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**EVALUATING THE IMPACT OF ATTENTION PROCESS TRAINING  
(APT) ON ATTENTION DEFICIT IN THE EARLY STAGES OF  
RECOVERY FROM STROKE**

**A thesis submitted in fulfilment of the requirements for the degree of**

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**in**

**Psychology**

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**by**

**Margaret Diana Dudley**



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## Abstract

Attention deficits are a prominent sequel of stroke and impact negatively on rehabilitation outcomes. However, rehabilitation efforts are almost entirely concerned with the remediation of physical impairments that result from the stroke despite the involvement of attention in physical functioning. Attention Process Training (APT) is a cognitive retraining programme originally designed for the remediation of attention deficit following traumatic brain injury. However, the efficacy of APT post-stroke is not yet known, as to date, few studies have been conducted with small sample sizes. This study evaluated the effectiveness of APT in improving attention in stroke survivors within the five to eight week period post-stroke. Seventy eight patients admitted to hospital with first-ever-stroke were identified as having an attention deficit by obtaining a score of one standard deviation below the normative mean on any of the following widely-used neuropsychological measures of attention; the Auditory Attention Quotient (AAQ) or Visual Attention Quotient (VAQ) of the Integrated Visual and Auditory Continuous Performance Test, (IVA-CPT), either trial of the Trail Making Test (TMT), the Paced Auditory Serial Addition Test (PASAT), or by three or more errors made on the left or right side of the Bells Cancellation Test. These measures were re-administered on completion of treatment. Participants were randomised to either the experimental group who received standard care and up to 30 hours of APT or to a control group that received standard care only. The primary outcome measure was the Full Scale Attention Quotient (FSAQ) of the IVA-CPT which is a measure of attention derived from both auditory and visual attention quotients. The secondary outcome measure was a health-related quality

of life measure, the SF-36, (Short-Form-36). Both measures were administered before treatment and again on the completion of treatment. The results showed that on the primary outcome, the APT group showed improvement from baseline to post-treatment whereas the SC group had not. Significant improvement by the APT group was also demonstrated on two other measures of the IVA-CPT including the Auditory Attention Quotient and the Full Scale Response Quotient (a measure of impulsivity). On the quality of life measure neither the APT group nor the SC group demonstrated a significant change in scores.

The results of this study provide further support for the efficacy of cognitive rehabilitation and in particular that APT is an effective cognitive treatment option for the remediation of attention deficit in the early stages of stroke recovery. The characteristics of stroke survivors who might benefit most from APT are identified as well as those factors that possibly influence the subjective experience of this particular intervention. The appropriateness of some measures such as the PASAT, the TMT, cancellation tests as well as continuous performance tests that are often used in research of attention deficit, are also discussed in the context of a stroke population.

It is hopeful that the optimistic outcomes of this study will encourage further needed research in this area in order to inform stroke rehabilitation specialists to incorporate cognitive rehabilitation into predominantly physically-focussed programmes.

*Maraea Dudley*  
*1923-2005*

You raised your eight children and worked full time throughout. You were the hardest working person I have known. I am so proud of you. This is for you mum.  
I wish you were here to enjoy it with me.

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## List of Abbreviations

AAN	American Academy of Neurology
AAQ	Auditory Attention Quotient
ACRM	American Congress of Rehabilitation Medicine
ADLs	Activities of Daily Living
AHA	American Heart Association
ANCOVA	Analysis of co-variance
ANOVA	Analysis of variance
APT	Attention Process Training
APQ	Auditory Prudence Quotient
BASIS	Boston Acute Stroke Imaging Scale
BDI	Beck Depression Inventory
BI	Barthel Index
BIAA	Brain Injury Association of America
BNT	Boston Naming Test
CCT	Clinical Control Trial
CFQ	Cognitive Failures Questionnaire
CHI	Closed Head Injury
COAST	California Older Adult Stroop Test
COWA	Controlled Oral Word Association
CPSP	Central Post Stroke Pain
CPT	Continuance Performance Test
CRP	Cognitive Remediation Programme
CTT	Consonant Trigrams
CVA	Cerebrovascular Accident
CVLT	California Verbal Learning Test
DALYs	Disability Adjusted Life Years
EBIQ	European Brain Injury Questionnaire
ED	Executive Dysfunction
EFNS	European Federation of Neurological Societies
fMRI	Functional Magnetic Resonant Imaging
FSAQ	Full Scale Attention Quotient
FSRQ	Full Scale Response Quotient
HRQoL	Health Related Quality of Life
IVA-CPT	Integrated Visual & Auditory Continuous Performance Test
LACS	Lacunar Stroke
LM	Logical Memory
MCI	Mild Cognitive Impairment
MCS	Mental Component Summary
MMSE	Mini Mental State Exam
MOANS	Mayo's Older Americans Normative Studies
mRS	modified Rankin Scale
NIHSS	National Institute of Health Stroke Scale
NINDS	National Institute of Neurological Disorders and Stroke
NINDS-CSN	National Institute of Neurological Disorders and Stroke-Canadian Stroke Network
OHI	Open Head Injury
OT	Occupational Therapy
PACS	Partial Anterior Circulation Stroke

PASAT	Paced Auditory Serial Addition Test
PCS	Physical Component Summary
PFO	Patent Foramen Ovale
PICH	Primary Intracerebral Haemorrhage
POCS	Posterior Circulation Stroke
PSHVF	Post-stroke Homonymous Visual Field
RCI	Reliable Change Index
RCT	Randomised Control Trial
ROCF	Rey-Osterrieth Complex Figure
SAH	Subarachnoid Haemorrhage
SD	Standard Deviation
SF-36	Short Form Questionnaire-36
SIS	Stroke Impact Scale
SSD	Single Subject Design
START	Stroke Attention Rehabilitation Trial
TACS	Total Anterior Circulation Stroke
TBI	Traumatic Brain Injury
TEA	Test of Everyday Attention
TLC-E	Test of Language Competence-Expanded Version
tPA	Tissue Plasminogen Activator
TIA	Transient Ischaemic Attack
UI	Urinary Incontinence
VAQ	Visual Attention Quotient
VPA	Visual Paired Associates
VPQ	Visual Prudence Quotient
WAIS	Wechsler Adult Intelligence Scale
WAIS-R	Wechsler Adult Intelligence Scale-Revised
WISC 111	Wechsler Intelligence Scale for Children-Third Edition
WHO	World Health Organisation
WMS	Wechsler Memory Scale

*“Suffering isn’t ennobling, recovery is”*

*Christiaan Barnard*

## **Chapter 1: Stroke**

Stroke is the most common disabling neurological condition of adults world-wide with survivors experiencing physical impairments, personality changes, disruption of family and community living, and decreases in vocational functioning (Anderson, 1992). Traditionally, rehabilitation from stroke has focused on recovery of physical abilities and speech/language rehabilitation (Paolucci et al., 1996). However, survivors of stroke commonly present with cognitive impairment that can create lifelong burdens for the individuals and their caregivers. Despite this common problem, relatively little research has been conducted into the efficacy of cognitive rehabilitation following stroke. Without this knowledge base the perception that cognitive retraining is ineffective and unrelated to physical recovery, continues to propagate. Indeed, even those patients who are aware of their cognitive deficits are more likely to focus their energy on physical rehabilitation with the expectation of achieving quicker and more observable outcomes, leaving little reserve for other rehabilitation pathways.

In the last few decades major forces that have helped fuel the advancement of cognitive rehabilitation have included, the exponential growth in new technology, new perspectives and findings with regard to neuro-plasticity, cutbacks in the health care sector, an increasing sense of self-empowerment regarding health issues, and a growing emphasis on functional outcomes of rehabilitation. Cognitive rehabilitation has become increasingly recognised as a method designed to reduce cognitive dysfunction and assist individuals in compensating for its impact on daily living (Wilson, 1997). Systematic reviews



of cognitive rehabilitation that have been conducted in the United States (Cicerone et al., 2000; 2005; Sohlberg et al., 2003) along with research conducted in Europe (Lincoln, Majid, & Weyman, 2000; Majid, Lincoln & Weyman, 2000) have added to a growing bank of evidence for the efficacy of cognitive rehabilitation (Halligan & Wade, 2005; High, Sander, Struchen, & Hart, 2005).

In response to this now substantial bank of evidence, the Brain Injury Association of America (BIAA) in 2009 urged the American government to include under the military healthcare insurance scheme, full access to cognitive rehabilitation for its military personnel who had suffered a traumatic brain injury. The BIAA stated that cognitive therapy is “one of the most widely accepted and critical rehabilitative treatments” for traumatic brain injury (BIAA, 2006).

The purpose of my thesis was to evaluate the efficacy of an attention rehabilitation programme in stroke survivors with attention deficit which has been found to be a common area of cognitive impairment following stroke (Hochstenbach, Mulder, van Limbeek, Donders, & Schoonderwaldt, 1998; Nys, 2005; Tuhim, 1993). Attention is a particularly important aspect of cognitive functioning as it is reported to be the basis for other areas of cognition including memory, communication, and executive functioning (Bennett, 1998; Whyte, 1992).

In order to place this study within its appropriate context, this review of the literature will begin with providing a definition of stroke followed by a presentation of the various taxologies of the disease. A summary of the epidemiology of the disease including the burden of stroke will then be presented followed by a summary of the risk factors associated with the disease. The subsequent chapter will then identify the after-effects of stroke leading to an

overview of the possible cognitive deficits that may result from this condition. A discussion on cognitive rehabilitation will then provide the background for a closer examination of attention deficit as a cognitive impairment following stroke, with an analysis of the interventions that have been utilised to date. Finally, there will be a discussion of the challenges often confronted when conducting research of behavioural interventions followed by a number of suggestions that will hopefully help researchers overcome those challenges.

### **Description of Stroke**

The terms stroke and cerebrovascular accident (CVA) are used interchangeably to describe brain damage or dysfunction that occurs as a result of some disruption in the vascular supply to or of the brain. The term stroke, however, conveys the suddenness and randomness with which it occurs and has now become the preferred term of use (Lindley, 2008). Stroke is a heterogeneous term incorporating a set of neurological symptoms that lead to damage to the brain and less commonly to the spinal cord (Caplan, 2000; Spengos et al., 2006). The World Health Organisation's (WHO) broad definition of a stroke as "rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 hours or longer or leading to death with no apparent cause other than of vascular origin", has sound practical application because of its non-reliance on imaging data and can therefore be used in countries where such technology is not readily available (Kwan, 2001).

Stroke comes in two major forms and is generally categorised as either ischaemic or haemorrhagic depending on the physiological antecedent (Lezak, 2005). An ischaemic stroke is the result of an interruption of the oxygen and nutrient carrying blood supply to the brain.

After a few minutes without that supply brain cells begin to die. Ischaemic strokes are more common accounting for approximately 80% of all strokes with haemorrhagic strokes accounting for the remainder. The different causes and location of the interruption of blood supply to the brain provides the basis for subtypes of ischaemic stroke including, thrombosis, embolism, and systemic hypoperfusion (Caplan, 2006; Rudd, Irwin, & Penhale, 2005). Strokes of an undetermined cause constitute 30-40% of all ischaemic strokes and are termed “cryptogenic” (Adams et al., 1993; Donnan, Fisher, MacLeod, & Davis, 2008).

