvement (see Figure 2).

Medium effect sizes were generated for the change in the items regarding: ability to retrieve a memory, pay attention to their environment, follow conversation, remembering when efforts are made to remember, remembering things that happened over one year ago, general alertness and ability to remember names. These items all showed an improvement from baseline to during the course of treatment. Insensitive to change were the items regarding retrograde amnesia for events many years prior to ECT.

Table 2

Effect Sizes of change in SSMQ items which occurred During the course of ECT to the Six Week Follow-Up

Item in SSMQ	Effect Size	Qualitative Size	Direction
Sensitive to Change			
My ability to remember what happened a few minutes ago:	-0.67	Medium	Improvement
My ability to make sense of what people explain to me is:	-0.60	Medium	Improvement
My ability to remember what I was doing after I have taken my mind off it is	-0.58	Medium	Improvement
In one month's time my ability to remember this questionnaire is:	-0.42	Medium	Improvement
My ability to remember the faces of people I meet is:	-0.41	Medium	Improvement
Moderately Sensitive to Change			
My ability to remember things when I really try:	-0.32	Small	Improvement
My ability to make a memory that is on the tip of my tongue available:	-0.31	Small	Improvement
My ability to remember things I have learned is:	-0.23	Small	Improvement
My ability to remember things that happened over a year ago	0.23	Small	Decline
My general alertness to things happening around me is:	-0.22	Small	Improvement
My relatives and acquaintances judge my memory to be:	-0.21	Small	Improvement
Insensitive to Change			
My ability to remember things that happened a long time ago is:	0.18	Very Small	Decline
My ability to remember things that happened during my childhood is:	-0.15	Very Small	Improvement
My ability to pay attention to what goes on around me:	-0.12	Very Small	Improvement
My ability to remember what I read and watch on television:	0.06	Very Small	Decline
My ability to follow what people are saying	-0.02	Very Small	Improvement
My ability to recall names	-0.01	Very Small	Improvement

As is shown on Table 3, further improvement occurred during the course to the six week follow-up. The items sensitive to improvement included the items regarding the individuals' short term memory, comprehension of conversation, long term memory and visual memory. Again,

retrograde memory for events many years prior to ECT was insensitive to change from during the course to follow-up.

Table 3

Effect sizes of change in SSMQ items which occurred from Baseline to Follow-Up

Item in SSMQ	Effect Size	Qualitative Size	Direction
<i>Highly Sensitive to Change</i>			
My ability to remember what I read and watch on television:	-1.13	Large	Improvement
My ability to make a memory that is on the tip of my tongue available:	-0.95	Large	Improvement
My ability to recall names	-0.94	Large	Improvement
My ability to make sense of what people explain to me is:	-0.85	Large	Improvement
My ability to remember things when I really try:	-0.80	Large	Improvement
<i>Sensitive to Change</i>			
In one month's time my ability to remember this questionnaire is:	-0.74	Medium	Improvement
My ability to pay attention to what goes on around me:	-0.74	Medium	Improvement
My general alertness to things happening around me is:	-0.70	Medium	Improvement
My ability to follow what people are saying	-0.60	Medium	Improvement
My ability to remember the faces of people I meet is:	-0.59	Medium	Improvement
My ability to remember what I was doing after I have taken my mind off it is	-0.48	Medium	Improvement
<i>Moderately Sensitive to Change</i>			
My relatives and acquaintances judge my memory to be:	-0.37	Small	Improvement
My ability to remember things that happened over a year ago	-0.36	Small	Improvement
My ability to remember things I have learned is:	-0.26	Small	Improvement
<i>Insensitive to Change</i>			
My ability to remember what happened a few minutes ago:	-0.12	Very Small	Improvement
My ability to remember things that happened during my childhood is:	0.04	Very Small	Decline
My ability to remember things that happened a long time ago is:	-0.01	Very Small	Improvement

Large improvements occurred from baseline to follow-up for the items regarding the

individual's ability to remember what they read and watch on television, retrieval of memories, recalling names, and remembering when efforts are made to remember. Medium improvements occurred in the items regarding long term memory, attention and alertness, comprehension of conversation, visual memory and short term memory. Insensitive to change from baseline to during the assessment and follow-up included an individual's ability to remember things that happened during their childhood and things that happened a long time ago. An individual's ability to remember things that happened only a few minutes ago remained unchanged from baseline to follow-up.

Mood and Subjective Reports of Cognition

To test the prediction that mood and subjective reports of cognitive function are correlated, a Pearson's correlation coefficient was conducted to test the relationship between scores on the SSMQ and scores on the BDI-II. The correlation revealed a moderate negative relationship ($r(88)=-.39, p=0.000$) and this correlation was statistically significant. The negative relationship suggests that when depression scores are high, patients tend to rate their cognitive function as poorer, when depression scores are low; patients tend to rate their cognitive function as better. The average SSMQ and BDI-II scores at each assessment phase are presented in Figure 4.

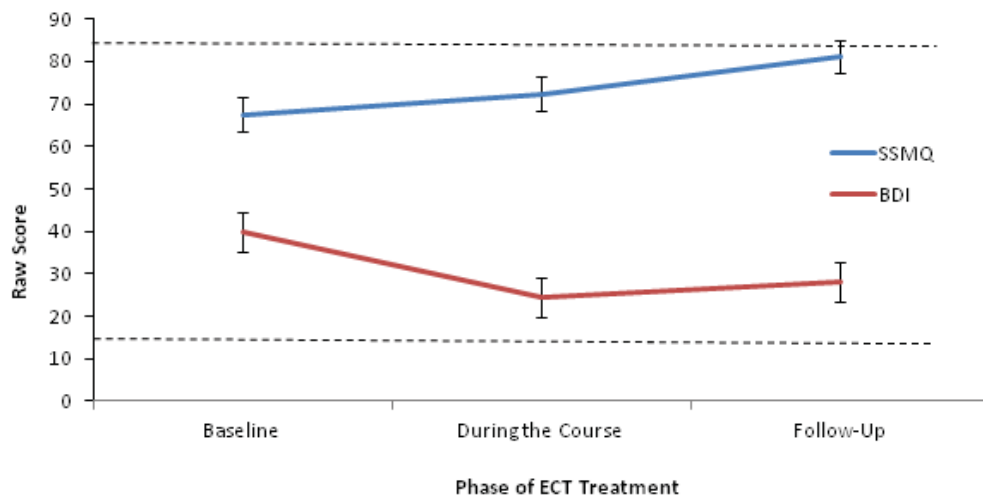


Figure 4. Subjective scores of depression and cognitive function as a function of treatment phase. The broken line above represents average SSMQ scores which would be seen if an individual indicated average or ‘ok’ cognitive function for all 17 items. The broken line below represents the cut off score for mild depression on the BDI-II.

Unexpectedly, the subjective reports of cognitive functioning as measured by the SSMQ actually improved from baseline ($M=67.53$, $SD=16.91$) to during the treatment ($M=72.42$, $SD=20.83$, $d=-0.26$). Again, SSMQ scores improved from during the course to the six week follow-up ($M=81.08$, $SD=15.82$, $d=-0.47$). Further analyses were conducted to determine which items in the BDI-II were sensitive to change during treatment. Effect sizes were conducted to determine the change in the 21 items in the BDI-II from baseline to during the treatment of ECT, and from baseline to the follow-up assessment.

Changes in Subjective Depression

Table 4

Scores on Beck Depression Inventory-II from Baseline to during the course of ECT

<i>Item in BDI-II</i>	<i>Effect Size</i>	<i>Confidence Interval</i>	<i>Qualitative Size</i>	<i>Direction</i>
<i>Highly Sensitive to Change</i>				
Agitation	1.16	0.33-1.93	Large	Decline ⁴
Concentration Difficulties	1.11	0.29-1.88	Large	Decline
Changes in Appetite	1.06	0.24-1.82	Large	Decline
Suicidal ideation	1.04	0.23-1.80	Large	Decline
Loss of Pleasure	1.01	0.20-1.77	Large	Decline
Tiredness/ Fatigue	0.99	0.17-1.74	Large	Decline
Self-Dislike	0.94	0.14-1.70	Large	Decline
Changes in sleeping patterns	0.87	0.14-0.70	Large	Decline
Worthlessness	0.86	0.08-0.63	Large	Decline
Irritability	0.85	0.06-1.61	Large	Decline
Loss of Interest	0.82	0.03-1.57	Large	Decline
Pessimism	0.81	0.01-1.55	Large	Decline
<i>Sensitive to Change</i>				
Loss of Energy	0.70	-0.07-1.46	Medium	Decline
Sadness	0.70	-0.09-1.43	Medium	Decline
Feelings of Punishment	0.62	-0.16-1.36	Medium	Decline
Loss of Interest in Sex	0.62	-0.16-1.36	Medium	Decline
Self Criticalness	0.52	-0.25-1.26	Medium	Decline
Indecisiveness	0.51	-0.26-1.25	Medium	Decline
Feelings of Guilt	0.48	-0.29-1.22	Medium	Decline
Feelings of Past Failure	0.42	-0.34-1.16	Medium	Decline
Crying	0.40	-0.36-1.14	Medium	Decline

⁴ Decline i.e, lowered score means *improvement* in mood

Table 5

Scores on the Beck Depression Inventory-II from Baseline to Follow-up

<i>Item in BDI-II</i>	<i>Effect Size</i>	<i>Qualitative Size</i>	<i>Direction</i>
<i>Highly Sensitive to Change</i>			
Loss of interest	0.96	Large	Decline
Concentration Difficulties	0.93	Large	Decline
Changes in sleeping patterns	0.92	Large	Decline
Suicidal ideation	0.86	Large	Decline
<i>Sensitive to Change</i>			
Tiredness/ Fatigue	0.78	Medium	Decline
Pessimism	0.73	Medium	Decline
Loss of Pleasure	0.72	Medium	Decline
Changes in Appetite	0.65	Medium	Decline
Loss of Interest in Sex	0.61	Medium	Decline
Worthlessness	0.61	Medium	Decline
Loss of Energy	0.57	Medium	Decline
Self-Dislike	0.55	Medium	Decline
Agitation	0.55	Medium	Decline
Irritability	0.53	Medium	Decline
Indecisiveness	0.49	Medium	Decline
Crying	0.45	Medium	Decline
Sadness	0.43	Medium	Decline
<i>Insensitive to Change</i>			
Feelings of Punishment	0.20	Very Small	Decline
Self-Criticalness	0.18	Very Small	Decline
Feelings of Guilt	0.16	Very Small	Decline
Feelings of Past Failure	0.03	Very Small	Decline

From baseline to during the course of treatment, large declines occurred for many of the items in the BDI-II indicating efficacy for ECT as a treatment for subjective depression. From baseline to the six-week follow-up, the effect sizes had declined somewhat indicating some relapse in symptoms from during the course, but improvement in depressive symptoms from baseline were still clearly evident.

Discussion

The current research sought to answer the following lines of enquiry:

1. What subjective cognitive changes occur during a course of ECT?
2. What is the nature of these changes?
3. Do the cognitive domains affected differ as a function of treatment phase?
4. And finally, what subjective changes in mood occur?

It was predicted that subjective report of cognitive dysfunction would be present at baseline; the severity of cognitive dysfunction will reduce at six week follow-up; subjective reports of cognitive function would vary as a function of severity of depression; therefore a negative correlation will exist between scores of subjective cognitive function and depression.

To determine what subjective cognitive changes occurred and the nature of these changes, patients were asked about ECT related cognitive change during their treatment and at a six week follow-up and also completed the SSMQ. Subjective memory as measured by the SSMQ improved from baseline to during the course of ECT, and again from during the course of treatment to follow-up.

From the qualitative content analysis it emerged that each phase of assessment yielded different types of cognitive complaints. Cognitive impairment existed prior to starting the course of ECT which is consistent with reports that depressive illness is manifested by cognitive impairment (Douglas & Porter, 2009). The types of cognitive impairment most typically reported prior to starting treatment were in the area of attention and concentration. General memory, (long and short term) was also commonly reported at this phase of assessment and patients also described having difficulty with decision making. Although concentration and attention difficulties remained a problem during the course of ECT, participants reported a broader range of cognitive functions being affected with general memory dysfunction being the most common

change, e.g. “my memory is quite bad at the moment”, without specifying the exact nature of the memory difficulty.

At the follow-up assessment, cognitive impairment remained for some individuals, and the types of impairment which remained included general memory, concentration and attention, episodic and personal semantic memory, prospective memory, general long term memory and executive function. Like Coleman and colleague’s (1996) study, subjective ratings of cognitive function were low at the baseline assessment increased at follow-up to just below average subjective memory function. Most cognitive improvement occurred in the categories of general memory and general cognitive function. The number of categories which emerged during this assessment phase decreased, and disorientation was no longer reported, neither were difficulties with decision making, visual memory, information processing speed and prospective memory. Reports of improvement occurred only for general cognitive function.

All ratings on the subjective memory items on the SSMQ improved from baseline to during the course of treatment, except the item “My ability to remember what happened a few minutes ago”. This item showed a small decline. From baseline to follow-up, there were a number of items which showed large effect sizes indicating significant change ranging from 0.80-1.13 standard deviations improvement from baseline. These improvements occurred for attention, retrieval, semantic memory, comprehension and general memory. Items insensitive to change from baseline to follow-up included retrograde amnesia for events many years prior to ECT which is congruent with the observation that memory loss due to ECT follows a temporal gradient with the course of ECT being the time point from which amnesia occurs (Calev et al., 1993; Cohen & Squire, 1981; Fraser et al., 2008; Peretti et al., 1996; Rami-Gonzalez et al., 2001; Sackeim et al., 1993).

When comparing the subjective reports of memory function measured by the SSMQ and the subjective cognitive dysfunction reported by the patients, one stark difference emerged.

Subjective cognitive impairment from the treatment was evident at each assessment point when reported by the patients, whereas subjective cognitive improvement was shown by the change in SSMQ scores. A reason for this may be due to pooling the SSMQ data which could have masked any individual reports of cognitive decline (Coleman et al., 1996), whereas qualitative content analysis reflected all cognitive complaints without any being lost during analysis.

It is important to note that during the course of treatment and at follow-up, patients were suffering from *moderate* depression and thus some of the cognitive complaints are likely to be attributable to the cognitive effects of depression. As detected by the BDI-II, subjective depression decreased significantly from baseline to during the course of ECT. Subjective scores of depression prior to ECT, on average, were in the *severely depressed* range, indicating high levels of depressive symptomatology. During the course and at the follow-up assessment, depression had dropped to the *moderately depressed* range. More in depth analysis of the depressive symptoms which changed from baseline to the six week follow-up revealed large improvements for the following items: loss of interest, concentration difficulties, changes in sleeping patterns, and of highest importance, suicidal ideation decreased significantly from baseline to during the course of treatment. Of the 21 items on the BDI-II, 17 were sensitive to change from baseline to follow-up.

Psychometric analysis of the items within the BDI have been found to segregate into two groups: cognitive-affective (items 1-3, 5-9, 13,14) and somatic-vegetative (items 4,10,11,12, 15-21) (Beck et al., 1996). ECT appeared to be most effective at alleviating the somatic-vegetative symptoms of depression for the current participant sample. The insensitive items mostly included those which tap into the cognitive depressive symptoms and individual core beliefs such as feelings of past failure, guilt, punishment, self criticalness. An implication of this may be that once ECT has alleviated the somatic-vegetative symptoms of depression, an individual may be in a better position to receive talking therapies which target the cognitive side effects of depression and the core beliefs which contribute to depression. A further implication of this

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finding is that if time were a major factor in restricting assessment of clinical efficacy of the treatment, items pertaining to somatic-vegetative symptoms of depression, in addition to suicidal ideation could be prioritised over the less sensitive cognitive symptoms.

As predicted, there was a significant correlation between subjective ratings of memory as detected by the SSMQ and subjective ratings of depression as detected by the BDI-II. A moderate negative correlation existed indicating that when scores of depression were high, ratings of subjective memory tended to be worse. It is unclear whether this is due to the fact that when people are in a state of depression they tend to be more pessimistic or whether their cognitive function was affected by the illness.

Limitations

There are some threats to the validity and reliability of patients' subjective reports of cognitive function during a course of ECT. For example an individual may not be aware that he or she is having difficulty with memory, unless prompted by someone else to remember what has been forgotten (Robertson & Pryor, 2006). Thus if a cognitive failure is not reported, it should not be interpreted as an absence of actual cognitive difficulty in that domain. The SSMQ takes away the risk that the patient will forget to report by providing cues, i.e. requires recognition rather than recall memory. Furthermore, cognitive failures which are more distressing to patients may be more likely to be reported than cognitive impairments which may be more prevalent. For example, forgetting somebody's name may be embarrassing or distressing for an individual and they may be therefore more inclined to remember this as a problem opposed to forgetting to post a letter. However, a cognitive failure which causes great distress may have more clinical significance than a cognitive failure which may be more profound or prevalent. Furthermore, when obtaining the subjective reports of cognitive function there may have been a selective attentional bias to cognitive change after starting the course of treatment as patients may be hyper sensitive to cognitive change after being informed that memory loss is a common side effect of ECT.

When interpreting the results of the qualitative content analysis, it is important to be aware that the reports of cognitive change are the patients' words and perceptions, and reflect an understanding of change which may not necessarily be congruent with a clinician's interpretation. For example, a patient may report "my short term memory is really bad" referencing forgetting something that they were told earlier that morning, whereas this type of memory failure would be considered a long term memory deficit given that short term memory typically refers to the ability to hold information for a very brief period of time, usually around 30 seconds (Goldstein, 2008). Like Koopowitz and colleagues' (2003) study participants in this study found it difficult to determine the etiologies of their cognitive difficulties, and often did not know whether impairments in cognition were due to ECT or depression. A further limitation was that only one baseline and follow-up assessment could be conducted. Increasing the number of assessments at these assessment points would have yielded more information regarding cognitive state during severe depression and the lasting effects of ECT six weeks post treatment, and may have better reflected the subjective cognitive function at each phase.