A thrombotic stroke is formed within the brain itself and often arises due to a localised occlusive process anywhere in the vascular system causing stenosis (an abnormal narrowing in a blood vessel) or blockage in the larger arteries that supply the brain, such as the carotid artery or the middle cerebral artery (Appel & Linas, 2007). Atherosclerosis (the thickening of an artery wall as the result of a build-up of fatty materials) is the most common disease that narrows the blood flow in the artery and is most common where arteries branch or bifurcate (Ponsford, 2004). This type of stroke is termed a large-vessel thrombosis while small-vessel thrombosis involves one (or more) of the brain's smaller, yet deeper penetrating arteries such as the anterior communicating artery or the posterior communicating artery. This latter type of stroke is also called a lacunar stroke (Donnan & Norrving, 2009; Hreib, 2008; Molina et al., 1999)

An embolic stroke, estimated to account for 15-20% of clinical stroke events, is caused by a clot that is formed outside the brain and travels the bloodstream until it becomes lodged and cannot travel any further. An embolic stroke can itself be further divided into cardiac embolism where the clot arises

proximally most commonly from the heart, and artery-to-artery embolism where the origins lie in major arteries and systemic veins (Bogousslavsky et al., 1991).

Systemic Hypoperfusion refers to the reduction in blood flow which can be caused by bleeding, dehydration, or loss of fluid into body tissues (shock) (Caplan, 1991). The permanent damage caused by such blockages is called an infarct. Damage to the larger arteries which supply blood to important parts of the brain such as those that control movement or speech can result in catastrophic outcomes. Signs and symptoms commonly seen when having a stroke include hemiparesis (weakness on one side of the body), aphasia (language disorder), dysphagia (difficulty in swallowing), dysarthria (speech problems caused by the muscles or nerves controlling them), hemianopia (visual field loss), ataxia (lack of coordination of voluntary movement), apraxia (a disorder of the nervous system which prevents the performance of tasks or movements despite having the physical ability to do so) severe headache, and/or disturbed consciousness (WHO, 2005).

Various classification systems exist for acute ischemic stroke based on factors such as clinical presentation and imaging as their distinguishing features. Classification systems have been found to be important as they provide evidence for determining prognosis, mortality rates and better management practices. For example, The Boston Acute Stroke Imaging Scale (BASIS) is a system that has been found to provide accurate prognostic data (Merino & Latour, 2008). However, determining the subtype of Ischaemic stroke is often accomplished using the criteria of TOAST (Trial of ORG 10172 in Acute Stroke Treatment), a system based on clinical findings supported by brain, vascular and cardiac imaging and includes the following five categories: (1) large-artery

atherosclerosis, (2) cardio-embolism, (3) small-artery occlusion, (4) stroke of other determined aetiology, (5) stroke of undetermined aetiology (Adams et al., 1993). The Oxford Community Stroke Project classification is another widely used system based on the site and extent of the symptoms and the categories include; Total Anterior Circulation Infarct (TACT), Partial Anterior Circulation Infarct (PACT), Lacunar Infarct (LACI), and Posterior Circulation Infarct (POCI) (Bamford, Dennis, Sandercock, Burn, & Warlow, 1990).

In a haemorrhagic stroke, as the name suggests, there is a leak or a bursting of a blood vessel and the blood spills into areas of the brain usually from a ruptured cerebral aneurysm or head injury, causing injury to the surrounding areas. Haemorrhagic strokes which account for 10-20% of all strokes can be further divided into categories; Primary Intracerebral Haemorrhage (PICH) and Subarachnoid Haemorrhage (SAH). The two subtypes present with dissimilar clinical problems and therefore necessitate different management methods. PICH is the most frequent form of haemorrhagic stroke and occurs when a small artery in the brain ruptures or leaks blood directly into the brain substance, often caused by hypertension. A SAH on the other hand, describes the leakage of blood (from aneurysms or arteriovenous malformations) onto the brain's surface which circulates around the brain via the spinal fluid pathways (Caplan, 2000; Warlow et al., 1996). SAH is a severe form of stroke with over 50% of patients dying in the first three months and 10-15% dying before reaching hospital (van Gijn, Kerr, & Rinkel, 2007).

A Transient Ischaemic Attack (TIA), although commonly referred to as a "mini stroke" is a temporary reduction in blood supply resulting in disturbance in body function lasting less than 24 hours and is therefore excluded from being

categorised as a stroke (Semple, 1998). However, TIA's have been shown to be predictive of a more major stroke in the future (Coull, Lovett, & Rothwell, 2004; Lovett et al., 2003).

### **Epidemiology of Stroke**

Epidemiological studies of stroke present an enormous challenge given the heterogeneity of the condition, a lack of readily available neuroimaging in resource-poor regions, the absence of primary data from many countries and differences in study design which make comparisons across populations difficult. Malmgren et al. (1987) published a list of 12 methodological criteria to standardise definitions and case ascertainment for the ideal study of stroke incidence and/or mortality. These criteria have evolved over time and were later updated by Bonita, Broad, Anderson and Beaglehole (1995), by Sudlow and Warlow in 1996 by Feigin, Lawes, Bennett, and Anderson in 2003 and again by Feigin and Vander Hoorn in 2004. In their population-based incident study of stroke covering two decades Rothwell et al. (2004) employed new methodological practises for the validation of completeness of case ascertainment for stroke. Those practises included monthly investigations of data bases of general practitioners in the study area to identify all patients coded with a cerebrovascular diagnosis, with subsequent reviewing of all patients identified. Also, all patients who were admitted to hospital with an acute vascular problem and all patients undergoing elective or emergency coronary, carotid, or peripheral vascular investigations or interventions, were reviewed on a daily basis. The utilisation of these two methods resulted in an additional 16% of new stroke events in the Rothwell study.

Based on these findings, Feigin and Vander Hoorn (2004) suggested that the “ideal” criteria for a study of stroke incidence and/or mortality consists of three categories including; Standard definitions, Standard methods, and Standard data-presentation. The WHO definition of stroke is recommended as the ideal criteria and is the definition that has been widely used in incidence studies (Donnan, et al., 2008; Feigin & Vander Hoorn, 2004). In addition to the WHO criteria, Feigin & Vander Hoorn suggest at least 80% verification by neuroimaging for diagnosis of ischaemic, intracerebral haemorrhage and subarachnoid haemorrhage, classification of ischaemic stroke into subtypes, and separate and combined data for first-ever-in-a-lifetime and recurrent stroke. Standardisation of methodological practises include, a prospective study design with population-based case ascertainment based on overlapping sources of information from hospital, outpatient clinics, general practitioner data bases and death certificates, and a large well-defined stable population with census data not more than 5 years old. Standard data-presentation should cover complete calendar years and no more than 5 years of data should be averaged together. Data on men and women should be presented separately and age-specific estimates should be presented in mid-decade age bands (e.g. 45-54 years) that include the oldest age group ( $\geq 85$  years) and have 95% confidence intervals around rates.

The available data suggests that the human toll resulting from the increase in the incident of stroke worldwide has reached pandemic proportions. In 2002, the WHO released data identifying stroke as the cause of approximately 10% of all deaths world-wide, ranking it as the second cause of death after ischaemic heart disease, (excluding neoplastic diseases as a group; Dewey et al., 2004; Feigin & Parag, 2007). In China, the most populous country on earth, stroke has

become the leading cause of death in many parts of the country (Xu, 2008). Approximately 16 million first-ever strokes occur annually in the world. Recent epidemiological studies report 85% of strokes now occur in low income and middle income countries, despite these populations having a shorter life expectancy (Feigin, Lawes, Bennett, Barker-Collo, & Parag, 2009; Johnson, Mendis, & Mathers, 2009). In their systematic review of world-wide stroke incidence spanning 28 countries, Feigin et al. (2009), provide data indicating that high income countries have witnessed a 42% decrease in stroke in the last four decades, however, in low to middle income countries over the same time period, the incidence of stroke has increased by 100%. Their study showed that in the years 2000 to 2008, stroke incidence rates in low to middle income countries for the first time, exceeded the stroke incidence rates in high income countries and has now reached epidemic proportions. Feigin et al. (2009) also found that over the last four decades there was a greater reduction in the incidence of primary intracerebral haemorrhage than of ischaemic stroke in high income countries although the incidence of subarachnoid haemorrhage had not changed during the same time period. Given that age is a major risk factor for stroke, more people are at risk in a globally aging population (Dewey et al., 2004), and of those that survive the stroke, between 15% and 30% are permanently disabled (Lloyd-Jones et al., 2009) placing enormous social and economic burdens on national resources.

### **Economics of Stroke**

The socioeconomic burden of stroke continues to escalate relative to a worldwide aging population and an increased number of survivors of stroke. The implications of this situation is no better illustrated than in Japan, a country that enjoys a rapidly aging population but must now face the drastic economic



consequences of healthcare and in particular mounting pressure for more efficient stroke rehabilitation (Liu, Chino, & Takahashi, 2000). A similar situation was also revealed by Tobias, Cheung, Carter, Anderson, and Feigin (2007), in their estimations and projections of stroke incidence, prevalence and mortality in New Zealand where they found stroke mortality falling faster than stroke incidence. While those results were representative of major public health achievements, paradoxically, unless effective prevention, management and rehabilitation services are developed accordingly, the burden of stroke will increase. Stroke remains the leading cause of physical disability in adults over the age of 65 years and accounts for one of the largest health burdens to face humanity. Stroke burden is projected to rise from around 38 million disability-adjusted life years (DALYs) lost globally in 1990 to 61 million DALYs in 2020 (MacKay & Mensah, 2004). In the United States in 2008, the total direct costs of stroke (such as ambulance services, hospitalisation, rehabilitation, nursing home costs, drugs and indirect costs like lost wages) in those under 65 is estimated to be \$65.5 billion and €38.1 billion in European countries. In the United Kingdom (UK) total societal costs were estimated at £8.9 billion which accounts for 5.5% of the total UK national health expenditure (Dewey et al., 2004). Tobias et al. (2007), report a parallel situation in New Zealand estimating a 4% reduction in stroke mortality between 1991 and 2003 against a 1% increase in stroke prevalence. Yet again, longer life expectancy and new and improved strategies for acute stroke management in New Zealand results in an increase in the number of stroke survivors thus placing ever-increasing demands on health resources. The average lifetime cost of each stroke in New Zealand for acute care, rehabilitation, support services, and institutional care is estimated to be NZ\$50,000 to NZ\$100,000 (Gommans, 2004), with a total

quantifiable cost to the country estimated at up to \$154 million per year (Anderson et al., 2005, Payne, Huybrechts, Caro, Green, & Klittich, 2002; Scott & Scott, 1994 ). The enormous drain stroke has on the annual health budget highlights the urgency for better prevention and management strategies of this health hazard.

### **Mortality**

Recent studies investigating world-wide stroke mortality rates indicate similar patterns of disparity to that found from incidence studies. For example, Johnson et al. (2009) in their compilation and analysis of data from the WHO Global Burden of Disease project (2002) found stroke mortality rates to be three to five times higher in low-income countries than in middle to high income countries. From a regional analysis they found national income to be a stronger predictor of stroke burden and mortality than risk factors commonly used to assess the cardiovascular health of a country such as diabetes, smoking, alcohol consumption and high body-mass index. They proposed access to care and better management of risk factors as reasons for the disparity. Epidemiological transitional influences such as mortality, fertility, and migration are also considered to be significant factors in the disparity between low and high income countries (Carandang et al., 2006; Feigin et al., 2009; Johnson et al., 2009).

### **Risk Factors**

Changes in the incidence and mortality rate for stroke are attributable to a range of risk factors. The 1984 Framingham Study researching heart disease coined the term “risk factor” (Wilson et al., 1998) to refer to those variables that predicted an individual’s likelihood of developing atherosclerosis or coronary heart disease, the precursors of heart attacks. The term is now commonly used to

refer to any variable associated with an increased risk of disease or infection although these are correlation and not necessarily causal relations. Upon follow-up, the Framingham Study found approximately 850 of the 5209 subjects included in the study, subsequently suffered a stroke. This provided a unique opportunity for the collection of a considerable data base on stroke risk factors. From this and other epidemiological studies, it can also be deduced that stroke is largely a preventable disease. Risk factors for stroke can be categorised into a dichotomy of either modifiable (i.e., those that can be altered either by intervention), or not. Non-modifiable risk factors include ageing, genetic predisposition, gender, low birth weight and ethnicity (Humphries & Morgan, 2004), while modifiable factors are mostly a combination of medical and behavioural causes. Medical risk factors include hypertension, high levels of cholesterol, atherosclerosis (hardening of the arteries), atrial fibrillation (irregular heartbeat), diabetes, sickle cell anaemia, and migraine (Moskowitz & Kurth, 2007). Behaviours or lifestyle factors that increase the risk of stroke include tobacco smoking (Feigin et al., 2005), insufficient physical activity (Ellekjaer, Holmen, Ellekjaer, & Vatten, 2000), alcohol abuse (Reynolds et al., 2003), unhealthy nutrition (Fisher, Lees & Spence, 2006) and obesity (Boden-Albala & Sacco, 2000; Walker et al., 1996). Other less well-documented factors associated with stroke occurrence include low socioeconomic status (Avendano et al., 2006; Cox, McKeivitt, Rudd, & Wolfe, 2006; Wolfe et al, 2002), stress (Truelsen, Nielsen, Boysen, & Gronbaek, 2003), infection (Lindsberg & Grau, 2003) , oral contraceptive use (Gillum, Mamidipudi, & Johnston, 2000), pregnancy (Kitner et al., 1996), and lack of hormone replacement therapy (Fieschi & Fisher, 2001; Li et al., 2008; Wolinsky et al.,

2009). A brief summary of the literature on modifiable and non-modifiable risk factors is presented in Appendix A.

### **Prevention of Stroke**

As the world's population ages, the collective risk of stroke increases so there is a critical need for knowledge of intervention and prevention strategies in order to reduce the rising prevalence of the disease. Awareness of epidemiology and the consequences of stroke are vital in order to press governments, health authorities, and patients into preventative action. Stroke is largely a preventable disease and does not have to be a death sentence or even a life-changing illness. In the United States, despite considerable public awareness campaigns, knowledge of warning signs and risk factors remains abysmally poor, suggesting a more proactive stance particularly within high risk populations is required (Kleindorfer et al., 2009). Modifiable risk factors such as hypertension, diabetes, atrial fibrillation, hyperlipidaemia, smoking, alcohol consumption, physical inactivity and obesity are by far the worst contributors of stroke in developed countries. Primary stroke prevention incorporating life style modifications to reduce risk factors would have a substantial affect on reducing stroke incidence, given that the American Heart Association identified 70% of strokes as being first-ever events (AHA, 2003; Chiuve et al., 2008; Goldstein et al., 2009). Patients with recurrent stroke have on average poorer outcomes than those with first time strokes (Samsa, Bian, Lipscomb, & Matcher, 1999). Stroke survivors have a 15-fold increased risk of stroke recurrence which is a consistent and independent predictor not only of death but of disability, institutionalisation and a decline into dependency (Burn et al., 1994; Hankey, Jamrozik, Broadhurst, Forbes, & Anderson, 2002). The maintenance of a healthy life style, medication

therapies, surgical intervention, stenting and in-hospital management programmes are all secondary stroke prevention techniques that have been found to reduce the incidence of subsequent stroke events (Antiplatelet Trialists Collaboration, 1994, 1998; Biller et al., 1998).

### **After-effects of Stroke**

Stroke fatality and stroke survival rates vary widely and are influenced by a number of variables including type of stroke, severity of stroke, site of lesion, expediency of medical intervention, comorbidity, previous health status, age, socioeconomic status, time since stroke, and so forth. For those that survive the stroke, the after-effects can be extensive, varying from person to person with either physical, psychological or cognitive impairments or a combination of two or all three dimensions.

In order to place this study in context, this chapter has provided a background to the disease of stroke. The next chapter will begin with a brief summary of the major physical, medical and psychological outcomes of stroke thereby setting the framework for the introduction of cognitive deficits post-stroke. The chapter will then lead to a discussion on attention deficit which is the area of cognition that is the main focus of this study. Chapter three will begin with an overview of the historical and current significance of cognitive rehabilitation followed by a summary of the literature on attention deficit after stroke and then the evidence for rehabilitation including Attention Process Training (APT), will be summarised and analysed. Finally, a critique of research conducted into cognitive rehabilitation will be conducted with some responses to those problems identified in this area of research.

*“There is no education like adversity”*

*Disraeli*

## **Chapter 2: Outcomes of Stroke**

Stroke is the most common disabling chronic condition which often results in the loss of independence in a significant proportion of survivors. Furthermore, the consequences of this disease can also have devastating effects on family and friends, a high percentage of whom are required to make significant adjustments to their lives in order to provide care for their loved ones with stroke (Anderson, 1992). Several scales have been developed to determine the neurological impact of stroke such as the National Institutes of Health Stroke Scale (NIHSS; Schlegel et al., 2003), the Canadian Neurological Scale (Cote, Hachinski, Shurvell, Norris, & Wolfson, 1986) and the European Stroke Scale (Hanston et al., 1994). These are often used in combination with health related quality of life (HRQoL) scales, functional scales and outcome assessments to determine recovery and disability after stroke.

Given that the focus of this study is concerning the field of cognition, only a brief overview of the physical, medical and psychological outcomes of the disease will be provided. There will however, be a greater focus on the cognitive problems, in particular attention deficit, that may result from this syndrome. An overview of the major theories of attention that have evolved will then be discussed. More detailed information gathered as part of this study on physical, medical and psychological outcomes of stroke are provided in Appendix B.

### **Overview of Physical Medical and Psychological Outcomes**

The onset of a stroke often results in cognitive, physical, medical and psychological impairment with devastating impact on aspects of daily life often

leading to a lack of social contact or social isolation. The identification of the outcomes of stroke facilitates the management and treatment of the sequelae of this disease and provides a rational basis for clinical guidelines that ultimately aim to reduce the burden of stroke on the individual, his or her family and society. An extensive range of classification systems for measuring stroke outcomes exist including the National Institutes of Health Stroke Scale (NIHSS), the Stroke Impact Scale (Duncan et al., 1999), the Barthel Index (Mahoney & Barthel, 1965), the Functional Independence Measure (Hall et al., 1996), the Modified Rankin Scale (Rankin, 1957), and the London Handicap Scale (WHO ICIDH, 1986) to name some of the more commonly used measures. There are numerous other measures specific to the various domains of impairment whether it is motor, sensory, vision, language, cognition or affect (e.g., the Beck Depression Inventory (Beck, Ward, Mendelson, Mock & Erbaugh, 1961). To provide some insight into the difficulties stroke patients may suffer, the most common impairments that can result from stroke are briefly described in Appendix B.

### **Cognitive Difficulties**

Cognitive difficulties following stroke are present in as many as 50% to 65% of survivors (Ballard, et al., 2003; Bonita, Solomon, & Broad 1997; Donovan et al., 2008; Hackett, Yapa, Parag, & Anderson, 2005; Hochstenbach et al., 1998; Kase et al., 1998; Pohjasvaara et al., 1998; Rasquin et al., 2004; Saxena, 2006; Snaphapaan, Rijpkema, van Uden, Fernandez, & de Leeuw, 2009; Srikanth et al., 2003), however, the real prevalence and natural course of post-stroke cognitive disorders remains unclear as a result of difficulties comparing across studies. Methodological features such as the heterogeneity of sample groups, the inclusion or exclusion of subjects with recurrent strokes, the exclusion of younger

patients and those with aphasia, timing of assessment and selection of those cognitive domains assessed, plus the use of different assessments, all contribute to divergent and inconsistent data.

Hachinski and Bowler (1993) coined the term “vascular cognitive impairment”, the use of which they suggested should include “the identification of cognitive impairment, the recognition of a potential vascular cause, and the establishment of a logical link between them”. It is a term which is commonly used in contemporary literature and will therefore be used in this overview.

Vascular cognitive impairment is far from homogenous and varies according to the location, number, size, and pathophysiology of the brain lesion (Greenberg, 2009; Nys, et al., 2005; Robinson et al., 1986). As Greenberg (2009, p. 214), succinctly puts it “the effects of strokes on cognition likely represents the cumulative effects of location, number, and volume”. Aminah, Normah, and Ponnusamy (2008) speculate, however, that patients with right hemispheric lesions are better able to cope than those patients with left hemispheric lesions because speech and language functions are left intact and the patient is more able to facilitate their recovery through verbal communication and positive self-talk.

There are other variables that have been found to influence vascular cognitive impairment. For example, Saxena (2006) and Zhou et al. (2005) found lesser education to be significantly correlated with higher vascular cognitive impairment levels although in a more recent study, Aminah et al. (2008) failed to find such an association. Increasing age has consistently been associated with vascular cognitive impairment (Aminah et al., 2008; Downhill & Robinson, 1994; Ebrahim, Nouri, & Barer, 1985; Saxena, 2006; Stephens et al. 2005; Tatemichi et al., 1994). Indeed, increasing age was also found to correlate with the progression



of vascular cognitive impairment at 2 years post-stroke (del Ser et al., 2005).

Brodaty et al. (2005) also found a negative association between age and cognitive decline.

Other factors that have been linked with increased risk of vascular cognitive impairment include previous stroke (Mok et al., 2004; Zhou et al., 2005), pre-stroke cognitive decline (Mok et al., 2004; Wagle et al., 2009), high diastolic blood pressure, number of prescribed drugs, hypotension and genetics (del Ser et al., 2005; Wagle et al., 2009). Moyer (2004) found that stroke survivors who were treated with statins for hypercholesterolemia prior to their stroke may be at less risk of vascular cognitive impairment than other stroke survivors. However, to date, research into vascular cognitive impairment is still in its infancy and therefore the research available is somewhat exploratory and in need of further verification.

Vascular cognitive impairment has also been shown to be a significant predictor of outcomes of stroke (Barker-Collo & Feigin, 2006; Ebrahim et al., 1985; McDowd, Filion, Pohl, Richards, & Stiers, 2003; Nurdan, Derya, Demet, Betul, & Caglayan, 2010; Nys et al., 2005; Ozdemir, Birtane, Tabatabaei, Ekukulu, & Kokino, 2001; Saxena, 2006) and quality of life (Mitchell, Kemp, Benito-Leon, & Reuber, 2010). A study of hospital-based stroke patients at three months found improved cognitive impairment, independent of physical impairment, to be positively associated with independent living and functional impairment (Tatemichi et al., 1994a). In a large cohort (n=486) of ischaemic stroke patients, Pohjasvaara, Erkinjuntti, Vataja, and Kaste (1998) also found cognitive decline to have an independent effect on dependence three months after stroke.

Indeed, cognitive deficit in stroke patients has consistently been associated with prolonged length of stay in hospital or rehabilitation settings (Galski, Bruno, Zorowitz, & Walker, 1993; Saxena, Koh, Ng, Fong, & Yong, 2007) as long as three years after the stroke (Pasquin, Leys, Rousseaux, Pasquier, & Henon, 2007). Conversely, a study of Chinese stroke patients found that a shorter stay in hospital was predicted by improved cognitive and self-care factors rather than mobility factors (Man, Tam, & Hui-Chan, 2006).

Vascular cognitive impairment has also been found to have an impact on the emotional state of the patient. Both depression and anxiety post-stroke have consistently been associated with a higher incidence of cognitive impairment (Kauhanen et al., 1999; Robinson et al., 1986; Saxena, 2006; Talelli et al., 2004) and in particular with slowed cognitive speed and poorer verbal memory (Barker-Collo, 2007). However, the effects of vascular cognitive impairment have been found to be even more far-reaching. For example, Patel, Coshall, Rudd, and Wolfe, in their 2002 population-based study found cognitive impairment to be significantly associated with death or disability at three and four years post-stroke.