A further limitation to subjective report is that insight and metacognition is affected by psychiatric state (David, Bedford, Wiffen, & Gilleen, 2012). Collateral informants may be useful when assessing subjective cognitive complaints, to assist in reporting cognitive changes not recollected by the patient. A final limitation to the current study is that of the low sample size, however, the current study shed some light on the subjective cognitive changes which occurred during a course of ECT for these 13 individuals.

Implications

Subjective reports of cognitive function are an important component of ECT treatment and should accompany objective cognitive assessment, as strong evidence suggests incongruence exists between the two (Berman et al., 2008). While most research into the objective cognitive effects of ECT report that cognitive impairment should resolve within weeks after treatment, a six week follow-up assessment revealed that subjective cognitive impairment were still present.

Subjective cognitive impairment beyond six weeks has also been found in other studies despite the resolution of objective cognitive function (Fraser et al., 2008). Subjective vegetative-somatic symptoms of depression were more sensitive to change than cognitive symptoms of depression, thus assessment could focus on these changes. As the results of this study provided further evidence to suggest that memory loss due to ECT is more highly concentrated for information and events learnt around the time of ECT, assessment of memory should focus on detecting amnesia for memories which would have been encoding around the time and during the course of ECT.

Future Research

The incongruence between objective performance in cognitive tests and subjective reports of cognitive dysfunction may be due to patients reporting cognitive complaints which are distressing or of high importance to the individual, rather than reporting a true reflection of the exact cognitive changes that the individual has endured. Future research should investigate the level of distress regarding reported cognitive failures, as from the patients' perspective it may be that the cognitive complaints which cause the greatest level of distress are of greater clinical importance to the patient. Future research should also update the SSMQ or develop a new subjective memory questionnaire which is sensitive to detecting subjective cognitive change.

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**STATEMENT OF CONTRIBUTION
TO DOCTORAL THESIS CONTAINING PUBLICATIONS**

(To appear at the end of each thesis chapter/section/appendix submitted as an article/paper or collected as an appendix at the end of the thesis)

We, the candidate and the candidate's Principal Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of Candidate: Anneke Thornton

Name/Title of Principal Supervisor: Janet Leathem

Name of Published Research Output and full reference:

Patients' Perspectives on the Short Term Cognitive Effects of Electroconvulsive Therapy

In which Chapter is the Published Work: Eight

Please indicate either:

- The percentage of the Published Work that was contributed by the candidate:
and / or
- Describe the contribution that the candidate has made to the Published Work:

The candidate had full responsibility for the design, data collection, analysis and write up of the research. Supervisors provided guidance and were involved in decisions made around research processes and provided input with data analysis and formatting of the thesis. For these reasons Professor Janet Leathem and Dr Ross Flett were included as co-authors for the manuscripts which comprise this thesis.

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The following chapter (Chapter Nine) suggests a screening measure The CASDECT – A Cognitive Screening Measure for use during ECT. This chapter will be turned into a manuscript subsequent to the studies in Chapters Seven and Eight being peer reviewed and published, as the proposed screening measure has been derived from findings from these studies. The manuscript will also not be published until the psychometric properties of the screen have been established. Furthermore, contact was made with Dr Nasreddine, the author of the Montreal Cognitive Assessment to seek permission for some of the items from the MoCA to be included within the CASDECT. Dr Nasreddine had concerns about the integrity of the MoCA being compromised if subtest were taken out in isolation. Therefore, alternate versions of the verbal fluency task and verbal learning items have been substituted to protect copyright of the MoCA. These alterations add to the importance of the psychometric properties of this task to be tested.

CHAPTER NINE

The Development of the CASDECT – a Cognitive Screening Measure for use during Electroconvulsive Therapy

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Abstract

Cognitive impairment remains a common side effect of electroconvulsive therapy (ECT) and accordingly frequent and thorough cognitive assessment during the course of treatment is recommended to detect cognitive change. Although recommended, the way in which cognition should best be assessed remains undetermined and under debate. The CASDECT, a new cognitive screening measure, is introduced here as a potential method to assess cognition during a course of ECT. The contents have been derived from the research conducted within this thesis, feedback from practitioners conducting cognitive assessments, feedback from consumers of the therapy, and from the existing literature into the cognitive effects of ECT and the assessment thereof. The screening measure is brief (20 minutes), covers a broad range of cognitive domains and has three alternate forms to control for practice effects. A limitation of the measure is that the psychometric properties are unknown, and future research should address this limitation by validating the measure.

Keywords: cognitive assessment, electroconvulsive therapy, screening measure

The Suggestion of a Cognitive Screening Measure to for use during Electroconvulsive Therapy

It is well established in the literature that electroconvulsive therapy (ECT) can cause cognitive impairment, and that frequent and thorough cognitive assessment can detect ECT related cognitive impairment (Nehra et al., 2007; Porter, Douglas, et al., 2008). Early detection of cognitive decline allows for changes to the way ECT is administered which can lessen the impact on cognitive function (Porter, Douglas, et al., 2008). Despite these recommendations, the way in which cognitive function should be assessed remains under debate (National Institute of Clinical Excellence, 2003; Verwijk et al., 2012).

There have been numerous publications to date which include suggestions of tests to comprise the cognitive assessment; however, no gold standard measure is available to monitor and assess cognition during a course of ECT (Gangadhar & Thirthalli, 2010). Resultantly the way in which cognition is assessed remains unsystematic (Porter, Douglas, et al., 2008) and many clinicians use short screening measures designed for the detection of dementia related cognitive decline, such as the Mini Mental Status Examination (MMSE; Folstein et al., 1975) at the expense of sensitivity (Falconer, Cleland, Fielding, & Reid, 2010). Also commonly used are structured tests of verbal learning such as the Auditory Verbal Learning Test (AVLT; Rey, 1964) and other forms of verbal paired associates which involve encoding and recollection of familiar verbal material are used during ECT. These tests assess memory post short retention intervals; however, there is a dearth of evidence which suggests ECT interferes with short term memory or well established vocabulary skills (Robertson & Pryor, 2006). As a result these types of tests may be insensitive to detecting the effects of ECT. A grave concern about using such measures is that patients will perform well on these tests and clinicians may subsequently conclude that cognitive impairment has not occurred (Robertson & Pryor, 2006). As simple standardised tests have been shown to be ineffective at detecting the deficits articulated by

individuals receiving ECT, there is a need for more appropriate tests to be devised (Robertson & Pryor, 2006).

As the importance of assessing cognitive function during a course of ECT is evident, some recommendations exist around what measures should be included within the cognitive assessment. Martin et al. (2013) examined the utility of the MMSE, the first and second recall items of the Modified Mental State Examination (3MSE; Teng, 1987), and several mental control items such as counting backwards from 20, saying the alphabet (forwards), counting forwards in 3s starting with 1 ending after 14 responses, saying the days of the week backwards, starting with 'Sunday', and saying the months of the year backwards starting with 'December'. This screen was administered 1-3 days before ECT and the day after the third treatment of ECT. Martin and colleagues also administered a more detailed cognitive battery which was administered 1-3 days before ECT, after the sixth treatment, and 1-3 days after the final treatment of ECT. This screen comprised of: the Medical College of Georgia Complex Figure (MCGCF; Loring & Meador, 2003), Hopkins Verbal Learning Test-Revised (HVLT-R; Benedict, Schretlen, Groninger, & Brandt, 1998), Letter Fluency (COWAT; Benton & Hamsher, 1976), Animal Fluency (Spreen & Strauss, 1998), Cross Out Task (Woodcock & Johnson, 1989), the Symbol Digit Modalities Test (SDMT; Smith, 1991) and the Autobiographical Memory Interview – Short Form (AMI-SF; M. McElhiney, Moody, & Sackeim, 2001). The items found to be most predictive of later changes in anterograde memory were: counting backwards from 20, and reciting the months of the year backwards (Martin et al., 2013). However, these items were not adapted for repeated administration which limits the use of these measures, as patients' performances are likely to improve over multiple assessments.

Porter and colleagues reviewed the sensitivity of a number of cognitive tests to the effects of ECT. From this review, a battery of tests was proposed to detect cognitive change in people receiving ECT. This 55 minute battery included the MMSE or 3MSE, Hopkins Verbal Learning

Test (HVLT; Brandt, 1991), Autobiographical Memory Questionnaire- Short Form (AMI-SF; Kopelman et al., 1989) and the Digit-symbol Substitution Task (DSST; Wechsler, 1997). These authors also encouraged reorientation be assessed after each individual treatment (Porter, Douglas, et al., 2008). This suggested battery is thorough in that it assesses a broad range of cognitive functions associated with change during ECT; however, a shortcoming of this selection was that it fails to assess visual learning and memory, a domain consistently associated with impairment from ECT (Semkovska & McLoughlin, 2010). In addition, the length of time required to carry-out this assessment is unrealistic for many health professionals, and despite the battery being well tolerated by patients receiving ECT, the authors noted that some patients refused the RAVLT and the autobiographical memory task because they were tiring (Porter, Heenan, et al., 2008). This is also suggestive that a one hour battery may be excessive for patients receiving ECT.

Authors of a recent article originating from India stated that in developing countries Porter et al.'s proposed battery is unsuitable, due to the high caseload of medical professionals (where typically 10-15 patients receive ECT per day) and low resources available to clinicians (Viswanath et al., 2013). In addition, they argued that the development of separate assessment measures is required to accommodate the cultural variation which exists globally. Viswanath et al. proposed a shorter (20-30 minute) assessment battery (Battery for ECT Related Cognitive Deficits (B4ECT-ReCoDe)) for the rapid assessment of cognitive deficits associated with ECT in India. Their battery comprised of both subjective and objective components, and included: a shortened version of the Rey Auditory Verbal Learning Test (Spreen & Strauss, 1998), a set of questions assessing subjective memory, the orientation section of the Hindi Mental Status Examination (Pandav et al., 2002), a culturally modified version of Squire's Autobiographic Memory Questionnaire (Squire et al., 1981), the Digit Symbol Substitution Test (Wechsler, 1981), Visual Memory Test (Strub & Black, 2000) and Letter Number Sequencing (Wechsler, 1997). They also included some questions from the Autobiographical Memory Interview (AMI;

Kopelman et al., 1989). Many of the items within this screen have been adapted to be culturally sensitive and for use with younger patients receiving ECT in India (Viswanath et al., 2013), and therefore may not be appropriate for use in the western world.

The current article will suggest a new cognitive assessment screen ‘The Cognitive Assessment Screen for use During ECT’ (The CASDECT) which aims to address some of the limitations of existing batteries. The CASDECT is based on following sources of information: studies included within this thesis which have investigated the subjective and objective cognitive changes which occurred during a course of ECT for 13 individuals; feedback regarding cognitive tests from consumers of ECT who took part in those studies; a questionnaire of current practice of cognitive assessment in New Zealand which also collected feedback from practitioners who conduct the cognitive assessments; and finally, the screen is informed by previous peer reviewed literature pertaining to the assessment of ECT related cognitive change. The pertinent results of these studies included within this thesis will be briefly restated, and the rationales for the selected items which comprise the screen are offered.

Results and Discussion

Study 1

Questionnaire of Current Practice of Cognitive Assessment in Aotearoa

Chapter Four of this thesis reported the results of a questionnaire sent to health professionals in New Zealand, which enquired about current practice of cognitive assessment during a course of ECT. These results summarised below informed the development of the screen:

- Of a sample of 24 respondents, it was determined that cognitive assessments are carried out by people from a range of different professional backgrounds. Many are conducted by nurses (38.9%). Assessments are also conducted by psychiatrists (22.2%) and clinical psychologists (22.2%). A small minority of assessments are conducted by junior doctors/ registered medical doctors (11.11%) and a data analyst (5.5%). Given this

finding, it is unlikely that the individual carrying out the assessment will have been trained in psychometric administration. Accordingly, tests should be simple to administer, score and interpret and thorough instructions should be provided to minimise variations in administration across the assessments.

- The average reported time spent conducting cognitive assessments with patients was 23 minutes, with large variation between respondents ($SD=16.8$ mins). Typically, 10 minutes (45%) was spent conducting assessments. When asked how long an ideal cognitive screen should take, respondents reported that on average, 17 minutes ($SD=8.26$) would be feasible.
- Restricting more thorough and frequent cognitive assessments was a lack of time (100%), resources (50%), and suitable screening measures sensitive to ECT related cognitive impairment (41.6%).

Study 3

Objective Cognitive Change during ECT

Chapter Seven of this thesis reported the results of a study which investigated objective cognitive change throughout a course of ECT. A broad range of cognitive domains were assessed, and the measures which were sensitive at detecting cognitive decline from baseline to during the course of treatment are shown in Table 1.

Table 1

Objective Cognitive Change which occurred from Baseline to during the ECT Course

<i>Variable</i>	<i>Assessment Measure</i>	<i>Effect Size</i>	<i>95% Confidence Interval</i>	<i>Qualitative Size</i>	<i>Direction</i>
Retrograde Amnesia	Autobiographical Memory Interview	1.07	0.47-1.65	Large	Decline
Short term Verbal Memory	Delayed recall phase - MoCa	0.62	0.04-1.19	Medium	Decline
Long term Visual Memory	Delayed Recall – (30min) MCGCF	0.57	-0.01-.14	Medium	Decline
Long term Verbal Memory	Delayed Recall (30 min)- MoCA	0.49	-0.08-.06	Medium	Decline
Verbal Fluency	MoCA	0.39	-0.19-.95	Small	Decline
Information Processing Speed	Coding Task	0.20	-0.39-0.76	Small	Decline

As retrograde amnesia showed a large decline during the course of treatment, the items which make up the Autobiographical Memory Interview (AMI; Kopelman et al., 1989) and the additional memory items were further analysed to determine which were sensitive to decline from the baseline assessment to during the course. The items most sensitive to decline yielded from this analysis are offered below:

Table 2

Change in items Assessing Autobiographical and Semantic Memory from Baseline to During the Course of Electroconvulsive Therapy

<i>Question</i>	<i>Effect Size</i>	<i>Confidence Interval</i>	<i>Qualitative Size</i>	<i>Direction</i>
What date did you start your treatment?	0.68	0.15-1.31	Medium	Decline
Can you tell me about something that has happened in the news over the past couple of weeks?	0.65	0.12-1.28	Medium	Decline
How long have you resided at your current address?	0.61	-0.31-0.83	Medium	Decline
What did you have for dinner the night before last?	0.61	0.05-1.20	Medium	Decline
The last holiday you went on – When was this? (Date?)	0.48	-0.27-0.87	Medium	Decline
Where did you spend last Christmas?	0.44	-0.09-1.06	Medium	Decline
Tell me about an incident involving a relative that's happened in the past year?	0.44	-0.10-1.06	Medium	Decline

Subjective reports of cognitive function and performance on objective cognitive tests were only congruent at the baseline assessment. During the course of ECT, subjective reports of cognitive function increased on the SSMQ despite a decline in cognitive function occurring.

Study 4

Subjective Cognitive Change during ECT

Chapter Eight of this thesis reported the results of a study which investigated patients' perspective of cognitive change throughout a course of ECT. Qualitative content analyses revealed that during the course of ECT, cognitive dysfunction occurred across a broad range of cognitive domains. The most common cognitive complaint was of difficulties with general memory. Patients also reported impairment in concentration and attention, personal semantic memory, general short term memory, decision making, executive functioning, orientation, information processing speed, general cognitive function, general long term memory, prospective memory, episodic autobiographical memory, visual memory and semantic memory.

Scores on the subjective memory questionnaire improved progressively throughout the course of treatment, which was likely due to the alleviation of depression. The items sensitive to change during the course of treatment are displayed in Table 3 below. The items insensitive to change, were those which assessed ability to recall things that happened a long time ago, remembering new information and remembering what the individual was doing after they took their mind off it.

Table 3

Effect sizes of change in SSMQ items which occurred from baseline to during the course of ECT

<i>Item in SSMQ</i>	<i>Effect Size</i>	<i>Confidence Interval</i>	<i>Qualitative Size</i>	<i>Direction</i>
My ability to remember what I read and watch on television:	-1.18	-1.69 - -0.50	Large	Improvement
My ability to make a memory that is on the tip of my tongue available:	-0.70	-1.30-0.015	Medium	Improvement
My ability to pay attention to what goes on around me:	-0.63	-1.18- -0.03	Medium	Improvement
My ability to follow what people are saying	-0.54	-1.10-0.04	Medium	Improvement
My ability to remember things when I really try:	-0.54	-1.12-0.02	Medium	Improvement
My ability to remember things that happened over a year ago	-0.52	-1.14-0.01	Medium	Improvement
My general alertness to things happening around me is:	-0.48	-1.06-0.08	Medium	Improvement
My ability to recall names:	-0.44	-0.394-0.20	Medium	Improvement

Subjective depression was also assessed using the Beck Depression Inventory (BDI II; Beck et al., 1996). The items most sensitive to decline are displayed in Table 4 below.