In some instances the post-stroke cognitive impairments are progressive in nature (Sachdev, Brodaty, Valenzuela, Lorentz, & Koschera, 2004), and reach the level of dementia in 5-7% of patients during the first six months increasing to 20-25% by five years. However, although post-stroke cognitive deficits can diminish during the first year (Rasquin et al., 2004), it is not unusual for late spontaneous and rehabilitative-related recovery (Cicerone et al., 2005; Moss & Nicholas, 2006).

## **Specific Cognitive Domains**

The relationship between specific cognitive domains and outcomes after stroke has also been investigated. The type of cognitive shifts demonstrated in stroke patients include varying degrees of impairment of executive functioning, memory, visuospatial and visuo-perceptual abilities, problem solving skills, orientation, attention, rate of information processing, anosognosia, and agnosia, singularly or a combination of two or more of these problems (Alladi, Meena, & Kaul, 2002; Tatemichi et al., 1994a). With increasing recognition of the importance of the role of cognitive function on treatment outcomes, the need for ecologically valid cognitive measures is becoming an increasingly useful requisite.

### **Memory**

Memory impairment is a common cognitive deficit that occurs following stroke. In a systematic review of the literature, Snaphaan and de Leeuw (2007) found that the incidence of memory impairment following stroke is 50% within the acute stage, reducing to 12% at 6 months post-stroke. In their study sample Riepe, Riss, Bittner, and Huber (2004) found 74% were impaired on a brief memory impairment test administered within 24 hours of stroke onset, and used this data as the rationale for providing memory specific rehabilitation for those patients. Other authors found a prevalence rate of 20% for memory impairment at 3 months post-stroke with varying degrees of severity of memory difficulties (Ferro & Martins, 2001). In their sample of 60 patients with ischaemic stroke, Usolteva, Dudarova, and Levin (2009), found a correlation at 6 months post-stroke between delayed verbal recall and logical memory scores with functional outcome (measured by the Rankin Scale). While different studies report the

presentation of memory problems at varying stages post-stroke, what does seem apparent, however, is that generally the incidence of memory impairment following stroke reduces over time as recovery occurs.

According to Cicala (1999), memory difficulties are more common when the stroke has affected the temporal lobes or structures near the thalamus. In their case study, Schott, Crutch, Fox, and Warrington (2003) also found thalamic involvement for memory impairment, although the deficit was in the verbal modality only. In a meta-analysis of memory impairment following right hemispheric stroke, the authors found evidence of verbal and nonverbal memory impairment thus challenging commonly held assumptions that the right hemisphere stroke produces nonverbal memory deficits only (Gillespie, Bowen, & Foster, 2006). Given the complex and extensive neural network involved in the memory system, Lim and Alexander (2009) suggest that the anatomical bases for impairments are most likely, quite variable.

### **Executive Functions**

Executive functioning is an umbrella term used to encompass a set of complex behaviours involved in the initiation, planning, sequencing, organisation, and regulation of behaviour. Executive functions consist of distributed interactive and overlapping networks and while mediated by structures of the frontal lobes, are also believed to involve other cortical and subcortical regions of the brain (Elliott, 2003; Sohlberg & Mateer, 2001; Stein, Harvey, & Macko, 2009).

Executive functions, are one of the most frequent domains of cognitive impairment following stroke. There is speculation that deficits of executive function are more prevalent in the early stages and indeed most of the available data has derived from studies conducted at three months post stroke (Au et al.,

2006; Ballard, Rowan, Stephens, Kalaria, & Kenny, 2003; Leskela et al., 1999; Stephens et al., 2004). Prevalence rates of between 40% and 52% have been found (Blake, McKinney, Treece, Lee, & Lincoln, 2002; Pohjasvaara et al., 2002). In a hospital-based study, Zinn, Bosworth, Hoenig, and Swartzwelder (2007), found that the rate of executive dysfunction in the acute stage of stroke was about 50% in 47 confirmed stroke patients, although the study was limited by the homogeneity of the sample, who were all relatively high functioning prior to the stroke, and had mild strokes.

Not surprisingly, stroke survivors with poor executive functioning have been found to have related to poorer outcomes from stroke (Barker-Collo, Feigin, Parag, Lawes, & Senior, 2010; Zinn et al., 2007). Mok et al. (2004) found impaired executive function but neither memory nor language impairment to be associated with performance of complex activities of daily living (ADLs) at 3 months post-stroke. In another study of patients with mild vascular cognitive impairment, Stephens et al. (2005) also found a decline in basic ADLs over six years was associated with deficits of executive functioning as well as impaired perceptual and spatial skills. Furthermore, those experience ongoing symptoms, despite preservation of other cognitive domains, often have difficulty re-entering the work force and indeed, adapting back into society in general (Caplan, 2005; Ownsworth & Shum, 2008).

Most studies that have investigated stroke site location relative to executive dysfunction, have found an association with the prefrontal lobe (Carey et al., 2008), which is unsurprising given that this part of the brain mediates higher cognitive thinking and behaviour (Alvarez & Emory, 2006). However, executive dysfunction has been reported in stroke studies involving other areas of the brain

(Carrera, Michel, & Bogousslavsky, 2004; Espay & Jacobs 2010; Vataja et al., 2003) thereby corroborating to some extent the evidence that executive function is mediated by areas of the brain other than the frontal lobes.

Research has also identified particular domains of executive functioning as being vulnerable to vascular disease or injury. For example, selective attention and cognitive flexibility, set shifting, and response inhibition have consistently yielded results that indicate a decline in performance when compared to base-line measures or controls (Bombois et al., 2007; Garcia, Haron, Pulman, Hua, & Freedman, 2004; Greve, Bianchini, Hartley, & Adams, 1999; Jokinen et al., 2005; Lu & Bigler, 2000; Murphy et al., 2007; Prins et al., 2005; Sachdev et al., 2003; Su, Wuang, Chang, Guo, & Kwan, 2006; Tang et al., 2009; Verdelho et al., 2007; Zinn et al., 2007). Other specific areas of executive functioning compromised in a stroke population are self-regulation (Amirian et al., 2010; Ownsworth & Shum, 2008), perseveration and problem solving (Su et al., 2006), working memory, and processing speed (Zinn et al., 2007).

Research has consistently demonstrated an association between executive dysfunction and depression (Channon, Baker, & Robertson, 1993; Mast, Yochim, MacNeil, & Lichtenberg, 2004; Pohjasvaara et al., 2002; Tang et al., 2009; Vataja et al., 2003). Narushima, Paradiso, Moser, Jorge, and Robinson (2007) found that antidepressants had a positive effect in both recovery and preventing further decline of executive functions in a stroke population. However, the sample in this study was comprised of high socio-economic people thereby restricting the scope to which their findings could be generalised.

## **Attention Deficit**

Of all the cognitive domains, attention appears to be the most frequent and prominent neuropsychological area affected in a stroke-related population (Marshall, Grinnell, Heisel, Newall, & Hunt, 1997; Rao, Jackson, & Howard, 1999) with rates of up to 46% to 92% reported in acute survivors (Hochstenbach, et al. 1998; Hermann et al., 2008; Hyndman, Pickering, & Ashburn, 2008; Lesniak, Bak, Czepiel, Seniow, & Ionkowska, 2008; Nys, 2005; Patel, et al. 2002; Tuhim, 1993). The implications of an attention deficit are potentially far-reaching given that attention is a precursor to other cognitive domains such as memory and language, and influences other executive functions (Ben-Yishay, Piasefsky & Rattock, 1987; Chen, Thomas, Glueckauf, & Bracy, 1997; Uomoto, 1992; White, 1992).

Some studies have investigated and distinguished impairment across the different components of attention. For example, Marshall, Grinnell, Heisel, Newall, and Hunt (1997) found significant impairment of divided attention in their sample of stroke patients one year post-onset compared to non-stroke controls and in a more recent study of older stroke patients, divided attention as well as alternating attention were areas of poorer performance compared to the performance of non-stroke patients (McDowd et al., 2003).

As with other areas of cognition, impaired attention abilities are also reported to influence post-stroke rehabilitation outcomes (Barker-Collo & Feigin, 2006; Hyndman, et al., 2008; Sohlberg & Mateer, 2001). For example, Robertson, Ridgeway, Greenfield, and Parr (1997) found that sustained attention measured at two months post-stroke significantly predicted functional status in individuals at two years post-stroke. Similarly, Nys (2005) found that cognitive

impairment at approximately one week post-stroke was a significant predictor of quality of life at six months post stroke (as measured by the Stroke Impact Scale-SIS), with visual hemi-attention being a major contributing factor ( $\beta = -.29, p < .01$ ). Indeed, attention as a key neuropsychological component in learning new motor skills, particularly in the early stages of learning has been consistently shown. For example, in one study, Stapleton, Ashburn, and Stack (2001) found that attention deficits were common among acute hospitalised stroke patients, and reported an association between distractibility, selective attention, balance, and functional impairment. In another study investigating the associations between attention, balance, function and falls in 48 community-based individuals with stroke, Hyndman and Ashburn (2003) found significant correlations between ADLs, balance and sustained and divided attention.

There is also an increasing bank of knowledge linking attention with social functioning abilities. McDowd et al. (2003) found that impaired attention had a negative impact on physical functioning and social outcomes in their sample of 55 older adults with ischaemic stroke. This area is particularly important in the field of neuropsychological rehabilitation where the relearning of appropriate social skills is often an objective (Addington et al. as cited in Combs & Gouvier, 2004; Bellack, Gold, & Buchanan, 1999; Penn & Combs, 2000).

One manifestation of attention problems which may be observed following stroke is hemineglect, also known as unilateral neglect, hemispatial neglect, spatial neglect or hemi-inattention. It is characterised by the “failure to report, respond or orient to novel or meaningful stimuli presented to the opposite side of a brain lesion, when this failure cannot be attributed to either sensory or motor defects” (Bailey & Riddoch, 1999; Churchland, 1989, p230). Most of the



cognitive dysfunction produced by hemi-inattention is because of an asymmetric distribution of attention, either with respect to extrapersonal space or to an object being viewed (Marshall, 2009). Hemineglect is a multimodal syndrome, usually resulting from a stroke and can affect, to varying degrees, visual, auditory, somatosensory and motor modalities in different combinations and severity (Robertson & Marshall, 1993; Stirling, 2002), depending on the lesion location (Mesulam, 1994). Visual neglect is the most common form of the disorder although neglect of the limbs opposite the brain-damaged side is also frequently seen. Both conditions for some result in serious rehabilitation difficulties (Hier, Mondlock, & Caplan, 1983; Ogden, 2005; Wade, Wood, & Hewer, 1988). While the syndrome usually manifests contralaterally, ipsilesional neglect has been reported (Kim, et al., 1999; Schwartz, Barrett, Kim, & Heilman, 1999). In their systematic review of 30 studies of contralesional unilateral spatial neglect, Bowen, McKenna, and Tallis (1999) found that neglect occurred predominantly with right brain damage as opposed to left brain damage. Indeed, Stirling (2002), purports that left-sided hemineglect is far more common and more severe, a finding that has been consistently observed in much of the previous literature (Heilman, Watson, & Valenstein, 1993, 2003; Hildebrandt, Spang, & Ebke, 2002; Kinsbourne, 1999; Vallar, 1998) giving rise to the hypothesis that the right hemisphere plays a more dominant role in the attention process (De Renzi, Faglioni, & Scotti, 1970). However, other explanations for this phenomenon, view attention as a result of hemispheric physiological arousal or activation (Heilman, et al., 1993) or that attention is a directional phenomenon with each hemisphere having greater influence to the opposite side of space (Kinsbourne, 1977). In contrast, Ogden (1985) found no difference in lateralisation of

hemisphere for hemineglect. Reported incidence rates of hemineglect range from 10% to as high as 95%, with variation again, largely due to different methodologies (Bailey & Riddoch, 1999; Bowen, McKenna, & Tallis, 1999; Lopes, Ferreira, Carvalho, Cardoso, & Andre, 2007; Ringman, Saver, Woolston, Clarke, & Adams, 2004; Schenkenberg, Bradford, & Ajax, 1980; Zoccolotti et al., 1989).

Attention is also a key link to the re-learning of motor skills and successful functional outcomes. It is a very basic function upon which other neurological/cognitive functions are predicated and as such is a determinant of positive outcomes in other cognitive areas (Barker-Collo, 2009; Hyndman et al., 2008). It is this central role to recovery from stroke that provides the impetus and incentive for further discussion of attention in this study.

### **Theories of Attention**

Attention is considered by many to be a precursory foundation for other more complicated cognitive skills such as memory and language skills (Cowan, 1995; Fisk & Schneider 1984; Langdon 2002; Whyte, 1992; Wood as cited in Raskin & Mateer, 1994), and as such it may be reasonable to assume that remediation of attention deficit needs to take place in order for other cognitive domains to improve.

The upsurge of scientific research into attention in recent decades has revealed a phenomenon that is so nebulous and complex that the common consent is that most definitions of this cognitive domain are now inapt, with some even arguing that a particular definition or description of attention is impossible (Leon-Carrion, 1997; Manly, 2003). Despite those sentiments, many definitions of

attention have nevertheless been articulated since William James wrote about it in 1890 when he stated:

It is the taking possession by the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought.

Focalisation, concentration, of consciousness are of its essence. It implies withdrawal from some things in order to deal effectively with others... (cited in Jagadeesh, 2006, p. 195)

Although James identifies that there is a relationship between attention and consciousness, even today the full extent of this relationship remains unclear and elusive (Posner, 1994; O'Regan & Noe 2001). Research at the neuronal and molecular levels to understand the mechanistic basis of attention and consciousness is required in order to gain a clearer understanding of the relationship between these two distinct yet intimately close processes.

The dominance of behaviourist theories in psychology in the early twentieth century meant a long delay before the first modern theory of attention developed. In 1958 during the 'cognitive revolution' (Harre, 2002), Broadbent presented the first comprehensive model of attention based on the human information processing approach. This work was largely built on and explained the dichotic listening experiments conducted by Cherry in 1953. Broadbent's single-filter, limited capacity model proposed that we could attend to only one stimulus at a time because target stimuli receive priority over concurrent non-target stimuli (Johnson & Wilson, 1980). That is, after an early low level perceptual analysis based on physical attributes, information is passed into a temporary sensory buffer where unattended information is simply lost and the one selected information is

then ‘evaluated’ via a filtering process that determines whether or not that information is passed into consciousness (Moray, 1995). (See Figure 1).

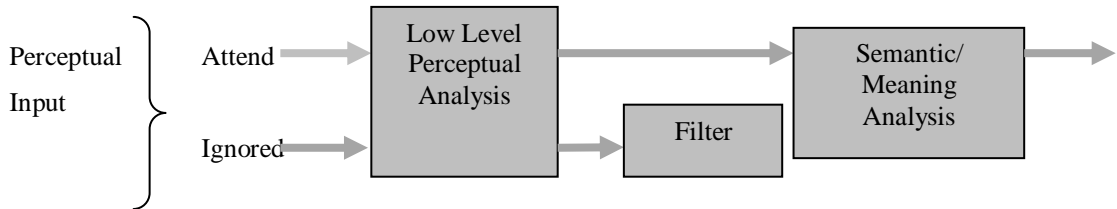


Figure 1. Early Selection Theory of Attention (Broadbent, 1957)

In a modified Early Selection Theory of attention, Treisman (1960) proposed a two-channel model of selective attention thus expanding on Broadbent's idea by proposing that non-attended stimuli are not completely filtered out but attenuated according to their subjective importance (See Figure 2). Treisman's model also emphasised the role that priming can have on psychological processes.

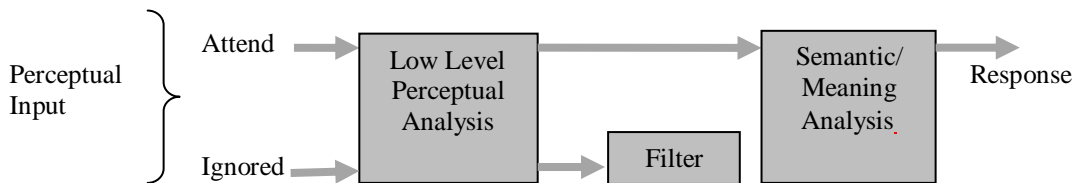


Figure 2. Modified Early Selection Theory of Attention (Treisman's Attenuator Theory)

Broadbent and Treisman's models were the first of the ‘Early Selection’ models in which the locus of the bottleneck for processing information occurs before the mind can analyze its semantic content i.e. at the perceptual level. However, subsequent theories of ‘Late Selection’ as developed by Deutsch and Deutsch (1963), and Norman (1968) purported that all stimuli is analysed with further processing afforded to pertinent stimuli just before entry into longer

lasting memory, effectively placing the locus of the bottleneck later in the process continuum. (See Figure 3).

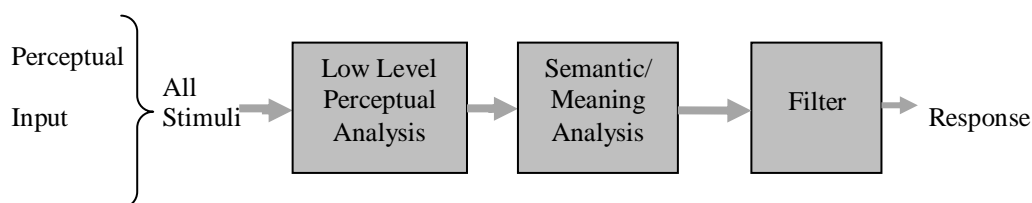


Figure 3. Late Selection Theory of Attention

At a time when the study of attention shifted from auditory to visual tasks, partly because of the more precision in controlling visual stimuli, Kahneman (1973) introduced his attentional resource theory that combined motivational, attentional and arousal processes. He argued for a finite cognitive capacity to devote to a task with each task requiring different capacity use. The number of activities which can be performed is determined by the capacity each requires and is controlled by a “central processor” that adjusts and allocates attention accordingly. He predicted that as a skill becomes more automated, it becomes more streamlined and takes up less of one’s attentional capacity. However, Kahneman’s model did not address a person’s ability for divided attention. It was Allport’s (1972) model of attention that provided a theoretical basis for this type of attention. This model argued for several separate modules for different kinds of input stating that attention can be divided between tasks that use different skills (e.g. speech and pattern recognition), although not between similar tasks where there is competition for resources from the same module (e.g. problem solving and decision making).

However, the selectivity models were limited in that they did not address the processing of information beyond the perceptual level. This lack of a more comprehensive model of the attention process was undertaken in 1974 when

Baddeley proposed an influential model of working memory which began to address the more comprehensive nature of attention. Baddeley's model included a 'central executive' that is primarily attentional in nature and responsible for directing attention to and from either of its two 'slave systems', the phonological loop (verbal stimuli) and the visuo-spatial sketchpad (visuo-spatial stimuli) while attention is temporarily shifted to other stimuli (See Figure 4). In 2000, Baddeley subsequently added a fourth slave system, the episodic buffer to this model. The purpose of the episodic buffer is to bind together all of the information from the other components of working memory with information about time and order. This assists in preparing memories for storage in episodic long term memory (Baddeley, 2000).

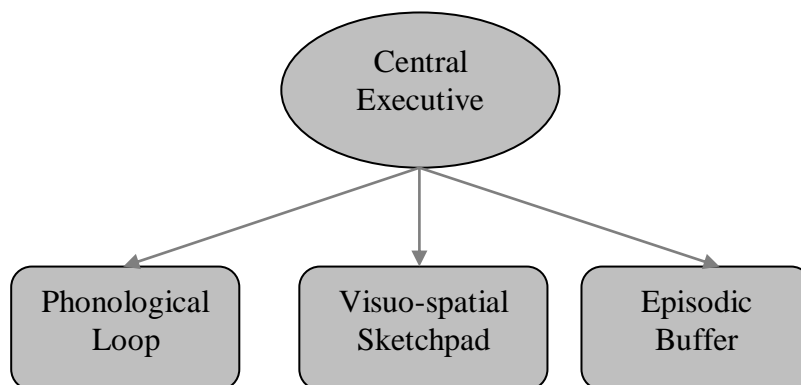


Figure 4. Baddeley's Model of Attention (Baddeley, 2000).

Since the early models of attention were introduced in the 1950's, the knowledge of attention has grown exponentially and many contemporary models not only attempt to integrate the early and late approaches but also extend their theory into the fields of neurophysiology, neuropsychology and computational modelling. In the 1960s, neuroscientist, Robert Wurtz, when recording electrical signals from the brains of macaques completing attentional tasks, was the first to demonstrate a direct neural correlate with a mental process, namely enhanced

firing in the superior colliculus (Goldberg, 2007). In 1935, John Stroop had published his work on the now famous Stroop Colour-Word task, although in the behavioural climate of the time, this work had little impact in the world of psychology. Stroop's work was 'rediscovered' in the 1960s and subsequently the Stroop effect has become one of the most valuable tools of cognitive psychology providing a fuller understanding of how attention works (McLeod, 1991). More recently, the utilisation of brain imaging technology has provided a functional anatomy of the human attentional system and now most researchers conceive attention as a system in which sequential processing occurs in stages that involve different brain systems (Lezak, 2004). This multi-modality and multi-resourced process explains, at least in part, why there is a lack of a common understanding of this phenomenon.

However, none of the theoretical models have been produced for clinical purposes to aid in the evaluation and remediation of attention deficit. Sohlberg and Mateer (1987, 1989), provide a clinical model which incorporates current theoretical thinking and is based on task performance, errors and subjective complaints by individuals with brain injury. The authors identify five different types of attention which are hierarchical in nature. Sustained attention is considered the least complex component followed by selective attention, then alternating attention and finally divided attention. The components of this model are summarised in Table 1.

Table 1  
*Sohlberg and Mateer's (1989) Clinical Model of Attention*

Focused Attention	The ability to respond discretely to specific visual, auditory or tactile stimuli.
Sustained Attention	The ability to maintain a consistent behavioural response during continuous and repetitive activity.
Selective Attention	The capacity to maintain a behavioural or cognitive set in the face of distracting or competing stimuli and therefore incorporates the notion of freedom from distractibility.
Alternating Attention	The capacity for mental flexibility that allows individuals to shift their focus of attention and move between tasks having different cognitive requirements.
Divided Attention	This is the highest level of attention and it refers to the ability to respond simultaneously to multiple tasks or multiple task demands.

This chapter has provided an overview of the outcomes of stroke with a more detailed review of the phenomenon of attention deficit following this disease. This was followed by a summary of the evolution of attention theory. The next chapter (Chapter 3) will begin with an explanation of cognitive rehabilitation and a summary of the literature on its efficacy. This will be followed by a review of the literature for cognitive rehabilitation narrowing to an investigation and discussion on the evidence for rehabilitation of attention, including APT. The closing stages of the chapter will provide a discussion of what the barriers to trials in cognitive rehabilitation are and how they might be overcome to ensure future high quality research into this area of health rehabilitation.