Table 4

Subjective Changes in Mood as measured by the Beck Depression Inventory-II from Baseline to during the course of ECT

<i>Item in BDI</i>	<i>Effect Size</i>	<i>Confidence Interval</i>	<i>Qualitative Size</i>	<i>Direction</i>
Agitation	1.16	0.33-1.93	Large	Decline
Concentration Difficulties	1.11	0.29-1.88	Large	Decline
Changes in Appetite	1.06	0.24-1.82	Large	Decline
Suicidal ideation	1.04	0.23-1.80	Large	Decline
Loss of Pleasure	1.01	0.20-1.77	Large	Decline
Tiredness/ Fatigue	0.99	0.17-1.74	Large	Decline
Self-Dislike	0.94	0.14-1.70	Large	Decline
Changes in sleeping patterns	0.87	0.14-0.70	Large	Decline
Worthlessness	0.86	0.08-0.63	Large	Decline
Irritability	0.85	0.06-1.61	Large	Decline
Loss of Interest	0.82	0.03-1.57	Large	Decline

Pessimism	0.81	0.01-1.55	Large	Decline
Loss of Energy	0.70	-0.07-1.46	Medium	Decline
Sadness	0.70	-0.09-1.43	Medium	Decline
Feelings of Punishment	0.62	-0.16-1.36	Medium	Decline
Loss of Interest in Sex	0.62	-0.16-1.36	Medium	Decline
Self Criticalness	0.52	-0.25-1.26	Medium	Decline
Indecisiveness	0.51	-0.26-1.25	Medium	Decline
Feelings of Guilt	0.48	-0.29-1.22	Medium	Decline
Feelings of Past Failure	0.42	-0.34-1.16	Medium	Decline
Crying	0.40	-0.36-1.14	Medium	Decline

All items in the BDI-II were sensitive to decline during the course of treatment; therefore the BDI would be a suitable adjunct to the assessment. However, there was some informal feedback given on the BDI-II which could be considered for future research into the assessment of mood during ECT.

One point frequently noted by consumers was: because ECT is a time of rapid change in mood and cognitive function, anchors on self reported measures should be bidirectional. For example, currently on the BDI-II, there are 4 options for every item, which focus on the symptoms of depression but this measure does not capture any improvement in depression beyond zero thus a significant floor effect exists. A suggestion made by a consumer of the treatment was to have a more dynamic approach to capture positive changes which inevitably occur for most individuals. For example, the following format the BDI-II could be adopted, as demonstrated by items 1 and 2 below.

1. Sadness

- 3. I am extremely happy all of the time
- 2. I am happy all of the time
- 1. I am happy much of the time
- 0. I do not feel sad
- 1. I feel sad much of the time

2. I am sad all of the time
3. I am so sad I can't stand it

2. Pessimism

- 3. I have a lot of hope for my future and things will only get better
- 2. I expect things to work out for me
- 1. I feel more hopeful about my future than I used to be.
0. I am not discouraged about my future
1. I feel more discourage about my future than I used to be
2. I do not expect things to work out for me.
3. I feel my future is hopeless and will only get worse.

However, doing so would have the limitation of scores being washed out by the pooling of the data (for example adding negative and positive responses together will cancel each other out).

An alternative to this would be adding in a measure which assesses positive mood states, such as the Affectometer (Kammann & Flett, 1983).

In addition, the BDI-II asks respondents to circle the answer which best describes how the individual has been feeling '*over the past two weeks including today*'. These instructions were difficult for the patients to follow, as often rapid changes in mood had occurred over the past two weeks and patients' self reports of mood were likely to be biased based on their current mood state. Thus, a further adaptation which could be put in place when using the BDI-II with patients receiving ECT would be to require responses based on how the patient is *currently* feeling.

Development of the Screening Measure

Considerations

It has been suggested that the ideal cognitive assessment tool would be brief, inexpensive, simple to administer, sensitive to change in attention and verbal and non-verbal memory and have alternate forms to control for practice effects (Coffey, 1998). In addition, as ECT affects a

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broad range of cognitive domains in addition to memory, cognitive assessments need to be comprehensive across domains. Cognitive screens need to be brief, and sensitive to detecting ECT related cognitive changes, and simple to administer and score as people from a wide range of professions carry out the assessments. Finally, any screening measure used with individuals receiving ECT in New Zealand and many other developing countries should be appropriate for use with older adults, as the majority of people receiving ECT (68.85%) are over the age of 55 (calculated from Ministry of Health, (2012). These considerations were taken into account when developing the CASDECT.

Inclusions and Exclusions of Items for the CASDECT

As the objective of the cognitive screening measure was to detect cognitive decline during a course of treatment, only items which showed a large or medium *decline* from baseline to during the course of treatment were included within the screen. Items which *improved* during the course (such as subjective memory items from the SSMQ) were not included for the sake of maintaining brevity.

The first task within the screen assesses orientation to time, as orientation to person and place did not tend to be effected during ECT when cognitive assessments were conducted 48 hours post ECT treatment (Study 3). The second task is the encoding phase of the verbal items which are used to assess verbal memory later on in the assessment. This item and its instructions are adopted from the Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005). To abide by the copyright restrictions which protect the MoCA, new words were generated. These words were matched as closely as possible for word frequency, number of syllables and semantic association, which follows the procedure of previous research which has generated alternate forms for the RAVLT (Geffen, Butterworth, & Geffen, 1994). These words were sourced from a website containing this information (Bringham Young University, 2014).

Visual memory is then assessed using the Medical College of Georgia Complex Figures (Loring & Meador, 2003) which has four alternate forms and good psychometric properties (Fred Ingram, Soukup, & Fishel Ingram, 1997). Typically, when complex figures are administered, a 30 minute delay occurs prior to the recall phase being conducted (Lezak et al., 2004). Due to the brevity of the current screen, only a 15 minute period lapses between the copy and recall phase; however, research has shown that the length of the delay does not make a significant difference to visual memory performance, so long as the delay does not exceed one hour (Berry & Carpenter, 1992).

The next component of the assessment is a digit symbol coding task, which assesses information processing speed, a cognitive domain sensitive to the effects of brain injury (Comerford, Geffen, May, Medland, & Geffen, 2002). To protect copyright of other tests which use the coding of symbols and numbers to tests information processing speed, three new alternate versions of a coding task were generated using new symbols. The instructions for this task were adopted from the Coding Task from Repeatable Battery for the Assessment of Neuropsychological Status (RBANS; Randolph, 1998). Another measure within the MoCA found to be sensitive to ECT related decline was verbal fluency, a measure of executive functioning. Three alternate versions of verbal fluency are offered, and verbal fluency is assessed after the Coding Task, and again, the instructions for this task were adopted from the MoCA. The letters included in the MoCA, are F, B and S. These letters have a frequency of 2.30%, 1.49% and 6.28% respectively. The letters selected for the verbal fluency task on the CASDECT were S, R and H which have frequencies of 6.28%, 6.02% and 5.92% respectively (Cornell University, 2014). These letters were selected as they were more comparable than the letters offered in the MoCA. The instructions for the verbal fluency task were based on those from Strauss and Spreen (2006). The task of reciting months of the year backwards, a task of working memory which was found to be sensitive in Martin et al.'s (2013) study, was included

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in the CASDECT after the Coding Task. This task was adapted for repeated use by starting with a different month of the year for each assessment.

In Study 3 which investigated objective cognitive change during ECT, attention did not appear to be adversely affected by ECT; however, previous literature has stressed the importance of including a measure of attention within the cognitive assessment (Coffey, 1998) and previous researchers have found attentional deficits to occur during a course of treatment (Semkowska & McLoughlin, 2010), therefore a measure of attention was included within the screen. The selected measure was digit span forwards, as this measure has alternate forms, and has been shown in previous literature to be sensitive to decline during ECT (Semkowska & McLoughlin, 2010).

Retrograde amnesia is a common complaint during ECT (Sackeim et al., 2007; Schulze-Rauschenbach et al., 2005), and a section assessing retrograde amnesia is included within the assessment. This section comprised of seven questions which were shown to be sensitive to decline determined from Study 3.

Study 4 revealed that subjectively, cognitive impairment can occur across a broad range of cognitive domains, and previous research has shown that objective and subjective cognitive function are often incongruent (Berman et al., 2008; Nehra et al., 2008; Prudic et al., 2000; Vamos, 2008) which adds to the importance of assessing both subjective and objective cognitive function. Therefore, a subjective report section is dedicated within the CASDECT for patients to freely describe to the clinician conducting the assessment the difficulties or changes in cognitive function the patient has noticed since starting the course of ECT. The final items of the CASDECT included the long term verbal and visual recall items from the MoCa and the Medical College of Georgia complex figure respectively.

Cognitive domains that did not change during ECT, and therefore are not included in the CADECT were abstract reasoning and language. This finding is in accordance with previous studies which have also found these domains to be insensitive to change (Jones, Henderson, & Welch, 1988; Tiller & Lyndon, 2003). Memory questions which assess memories formed further back in time were less sensitive to change than memory questions which occurred closer in time to ECT treatment. This finding also mirrors what has previously been established in the ECT literature (Calev et al., 1993; Cohen & Squire, 1981; Fraser et al., 2008; Peretti et al., 1996; Rami-Gonzalez et al., 2001; Sackeim et al., 1993). Thus only the memory items assessing memories formed closer in time to treatment were included in the screen.

Applications, Implications and Limitations of the CASDECT

The CASDECT has three alternate forms and is formatted so that three cognitive assessments can be conducted consecutively during a course of ECT treatment. Any cognitive change which has occurred from one assessment to the next can be directly compared on the record form or the scoring table provided at the end of the measure. The screening measure takes approximately 20 minutes to administer, includes thorough instructions, is simple to score, will be free for practitioners to use and has been designed to be sensitive to detecting cognitive changes across a broad range of cognitive functions as they occur during a course of ECT. Thus, this screen has potential to be used to monitor cognitive change over a course of ECT treatment, to assess whether cognitive decline is occurring.

The greatest limitation of the proposed screening measure is that the psychometric properties are currently unknown. There is a need for future research to establish the psychometric properties of the measure to facilitate its use. In particular, the sensitivity of the screen to detect ECT related cognitive change should be further assessed. Although the BDI-II proved sensitive to changes in mood throughout the course of ECT as determined by Study 1, consumers of the measure noted shortcomings which may affect the sensitivity of the measure during ECT.

Future research should adapt the BDI-II or develop a new mood measure which assesses current

mood state and has bidirectional anchors, so is able to capture both the positive and negative changes in mood which typically occur during a course of treatment.

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Cognitive Assessment Screen for use during ECT (CASDECT)

Patient Name _____ D.O.B: _____

Note to practitioner: Conduct a baseline assessment prior to any ECT treatment. Assess cognition again intermittently throughout the course of ECT, approximately 48 hours post treatment. The screen can be used for three assessments, practice effects may occur thereafter. Augment the assessment with a measure of mood. Score assessment on final page.

Additional Materials required: 2x A4 paper, pencil, pen, eraser, stopwatch and Medical College of Georgia Complex Figure and scoring sheet.

Introducing the CASDECT:

“I am going to ask you some questions and get you to do some short tasks, some of them you may find hard and some you may find easy. The purpose of doing these tests is to track any changes in memory or other functions that might be happening over your course of ECT. The most important thing is that you do your best. Do you have any questions?”

Q1. Date:

Can you tell me today’s date including the day of the week, the month and the year?

Assessment A: D____D____M____Y____ (/4)

Assessment B: D____D____M____Y____ (/4)

Assessment C: D____D____M____Y____ (/4)

Q2. Verbal Memory:

Instructions: “I am going to read you a list of words, twice. After each time, I would like you to repeat the words back to me, in any order”: (Tick box for each correct repeat, do not score this item).

Assessment A:	Door	Olive	West	Toad	Hair
Trial 1					
Trial 2					

“Try and remember those words, as I will ask you to recall them again at the end of the test.”

Assessment B:	Mountain	Palace	Eye	Fruit	Seat
Trial 1					
Trial 2					

“Try and remember those words, as I will ask you to recall them again at the end of the test.”

Assessment C:	Visitor	Coffee	Grass	Fish	Prize
Trial 1					
Trial 2					

“Try and remember those words, as I will ask you to recall them again at the end of the test.”

Q3. Complex Figure Copy

Place one of the four Medical College of Georgia Complex figures in front of the patient (alternate version every assessment). Give the patient a blank A4 piece of paper, a pencil and an eraser. Orientation of paper should be portrait. Minimum exposure time: 2.5 min, maximum: 5 min.

Instructions: *“See this figure, I would like you to copy this figure onto this blank sheet of paper. I would like you to do a good job so I know that this figure is the one that you have copied.”*

Q4. Coding Task

Give patient correct version of coding task (A,B or C included within screen).

Instructions: *“Look at these boxes”* (point to key). *“For each one of these symbols there is a number that goes with it. Down here there are symbols, but no numbers. I want you to fill in the number that goes with each symbol.”* (Demonstrate the first three). *“Now I would like you to fill in the rest of these boxes up to the double lines (indicate) for practice”.* (Correct any errors as they are made. Make sure that the examinee understands the task and has completed the sample items before you begin timing.) *“Now I would like you to continue to fill in the numbers that match the marks. Go as quickly as you can without skipping any. When you reach the end of the line, go on to the next one. Ready? Begin.”*



Time 90sec.

Q5. Verbal Fluency

Instructions: “I am going to give you a letter of the alphabet, and I would like you to tell me as many words as you can think of starting with that letter as quickly as you can. For example if I say “B” you could say “bad, battle, bug...” There are a few rules. Don’t say the names of people or places like “Bob, or Bluff or Belgium” Also do not use the same word with different endings such as “eat” “eats” “eating”. Also don’t say numbers. I will tell you to stop after 60 seconds.

Do you have any questions?” “The letter is ____ . Begin!”



Time 60sec.

Assessment A: **S**

Assessment B: **R**

Assessment C: **H**

Total: ()

Total: ()

Total: ()

Q6. Months of the Year Backwards

Instructions: “Now I would like you to say all 12 months of the year backwards, starting with _____. Remember to say all 12 months” (Choose starting month indicated below).

Assessment A (/12)

October	September	August	July	June	May	April	March	February	January	December	November

Assessment B (/12)

August	July	June	May	April	March	February	January	December	November	October	September

Assessment C (/12)

June	May	April	March	February	January	December	November	October	September	August	July

Q7. Digit Span Forwards

Instructions: “I am going to read you some numbers, and when I am finished, I would like you to repeat them back to me exactly as I have said them”

Assessment A (/3)

Assessment B (/3)

Assessment C (/3)

42731

54187

21854

392847

628175

59736

4173386

83754216

7392864

Q8. Autobiographical Memory

A1 (_/7)

A2 (_/7)

A3(_/7)

Instructions: “Now I am going to ask you some questions, please answer them as accurately as you can.”

1. What date did you start your ECT treatment? Or, what date did you arrive at the hospital?
2. Can you tell me about something that has happened in the news over the past couple of weeks?
3. How long have you resided at your current address?
4. What did you have for dinner the night before last?
5. Think about the last holiday you went on – What was the date of this holiday?
6. Where did you spend last Christmas?
7. Can you tell me about an incident involving a relative that has happened in the past year?

Q9. Subjective Report

Instructions: “Since starting ECT, have you had any difficulty with your memory, or any other changes in mental processes (attention, concentration, decision making etc) that you have noticed? Please explain.”

A1 _____

A2 _____

A3 _____

Q10. Long Term Verbal Memory

Instructions: “I read some words to you earlier, which I asked you to remember. Tell me as many of those words as you can remember.”

Assessment A:

(_/15)

	Door	Olive	West	Toad	Hair
Free Recall					
Category Cue	Part of a building	Type of food	Type of Oreintation	Type of animal	Part of the body
Multiple Choice	Window, Door, Room	Onion, Gherkin, Olive	West, North, East	Snake, Toad, Lizard	Hair, nose, eyes

Assessment B:

(_/15)

	Mountain	Palace	Eye	Fruit	Seat
Free Recall					
Category Cue	A geographical feature	Type of building	Part of the body	Category of food	A piece of furniture
Multiple Choice	Lake, Mountain, Forest	Castle, Mansion, Palace	Eye, Nose, Mouth	Vegetables, fruit, meat	Seat, Sofa, Chair

Assessment C:

(_/15)

	Visitor	Coffee	Grass	Fish	Prize
Free Recall					
Category Cue	Type of person	Something you can drink	Something that grows	Type of animal	Something a person can receive
Multiple Choice	Tourist, visitor, guest	Tea, juice, coffee	Grass, lawn, field	Lizard, fish, bird	Prize, reward, trophy

Q11. Complex Figure Recall

Instructions: “A short time ago I asked you to copy a figure for me; I would like you to draw that figure again but this time from memory. Draw that figure here.” (Give patient A4 piece of paper in portrait orientation, pencil and eraser.)

1	2	3	4	5	6	7	8	9
Δ	\diamond	Θ	Π	U	O	\wedge	X	=

SAMPLE _____

\wedge	U	Δ	X	\diamond	Π	Θ	U	O	Θ	=	\diamond	X	U

Π	=	\diamond	\wedge	Θ	U	X	\diamond	Δ	\wedge	U	O	=	\wedge

X	=	\wedge	Θ	\diamond	O	Δ	U	Π	Θ	X	Δ	U	=

Δ	U	Π	=	\diamond	Θ	O	Δ	Π	O	\diamond	X	\wedge	=

\diamond	X	Δ	=	O	Δ	\wedge	=	Π	Θ	\wedge	Δ	U	O

X	Θ	\diamond	Δ	=	O	Θ	Π	U	\diamond	\wedge	X	O	Π

1	2	3	4	5	6	7	8	9
=	Δ	\diamond	Θ	Π	U	O	\wedge	X

SAMPLE _____

\wedge	U	Δ	X	\diamond	Π	Θ	U	O	Θ	=	\diamond	X	U

Π	=	\diamond	\wedge	Θ	U	X	\diamond	Δ	\wedge	U	O	=	\wedge

X	=	\wedge	Θ	\diamond	O	Δ	U	Π	Θ	X	Δ	U	=

Δ	U	Π	=	\diamond	Θ	O	Δ	Π	O	\diamond	X	\wedge	=

\diamond	X	Δ	=	O	Δ	\wedge	=	Π	Θ	\wedge	Δ	U	O

X	Θ	\diamond	Δ	=	O	Θ	Π	U	\diamond	\wedge	X	O	Π

1	2	3	4	5	6	7	8	9
◇	Θ	Π	U	O	^	X	=	Δ

SAMPLE _____

^	U	Δ	X	◇	Π	Θ	U	O	Θ	=	◇	X	U

Π	=	◇	^	Θ	U	X	◇	Δ	^	U	O	=	^

X	=	^	Θ	◇	O	Δ	U	Π	Θ	X	Δ	U	=

Δ	U	Π	=	◇	Θ	O	Δ	Π	O	◇	X	^	=

◇	X	Δ	=	O	Δ	^	=	Π	Θ	^	Δ	U	O

X	Θ	◇	Δ	=	O	Θ	Π	U	◇	^	X	O	Π

Scoring Sheet

Question Number. Maximum points in brackets.	Cognitive Domain Assessed	Baseline Assessment Date: Number of Treatments:	Second Assessment Date: Number of Treatments:	Third Assessment Date: Number of Treatments:
Q1: Orientation (4pt)	Orientation to time.			
Q3: Complex Figure Copy Phase (36 pt)	Planning, organisation, attention.			
Q4: Coding Task (total correct)	Information processing speed.			
Q5: Verbal Fluency (total# words produced)	Executive Function			
Q6: Months of year backwards (12 pt)	Working memory, executive functioning.			
Q7: Digit Span (3pt)	Attention			
Q8: Autobiographical Memory (7 pts)	Retrograde Amnesia for Autobiographical Memories.			
Q10: Recall of 5 words (15pt)	Anterograde amnesia. Long term verbal memory.			
Q11: Recall Complex Figure (36 pt)	Anterograde Amnesia. Long term visual memory.			