*“A journey of a thousand miles begins with a single step”*

*Lao Tzu*

### **Chapter 3: Cognitive Rehabilitation**

There are a multitude of definitions to describe cognitive rehabilitation and Prigatano’s (2005) explanation that it “refers to non-pharmacological and non-surgical intervention by healthcare providers that aim to improve or restore problem solving capabilities of brain function that have been disturbed by a known or suspected brain lesion(s)”, (cited in Halligan & Wade, 2003, p3), provides an indication of the diversity and complexity of this field of therapeutic knowledge. Gaylins’ (1977, p. 2) definition that “...the rehabilitation of a head-injured person is, principally, a task of aiding that person to speak and act in a way which optimises his or her adaptability and sense of belonging”, also alludes to the eclectic nature of the discipline. Indeed, Sohlberg and Mateer (2001), advocate for a wider appreciation of the aims of cognitive rehabilitation to include personal, emotional and social contextual variables in treatment plans and goals. Nevertheless, within this holistic framework, there is a strong emphasis on the retraining or alleviation of problems caused by cognitive “deficits in attention, visual processing, language, memory, reasoning/problem solving, and executive functions” (Sohlberg & Mateer, 1989, p. 4), as being fundamental to cognitive rehabilitation. Cicerone et al. (2000) provide a more prescriptive definition of cognitive rehabilitation as;

a systematic, functionally-orientated service of therapeutic cognitive activities, based on an assessment and understanding of the person’s brain-behaviour deficits. Services are directed to achieve functional changes by (1) reinforcing, strengthening, or re-establishing previously

learned patterns of behaviour, or (2) establishing new patterns of cognitive activity or compensatory mechanisms for impaired neurological systems. (p. 1696)

Tasks designed to reinforce or re-establish previously learned patterns of behaviour or to establish new compensatory mechanisms for impaired neurological systems are the mainstay of this approach. Cognitive rehabilitation can be regarded as a particular aspect of the broader field of neuropsychological rehabilitation, the latter being a method of restructuring lives in a social context that does not represent training of cognitive abilities only but also includes addressing emotional and psychosocial problems, particularly as difficulties in these areas can exacerbate cognitive difficulties (Wilson, 2008) .

The discipline of cognitive rehabilitation has been practised for well over a century with its origins lying in language recovery programmes developed by Broca in the mid-1800s, for people with speech disorders. Modern cognitive rehabilitation is, however, largely attributable to the work of physician Kurt Goldstein (1942), who treated brain-injured German soldiers during World War I and recognised the importance of working with both cognitive and personality processes simultaneously. Psychologist and physician, Alexandria Luria and Neuropsychologist, Oliver Zangwill are two other important figures in the evolution of cognitive and neuropsychological rehabilitation. Indeed, Luria has become universally known as one of “the founding fathers of neuropsychology” (Christensen, 1996, p. 279; Goldberg, 2009, p. 10). In her review of the history of cognitive and neuropsychological rehabilitation, neuropsychologist, Freda Newcombe, (2002), refers to Zangwill as the “Great Precursor” of clinical and neuropsychological rehabilitation. During World War II when working with

victims of missile wounds, Alexander Luria acknowledged the presence of functional systems mediating cognitive functions located in different brain regions (Ponsford, 2004), while it was Oliver Zangwill (1947), who first identified three main approaches to rehabilitation including compensation, substitution and direct training; methodology that continues to be widely practised in neuropsychology today (Wilson, 1997). Indeed, the efforts of these two practitioners provide much of the foundation and rationale for contemporary cognitive and neuropsychological rehabilitation (Boake, 2003).

In the two decades spanning the 1980's and 1990's, against a backdrop of consumer health reforms, an exponential amount of research into recovery from brain injury took place and there now exists a substantial body of evidence to support the effectiveness of cognitive rehabilitation for the improvement of cognitive deficits following trauma to the brain (Cappa et al., 2005; Cicerone et al., 2000; Kreutzer, 1999; Park, Proulx, & Towers, 1999; Ponsford, 2004; Sohlberg & Mateer, 2001). Additionally, technological advancement has accelerated improvement and growth of cognitive rehabilitation with published research studies and neuroimaging of both humans and animals having contributed to this store of data (Cicerone et al., 2005).

Knowledge of neuroplasticity, (the process by which neurons create new connections among themselves), has risen dramatically since the notion was first introduced in the 1950's by researchers who found that environment had an effect on the structure and function of the animal brain. Evidence from functional Magnetic Resonance Imaging (fMRI) studies provides compelling evidence of the human brain's ability for reorganisation of structure and function (plasticity) following pathology (Benton & Tranel, 2000). Furthermore, it is now known that

neuroplasticity is not the prerogative of the young but is a phenomenon that can occur throughout the human lifespan, albeit more slowly as the organism ages. Using functional Magnetic Resonance Imaging (fMRI), a small number of researchers have found evidence of neural plasticity when testing for the efficacy of cognitive rehabilitation in patients with Traumatic Brain Injury (TBI; Kim, Yoo, Ko, Park, & Na, 2009; Laatsch, Thulborn, Krisky, Shobat, & Sweeney, 2004). Recent insights into the neurobiology of repair after stroke also provide evidence for the guidance of optimal prescription of therapeutic interventions (Cramer & Riley, 2008). These preliminary findings of brain reorganisation following brain trauma present potentially major implications for cognitive rehabilitation. In principle, if neural circuits can be modified after injury, then it is feasible to assume that functional change may also occur (Kolb & Gibb, 1999; Sohlberg & Mateer, 2001).

### **Approaches to Cognitive Rehabilitation**

The management of cognitive impairment is multi-faceted with different problems presenting at various stages requiring different approaches. This section will present an overview of the models of the more common approaches used in cognitive rehabilitation today.

Models provide a rationale and a direction for increasing the specificity and efficiency of clinician-targeted interventions. Bracy (1986), states "... a theory of brain functioning and of rehabilitation is necessary for assessment diagnosis, treatment planning, goal setting and providing therapy. Without a unifying and guiding theoretical framework, our efforts would not amount to much more than random stabs in the dark". In his description of cognitive rehabilitation as "an assortment of procedures to improve or restore a diverse collection of abilities and

skills”, Wood (1990, p3), identified four main approaches for those procedures as being; restorative, compensatory, environmental, and behavioural. These approaches had been alluded to much earlier when Zangwill (1947) proposed substitution (therapy in which a substitute substance is used), restoration and compensation as the three activities that constitute cognitive rehabilitation. Mateer, Kerns, and Eso (1996), identified three broad categories for the management of attention and memory impairments in children following traumatic brain injury. The first approach is externally focussed in that it attempts to alter aspects external to the individual. The second approach is designed to improve or restore cognitive abilities, and the third category involves the training and implementation of procedures or strategies that compensate or lessen the functional impact of cognitive deficits. The second two approaches can be considered internally based as they are designed to directly change the individual’s abilities and/or behaviours. The four approaches widely utilised in cognitive rehabilitation are:

### **Restorative Therapy.**

Restorative Therapy is based on the theory that systematic repetitive exercises can restore lost functions which have been identified by neuropsychological or psychometric assessment (Cicerone & Tupper, 1990; Coelho, De Ruyter, & Stein, 1996; Sohlberg & Mateer, 1989). This approach is most effective when the remediation has been tailored to address the pattern of deficits of the particular individual. Such exercises are available in the Attention Process Training programme which is based on a clinical model of five components of attention; namely focussed, sustained, selective, alternating and divided attention (Sohlberg & Mateer, 1989). An example of a task designed to

improve sustained attention requires listening for target letters or numbers on an attention tape and pressing a buzzer when the target is identified. Pretesting identifies the appropriate level at which therapy starts and the parameters of the exercises (e.g., complexity, quantity, speed of presentation, or the amount of cueing given) are incrementally modified in accordance with 'mastery' of each exercise, thereby extending the goal of therapy (Ponsford, Sloan, & Snow, 1995). Thus, the more basic processes of attention are trained before attempting the more complex processes. The goal of restoration therapy is for the patient to perform the activity in the same way and using the same functions whether it is cognitive, motor or perceptual (Shiel, 2003).

#### **Compensatory Therapy.**

Compensatory therapy in contrast, strives to improve functioning by recruiting relatively intact cognitive processes to fulfil the role of impaired functions or by using external prosthetic aids (e.g., diaries, and social supports) to compensate for the loss of function (Lieberman, 2008). Visual mnemonics is a common effective strategy used in the treatment of impaired verbal memory (Wilson & Evans, 2003). An example of a verbal mediation strategy often used is the PQRS (Preview, Question, Read, State, and Test) method which requires deeper processing of the material. The assumption underlying this technique is that the broader encoding will enhance later recall (Barker-Collo & McCarthy, 2007). Generally, restorative techniques are used to focus on the patient's weaknesses, while compensatory techniques are often driven by a person's strengths.

### **Environmental Therapy.**

Environmental therapy in the acute stage of rehabilitation involves modification of the individual's environment with much of the focus being on safety. In later stages of recovery the therapist often engages with family members and others in order to tailor the environment so as to facilitate adaptive learning. For example, individuals with significant memory or executive function deficits may benefit from an environment that is clutter-free and has been manipulated with cues such as labelling, a central message centre, and posting checklists reminders, to initiate a certain behaviour, or clocks and calendars for orientation purposes (Mateer, 2005).

### **Behaviour Therapy.**

Behavioural approaches are advocated by some authors who have found some success when working with neurologically damaged patients (Cotrell, 1997; Goldstein & Ruthven, 1983; Wilson, Herbert, & Shiel, 2003). Behaviour therapy encompasses a diverse range of procedures to attain its overall aim which is the modification of behaviour. In patients who have been cognitively compromised, task-specific, simple-response behavioural techniques such as token reinforcement have been shown to be effective (Goldstein & Ruthven, 1983). For example, in an auditory listening task, the person receives a token for each correct response and is fined a token for each incorrect response. All participants in a study using this procedure demonstrated improved performance of sustained attention and more efficient information processing ability (Wood, 1986).

However, a major challenge for any rehabilitation specialist is to motivate the patient to engage in therapy and when working with neurologically or psychologically compromised patients, this goal can be particularly difficult to

achieve (Marin & Chakravorty, 2005; Prigatano & Fordyce, 1986). Motivation is often a problem of executive dysfunction as patients may lack insight into their condition (anosognosia), and because of this reason these individuals are sometimes excluded from cognitive therapy. However, such problems do not necessarily constitute a barrier to the effective use of behaviour therapy (Sohlberg & Mateer, 2001). Indeed, Craighead, Kazdin, and Mahoney (1976), Lietenberg (1976), and Goldstein and Ruthven (1983), all found behaviour therapy to be effective with people who present with aggression, anxiety, depression or who are non-compliant (Horton & Howe, 1981; Turner, Green & Braunling-McMorrow, 1990), difficulties that are frequently present, in varying combinations, in people who have suffered TBI.

However, many aspects of rehabilitation modes of therapy overlap and it is not unusual for therapists to adopt more than one approach (Ylviskaer & Feeney, 1998). Indeed, Wilson (2005) suggests that because models of cognitive rehabilitation are always evolving and often draw on many fields of psychology, it would be prudent for clinicians to utilise more than one approach. Multiple approaches may become particularly pertinent as therapy progresses, and the needs of the individual change with improved functioning, resulting in the renegotiation of the goals of rehabilitation.

Nonetheless, a number of psychologists are critical of some neuropsychological and cognitive rehabilitation models for the lack of a relationship with daily-tasks and functional capacities. Wilson (1997, 2008), for example, disapproves of cognitive neuropsychologists who typically treat the impairment rather than the disability and asserts that any reduction in impairment needs to be demonstrated in a reduction of disability if it is to be of any use to the



patient. The World Health Organisation defines impairment as “a limitation of a physical or mental function because of disease or injury” and disability as “the loss of the ability to participate in some activity because of an impairment” (Schefft, Malec, Lehr, & Kanfer, 1997, p238). The National Institute of Neurological Disorders and Stroke (NINDS) impressed the need for a paradigm shift from one focussing on diagnosis and descriptive analysis of neuropsychological assessment and cognitive impairments towards a linking of diagnosis and interventions that improve functional outcomes (Paolucci, et al., 1996).

Similar sentiments have been conveyed by other researchers (Cicerone, et al., 2005; Rohling, Faust, Beverly, & Demaki, 2009; Uzzell, 2000). Prominent pioneers of the holistic approach to neuropsychological rehabilitation include Goldstein (1942), Luria (1963), Ben-Yishay (1975), Diller (1976), and Prigatano (1986). In addition to the battery of therapeutic interventions for neurologically impaired patients, the provision of psychotherapeutic interventions as an important key ingredient to assisting both patients and their families deal with their personal emotional distress is widely recommended (Christensen, 2000; Leathem & Christianson, 2006; Prigatano and Ben-Yishay, 1999; Sohlberg & Mateer, 2001).

Other factors can also influence the success of the intervention. For example, many clinicians and researchers in the field of neuropsychological rehabilitation advocate for an individualised programme designed and managed by a multi-disciplinary team. This tailored approach considers personal aspects such as time post-injury/onset, the preferred learning style, cognitive status, emotional and personality factors both before and after injury, the type of injury

sustained, co-morbidity, motivation, age and education, and the role of the family and significant others including employers (Christensen & Uzzell, 2000). It is also important that the characteristics of the treatment programme incorporate duration, intensity, frequency and the setting of the programme.

As neuropsychological rehabilitation is becoming a more standard component of care after brain injury, ongoing research to achieve better understanding of how cognitive rehabilitation interventions improve recovery and functioning, is needed. The following section will provide a summary of the major publications available in the literature that have attempted to investigate the quality of cognitive rehabilitation.

### **Efficacy of Cognitive Rehabilitation**

The steep rise in neuropsychological research and cognitive rehabilitation has generated a large number of reviews and analyses evaluating the effectiveness of its use with individuals who have suffered neurological insult, over the last decade. Overall the information that follows demonstrates that while cognitive rehabilitation is not without its critics or inconsistencies there has nevertheless been an accumulation of evidence suggesting that it does have value in the rehabilitation process.

Influential investigations that have produced positive findings for cognitive rehabilitation date back to 1999, when the European Federation of Neurological Societies (EFNS), Task Force on Cognitive Rehabilitation was established for the purpose of producing recommendations for neurological practice. In their review and subsequent update of the literature on cognitive rehabilitation, the EFNS found evidence, albeit limited, to support cognitive rehabilitation for visual neglect and apraxia after stroke, attention deficit after TBI, and memory

dysfunction after either TBI or stroke (Cappa et al., 2003; Cappa et al., 2005). In the same year Carney et al. (1999) in their systematic review of the literature, also found evidence for a domain-specific effect, namely memory for cognitive rehabilitation.

Further evidence promoting cognitive rehabilitation was published in Park and Ingles' (2001) meta-analysis of the efficacy of 30 studies using interventions for attention disorders following brain damage. Unlike systematic reviews, a meta-analysis requires that effect size for each study is included and as such provides statistically rigorous data integrated from across studies (Egger & Smith, 1997). The authors' analysis concluded that acquired deficits of attention are treatable using specific-skills training, although they found that the learning that occurred did not generalise to tasks outside those used in training (Park & Ingles, 2001).

Furthermore, in 2000, the American Congress of Rehabilitation Medicine's (ACRM) published a comprehensive review of the literature of evidence-based cognitive rehabilitation for the purpose of providing evidence-based practice guidelines for persons with brain injury (Cicerone et al. 2000). This review was updated in 2005 (Cicerone et al. 2005). These reports evaluated the results of studies of cognitive rehabilitation versus alternative treatments and found that approximately 79% of the comparisons demonstrated a benefit of cognitive rehabilitation over the alternative treatment (Cicerone et al. 2005). Indeed, such was the influence of the review, that The Brain Injury Interdisciplinary Special Interest Group of the ACRM published practice guidelines with an emphasis on the findings of these two reviews. In the 2005 review, Cicerone and his colleagues declared that "there is now a substantial body of evidence

demonstrating that patients with TBI or stroke benefit from cognitive rehabilitation” (p. 1689).

A meta-analysis of the systematic reviews of Cicerone et al. (2000, 2005), was conducted by Rohling et al. (2009) which yielded reasonably positive conclusions. The analysis of the 97 studies found the effects of cognitive rehabilitation on global cognitive function to be relatively modest yet statistically significant with treatment effects moderated by time post-injury, type of brain injury, and age. The strongest evidence to emerge from the analysis was for the effectiveness of attention training after TBI and cognitive training of language and visuospatial deficits for aphasia and neglect syndromes after stroke. It was also concluded that visuospatial training tended to improve performance in other cognitive domains. However, the authors did not find any evidence to support memory training as being effective or any treatments that attempted to improve holistic cognitive problems. As a result of this analysis the authors went on to make recommendations for clinicians to focus their efforts on direct cognitive skills training (Rohling et al., 2009).

A number of recent studies have attempted to establish the effectiveness of cognitive training for memory, attention, perception and language in older adults with mild cognitive impairment (MCI) who are at risk of developing Alzheimer’s or other types of dementia. In her review of seven of those studies Belleville (2008) found that six reported positive results although variability in research design and heterogeneity of the MCI population undermined those findings. This flaw in the methodology prompted the author to emphasise the need for studies that use larger samples of participants and randomised controlled designs.

Less conclusive findings regarding the efficacy of cognitive rehabilitation have also been published. For example, a Cochrane review of the literature to evaluate the effectiveness of cognitive training on attention deficit following stroke was published by Lincoln and colleagues in 2000 (Lincoln et al., 2000). The authors' findings were somewhat tentative in that although they found attention training improved alertness and sustained attention, only two studies were evaluated and in neither study was the assessment of outcome carried out blind to the intervention. Furthermore there was insufficient evidence to support or refute the effect of attention training on functional abilities. In two other separate Cochrane reviews of the literature on memory rehabilitation following stroke, the authors were unable to find evidence to either support or refute the effectiveness of such treatment (Majid, Lincoln, & Weyman, 2002; Nair & Lincoln, 2007). A more recent review of the literature also found inconclusive evidence for or against cognitive approaches for recovery of memory following stroke (Coulas, 2007).

However, despite its critics and controversy, cognitive rehabilitation has become a standard component of rehabilitation after neurological trauma, particularly traumatic brain injury (Rohling et al., 2009). Overall, the evidence for the efficacy of cognitive rehabilitation is positive thereby providing confidence not only for its inclusion in rehabilitation programmes but also for providing impetus for further research. However, in the current era of evidenced-based practice, further research needs to be more scientifically rigorous with class 1 evidence (randomised control trials). The question of whether or not cognitive rehabilitation should aim to reduce impairment or compensate for the impairment, are issues that also need further investigation. Moreover, given the vast array of

cognitive impairment, future research into specific cognitive areas such as attention, memory, visuospatial skills, and executive functioning is essential.

### **Cognitive Rehabilitation following Stroke**

The focus of stroke rehabilitation tends to be on remediation of physical deficits; language/speech therapy; and to some extent functional activities that, though impacted by cognition, are treated without a focus on their cognitive basis. This is despite the finding that long-lasting neuropsychological sequels; such as deficits in attention, planning, problem solving, memory, and speed of information processing; occur in nearly half of stroke survivors. For example, using measures of overall cognitive status (e.g., Mini Mental Status Exam; MMSE; Folstein, Folstein, & McHugh, 1975), it has been reported that the majority of individuals referred to in-patient rehabilitation (55.2%) will exhibit cognitive deficits, with many individuals obtaining scores at or near the cut-off for cognitive impairment (Bonita et al., 1997). Unfortunately, the general nature of measures such as the MMSE does not allow them to provide an understanding of the complex and heterogeneous nature of post stroke cognitive deficits (Donovan et al., 2008).

In addition to their prevalence, there is evidence that neuropsychological factors play a significant role in determining functional outcomes after stroke with some arguing that cognitive impairment is more of a determinant of outcomes than physical disability (Bays, 2001; Hochstenbach et al., 1998; Heruti et al., 2002; Labi, Phillips, & Greshman, 1980; Paolucci et al., 1996; Robertson, Ridgeway, Greenfield, & Parr, 1997; Zhu et al., 1998), and primarily accounts for strain in stroke caregivers (Zak, 2000). This accumulating body of evidence

strongly suggests that appropriate neuropsychological rehabilitation may improve functional outcomes and reduce the burden of stroke.

In an initiative to understand the impact of cognitive deficits and functional recovery after stroke, NINDS and the Canadian Stroke Network have established domains for studying 'functional cognition' following stroke. The ten functional cognitive domains identified include; language, reading and writing, numeric/calculation, limb praxis, visuospatial function, social use of language, emotional function, attention, executive function and memory. It is within these realms that the impact of stroke on daily activities can be evaluated leading to an increase of the potential benefits of rehabilitation and accordingly enhancing the ecological validity of neuropsychological assessments (Donovan et al., 2008).

### **Evidence for Rehabilitation of Attention**

Attention is one of the specific domains where there is a pressing need for further research given the prevalence of attention deficits in post-stroke impairment. Furthermore, attention is inextricably involved with memory and executive functioning and is sub-served by shared neural circuitry (Sohlberg & Mateer, 2001). Impairment of this domain sometimes leads to problems of a wider and more complex nature which makes attention deficits one of the greatest impediments to rehabilitation.

In their review of the literature of attention rehabilitation following stroke and TBI, Michel and Mateer (2006), included three major approaches in which to present their findings. The first approach embraces the adoption of a specific-skills training regime. That is, people with an attention deficit learn or relearn how to perform specific skills of functional significance, with the underlying rationale of developing alternative neuropsychological processes that rely on

preserved brain areas, to improve performance of the skill (Carpenter, 2001). The second approach is the so-called “direct route” that requires the patient to practice abstract cognitive exercises designed to directly restore impaired attention processes. The third approach may be more relevant further along in the rehabilitation process, when the patient reintegrates into the home or work environment. This approach focuses its efforts on environmental modification, developing self-management strategies and building environmental support. However, in practice, interventions from a combination of approaches are often utilised to address the fluid, multi-faceted needs of the patient (Michel & Mateer, 2006).

The question that needs to be addressed is why then, given the extent of post-stroke attention deficits and impact on functional outcomes, have there been so few good quality controlled trials of cognitive rehabilitation for attention? In the Cicerone et al. (2005) updated review of the literature for evidenced-based cognitive rehabilitation of people with traumatic brain injury and stroke, only 17 of the 87 studies evaluated were Class I design (i.e., well-designed, prospective Randomised Control Trials (RCTs) and of these only two looked specifically at remediation of attention deficits.

The primary objective of intervention studies is to determine whether or not a particular treatment is effective in bringing about positive change in a given population. In accordance with the American Academy of Neurology 2004 criteria, the highest level of evidence (Class I study) comes from RCTs (Sacco et al. 2006). The degree to which changes occurring during any study can be attributed to the treatment of interest depends on the extent to which the research is able to control for alternative explanations. Control may be exerted, for



example, by making sure that the individual(s) who conduct baseline and follow-up assessment are blind to the recipient of the treatment, by being explicit in defining the sample population, using reliable measures, and providing a detailed account of the treatment employed (Coolican, 1994). Class II studies are those which do not randomly assign participants to treatment conditions, but meet all other requirements of an RCT. These studies include masked outcome assessment and are termed prospective matched group cohort studies (Chalmers et al., 1981; Matthews, 2006).

The following section will present evidence from studies which evaluated attention rehabilitation interventions following stroke. Attention was selected as the area of deficit on which to focus because it allows examination of interventions that are geared towards addressing attention more generally, including interventions for visual neglect and other specific forms of attention deficit. There are also products designed for the rehabilitation of attention deficits that have been commercially available for a number of years, but have not yet been robustly evaluated in this population.

For the sake of clarity, scanning and general attention interventions will be discussed separately. A literature search of studies investigating scanning interventions used for visual neglect was carried out. PsychInfo and the Cochrane Data Base were the search engines used with the keywords: unilateral neglect, attention, brain injury, stroke, CVA, scanning, rehabilitation, and cognitive rehabilitation. Visual neglect is strongly associated with impairments of the ability to sustain attention (Posner, 1993). The aim of scanning strategies is to compensate for difficulties attending to the left by encouraging full scanning of the environment (Bowen & Wenman, 2002). A summary of these studies is

presented in Table 2. As the focus here is on cognitive rehabilitation, pharmacological interventions were not reviewed.