MASSEY UNIVERSITY
GRADUATE RESEARCH SCHOOL

**STATEMENT OF CONTRIBUTION
TO DOCTORAL THESIS CONTAINING PUBLICATIONS**

(To appear at the end of each thesis chapter/section/appendix submitted as an article/paper or collected as an appendix at the end of the thesis)

We, the candidate and the candidate's Principal Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of Candidate: Anneke Thornton

Name/Title of Principal Supervisor: Janet Leatham

Name of Published Research Output and full reference:

The Development of the CASDECT – a Cognitive Screening Measure for use during Electroconvulsive Therapy

In which Chapter is the Published Work: Nine

Please indicate either:

- The percentage of the Published Work that was contributed by the candidate:
and / or
- Describe the contribution that the candidate has made to the Published Work:

The candidate had full responsibility for the design, data collection, analysis and write up of the research. Supervisors provided guidance and were involved in decisions made around research processes and provided input with data analysis and formatting of the thesis. For these reasons Professor Janet Leatham and Dr Ross Flett were included as co-authors for the manuscripts which comprise this thesis.

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CHAPTER TEN

General Discussion and Reflections on the Research

This thesis entitled “Cognitive Change and Assessment during Electroconvulsive Therapy” has involved conducting a number of different studies investigating different populations in order to improve the standard of cognitive assessment with patients receiving ECT. The first study was a questionnaire to health professionals investigating current practice of cognitive assessment during a course of ECT in Aotearoa, New Zealand. The second study involved collecting normative data for a test of motor dexterity, and the two main studies investigated the objective and subjective cognitive changes which occur during ECT. From the results of these studies and the peer reviewed literature, a screening measure was suggested. This final chapter will offer a personal reflection on the research. This reflection will include some ethical considerations which were made when conducting the research and will also discuss some challenges and benefits of conducting research with this population.

Ethical Considerations

When conducting this research with the 14 individuals receiving ECT for severe psychiatric illness, some ethical considerations inevitably resulted. These included ethical considerations surrounding the construction of the assessment procedure, determining patients’ ability to provide informed consent to carry out the assessment and contribute to the research, managing patient risk, and sharing the assessment results with the patient.

Assessing cognitive function during a course of ECT carried a large responsibility, as the way in which the treatment was administered or whether or not the treatment was continued was, in many cases, informed by the results of the cognitive assessments. Therefore, it was critical that the measures selected for the assessments were sensitive and psychometrically sound, that good rapport was developed between myself and the patient and that the cognitive assessment was

only conducted if the patient was able to exert their best efforts. The assessments had to control for practice effects, and subjective reports of cognitive change were taken at each assessment point.

Informed consent to conduct the assessments for the research was obtained prior to the patient commencing the course of ECT; however, it soon became apparent that for some patients informed consent needed to be obtained prior to *each* assessment as even though the research had been explained in detail, previous assessments had been conducted and the patient was in possession of an information pack outlining the purpose of the research, this did not mean the patient would remember this information at each assessment point, particularly if they were prone to anterograde amnesia during the treatment. On a number of occasions, patients were able to recognise me but not remember who I was, not remember what the research was about and what the purpose of the assessments were, thus I had to consider on a case by case basis whether informed consent needed to be obtained prior to every assessment.

A further consideration was whether or not the individual was *able* to give informed consent to participate in the research, particularly if they were receiving ECT under the mental health act. Thus all patients were recruited first through the treating psychiatrist or their mental health nurse. These treating professionals made the decision as to whether or not the individual was competent to make the decision to take part in the research. Only after they were deemed competent were they referred onto me.

From administering over 85 cognitive assessments to 14 individuals, a trend was observed that the cognitive assessments were well received and tolerated; however, many individuals found it distressing to fill out the depression scale (BDI-II). Possibly because it required the patient to scrutinise their symptoms of depression. Although distressing, the BDI-II was an invaluable measure for gauging the efficacy of ECT and provided an additional measure of suicidal ideation. Given that ECT is a treatment reserved for those suffering from severe treatment

resistant depression and bipolar disorder, it was not uncommon for patients to engage in suicidal ideation or attempts during the course of treatment. Although I was solely responsible for the assessment of the patients' cognition, administering the BDI-II was beneficial as it allowed for suicidal ideation to be assessed each time I met with the patient. Thus throughout the course of research, numerous emails were sent to patients' treating psychiatrists when concerns of risk were raised, especially if the individual was an outpatient. After every cognitive assessment conducted, a report was sent to the patients' treating psychiatrist, prescribing psychiatrist, ECT nurse, and if applicable their psychologist and community nurse outlining any cognitive change and concerns for safety.

A further ethical consideration was whether or not the results of the cognitive assessments should be shared with the person receiving the treatment. There are some advantages and disadvantages of doing so. Sharing the results could be advantageous for a person who is voluntarily receiving ECT treatment (i.e. not under the mental health act), as knowledge of any cognitive change could help inform their decision whether or not to continue with the treatment. Objective evidence of impairment or cognitive change could be validating for the person if he or she is suffering some subjective cognitive impairment. It may also be useful for the person to see objectively, any clinical benefits he or she is receiving from the treatment. The disadvantages of sharing assessment results with the patient could be that the patient could prematurely decide to withdraw from treatment if ECT was not immediately effective and misunderstanding or catastrophising about cognitive change which is likely to resolve over a short period of time. Furthermore, ECT is often referred to as a 'last resort treatment', for a person who is severely unwell and desperate to get well, hearing news that ECT, their last hope for improvement, is not 'working' for them could be extremely disheartening.

A situation arose during the course of the research which raised this ethical consideration. I was conducting repeated assessments with a woman who was voluntarily receiving ECT for severe treatment resistant depression. After nine treatments of ECT, she was still not receiving any

clinical benefit from the treatment and was complaining of memory loss which was interfering with her occupational functioning. After a request from her, I agreed to share with her the results of her cognitive testing, which indeed showed some objective cognitive decline and also increases in depression severity. Despite this, the woman decided to continue with further treatment and fortunately after a few more treatments of ECT, did receive some clinical benefit and her cognitive function returned back to normal by the six week follow-up assessment. On another occasion, a patient who was receiving ECT under the Mental Health Act, and who had very little insight into her clinical state had requested to stop ECT treatment as she believed that ECT was not helping her. Outcomes collecting using the BDI-II showed stark improvements in her symptoms of depression occurring after every three treatments, and the ECT administering psychiatrist called me in to share these results with her. This was reassuring for her to see. She decided to continue with the treatment and benefitted clinically from three more treatments of ECT.

Challenges of Conducting Research with People receiving ECT

Conducting cognitive assessments with people receiving ECT posed some challenges, which included: difficulty conducting baseline assessments from which further assessment should be compared; the variation in the length of assessments, communication difficulties; and considerations for assessments which differ from assessing cognition with the general population.

One of the limitations of the research was that only one baseline cognitive assessment could be conducted and only one follow-up assessment. On two occasions, a baseline cognitive assessment was not able to be conducted. This was largely due to the acuity in which patients received ECT, and it was not uncommon to receive a call on Wednesday afternoon, requesting a baseline cognitive assessment for a patient starting a course of treatment the following morning. Repeated follow-up cognitive assessments could not be ethically justified, as excessive cognitive assessments would not have benefitted the patient, only the research.

Although the cognitive assessments were designed to take 50 minutes, the actual length of assessment fluctuated across participants. Each patient was highly variable in terms of how their illness affected them and their cognitive capabilities. Some patients had severe psychomotor slowing from the depression, and cognitive assessment long exceeded the predicted time. For one patient, concentration and attention difficulties were so profound that in order to obtain a response I needed to read out each item of the SSMQ and BDI-II. This significantly lengthened the duration of the assessment. Some patients remembered the instructions for the cognitive tasks at subsequent assessments, and for some patients it appeared as though they were seeing each task for the very first time, despite having done numerous assessments. Thus the timing for the assessments was variable and ranged from about 35 minutes to over 100 minutes.

As the assessments were frequently repeated, tests with alternate forms were important. It was also important to keep the cognitive assessment to under an hour, so the individual could sustain concentration and not subject them to undue distress. It was also important for the patient to view the cognitive assessment as a positive component to their treatment put in place to protect the patient's cognition rather than a tedious or anxiety provoking procedure. Some education around why the assessments were beneficial for the patient was important and assisted in the patients' motivation to complete the tasks.

Another challenge in conducting research with this population was scheduling appointments for assessments. It was not uncommon for patients to move from the inpatient unit, to respite, back home then back to the inpatient unit over the period of two weeks. Therefore each assessment often required talking to multiple sources to locate the patient. As ECT was typically administered on a Monday and Thursday, and cognitive assessments took place two days post treatment, half of the assessments took place on a Saturday. This may not be practical for patients in an outpatient setting, and staff may not always be available to conduct assessments during the weekend. Another difficulty was locating patients for the six week follow-up assessment. Although for the current research, I was able to drive to the patients' house and

meet them which facilitated collecting this information, in hospital and other research settings it may be difficult to get the person to come back for a follow-up assessment, particularly if they are experiencing a relapse in depression.

Benefits of Conducting Research with People receiving ECT

Working with people receiving ECT and being responsible for the assessment of their cognition was an honourable and rewarding undertaking. It allowed me to get to know each person quite well, particularly if they ended up getting multiple or long courses of treatment. Talking with each person receiving the treatment revealed different attitudes about the treatment and its side effects. For some people, cognitive function, particularly their memories, were an integral component to their wellbeing and their identity. Thus for these people any cognitive decline was extremely distressing and often depersonalising. For other people, cognitive impairment was a small price to pay for the improvement in mood, and was perceived as a small price to pay for the alleviation of the suffering from depression.

The people taking part in the study and many of their families showed immense support by taking part in the research, travelling to undertake the assessments or inviting me to their homes to conduct the assessments. In addition, the support received from the patients' psychiatrists and other health professionals was also very humbling. Conducting research into the subjective experience of cognitive change, allowed each person to express their concerns about any cognitive changes they were enduring, and allowed them to have their voices heard. I was also able to spend more time with each person than a medical professional would have been able to, ensuring that the individual had sufficient time to voice their concerns and ask any questions. Conducting my doctoral thesis in this area put me in a position where I was aware the recent research findings into what the duration and nature of the cognitive changes were, and put me in a position to share this knowledge with the people who enquired about it.

General Conclusion and Reflections

An observation from conducting this research was that for some individuals, ECT was extremely effective in reducing symptoms of depression, for others, ECT was effective but it took much longer to work and for a small number of participants, ECT appeared to have very little clinical benefit.

From this research, it is evident that a course of ECT is a time of rapid cognitive change; both in the form of impairment and improvement. All of the participants included in this study reported cognitive change due to ECT in the short term, and cognitive recovery or improvement occurring after finishing the course of treatment. Objective tests showed that ECT related cognitive impairment was generally short term; however, subjective report showed impairment still persisted for many at the six week follow-up. Thus one conclusion which could be drawn from this research is that obtaining a subjective report of cognitive function from the patient is of high importance. In addition, if the objective measures being used to detect cognitive impairment from ECT are insensitive to detecting impairment, subjective reports will increase the sensitivity of the assessment; however, as subjective reports of cognitive function are influenced by a patient's mood state, both subjective and objective cognitive outcomes should be documented.

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APPENDICES

Appendix A

Electronic Questionnaire Sent to Health Professionals Nationwide



INFORMATION SHEET

Part 1 of 2

Monitoring the Short term Outcomes of Electroconvulsive Therapy

Introduction

My name is Anneke Thornton and I am conducting doctoral research in clinical psychology at Massey University. The research is into assessing the short term effects of electroconvulsive therapy (ECT).

From my research I hope to develop an ideal screening measure for memory and cognition pre, during and post ECT which would be brief, relevant and simple to administer by a busy Psychiatrist who is prescribing ECT.

I am interested in finding out about the procedures currently in place around measuring cognition in your work place and whether you have any recommendations for the screening measure.

There are two short components to this questionnaire.

The first section asks questions regarding the practices around assessing cognition during a course of ECT and your answers to this section remain anonymous. The second section is separate from the first and asks some demographic information.

If you agree to take part in this survey, participation takes around 5-10 minutes to complete both sections.

Your participation in this research is hugely appreciated.

If you would like to know more about this research after reading this information, please contact:

Thank you,
Anneke Thornton

Researcher

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*This project has been reviewed and approved by the
Health and Disability Ethics Committee: Central Region
If you have any concerns about the conduct of this research,
please contact Central Region Ethics Committee,
Ph: (04) 816 2403, Email: central_ethicscommittee@moh.govt.nz*

What assessment measures are utilised in your workplace for monitoring cognition during ECT?

- Mini Mental Status Examination (MMSE)
- Extended Mental Status Examination (EMSE)
- Modified Mental Status Examination (3MSE)
- Montreal Cognitive Assessment (MoCa)
- Addenbrooke's Cognitive Assessment –Revised (ACE-R)
- Frontal Assessment Battery (FAB)
- Other (Please specify)

Do you include a mood measure alongside your cognitive assessment?

- No
- Yes (Please specify)

If anxiety or psychosis is assessed, what measures are used to assess these?

Who conducts the cognitive assessment?

- Nurse
- Psychiatrist
- Other (please specify)

How long do you spend doing each cognitive assessment with patients receiving ECT?

- <10 minutes
- <20 minutes
- <30 minutes
- <40 minutes
- <50 minutes
- <60 minutes
- >1 hour
- Other (Please specify)

How frequently do you monitor cognition during a course of ECT?

(Please check all that apply.)

- Cognition is not monitored
- Before any ECT
- After the fourth session
- After the fifth session
- After the eighth session
- After the ninth session

<input type="checkbox"/> After every session	<input type="checkbox"/> After the sixth session	<input type="checkbox"/> After ECT treatment has ceased
<input type="checkbox"/> After the second session	<input type="checkbox"/> After the seventh session	<input type="checkbox"/> Other (Please specify) <input type="text"/>
<input type="checkbox"/> After the third session		

What is stopping you from doing more frequent or thorough cognitive assessments?

(Please check all that apply.)

- Time
- Resources (such as money, availability of tests to use etc)
- A lack of sensitive assessment measures
- Nothing. Cognition is assessed frequently and thoroughly enough.

How long after a session of ECT do you assess cognition (not including reorientation)?

(Please check all that apply.)

- | | |
|--|--|
| <input type="checkbox"/> Immediately | <input type="checkbox"/> 48 hours post ECT |
| <input type="checkbox"/> One hour post ECT | <input type="checkbox"/> Cognition is not assessed after treatment |
| <input type="checkbox"/> 12 hours post ECT | <input type="checkbox"/> Other (Please specify) <input type="text"/> |
| <input type="checkbox"/> 24 hours post ECT | |

How long would the 'ideal' screening measure take to administer?

- | | |
|-------------------------------------|---|
| <input type="radio"/> 10 minutes | <input type="radio"/> 40-50minutes |
| <input type="radio"/> 10-20 minutes | <input type="radio"/> 50-60 minutes |
| <input type="radio"/> 20-30 minutes | <input type="radio"/> Other (Please specify) <input type="text"/> |
| <input type="radio"/> 30-40 minutes | |

Any other comments or suggestions?

Block 3

Thank you for your responses.

This section of the survey will be closed to anonymise responses.




A separate survey will then appear, to ask a couple of questions to ensure I have an adequate selection of respondents to develop a suitable screening measure.

You will also be able to provide a contact address if you wish to receive the screening measure.




Appendix B

Adapted Version of the Squire Subjective Memory Questionnaire

*This is a questionnaire on your memory.
Since having ECT, how would you now rate your...*

	Worse		No Change		Better				
									
Ability to recall...									
Names:	1	2	3	4	5	6	7	8	9
Things when I really try:	1	2	3	4	5	6	7	8	9
Things that happened a long time ago:	1	2	3	4	5	6	7	8	9
Things that happened during my childhood:	1	2	3	4	5	6	7	8	9
What happened a few minutes ago:	1	2	3	4	5	6	7	8	9
Ability to remember...									
The faces of people I meet:	1	2	3	4	5	6	7	8	9
What I was doing after I have taken my mind off it:	1	2	3	4	5	6	7	8	9
Things that have happened over a year ago:	1	2	3	4	5	6	7	8	9
What I read and what I watch on television:	1	2	3	4	5	6	7	8	9

Since having ECT, how would you now rate your...

	Worse		The Same		Better				
									
Ability to...									
Make a memory that is on the 'tip of my tongue' available:	1	2	3	4	5	6	7	8	9
Make sense of what people explain to me:	1	2	3	4	5	6	7	8	9
Pay attention to what goes on around me:	1	2	3	4	5	6	7	8	9
Follow what people are saying:	1	2	3	4	5	6	7	8	9
Other									
My relatives and acquaintances judge my memory to be:	1	2	3	4	5	6	7	8	9
My general alertness to things happening around me is:	1	2	3	4	5	6	7	8	9
In one month time, my ability to remember facts about this questionnaire would be:	1	2	3	4	5	6	7	8	9

Appendix C
Summary of Patient Characteristics

Participant	Primary Diagnosis	Specifier	Age	Sex	Ethnicity	Courses of ECT	Total ECT treatments	Electrode Placement	Inpatient/Out-patient	Medication
P1	Major Depressive Episode,	Recurrent, Severe. Without psychosis.	78	Male	NZ/European	1	8	Right Unilateral	Inpatient	Unknown
P2	Major Depressive Disorder	Recurrent, Severe. Without psychosis.	56	Male	NZ/Dutch	3	25	Bilateral	Inpatient	Lithium carbonate 230mg 3x daily Mirtazapine 30mg 2x daily
P3	Major Depressive Disorder	Recurrent, Severe. Without psychosis.	36	Female	NZ/ Māori	1	12	Right Unilateral	Outpatient	Zopiclone Parnate
P4	Bipolar I	Most recent episode hypomanic	39	Male	NZ/Chinese	1	15	Right Unilateral	Outpatient	Fluoxetine 40mg, Carbamazopine 300mg
P5	Major Depressive Disorder	Recurrent, Severe. Without psychosis.		Male	Māori	1	12	Right Unilateral	Inpatient	Nortriptyline
P6	Major Depressive Disorder	Recurrent, Severe. Without psychosis.	51	Male	NZ/European	1	11	Right Unilateral	Outpatient	Nortriptyline 200mg and Quetiapine 50mg
P7	Major Depressive Disorder	Recurrent, Severe. Without psychosis.	18	Female	NZ/European	1	7	Right Unilateral	Inpatient	None
P8	Major Depressive Disorder	Recurrent, Severe. Without psychosis.		Female	Māori	3	47	Right Unilateral	Inpatient and Outpatient	Zopiclone 7.5mg
P9	Bipolar-I disorder		44	Female	NZ European	1	9	Right Unilateral	Outpatient	Unknown
P10	Major Depressive Disorder	Recurrent, Severe. Without psychosis.		Female	British	1	12	Right Unilateral	Outpatient	Venlafaxine 300mg Metazapine 30mg
P11	Major Depressive Disorder	Recurrent, Severe. Without psychosis.	56	Female	NZ/European	2	10	Left Unilateral	Outpatient	Unknown
P12	Bipolar-I	Most recent episode manic	45	Female	NZ/European	1	12	Right Unilateral	Outpatient	Lithium, 600mg Tipiramate, 400mg
P13	Major Depressive Disorder	Recurrent, Severe. Without psychosis.		Female	South African	1	10	Right Unilateral	Inpatient	Prazosin 2mg nocte Lorazepam 1mg, promethazine 25mg, riperidone 0.5mg, Mirtazapine 15mg, Venlafaxine 187.5mg

Appendix D
Participants' Information Packs



MASSEY UNIVERSITY
COLLEGE OF HUMANITIES
AND SOCIAL SCIENCES
TE KURA PŪRINGA TĀNGATA

**Monitoring the Short term Effects of Electroconvulsive Therapy on Memory
and Thinking**

Cover Letter

Dear.....

My name is Anneke Thornton and I am conducting a study as part of a Doctor of Psychology qualification at Massey University. Over the next 18 months I will be working with psychiatrists to develop a new quick method for monitoring memory and thinking for those receiving electroconvulsive therapy (ECT).

You have been given this information pack as you have been prescribed a course of ECT and this letter is to ask you to participate in this study. When your psychiatrist discussed ECT with you he/she will have explained that some people experience difficulties with memory and concentration afterwards. Although these changes are temporary and do not happen to everyone, short tests of memory and concentration are given regularly to keep track of any change. If any difficulty is detected, the way ECT is administered may be changed or stopped.

Usually the tests are given by your psychiatrist but for the next 6 months, I will be assisting him/her. As well as the usual tests, some new ones will be added in an effort to develop the best way of monitoring memory and concentration. This letter is to ask you whether you would agree to allow me to give you the usual tests as well as the newer ones –just to see which ones are best.

If you would like to accept this invitation, please sign the consent form in the ECT information pack that your psychiatrist will go over with you.

Inside this information pack you will be able to find more information about the assessment and the procedure. The contents of the pack include:

- Letter of Support from Dr Alison Masters
- Information Sheet
- Consent Form

Thank you for considering this request

Yours sincerely,
Anneke Thornton BSc (Hons) Psychology
C/O Massey University Psychology Clinic, Massey University
PO Box 756, Wellington
email: anneke_thornton@hotmail.com

Ph: (04) 8015799 extn 62528

Dear Sir/Madam

I would like to invite you to participate in a study being undertaken by Anneke Thornton, a Clinical Psychology Doctorate student from Massey University.

Anneke is interested in finding out more about the short term side effects of Electroconvulsive therapy (ECT), and she is offering the patients through the Capital and Coast District Health Board thorough assessments of cognitive functioning throughout their treatment of ECT.

Cognition refers to your ability to think and process information and your memory. The assessment involves tests of memory and different thinking processes, as these have been associated with change during ECT for some but not all patients. The main objectives of these tests is to carefully monitor any side effects you may experience from receiving ECT as a treatment and to enable Anneke to better understand what types of difficulties (if any) patients receiving ECT may encounter. It is beneficial to carefully monitor any changes in these functions, as your psychiatrist is able to change the way ECT is administered to reduce these effects or stop the course completely if needed.

In addition, this study is being carried out so that we may be able to improve on current methods of monitoring patients' cognition, and provide the best ongoing care and support to present and future patients who will receive electroconvulsive therapy as a treatment.

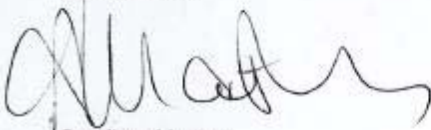
Included with this letter is a pack containing information for you to read to aid you in deciding whether or not you would like to receive routine cognitive assessments from Anneke and to take part in this study. You are under no obligation at all to agree to take part and participation is entirely your decision.

We support Anneke's study through the Capital and Coast District Health Board and see this as a worthwhile opportunity. If you are interested in participating, please fill in the consent form.

Your contribution would be greatly appreciated by both the Doctorate student and by those who will benefit from the knowledge generated from this project.

For more information you can ask Dr Contractor or contact Anneke directly. Anneke's contact details are contained in the information pack.

Yours sincerely



Dr. Alison Masters
Executive Director Clinical
Mental Health Directorate
C&CDHB

Monitoring the Short Term Effects of Electroconvulsive Therapy

INFORMATION SHEET

Recruitment

It is important for you to know that you are not obliged to take part in this study and you do not have to consent to meeting with me. By signing the consent form you will be giving me permission to come to the hospital and meet with you to do some assessments on your memory and thinking before and during your course of ECT. If you are an outpatient, you may wish to return the consent form by post. I have included a prepaid envelope with the information pack, with my address written on it.

What will happen during the assessment?

Once you have signed the consent form, I will arrange a time with you or your psychiatrist to come and meet with you before any ECT has taken place. We will take a baseline measure of your memory and concentration. This will involve me asking you some questions, asking you to copy a drawing, to do a short task of motor-speed and asking you to remember some things which I will ask you to recall later on in the assessment. I will also ask you to fill out two questionnaires – one about your mood and another about your memory. I am interested in hearing your comments around any impairments or improvements you have noticed since receiving ECT. I will also meet with you after every three treatments of ECT to monitor any change that may be occurring. Each time we meet should not take longer than one hour but extra time will be permitted if you need it.

What benefits will you receive from participating?

This study has three main purposes: To will enable you to voice your opinion about practices in ECT and how ECT has affected you, to closely monitor your thinking processes and memory (this is often referred to as cognition) during your treatment to ensure you are getting the best possible treatment, and from the results of assessing a number of individuals receiving ECT, a screening measure will be developed so that future patients receiving ECT will also have adequate monitoring of their cognition.

The biggest benefit to you is that you will receive a thorough cognitive assessment conducted by a trained Clinical Psychology student. This means that any effects on your thought processes and memory caused by receiving ECT will be able to be picked up early on in your treatment and appropriate changes can be put in place. Information from these assessments will inform how you are responding and how ECT is benefitting you. This is also an opportunity for you to contribute towards the improvement of cognitive assessment during a course of ECT.

Project Procedures

This study is being conducted under the supervision of Professor Janet Leathem of Massey University. The information I gather about you, and the results of your assessment will be kept confidential and you will not be able to be identified from your data. However, I will be working closely with your psychiatrist so if I have any concerns about deterioration in memory and concentration, I will let your psychiatrist know so that suitable changes can be made to your ECT treatment if necessary. Data and analyses of this study will be included in a journal publication, conference presentation and a Doctorate thesis. No one will be allowed to see the raw data except for Anneke Thomson and Professor Janet Leathem. Data collected electronically will be stored on a computer under password protection. Any hard copy personal information or results collected will be locked in a secure cabinet in a locked room at Massey University, Wellington Campus. Massey has a 10 year policy for storage and disposal of data. After 10 years all the raw data will be destroyed.

You may request a summary of the findings by indicating so on the consent form. The summary will be posted to you at the conclusion of the project.

If you decide to participate, you have the right to:

- Decline to answer any particular question;
- Withdraw from the study at any time
- Ask any questions about the study at any time during participation;
- Provide information with the understanding that your name will not be used and you will not be identified
- Be given access to a summary of the project findings when it is concluded

If you have any questions regarding anything in this information pack please do not hesitate to contact me.

My contact details are listed on page 1 of this information pack.

This study has received ethical approval from the Central Region Health and Disability Ethics Committee CEN/12/EXP/032.

Monitoring the Short Term Effects of Electroconvulsive Therapy

PARTICIPANT CONSENT FORM

I have read the Information Sheet and understand the details of the study. I have sought explanation about any details of the study I do not understand.

I have had time to consider my participation and my questions have been answered to my satisfaction. I understand that I may ask further questions at any time.

I agree to participate in this study under the conditions set out in the Information Sheet and I understand that I can withdraw myself and any data that has been collected from the study at any time.

I am happy for my doctor or psychiatrist to be contacted if there are any concerns regarding cognition which arise during the assessment.

YES NO

I wish to receive a summary of the study when it is completed

YES NO

Postal Address for Summary:

.....
.....
.....
..... Postal Code.....

Contact phone number:

.....

Signature:..... Date.....

Full Name Printed

Appendix E

Case Report: Cognitive Improvement without Clinical Improvement after Bilateral ECT for Schizoaffective Disorder

Abstract

This article reports the cognitive changes observed in a 56 year old man receiving bilateral electroconvulsive therapy (ECT) for treatment resistant schizoaffective disorder. Cognitive assessment conducted prior to starting the course of bilateral ECT, post-course and at a four week follow-up. Cognitive changes which occurred during the first course of three treatments included improvements in: verbal fluency, abstraction, delayed verbal memory and delayed visual memory, digit span backwards, attention and global cognitive functioning. Impairment from baseline to follow-up included serial sevens, orientation and motor speed. At a four week follow up, most domains of cognitive function either improved from baseline or showed no change. In spite of these changes, ECT was not successful in ameliorating the psychotic or mood effects of the schizoaffective disorder for this man, either in this course of treatment or in a subsequent second course of ECT.

Cognitive Improvement without Clinical Improvement after Bilateral ECT for Schizoaffective Disorder

JQ, a 56 year old Caucasian man suffering from treatment resistant schizoaffective disorder was referred for inclusion into a study which investigated the cognitive side effects of ECT amongst patients who were receiving ECT treatment for mood disorders. JQ's underlying mood disorder was depressive, and he suffered from positive psychotic symptoms such as auditory hallucinations of a religious content and persecutory delusions. Negative symptoms included mutism and severe catatonia. JQ had been trialed on a number of antipsychotics, however, was non-responsive to psychopharmacology and was eventually prescribed a course of Right Uni-

Lateral (RUL) ECT. His psychotic and depressive symptoms were unresponsive to RUL ECT, and a decision was made to trial JQ on a course of bilateral ECT. Due to the concerns around memory loss and cognitive impairment associated with bilateral ECT, JQ was referred to the study (see Chapter 11). Due to JQs prominent psychotic features and the uniqueness of his presentation which resulted in modifications of the assessment battery, the results of JQ's cognitive assessments were not included within the main analysis. As few studies into the cognitive side effects of people receiving bilateral ECT for schizoaffective disorder exist, the current report presents the cognitive changes of JQ, a 56 year old male receiving bilateral ECT for schizoaffective disorder.

Method

Research Design

A naturalistic prospective single case study design was adopted for the current research.

Measures Administered

The Montreal Cognitive Assessment.

The Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005) is a one page 30 point test of global cognitive function taking approximately 10 minutes to administer. It assesses short term memory, visuospatial abilities, executive functioning, attention, abstraction, orientation, concentration, working memory, language, short term memory recall and delayed recall after approximately five minutes.

Medical College of Georgia Complex Figures.

The Medical College of Georgia (MCG) Complex Figures (Ingram, Soukup, & Ingram, 1997) are scored on a 36 point scoring system comparable to the RCFT. The four figures show equivalent performances on the initial copy and recall tests (Loring & Meador, 2003; Yamashita

& Yasugi, 2008) and no practice effect is observed across the four conditions (Yasugi & Yamashita, 2010).

RBANS Coding Task.

This test of processing speed with four alternate forms from the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS; Randolph, 1998) the RBANS requires the respondent to code as many symbols as possible into numbers within a 90 second time limit.

Coin Rotation Task.

The coin-rotation task (CRT; Mendoza, Apostolos, Humphries, Hannah-Pladdy, & O'Bryant, 2010) measures psychomotor speed and motor dexterity in a task requiring the participant to rotate a coin 180 degrees using the thumb, index and middle fingers as quickly as possible for 20 rotations (Minor, Jones, Stewart, Hill, & Kulesza, 2010).

Autobiographical Memory Interview.

The Autobiographical Memory Interview (AMI; Kopelman, Wilson, & Baddely, 1989) is the most commonly used clinical test in the ECT literature to assess autobiographical memory. The AMI, involving the assessment of episodic and semantic autobiographical memories has shown strong reliability and validity as a measure of retrograde amnesia and is sensitive to variation in ECT technique (Lisanby, Maddox, Prudic, Devanand, & Sackeim, 2000; Sobin et al., 1995). Only the recent memory section, section C, was used in this research.

Procedure

Assessment Schedule.

A cognitive assessment was conducted prior to JQ starting the course of bilateral ECT treatment, after the third treatment and again at a 4 week follow-up. Assessments were conducted at least 48 hours after a treatment of ECT to allow for any residual effects of the ECT to resolve, such as disorientation (Porter, Douglas, & Knight, 2008).

This study received ethical approval from the Health and Disability Ethics Committee, New Zealand. The current study was conducted as part of a larger study investigating the short term cognitive side effects of ECT

Assessment Procedure.

During the initial assessment, the purpose of the study was explained to JQ and an information pack containing information about the study was provided (see Appendix X). To obtain informed consent, JQ's understanding of the research procedure was assessed by asking him to repeat back what he understood of the research. This ensured that he was able to comprehend the purpose of the assessments. JQ's understanding was reassessed at the end of the assessment. Each assessment was voluntary and JQ was able to terminate the assessment at any time. Therefore, the amount of the cognitive assessment completed varied across each assessment varied depending on JQs clinical state. A nurse was present during each assessment.

During the initial assessment, the AMI was administered. JQ was unable to answer many of the questions of the AMI, and instead of responding "I don't know" would confabulate. As his answers were invalid, the questionnaire was not repeated in any further assessments. Thereafter, the assessment procedure was as follows: MoCA, copy phase of the MCG Complex Figure, RBANS coding task, CRT and finally the recall phase of the complex figure.

ECT Treatment.

ECT was administered bilaterally twice weekly via a MECTA SpECTrum Q5000 machine using an ultra-brief 0.3ms pulse width. Dose titration was initially done using the limb isolation technique to establish seizure threshold. This technique involves isolating a limb from the effects of the muscle relaxation, by inflation of a blood pressure cuff to above systolic pressure. As the muscle relaxant does not affect that limb, the seizure can be observed in that limb while the rest of the body is paralysed. Once JQ's seizure threshold was determined, the treatment dose delivered was 1.5 to 2.0 times seizure threshold.

Results

JQ was seen for a cognitive assessment a total of 5 times over two courses of bilateral electroconvulsive therapy. The timing between the courses of ECT was six weeks. No assessments could be conducted over the second course of ECT due to a decline in JQ's mental state. An initial cognitive assessment was conducted with JQ at baseline, after three treatments and at a four week follow-up. JQ then received a further three treatments, to which he was unresponsive and he became catatonic. A post treatment and follow up assessment for his second ECT could not be conducted, as JQ was catatonic during these times. The results of his cognitive assessments were analysed visually, and changes in scores across assessment times are discussed.

Table 1

Results of Cognitive Assessments during the First Course of ECT

<i>Number of ECT</i>	<i>Baseline</i>	<i>3 ECTs</i>	<i>Follow up</i>	<i>Positive Change</i>
MoCA total (30)	11	10	14	YES
Trail Making Task (1)	0	0	0	N/C
Figure Copy (1)	0	1	1	YES
Clock (3)	1	1	1	N/C
Naming (3)	3	3	3	N/C
Immediate Recall (10)	7	10	10	YES
Digit Span Forwards (4)	0	0	3	YES
Digit Span Backwards (4)	0	1	1	YES
Vigilance (1)	0	0	0	N/C
Serial Sevens (5)	4	3	3	NO
Sentence Repetition (2)	0	0	0	N/C
Verbal Fluency	3	2	7	YES
Abstraction (2)	1	1	2	YES
Delayed Memory (15)	3	2	4	YES
Orientation (6)	4	2	2	NO
Coding Task total	10	12	20	YES
Coding Task total correct	4	10	19	

Complex Figure Copy (36)	28	31.5	33	YES
CRT Dominant Hand	10	8	7	NO
CRT Non-Dominant Hand	13	8	8	NO

Under the column 'Positive Change' Yes indicates a positive change from baseline occurred, No indicates a negative change from baseline occurred, and N/C indicates that there was no change from baseline to follow up. The numbers in brackets after the cognitive tasks indicate the highest score possible for the task. From the 19 cognitive domains or cognitive tasks, 10 improved from baseline (53%), 5 showed no change (26%), and 4 showed declines (21%).

Language

JQ's ability to produce language as measured by the sentence repetition component of the MoCA was impaired at all assessment points. When repeating a sentence, he would often switch words with similar semantic meanings (e.g. grandmother to grandfather) or lose track of where he was and would stop part way through the sentence, or at other times JQ would repeat back a totally different unrelated sentence (e.g. "A bird can fly into closed windows when it's dark and windy" was repeated back as "coast, time, thirty"). JQ's comprehension of instructions was also impaired, for example, when asked to tap his hand on the table each time he heard the letter A in the vigilance task, JQ would tap his hand as soon as he heard the letter A and would not stop tapping.

Attention

JQ's inability to repeat back a sentence and inability to correctly follow instructions may have been due to a deficit in attention and concentration. He was often distracted during the assessment and may have been having auditory hallucinations. He was however, able to immediately recall words repeated to him, and his performance on digit span forward improved from baseline.

Memory (verbal delayed)

JQ was unable to remember any words which were repeated to him 30 minutes earlier, however,

his ability to correctly recognise the words improved slightly over time, suggesting a deficit in memory retrieval during the course of ECT, and a slight recovery of this function at follow-up.

Information Processing Speed

JQ's psychomotor speed increased significantly at the 4 week follow up, and the number of errors made decreased.

Motor speed

JQ's motor speed as measured by the coin rotation task decreased from baseline, which may have been a reflection of his deteriorating clinical state.

Visuo-constructional skills

JQ's visuo-constructional skills and visual planning improved from baseline, as shown by the complex figure copy phase and the figure copy on the MoCa. Quantitative scoring of the Clock Drawing Task showed no change, however, qualitative analysis of the clock drawing task showed an improvement in visual planning, and conceptualization over time (see Figure 1).

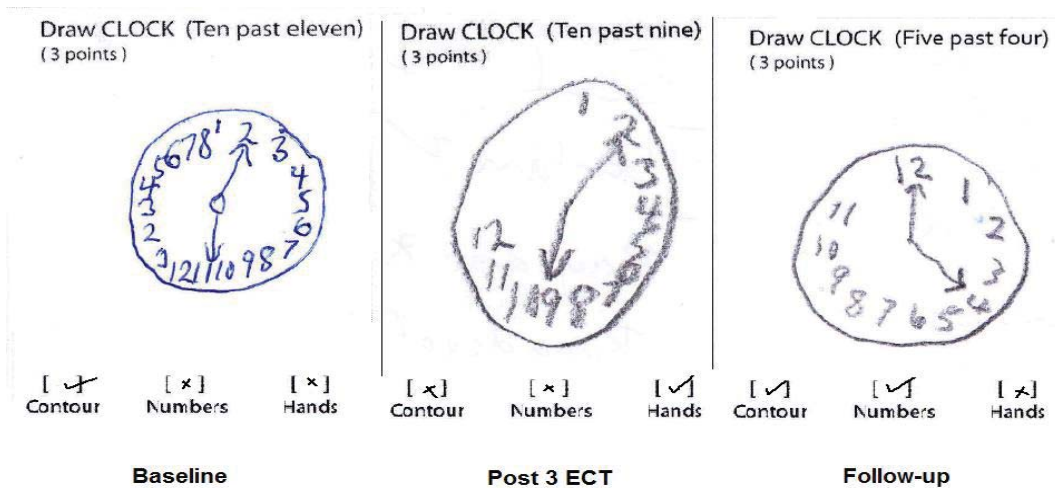


Figure 1. JQ's Performance on the Clock Drawing Task at baseline, post treatment and at a 4 week follow-up.

Cognitive Flexibility and Divided Attention

JQ did not appear to comprehend the requirements of completing the trail making task, and interestingly his performance differed on this task at each assessment time. Visual analysis of assessments two and three suggest that the poor performance is likely due to an inability to comprehend the instructions of the task, rather than the inability to switch between responses.

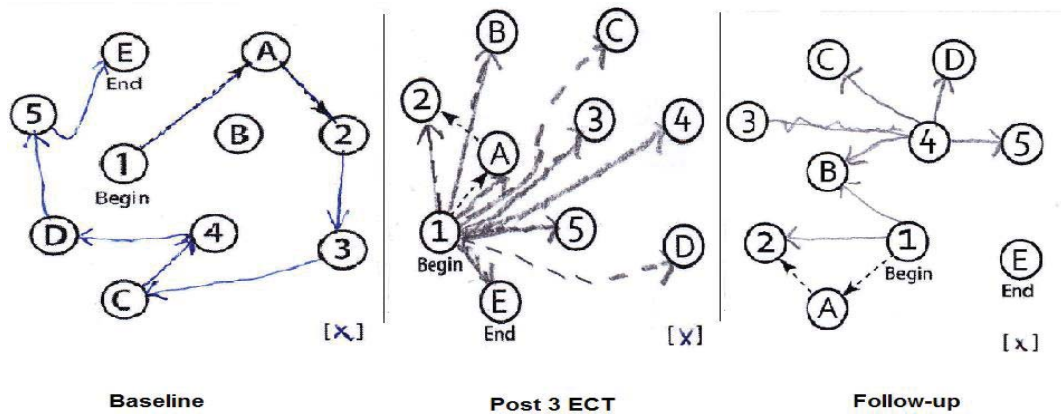


Figure 2. JQ's Performance on the Trail Making Task at baseline, post treatment and at a four week follow-up.

Verbal Fluency

JQ's ability to fluently produce words starting with a specific letter of the alphabet while adhering to a set of rules improved throughout from baseline to follow up. There was a slight decrease in number of words generated after three ECTs but this improved beyond baseline at the follow-up assessment.

Discussion

JQ, a 56 year old Caucasian male received two courses of Bilateral ECT, each containing three treatments, for medication resistant schizoaffective disorder, depressive type. For the first course of ECT, cognitive assessments were conducted prior to any Bilateral ECT, after his third treatment and at a 4 week follow up. The most salient change which occurred across the

assessments was JQ's change in ability to undergo cognitive assessment during his first course of ECT, and his decline in health which led to him being in a catatonic state during the second course, despite having further treatments of ECT.

Both schizoaffective disorders and depression are associated with cognitive deficits (Bora, Yücel, & Pantelis, 2010; Douglas & Porter, 2009; Meier et al., 2014; Reichenberg & Harvey, 2007). Greater neuropsychological impairment has been observed in people with psychotic depression than in people with non-psychotic depression (Schatzberg et al., 2000), and the degree of dysfunction observed in people with psychotic depression has been found to parallel that of schizophrenia, albeit less severe (Hill, Keshavan, Thase, & Sweeney, 2004). Thus the finding that the majority of cognitive domains assessed over the course of JQ's initial course of ECT either improved or did not change may indicate that ECT had some efficacy in treating the schizoaffective disorder, as if this were not the case, a worsening in cognitive performance would be anticipated due to the ECT. A measure of mood and psychosis would have assisted in confirming this observation.

A major limitation of this case report is that no formal measure of clinical state was obtained; this was due to the limited time in session, the long length of time which it took for JQ to complete a task and the lack of insight into his disorder. This indicated that he would be unable to fill out a questionnaire or report accurately on his mood or psychotic symptoms. Having a formal measure of mood or psychosis would have allowed the results of his cognitive assessments to be understood within the context of his clinical state (Porter et al., 2008), and would have provided an indication of how much his clinical state would be impacting on his cognitive function, and thus allow for the cognitive effects of the treatment to be better teased out from the cognitive effects of the disorder.

A further limitation which may have affected the validity of the assessments was that as JQ was not in touch with reality, it is likely that he was not motivated to carry out the assessments and did not give his best efforts. In addition, JQ had previously received numerous courses of Right Unilateral ECT, which may have had a residual effect on his cognitive function. As only one baseline assessment was able to be conducted, it is possible that this baseline assessment may not be entirely reflective of his pre- Bilateral ECT cognitive function, and further baseline assessments would have allowed for a stable baseline level of functioning to be obtained (Center & Leach, 1984). More research is needed which investigates the cognitive effects of ECT while controlling for clinical changes which occur during the course of treatment.

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Appendix F

Content Analysis of Subjective Reports at Baseline

Table 2

Categorisation of subjective reports of cognitive function prior to commencing a course of ECT

Domain	Operational Definition	f
<i>Cognition</i>		
<i>General Memory</i>		
<i>Impaired</i>	<p>References made to general/non-specific function of memory.</p> <p>My memory isn't as good as it used to be but it's not too bad.</p> <p>The last fortnight I have been having trouble with my memory.</p> <p>I have had lapses in my memory.</p> <p>I find things harder to remember.</p> <p>I definitely have memory problems now. I have previously always taken pride in my memory.</p> <p>My memory is not very good for almost everything.</p> <p>I have a really bad memory.</p>	f=7
<i>Functioning</i>	<p>My memory is generally pretty good.</p> <p>I have no problems with my memory.</p> <p>My memory is fine.</p>	f=3
<i>General Short Term Memory</i>	References made to general/non-specific function of short term memory.	
<i>Impaired</i>	<p>It is mainly my short term memory that I struggle with.</p> <p>I have problems with my short term memory</p> <p>I can follow what is happening in the book but I will forget the characters and the plot quickly</p> <p>My short term memory has gotten worse the past couple of months.</p> <p>My short term memory has gone.</p> <p>I can read but I won't remember what I was reading</p> <p>I can watch a movie but then I forget what it was about.</p> <p>I have trouble remembering things I read or things I have done recently.</p> <p>It is mainly my short term memory that I struggle with.</p>	f=9

<i>General Long Term Memory</i>	References made to general/non-specific function of long term memory.	
<i>Impaired</i>	My long term memory is the worst. It wavers day by day; I'm not struggling with it all the time. I forget things that I have done the day before I have difficulty remembering things that happened ½ an hour ago. I can comprehend and follow information but I can't remember things. I self medicated for about 11-12 years and drank a bottle of wine a day so I don't have a good memory for things that happened around this time. I have worked for 10 years in the same job, but I still have to ask people for help sometimes. I don't remember anything from when I was really young.	f=7
<i>Functioning</i>	My long term memory is fine I can remember things from the past but I try not to. My long term memory is fine.	f=3
Retrograde Amnesia	References made regarding the ability to remember facts and information learned prior to starting ECT.	
<i>Episodic Autobiographical Memory</i>	References made regarding the ability to remember personal episodic information learned prior to starting ECT. Episodic autobiographical memory is the conscious recollection of temporally and spatially specific events from one's past (Levine, 2004). This conscious state of mental time travel is achieved through recollection of events, sensory-perceptual details, thoughts, and feelings such that the rememberer becomes aware of an event as belong to his or her own past (Tulving & Craik, 2000).	
<i>Impaired</i>	I would struggle with telling you what I did last week. My memory is also bad for remembering what I did yesterday. I find it really hard to remember things that happened between now and last fortnight. I have trouble remembering things that happened a few years ago, things I have done with my children, and places I have gone with the kids I have trouble remembering things I read or things I have done recently.	f=5
<i>Functioning</i>	I can remember big things like my son and daughter being born	f=1
<i>Semantic Memory</i>	References made regarding the ability to remember knowledge and facts about the world that is not tied to any specific personal experience (Goldstein, 2008). This information was learned prior to starting ECT.	
<i>Impaired</i>	My memory is especially bad for people's names Remembering names is hard.	f=2
<i>Personal Semantic Memory</i>	References made regarding the ability to remember semantic memories that have personal significance (Goldstein, 2008) learned since starting ECT.	
<i>Impaired</i>	I couldn't tell you what I had for dinner last night. I can't remember how old my grandmother is	f=4

	I couldn't remember my post code when I was writing my address. The other day I had a shower twice because I had forgotten that I had already taken one.	
Prospective Memory	References regarding the ability to remember to perform intended actions (Goldstein, 2008)	
<i>Impaired</i>	I have trouble keeping appointments. I need to set alarms on my phone to remind me, this is because of my depression. I forget what time my kids' extracurricular activities start and finish	f=2
Executive Functioning	References made regarding the ability to plan, organise, sequence, regulate inhibit responses, and use judgement (Schoenberg & Scott, 2011)	
<i>Impaired</i>	I used to be able to do big sums in my head, I was an accountant. Now I have trouble doing simple sums in head.	f=1
Concentration/ Attention	Refers to the ability to focus on specific features of the environment or on certain thoughts or activities (Goldstein, 2008).	
<i>Impaired</i>	I am not able to remember things like what has been happening on the news but this is likely due to inattention when watching the news. My concentration is really bad. I have lots of books that are just sitting waiting to be read I am rereading books but not taking anything in. I struggle to focus on anything on TV. My concentration isn't too good. My concentration and decision making is worse that it was previously when I wasn't so depressed. My concentration is worse now than it was a few years ago. I have problems with my concentration, I can't read. I am not able to remember things like what has been happening on the news but this is likely due to inattention when watching the news. My concentration isn't good. I forget what I have done or what I am doing. I can't read books and movies are too long. I have had lapses in my concentration. When I watch TV I stare at it, and don't take anything in. My work was calling me up telling me that I had left things out. I have trouble concentrating when I watch TV and read the newspaper I have trouble remembering things I read or things I have done recently. I am rereading books but not taking anything in. I feel exhausted after concentrating for a while I can read books ok I write poetry. I can read but I won't remember what I was reading	f=18
<i>Functioning</i>		f=7

	<p>I can watch programmes on TV fine and will remember nearly everything I watch.</p> <p>I can follow what is happening in the book but I will forget the characters and the plot quickly</p> <p>I can comprehend and follow information but I can't remember things.</p> <p>I can watch a movie but then I forget what it was about.</p> <p>References regarding the ability to decide between two or more choices.</p>	
Decision Making		
<i>Impaired</i>	<p>When I make decisions I source other people's opinions because I don't feel confident in my own judgment now.</p> <p>I have trouble making decisions and feel like everything is too hard. I get overwhelmed quickly.</p> <p>Decision making is harder.</p> <p>I'm ok at making decisions. Bigger ones are more difficult than others. Day to day decisions are fine.</p> <p>I am not indecisive about everyday things, but am indecisive about treatment.</p> <p>My concentration and decision making is worse that it was previously when I wasn't so depressed.</p> <p>I am fine with making decisions.</p> <p>I'm good at making decisions.</p> <p>I don't have any trouble making decisions</p>	f=6
<i>Functioning</i>		f=3

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Appendix G

Content Analysis of Subjective Reports during a Course of ECT

Table 3

Categorisation of subjective reports of cognitive, clinical and somatic changes reported during a course of ECT

Domain	Operational Definition	Frequency
Cognition		
General Cognition		
<i>General Cognitive Function</i>	References made to general/non-specific function of cognitive processes.	
<i>Impairment</i>	<p>When I look at words on a page, the letters come up it is like they are lifting up off the page. I used to have a thought and not think about it, but now when I think about thinking, I visualize my brain as a mucky scrambled brown muscle that is damaged. Work is more difficult. I am not as sharp cognitively. I feel a general fuzziness in my head. Cognitively – I don't feel very sharp or onto it. This is due to both my mood and probably a bit of the ECT. It is as if everything is just too hard and it is too hard to think.</p>	f=7
<i>No Change</i>	<p>I haven't noticed any changes I haven't really noticed any negative effects! No negative changes. I have been really lucky with side effects.</p>	f=3
<i>Improvement</i>	<p>I am able to think more clearly. My thinking is quite a bit clearer. I am not in a fog. I am thinking more clearly. My head is clearer now and it is easier to think. It is like the fog has been taken away.</p>	f=5
<i>General Memory</i>	References made to general/non-specific function of memory.	
<i>Impairment</i>	<p>I feel like my memory is being played around with. I spend a lot of time rethinking things, and trying to remember things, but they do come back to me eventually.</p>	f=30

	<p>It is hard to remember things. My memory is not the best. I am forgetful. I forget a lot. My memory is quite bad at the moment. My memory is a bit rough. My memory is a bit vague about some things. I am forgetful. I forget things quickly. There was a whole period of when I was having ECT that I don't remember. My memory is affected quite considerably, especially around the time of the ECT and sometime prior. I forget things all the time. My memory has definitely been altered temporarily. It is always slightly worse after ECT. My memory isn't as good. ECT isn't doing my memory any good. I have some memory loss. I can't remember certain things. My memory has been affected in a negative way. My memory is not as good. My memory is much worse straight after ECT. I found the memory test really difficult to do today. My memory the day of ECT isn't very good. I am forgetful. My memory is worse. My memory is playing up a bit. My memory has been affected a bit. My memory has gotten worse. I'm not remembering, progressively getting worse. There are a range of things that I am not remembering.</p>	<p><i>No Change</i></p>	<p>f=6</p>
	<p>I haven't had any memory problems like I did during my last course of ECT. My memory is ok. There haven't been any positive changes at the moment. I haven't noticed any negative effects with my memory. My memory is not better but not worse – I don't think it has changed. My memory is the same. My mood hasn't gotten any better it has only gotten worse.</p>		

<i>Improvement</i>	<p>Sometimes my memory is better. My memory is much better now that my mood is better.</p>	f=2
<i>General Long Term Memory</i>	References made to general/non-specific function of long term memory.	
<i>Impairment</i>	<p>Last week I went home and opened a photography programme on my computer and I could not remember how to use it. I didn't know what to do, it was like the first time I had ever seen the programme! I go into a room and forget why I am there. I go to my room and forget why I am there. This happens 2-3 times per day. I forget what I am doing half way through doing something. I go into rooms and forget why I am there. We have had neighbours who moved in April (2 months prior to ECT course) and I couldn't remember them! Today my memory was worse at work. I couldn't remember things I was trained to do 10 years ago. Some important memories have gone but do come back. I also forgot something quite major that my sister picked up on but I can't remember what it was. I forgot my flatmates friend had died. I asked how her flatmate was and she got upset. Memories laid down since moving to New Zealand (past year) seem to be more fragile than memories from back in the UK. I read My Sister's Keeper last week and now I have totally forgotten what it was about and how it ended.</p>	f=11
<i>No change</i>	My long term memory has stayed the same.	f=1
<i>Improvement</i>	<p>My childhood memories have improved. My long term memory is much clearer and things from my childhood are much clearer. I can remember much more of my childhood. I can remember what togs I wore when I was three; I never remembered this before ECT. I seem to be able to recall some of my childhood memories that I had previously forgotten. My long term memory is better a few days after ECT I'm starting to remember more- long term memory.</p>	f=7
<i>General Short Term Memory</i>	References made to general/non-specific function of short term memory.	
<i>Impairment</i>	<p>I mainly have trouble with my short term memory. Short term memory is worse. My memory is bad – short term. My short term memory is bad. I have a slight short term memory problem.</p>	f=18

	<p>I have trouble remembering some things – mostly short term. Something will be in my head and then I've forgotten it.</p> <p>If someone tells me something I am likely to ask a question which has already been covered off.</p> <p>My memory is slightly worse for things that are happening in the moment.</p> <p>My short term memory has deteriorated</p> <p>My short term memory is playing up. Someone will ask me to do something and I will forget straight away.</p> <p>I have experienced a little short term memory loss but nothing major.</p> <p>My short term memory around the time of ECT isn't very good.</p> <p>My short term memory is worse.</p> <p>Someone will say something to me and I will completely forget.</p> <p>My short term memory is bad.</p> <p>Short term memory loss.</p> <p>My short term memory is bad around the period of ECT</p> <p>I forget what I am doing half way through doing something.</p>	
<p><i>No Change</i></p>	<p>I am forgetful with short term memory but I don't feel it is worse than before the treatment began.</p>	
<p><i>Improvement</i></p>	<p>I can retain information longer.</p> <p>My short term memory is a little bit better.</p> <p>Short term memory is better, as long as it has been a few days since ECT</p>	<p>f=3</p>
<p><i>Anterograde Amnesia</i></p> <p><i>Impairment</i></p>	<p>References made regarding the ability to remember facts and information learned since starting ECT.</p> <p>I forget people who I have met over the past few months.</p> <p>My concentration is ok and I am able to read a book but I don't remember anything I read.</p> <p>I went home last week to see my sons but I can't remember the day. It is like the day has been erased from my memory.</p> <p>If I get told something I will forget it quickly.</p> <p>I find it hard remembering new things that I have learnt.</p> <p>I can't remember you coming in a week and a half ago.</p> <p>Apparently I walked from here (Ward 27) to te papa and got lost and called my husband and asked him to come and pick me up, I only know this because I was told; I don't remember it.</p> <p>I don't remember people that I have met recently. Both their faces and their names – but names are harder. For example, a lady on the ward asked me to bake her cake. I can picture her face but I have no idea what her name is.</p> <p>I have recently had to cancel my studies, I was studying extramurally. I cancelled because of concerns around how much I would remember.</p> <p>I find it really hard to form new memories. I can't remember meeting people and things that I have discussed with people. Even if I have been told about someone, I then need to ask again who is this person?</p> <p>Things that have happened before and after ECT are really fuzzy or gone.</p> <p>People telling me things and I keep stuffing things up. It is really embarrassing. I told one client that I would hand deliver</p>	<p>f=13</p>

	<p>an invoice. I then rang them up to remind them to pay the invoice, and they told me I said I would deliver it but I didn't remember saying that.</p> <p>On the day of treatment I seem to be able to recall more than a few days after treatment.</p> <p>I saw someone right before my treatment, and I had absolutely no recollection of seeing her.</p>	
<i>Retrograde Amnesia</i>	<p>References made regarding the ability to remember facts and information learned prior to starting ECT.</p>	
<i>Episodic Autobiographical Memory</i>	<p>References made regarding the ability to remember personal episodic information learned prior to starting ECT. Episodic autobiographical memory is the conscious recollection of temporally and spatially specific events from one's past (Levine, 2004). This conscious state of mental time travel is achieved through recollection of events, sensory-perceptual details, thoughts, and feelings such that the rememberer becomes aware of an event as belong to his or her own past (Tulving & Craik, 2000).</p>	
<i>Impairment</i>	<p>I find it hard to remember things that have happened.</p> <p>I forget things that have happened.</p> <p>When my nurse talks about things that we have done, I often won't remember. He will say do you remember going here, or doing this but I can't.</p> <p>I have trouble remembering things I have done.</p>	f=4
<i>Semantic Memory</i>	<p>References made regarding semantic memories; memories of facts and knowledge about the world that is not tied to any specific personal experience (Goldstein, 2008) and learned prior to starting ECT.</p>	
<i>Impairment</i>	<p>I always have things on the tip of my tongue but just can't quite get to them.</p> <p>I forget the names of day to day things like animals. I can usually remember them a couple of days later. It is always worse straight after ECT.</p>	f=2
<i>Improvement</i>	<p>My memory has improved! Events and people are much clearer now.</p>	f=2
<i>Personal Semantic Memory</i>	<p>References made regarding the ability to remember semantic memories that have personal significance (Goldstein, 2008) learned since starting ECT.</p>	
<i>Impairment</i>	<p>I have had some minor problems with my memory. Yesterday I couldn't remember where we kept the vacuum cleaner so I had to ask the kids to get it. We make a joke of it now.</p> <p>I was at my sisters setting the table and I couldn't remember where my niece usually sits.</p> <p>People telling me things and I keep stuffing things up. It is really embarrassing. I told one client that I would hand deliver an invoice. I then rang them up to remind them to pay the invoice, and they told me I said I would deliver it but I didn't remember saying that.</p> <p>After ECT, the nurse gave me my shoes; I couldn't recognise them and didn't believe they were mine. It took a while for</p>	f=19

	<p>me to come to the conclusion that they were in fact mine. I can't remember which dentist I go to. I have more difficulty recalling who I have had conversations with. I am able to remember details of the conversation but not who I had the conversation with. This is worse on the day of ECT. I forgot where my favourite shop was, and I go there all the time. I'm forgetting little things like names. This usually resolves a couple of days after ECT. I have trouble remembering people and their names. I forget names and that's of people who I know well. If I think about my home here in New Zealand and my home in South Africa they are meshing – I am not sure which one is which. I forget people who I have met over the past few months. I have trouble remembering people's names. I can't recall names. I can't remember names. Sometimes people who I have known for a while, I can't remember their names. Straight after the last session of ECT I couldn't remember my address. This came back a few hours later. I couldn't remember when my depression started when asked by my doctor and I should know my background, well, I did anyway. I forget names of people who I don't know all that well. I have trouble remembering people and their names.</p>	
<i>No Change</i>	I can still remember names of people who I know well.	f=1
<i>Visual Memory Impairment</i>	<p>References made regarding the ability to remember visual facts or information learned prior to starting ECT.</p> <p>I forgot where my favourite shop was, and I go there all the time. The other day I was driving my car, I couldn't concentrate I didn't know where I was going. I got lost on my way home even though I have lived there for so long. I couldn't remember who you were but I knew I recognised you from somewhere I just don't know where from. The other day I was driving my car, I couldn't concentrate I didn't know where I was going. I got lost on my way home even though I have lived there for so long.</p>	f=4
<i>Prospective Memory Impairment</i>	<p>References regarding the ability to remember to perform intended actions (Goldstein, 2008)</p> <p>I find it hard to remember things that I need to remember. I can't remember what to do without lists or calendars. I had forgotten you were coming. I will forget things my sister has asked me to do. She will talk to me about something and I will totally forget that she</p>	f=5

	<p>even told me.</p> <p>I have to write stuff down and put alarms on my phone, when I previously have never had to do this.</p> <p>I write notes for myself to remind me of things around the time I have treatment but stop using them after a few days after treatment</p>	
Executive Functioning	<p>References made regarding the ability to plan, organise, sequence, regulate inhibit responses, and use judgement (Schoenberg & Scott, 2011)</p>	
Impairment	<p>I find it harder to express myself.</p> <p>I feel really disinhibited.</p> <p>I am not able to get the words out. I get a complete blank.</p> <p>I keep forgetting words.</p> <p>The other day I was driving my car, I couldn't concentrate I didn't know where I was going. I got lost on my way home even though I have lived there for so long.</p> <p>I sometimes find it hard to string sentences together.</p> <p>I find it hard to remember the way to get to places.</p> <p>I have made some not so good decisions, like driving when under doctor's orders not to drive. I crashed my car into the fence and now I have to pay for damages myself because I am not insured because I was under instructions not to drive.</p>	f=8
Improvement	<p>I am able to think in the big picture now, rather than only in the little picture.</p>	f=1
Orientation	<p>References regarding one's awareness of time, place and person.</p>	
Impairment	<p>I am disorientated.</p> <p>Sometimes I forget what day it is and what date.</p> <p>I'm not feeling quite with it I feel very dazed and spaced out.</p> <p>I don't know recent things like what time it is and what the date is or what day it is.</p> <p>I get confused as to where I am and where I am going.</p> <p>I forget what day of the week it is.</p> <p>My short term memory is a bit fuzzy, like knowing where I am.</p> <p>I forget what the date is and what day of the week it is</p>	f=8
Information Processing Speed	<p>References regarding the speed at which one is able to process information.</p>	
Impairment	<p>I go to the shops and forget why I am there.</p> <p>Sometimes I go into a room to do something and forget why I am there.</p> <p>I start a job and get distracted and not remember what I was doing. This is worse since ECT. This is still happening a lot.</p> <p>I go into a room and forget why I am there.</p> <p>I go to my room and forget why I am there. This happens 2-3 times per day.</p> <p>I forget what I am doing half way through doing something.</p>	f=8

	<p>I go into rooms and forget why I am there.</p> <p>I find comprehension of conversations difficult and my attention is poor. This might be from the depression too.</p>	
Concentration/ Attention	Refers to the ability to focus on specific features of the environment or on certain thoughts or activities (Goldstein, 2008).	
<i>Impairment</i>	<p>I go to the shops and forget why I am there.</p> <p>I'm finding it difficult to concentrate on things.</p> <p>I am finding it hard to engage.</p> <p>My concentration is worse. I will be having a conversation and lose track of what I am saying quickly.</p> <p>Sometimes I can't concentrate.</p> <p>My concentration has been affected.</p> <p>Sometimes I go into a room to do something and forget why I am there.</p> <p>The other day I was driving my car, I couldn't concentrate I didn't know where I was going. I got lost on my way home even though I have lived there for so long.</p> <p>I start a job and get distracted and not remember what I was doing. This is worse since ECT. This is still happening a lot.</p> <p>I go into a room and forget why I am there.</p> <p>I go to my room and forget why I am there. This happens 2-3 times per day.</p> <p>I can't concentrate so I can't read anything. I used to be able to read magazines.</p> <p>I won't remember what I watch on TV. I stare at it but don't take anything in.</p> <p>I find comprehension of conversations difficult and my attention is poor. This might be from the depression too.</p> <p>My concentration is a bit worse at the moment</p> <p>My concentration is poor but I think this is because of the depression not because of the ECT.</p> <p>Concentration not clear.</p> <p>My concentration is bad.</p> <p>I forget what I am doing half way through doing something.</p> <p>I put things down and forget where I have put them. This has started happening since having ECT.</p> <p>I go into rooms and forget why I am there.</p> <p>I forget what I am doing half way through doing something.</p> <p>I find myself absent minded doing things, because I can't pay attention to things.</p>	f=23
<i>No Change</i>	<p>My concentration is the same.</p> <p>My concentration is ok and I am able to read a book but I don't remember anything I read.</p> <p>My concentration is ok.</p> <p>I still can't read books but nothing has changed.</p> <p>My attention is the same.</p>	f=5
<i>Improvement</i>	<p>I find it hard to concentrate but can read a book now. I couldn't before.</p> <p>I can read books again, reading is easier.</p>	f=16

	<p>My concentration is getting better. I am reading lots of books. I can sustain attention for longer. I can concentrate for longer. My mind is clearer. My concentration is slightly better. I am not daydreaming as often as I used to. My concentration is more focused. My concentration has gotten better. My attention span has slightly improved. My concentration has improved with my improvement in mood My concentration is better. I can concentrate a little bit better, I can read now and I couldn't read at all when I was feeling really depressed. I can concentrate better and think more clearly. Reading is easier. I can sustain attention longer. References regarding the ability to decide between two or more choices.</p>	
<i>Decision Making Impairment</i>	<p>I am indecisive. Decision making is harder but I can do it with my husband's support. I cover things off with him. It is more because I am lacking confidence than it is that I have trouble with decision making. I can make a decision but not feel confident in it. I can't make decisions. I find it hard to make decisions. I'm not sure about things anymore. It is harder to make decisions. I still have trouble making decisions. I still can't make decisions. I am still finding it hard making decisions. I find it hard making decisions. I find it difficult to make decisions. I rely on people for help. Decision making is the same. My ability to make decisions and my memory varies with my mood.</p>	f=11
<i>No Change</i>	<p>My decision making is better. I seem to be more precise in my decision making but that might be because of my mood. I am able to make more rational decisions I can make decisions quicker</p>	f=2
<i>Improvement</i>	<p>References to one's emotional and clinical state. My depression is getting worse and I am getting desperate. I burst into tears after ECT. I am more emotional.</p>	f=4
Depression Worsening		f=15

	<p>My mood is still swinging markedly. Last week I was feeling more positive but this week I feel terrible. I'm getting quite anxious about the negative changes that are happening. I am more depressed than when I started. My mood has gotten worse. I am self harming again. Sometimes I feel more alert but then it feels like I have a comedown and feel worse. I feel more emotional but not in a good way. I find I am talkative to people about things that I wouldn't usually talk to them about. I am more emotional and things are getting on top of me more. I get upset and stressed more easily than I have in the past. This level of emotion has occurred since ECT. I have trouble sleeping. My mood has been very up and down. Yesterday (the day of treatment) I felt a little bit witty, I cracked a few jokes but now I feel a bit worse overall.</p>	
<p><i>No Change</i></p>	<p>I have really bad days where I want to end it all. I feel so awfully depressed. My mood hasn't changed. My appetite is still really poor. I haven't noticed any positive change in mood. The first few treatments felt great. Now it is like it's not working. I've had no major noticeable positive changes in my mood. I am still having negative thoughts but they are not as intense. It is slightly better on the day of the treatment, but the next day is the same again. After Monday's treatment I felt a bit more with it, and positive about it, but this did not last longer than one day. My mood is about the same as it was before treatment. My mood has stayed the same. My outlook on life hasn't changed. I still feel so pessimistic about my future I can't stand it. I can't see things ever getting any better. I haven't really noticed any change in mood yet. I just feel a burst of energy but do not feel any better. The first day I had a really good day for the whole day. The second treatment I felt good until the evening. The third treatment didn't change my mood all that much. Overall my mood is about the same I feel pretty flat all the time. I haven't really noticed a change in my mood. I don't know if it is working. The first few treatments felt great. Now it is like it's not working. I get a slight lift in mood but it goes back down again. For the day I have ECT I have the lift for most of the day but only</p>	<p>f=19</p>

<p><i>Improvement</i></p>	<p>for a few hours.</p> <p>Something is making me feel much better</p> <p>My mood has lifted a little.</p> <p>I feel stimulated after treatment but am not noticing a lift in my mood.</p> <p>I have noticed a slight change in my mood in the positive direction.</p> <p>I am much more alert now.</p> <p>I have learnt to appreciate my children.</p> <p>I can cope better with life's challenges better now.</p> <p>The ECT is making me feel better.</p> <p>I now want to live, I have hope.</p> <p>I don't get upset at little things anymore.</p> <p>I don't have so many negative thoughts going through my head anymore.</p> <p>My kids make comments that I am better because I am not sleeping as much.</p> <p>Last week I was feeling more positive but this week I feel terrible.</p> <p>I have my sense of humour back.</p> <p>I am relieved that I have finally found something that works to treat my depression.</p> <p>I am less irritable.</p> <p>I am more alert.</p> <p>I know that my mood is better because my chains of thoughts are different.</p> <p>I can get thought my days easier.</p> <p>Life is more enjoyable.</p> <p>It is slightly better on the day of the treatment, but the next day is the same again. After Monday's treatment I felt a bit more with it, and positive about it, but this did not last longer than one day.</p> <p>My mood has improved.</p> <p>Other people notice that I am better and I am smiling more.</p> <p>I feel more motivated and want to get out of hospital and get lots of stuff done around the house.</p> <p>I hit the golf course, I would've said no previously. I actually enjoyed myself. Quite often I would make myself do stuff and wont enjoy it but this time I did, and I think that is due to the ECT starting to work.</p> <p>I am a little bit more motivated.</p> <p>On the day of treatment I feel more engaged.</p> <p>My mood is getting better, my mood swings are not so intense and my thoughts are not as intense.</p> <p>I can handle stress better.</p> <p>I have cut down on my medication because my mood is better.</p> <p>The first few treatments felt great. Now it is like it's not working.</p> <p>My mood isn't swinging uncontrollably.</p>
	<p>f=94</p>

	<p>I feel brighter straight after ECT and this lasts for most of the day. I have less negative thoughts. I am staying up longer; whereas I would previously go to bed as soon as I got home from work. I have my motivation back. Yesterday (the day of treatment) I felt a little bit witty, I cracked a few jokes but now I feel a bit worse overall. I think now that I have had a few treatments that I am starting to notice the effects. I've recently noticed that over the long term I am a bit more motivated over all. I am a little more motivated. I am able to get more work done. I have a little more energy. I sleep better. More sound sleep. I am not waking up at 2 or 3 in the morning. I am taking zopiclone, a sleeping medication. Previously when I would wake up at 2am I wouldn't go back to sleep. The way I am feeling doesn't feel as bad. I'm able to sleep better and sleep for longer. I don't think about suicide so often. I have had a really positive change in my mood, and the effects are lasting longer this time. I am feeling so good I have been busy planning things to do this week. My mood has been very up and down. My mood has improved a lot. I want to get the house sorted out and clean up. My mood is elevated straight after treatment. On the day of treatment I feel euphoric but this decreases the following day. I am much calmer. My mood is lighter I am not angry anymore. My anger has gone completely. I don't have to take olanzapine any more my symptoms are now manageable with only diazepam. On the day the day of treatment I feel euphoric. It is an awesome feeling. For the whole day. The following day is normal again. I am seeing things in a more positive light. I feel a little bit more alert but this is mostly on the day and isn't lasting. My mood is a bit better, lighter, not as heavy. I'm not so sad. I can smile and mean it. I can see hope now. I laugh more. I get a slight lift in mood but it goes back down again. Mostly for the day I have treatment for a few hours. I have noticed a positive change to my mood and my thoughts are more positive.</p>

	<p>I laugh more now. I don't dwell on the bad things as much after ECT. This lasts about 4 days. My mood has changed in a positive direction. My mood is much much better. I get a slight lift in mood but it goes back down again. For the day I have ECT I have the lift for most of the day but only for a few hours. My mood is better. I don't feel quite so down. Funnily enough I feel more alert after ECT. The positive effects of ECT on my mood are lasting about 5 days. It is such a shame it doesn't last longer! My mood has lifted and it lasts longer. My mood increased which lasts all day for about 2-3 days. I have noticed a positive change in my mood since having ECT on Monday. I'm not as stressed and I feel happier. Good change in mood and it is lasting longer. Sometimes I feel more alert but then it feels like I have a comedown and feel worse. Sometimes I am really alert and positive straight after ECT, but it doesn't last, it usually goes down the day or day after ECT. My mood is better. I am still having thoughts of self harm but they are less frequent and I don't act on the thoughts anymore. I have had a lift in mood. I haven't self harmed since last week. I think my mood is a little bit better now. My mood has improved. My mood is slightly up. I think I'm less depressed. My mood has become more even I have become more active and I have a better frame of mind. My family relationships have improved because I have more energy for my family My outlook on life is better</p>	
<p>Somatic Impairment</p>	<p>References to physiological or somatic change. Last time I had ECT my heart stopped and I cardiac arrested from the anaesthetic so I didn't have a treatment. I feel tired but have to be doing something. I have gained weight since having ECT I am eating more and am hungry more often. I get quite bad headaches. I have a headache after treatment. I get bad headaches after treatment.</p>	<p>f=14</p>

	<p>Get headaches on the day of treatment which last 3 hours. I have bad headaches the day of ECT. I have more physical symptoms. My balance has gotten bad. I feel more fatigued. I get a dry mouth after ECT and tiredness. I have been sleepwalking really badly. More frequently than usual. Doctor said it may be due to the ECT stimulating my frontal lobes. My body is exhausted. I get headaches after treatment. I take pills to resolve it half an hour after ECT. It is a throbbing headache.</p>	
<p><i>Improvement</i></p>	<p>I have less headaches and less severity in my headaches.</p>	<p>$f=1$</p>

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Appendix H

Content Analysis of Subjective Reports at the Six-Week Follow-Up Assessment

Table 4
Categorisation of subjective cognitive, clinical and somatic changes six weeks after finishing a course of ECT

Domain	Operational Definition	f
Cognition		
General Cognition		
<i>General Cognitive Function</i>	References made to general/non-specific function of cognitive processes.	
<i>Improvement since finishing ECT</i>	<p>My cognition is so much better. I can work through my memory loss now and I do homework to refresh my memory. My thinking is clearer. After my last course of ECT in my early thirties, I also had some cognitive difficulties which subsided soon after treatment. During my treatment, my memory and my ability to think were affected and that has improved a bit. I am able to listen now. I can think things through again. I feel more onto it. Since finishing ECT, my responses are sharper if people ask me questions.</p>	f=9
<i>General Memory</i>	References made to general/non-specific function of memory.	
<i>Improvement since finishing ECT</i>	<p>My memory is much better. My memory is about the same as it always was (before ECT). My memory is getting back to where it was before starting ect. During my treatment, my memory and my ability to think were affected and that has improved a bit. Since I have finished ECT I haven't had many problems with forgetting things but during the course had some incidents with forgetting. Memory isn't as bad as what it was during ECT</p>	f=6
<i>Impairment remained</i>	<p>My memory is worse since having ECT. My memory is still poor for things that happened around the time of ECT. I have forgotten a lot that happened during that time.</p>	f=2
<i>General Long Term</i>	References made to general/non-specific function of long term memory.	

<i>Memory</i>		
<i>Improvement since finishing ECT</i>	I don't have any problems with my memory, but I never really did during my treatment either. Both my short term and long term memory seems to be the same as it always was.	f=2
<i>Impairment remained</i>	Long term memory still bad.	f=1
<i>General Short Term Memory</i>	References made to general/non-specific function of short term memory.	
<i>Improvement since finishing ECT</i>	My memory for 'recent' things is back to where it was before ect. Both my short term and long term memory seems to be the same as it always was. I have had a slight but not complete recovery in my memory function Some things completely gone and other things are slowly coming back to me when I am reminded of them.	f=4
<i>Impairment remained</i>	My short term recall is still compromised. My short term memory isn't as good. My memory is shocking. Some things completely gone and other things are slowly coming back to me when I am reminded of them.	f=4
<i>Anterograde Amnesia</i>	References made regarding the ability to remember facts and information learned since starting ECT.	
<i>Improvement since finishing ECT</i>	Now when I do things I do remember them later.	f=1
<i>Retrograde Amnesia</i>	References made regarding the ability to remember facts and information learned prior to starting ECT.	
<i>Episodic Autobiographical Memory</i>	References made regarding the ability to remember personal episodic information learned prior to starting ECT. Episodic autobiographical memory is the conscious recollection of temporally and spatially specific events from one's past (Levine, 2004). This conscious state of mental time travel is achieved through recollection of events, sensory-perceptual details, thoughts, and feelings such that the rememberer becomes aware of an event as belong to his or her own past (Tulving & Craik, 2000).	
<i>Improvement since finishing ECT</i>	Now when I do things I do remember them later.	f=1
<i>Impairment remained</i>	I can't remember things that I did with my kids just before the course of ECT. I also can't remember places that I visited while I was having ECT. I couldn't remember my sister's wedding reception. Somebody asked me where it was and I couldn't tell them. It was at a beautiful venue and we have lots of photos, but I still couldn't remember. I worked hard trying to think of it, and eventually it came back to me. This is happening all the time.	f=3
<i>Semantic Memory</i>	References made regarding the ability to remember knowledge and facts about the world that is not tied to any specific personal experience (Goldstein, 2008). This information was learned prior to starting ECT.	
<i>Impairment remained</i>	I forgot that my friend's wife was pregnant with their second child. I have forgotten some of the streets in the city and what they are called. But it might be because I haven't driven to town for 3	f=2

	months.	
<i>Personal Semantic Memory</i>	References made regarding the ability to remember semantic memories that have personal significance (Goldstein, 2008) learned since starting ECT.	
<i>Impairment remained</i>	I had forgotten my bank number. I still forget little things like my siblings ages. There are some colleagues from works whose names I couldn't remember and I have been working with them for almost a year. One in particular looked familiar but I couldn't place her. That happened when I came back here too, there were a couple of nurses who I knew looked familiar, but I couldn't place them.	f=3
<i>Prospective Memory</i>	References regarding the ability to remember to perform intended actions (Goldstein, 2008)	
<i>Impairment remained</i>	I have some lapses in my memory like the other day I left the car door open, and left the tap going full bore. I still can't do without lists or calendar I am still needing reminders on my phone.	f=3
<i>Executive Functioning</i>	References made regarding the ability to plan, organise, sequence, regulate inhibit responses, and use judgement (Schoenberg & Scott, 2011)	
<i>Improvement since finishing ECT</i>	Before ECT I was more impulsive, I am less impulsive now. I can do Sudokus now, never used to be able to do them while I was in hospital. I had forgotten the steps in how to drive, but I remember it again now My thoughts are always negative and they are constantly negative but after having ECT my thoughts are never as negative.	f=4
<i>Impairment remained</i>	I'm not as good as I was at crosswords	f=1
<i>Concentration/ Attention</i>	Refers to the ability to focus on specific features of the environment or on certain thoughts or activities (Goldstein, 2008).	
<i>Improvement since finishing ECT</i>	I can think things through now.	f=1
<i>Impairment remained</i>	My concentration is still not where it should be. My concentration is really poor but that might be my bipolar. Concentration is still not great. The other day I went into wellington, I parked my car and had trouble finding my car! I found it in the end but I had forgotten what street it was parked on. This might have been because I was quite manic and had a lot of thoughts racing through my head that I didn't pay much attention to where I had parked it.	f=4

<p>Depression</p>	<p>References to one's emotional and clinical state.</p>
<p>Duration of Clinical Improvement</p>	
<p><i>Improvement maintained</i></p>	<p>The ECT didn't really work for me but about three weeks after ECT I had an improvement in my mood and became hypomanic. My mood has stayed the same. Since I have had ECT I have been more aware of what is going on in my life. I'm more alert. I am more motivated now; I am going to the gym. I feel much better and the ECT has taken away my anxiety. I am feeling much more enthusiastic about myself and positive about life My mood isn't fantastic but it has definitely lightened up – I'm not as heavy.</p> <p style="text-align: right;">f=8</p>
<p><i>Re-emergence of depressive symptoms</i></p>	<p>My mood had improved from ECT but it is a little bit worse now that I haven't had ECT for a while. The positive effects on my mood only lasted a couple of days after my last ECT, and then I went downhill again. The first few weeks after ECT I was happy, but it didn't last. I was really motivated and I liked my life. I feel silly about that now, I might have been a bit careless, but after being so depressed I was just so relieved to be happy. ECT worked really well but the effects only lasted for about 1.5 weeks and then I got really sick. My sleep is still out of whack. I'm still irritable. I had a suicide attempt last week. My mood has gone down a lot. I am having rapid mood swings which are much worse since stopping the treatment and much more extreme. The highs are really high and the lows are really low. I have really bad days where I want to end it all. I have trouble sleeping. My body is exhausted but my mind is still awake. I am quite suicidal. I thought about hanging myself but then my psychiatric nurse talked me out of it. I am on weekly pick up for my meds so I don't overdose. I have really bad mood swings</p> <p style="text-align: right;">f=13</p>

Goldstein, B. E. (2008). *Cognitive psychology Connecting the mind, research and everyday experience* (2nd ed.). Belmont, CA Wadsworth.

- Levine, B. (2004). Autobiographical memory and the self in time Brain lesion effects, functional neuroanatomy, and lifespan development. *Brain and Cognition*, 55(1), 54-68. doi 10.1016/s0278-2626(03)00280-x
- Schoenberg, M. R., & Scott, J. G. (2011). *The little black book of neuropsychology A syndrome based approach*. New York Springer.
- Tulving, E., & Craik, F. (2000). *The Oxford Handbook of Memory*. Oxford Oxford University Press.

Appendix I

Ethics Approval



Central Regional Ethics Committee
c/- Ministry of Health
PO Box 5013
1 the Terrace
Wellington
Phone: (04) 816 2403
Email: central_ethicscommittee@moh.govt.nz

16 May 2012

Ms Anneke Thornton
Massey University
2/259 Jackson Street
Petone, Lower Hutt

Dear Ms Thornton

Ethics ref: **CEN/12/EXP/032** (please quote in all correspondence)
Study title: An Evaluation into the Short Term Cognitive Effects of Electroconvulsive Therapy, and the Development of a Screening Measure to Monitor these Cognitive Changes.

This study was given expedited ethical approval by the Chair of the Central Ethics Committee on 15 May 2012.

Approved Documents

- Expedited Review of Observational Studies Application Form signed and dated 8 May 2012 by Anneke Thornton
- Data Collection Form
- Participant Information and Consent Pack
- Questionnaire for ECT practitioners
- Study Protocol Version 1 dated 15 April 2012

This approval is valid until 1 June 2013, provided that Annual Progress Reports are submitted (see below).

Amendments and Protocol Deviations

All significant amendments to this proposal must receive prior approval from the Committee. Significant amendments include (but are not limited to) changes to:

- the researcher responsible for the conduct of the study at a study site
- the addition of an extra study site
- the design or duration of the study
- the method of recruitment
- information sheets and informed consent procedures.

Significant deviations from the approved protocol must be reported to the Committee as soon as possible.

Annual Progress Reports and Final Reports

Programme & Abstracts, Sunday 8th

context of post-structural ideas, with a focus on students as 'subjects-in-process' and 'subjects-in-relation'. This presentation will be of interest to educators, trainee psychologists/counsellors, and those with an interest in understanding ideas of transformation through attention to the affective and discursive in moments of lived experience.

shane.barracough@canterbury.ac.nz

Neuropsychology Symposium continued

Chair: Janet Leatham

3.00pm - 4.30pm Case Room 3

3.00pm

Coin rotation task, a test of motor speed and dexterity: Norms for New Zealand

Anneke Thornton, DCLinPsych candidate, Massey University

Janet Leatham, Massey University

Ross Flett, Massey University

Background: The Coin Rotation Task (CRT) is a validated test of psychomotor speed and motor dexterity. It is quick, convenient and inexpensive. However it is based on the US nickel, normative data is limited and no norms exist using any NZ coin.

Aims: To collect New Zealand norms for the use of the CRT and to compare the US nickel against the NZ 20 cent coin to examine equivalency between the two coins.

Method: As quickly as possible, 215 participants aged 16-87, rotated the 20 cent coin 180 degrees in their dominant and non-dominant hands - 20 seconds per hand.

Results: Normative data was collected for number of rotations per 20 seconds with the NZ 20 cent coin. No significant difference in the number of coin rotations was found between the nickel and the 20c coin.

Conclusion: Norms are now available to facilitate use of the CRT in New Zealand and the US.

anneke_thornton@hotmail.com

3.30pm

Cheap, fast, good: Pick Two - assembling a practical comprehensive assessment for a Memory Service

Dryden Badenoch, Consultant Clinical Psychologist, Relaxed Therapy

Providers of Memory Services are increasingly opting to forego neuropsychological assessment (seen as costly and time-consuming) in favour of simple screening measures (seen as quick & convenient, despite being imprecise and unhelpful when planning interventions): choosing "cheap & fast" over "good".

The challenge for over-worked clinicians is to identify or assemble a test battery sufficient to diagnose dementia by sub-type and rule out or support differential diagnoses, as well as inform future management, without demanding too much of either the client or the clinician.

This presentation describes the process of assembling such a battery. It is suitable for any Clinical Psychologist or intern with an interest in neuropsychology.

We review the relevant areas of cognitive functioning, the available tests of these functions and the tests' suitability for use together in an assessment process which is as "cheap & fast" as possible while remaining "good".

drydenbadenoch@relaxedtherapy.com

4.00pm

Monitoring cognitive function during ECT: Current New Zealand practice.

Anneke Thornton, DCLinPsych candidate, Massey University

Janet Leatham, Massey University

Ross Flett, Massey University

Objective: To determine current practice regarding cognitive assessment during electroconvulsive therapy among ECT treating professionals across New Zealand.

Design/Participants: A questionnaire sent to 45 professionals resulted in a response rate of 35.5% from 14 district health boards across New Zealand.

Results: Most assess cognitive function at least once during a course of ECT. A third assess at baseline, at least once during and then after the course. Assessments are being conducted by people from various professions. Over two thirds said that a lack of time, resources and sensitive tests were restricting more frequent and thorough cognitive assessments.

Conclusion: Respondents recognise that assessing cognitive functioning during a course of ECT is important, though large variations in the nature, frequency and length of the assessments exist. Future research should develop a standardised, sensitive, inexpensive screening measure tailored for use with patients receiving ECT to help overcome the current restrictions to cognitive assessment.

anneke_thornton@hotmail.com

Forensic Psychology Symposium

Chair: Bronwyn Rutherford

3.00pm - 5.00pm OGGB 5

3.00pm

Development and evaluation of a dynamic risk assessment measure for prison case management

Nick Wilson, Principal Advisor Psychological Research, Department of Corrections, NZ

One of the key tasks for offender case management is the creation and implementation of an individualised dynamic offender management plan that covers both offender risk variables and protective assets empirically linked to desistance from offending. The Structured Dynamic Assessment Case-management-21 item (SDAC-21; Serin & Wilson, 2012) is a 21 item dynamic risk structured assessment approach developed to assist New Zealand case managers to systematically assess three domains reflected in the measure that are empirically related to risk of re-offending, responsibility, and desistance (Protective) in relation to viable individual risk scenarios.



Asociación Española de
Psicología Conductual
(AEPC)

VI CONGRESO INTERNACIONAL Y XI NACIONAL DE PSICOLOGÍA CLÍNICA

**Santiago de Compostela, España
6, 7 y 8 de Junio 2013**



El Comité Científico del "VI CONGRESO INTERNACIONAL Y XI NACIONAL DE PSICOLOGÍA CLÍNICA" celebrado en Santiago de Compostela, España, del 6 al 8 de Junio de 2013, informa que

Anneke Thornton y Janet Leathem

Ha(n) presentado en éste Congreso la Comunicación titulada

COIN ROTATION TASK. THE DEVELOPMENT ON NORMS FOR NEW ZEALAND AND THE UNITED STATES

Santiago de Compostela, España, 8 de Junio de 2013



Fdo.: Juan Carlos Sierra
Presidente del Comité Científico