A summary of the twenty three papers of scanning interventions examined in Table 2 cover fourteen randomised controlled trials (RCTs), eight controlled clinical trials (CCTs) and one pre-post design. All studies recruited stroke patients with unilateral neglect. Outcome measures cover a wide range of domains of impairment including neglect, scanning ability, visual attention, cognition, disability, functional abilities, and activities of daily living and some well-known measures such as the Barthel Index, the WAIS and various cancellation tests were used. Generally, sample sizes were small with the largest study providing outcome data on 80 participants. Those studies, using measures of overall functioning such as activities of daily living and functional outcomes, consistently show improvements in level of disability and or independence. However, the findings for neuropsychological outcomes are less consistent.

Limitations in methodology was an issue raised by Bowen and Wenman (2002) when they concluded that only three out of the fifteen studies they reviewed were sufficiently well-designed and controlled to provide conclusive evidence for the effectiveness of attention rehabilitation.

Table 2  
*Studies evaluating scanning interventions for neglect*

Authors	Design & Sample	Interventions	Outcome measures	Conclusions
Bowen, and Wenman, (2002)	SR & MA of 15 studies  RCT = 8. CCT = 7.  N = 400 10 studies from rehabilitation 5 studies from hospital admission  Only 3 given A grade.	Any activity to reduce neglect or resulting disability vs alternative treatment or no treatment. Drug trials were excluded. Therapy length varied from <2 to 30 hrs.  Several provide no detail about the intervention.	Impairment (e.g., line cancellation or bisection); disability (Barthel, FIM, ADL); discharge setting (home or not)	Impairment level improved significantly and these effects persist. Insufficient evidence for impact on ADLs or discharge setting. Few studies examined disability / participation.
Robertson, McMillan, MacLeod, Edgeworth, and Brock, (2002)	RCT N = 39 Right hemisphere stroke with left neglect, right – handed, from in and out-patient rehabilitation.  ≥7 Hodgkinson Test for Dementia	12 x 45 minutes of: (1) n = 19 Limb Activation Device (LAD) - worn on wrist, makes auditory tone if no left movement made + Perceptual Training (PT) – workbook exercises (e.g., visuospatial puzzles)  (2)n = 19 PT only + placebo LAD	At post-treatment and 3-month follow-up: Barthel, self- and other-ratings of neglect, Motricity Index, BIT, Comb & Razor personal neglect tests, Modified landmark test.  At 18-24 month follow-up: Nottingham Extended ADL, Motricity Index, Balloons test, self- and other- ratings of neglect.	Significant Time x Treatment effect on Motricity Index at 18-24 months with LAD+PT performing better than PT only. Analyses used are questioned.
Edmans and Webster, (2000).	RCT N = 80 from a larger RCT of consecutive stroke admissions, mean 34 days post onset.	6 weeks x 2.5 hours per week (1) n = 40 Transfer of training to ‘treat the cause’ of perceptual problems (2) n = 40 Functional treatment of ‘symptom rather than cause’	Post-treatment: Barthel, Edmans Extended ADL, RPAB, RMA Gross Functioning Scale	Improvement in perceptual abilities not related to group.

Weinberg et al. (1977)	RCT N=32 Experimental group 25= Control group Stroke patients with right-brain injury ≥4 weeks post injury	20 hours graded intervention designed to train compensation for impaired left sided scanning abilities vs standard rehabilitation	Scanning ability Achievement test – reading tasks	Treatment resulted in significant improvement on measures of scanning and academic reading tests thought to rely on scanning ability.
Young, Collins, and Hren, (1983)	CCT Non-randomised, not-blinded N = 27 right hemisphere stroke with left visual neglect assigned to 3 different groups	All receive 1 hr treatment/day for 20 days Gp 1) Standard OT Gp 2) 20 minutes Standard OT + 20 minutes paired cancellation + 20 minutes visual-scanning training Gp 3) 20 minutes cancellation + 20 minutes visual-scanning training + 20 minutes block-design training	WAIS- Digit Symbol Coding, Block Design, Picture Completion, Object Assembly; letter cancellation; WRAT Reading test; copying an address and counting faces.	Groups 2 and 3 improved significantly on visual scanning, reading and writing compared to group 1, with group 3 showing greatest improvement.
Robertson, Gray, Pentland, and Waite, (1990)	RCT N = 36 with behavioural inattention, 32 post-stroke, remainder with head trauma or meningioma	Control Group (N=16) 14 x 75 minute sessions over 7 weeks of unspeeded computer activities.  Experimental Group N=20) 14 x 75 minutes sessions over 7 weeks 4 levels of speeded scanning training using touch screen	WAIS, Neale Reading Test, letter cancellation, ROCF, observer reports; BIT (primary outcome)	No significant changes on the BIT for either group although controls showed greater improvement at 6 months than the experimental group
Wuart et al. (1997)	RCT N = 22 < 3 months post stroke	1) traditional stroke rehabilitation 2) usual care + 20 hours over 1 month of visual scanning with trunk-control rotation	Battery of tests of neglect (e.g., bells, line cancellation) and FIM	Improvement of those in experimental group was significantly better than in controls for both neglect and FIM tests.
Antonucci, Guariglia, and Judica, (1995).	RCT N = 20 ≥ 2 months post right hemisphere first-ever stroke	Group 1) 2 months of neglect treatment Group 2) General cognitive stimulation for 2 months, then rehabilitation training for neglect for 2 months.	Standardised test battery and functional scale	At end of neglect treatment there was significant improvement that generalised to everyday life.

Bergego, et al. (1997)	Pre-post N = 7 right hemisphere ischemic stroke	45 mins per day for 2 weeks on each task: 1) Visual Scanning Training; 2) Level Comparison of 2 vertical bars; 3) Dynamic visual matching to sample 4) 150 words text reading.	Scene drawing, 3 cancellation tasks, letter cancellation, line cancellation, bells test, writing 5 lines.  Two computerised tasks using words or non-words and determining if 2 stimuli are same//different	No specific treatment effects on static paper and pencil or computerised tests.
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ADL = Activities of Daily Living, BDAE = Boston Diagnostic Aphasia Examination, BIT = Behavioural Inattention Test, CCT = Controlled Clinical Trail, FIM = Functional Independence Measure, MA = Meta-analysis, MIT = Melodic Intonation Therapy, OT = Occupational Therapy, PASAT = Paced Auditory Serial Attention Task, PT = Physiotherapy, RCPM = Raven's Coloured Progressive Matrices, RCT = Randomised Controlled Trial, RH = right hemisphere; RMA = Rivermead Motor Assessment; ROCF = Rey Osterreith Complex Figure, RPAB = Rivermead Perceptual Assessment Battery, SR = Systematic Review,; WAIS = Wechsler Adult Scale of Intelligence, WRAT = Wide Range Achievement Test

In contrast to the studies addressing visual neglect only, three RCTs, one CCT and three pre-post design studies were found which examined more general attention rehabilitation, five of which had small samples ( $N < 39$ ). These are summarised in Table 3. Two trials involving general attention training, reported significant improvements in alertness (Sturm & Willmes, 1991) and sustained attention (Schottke, 1997), although these improvements were not reflected on measures of functional independence. Using computerised attention training, however, the experimental groups performed better on tests related plausibly to attentional functions (Gray et al., 1992; Schottke, 1997; Sturm et al., 1997; 2004). A programme consisting of hierarchical linguistic and non-linguistic tasks targeting sustained, selective, and alternating attention resulted in improvement on a measure of aphasia and a measure of abstract reasoning when compared to baseline performance (Helm-Estabrooks, Connor, & Albert, 2000). Only one study failed to produce an encouraging outcome. In the Mazer et al. (2003) study, two different visuo-perceptual training techniques, resulted in no significant differences, although the lack of a no-treatment or standard care control group means that it is not possible to determine if either intervention might have been equally and significantly effective. Otherwise, of the eight studies outlined in the table, seven provide encouraging evidence for the positive effects of general cognitive interventions for individuals with attention deficit.

Table 3.

*Studies evaluating general attention interventions*

Authors	Design and Sample	Interventions	Outcome Measures	Conclusions
Sturm and Wilmes (1991)	RCT N = 27 (LHD) 8=(RHD)	14 x 15 min sessions over 3 weeks WDG and Cognitron Training programme for attention and perceptual speed	10 standardised psychometric tests similar to the training procedure	Attention training improves alertness (Standard mean deviation 0.77, 95% CI 0.21 to 1.33); sustained attention (SMD 1.03, 95% CI 0.44 to 1.61).
Gray, Robertson, Pentland, and Anderson, 1992	RCT N = 31 TBI patients with attention deficit $\approx$ 20 months post-stroke	17 received 14 sessions x 75 mins computerised attention training vs 14 received 14 sessions x 75 mins recreational computing	PASAT, Arithmetic, Picture Completion, Digit Span subtests of The WAIS-R, word fluency, WCST, finger tapping, GHQ	Improvement of those in experimental groups was more significant on the PASAT and Arithmetic subtest and GHQ.
Sturm, Willmes, Orgass and Hartje (1997)	Pre-post design N = 38 with left and right hemisphere vascular lesions > 2 months post-onset	2 x 14hrs computerised training programme	Improved Intensity and Reduced Response Time and Error Rate on Attention Tasks	There were significant specific training effects for both intensity aspects (alertness and vigilance), and also for response time in the selective attention and error rate in the divided attention task.
Schoettke (1997)	CCT N = 29 no blinding, matched allocation 38-52 days post-stroke	Computer, paper pencil, and scanning training 13 sessions over 3 weeks vs. standard care.	Measure of sustained attention Barthel Index	Significant effect on sustained attention. No evidence to draw conclusions about information processing or ADL.
Helm-Estabrooks, Connor and Albert (2000)	Pre-post design N = 2 LH stroke patients with aphasia >12 months post-onset	Case 1=17 twice weekly sessions ATP plus 16 sessions of MIT  Case 2=16 twice weekly sessions ATP only	BDAE, RCPM	Significant improvement on both measures for both patients.
Mazer, Sofer, Korner-Bitensky, Gelinias, Hanley and Wood-Dauphinee, (2003)	RCT N = 84 referred for driving evaluation	20 sessions of "field of view" training of visual processing speed, divided attention compared to traditional computerised visuoperceptual training	On-road driving test, visuoperceptual tests and TEA	No difference in driving skill, some benefit for those with right hemisphere lesions.

Sturm et al. 2004	Pre-post N = 8 right-handed right-hemi vascular lesion patients >5 months post stroke	Grp 1 (N = 4) computerised alertness training Grp 2 (N = 4) memory training Both groups received 14 x 45min sessions over 4 weeks	TAP, PET/fMRI	3 ppts in Gp 1 improved on alertness compared to only 1 of Gp 2. Scans showed restitution of the RH observed in ppts with behavioural improvement
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ADL = Activities of Daily Living, ATP = Attention Training Programme, BDAE = Boston Diagnostic Aphasia Examination, CCT = Controlled Clinical Trail, fMRI = functional Magnetic Resonance Imaging, GHQ = General Health Questionnaire, LHD = Left Hemisphere Damage, PASAT = Paced Auditory Serial Attention Task, PET = Positron Emission Topography, RCPM = Raven's Coloured Progressive Matrices, RCT = Randomised Controlled Trial, RH = Right Hemisphere, RHD = right hemisphere damage; TAP = Test of Attentional Performance; TEA = Test of Everyday Attention, WCST = Wisconsin Card Sorting Test, WDG = Wiener Determinationsgerat; WAIS = Wechsler Adult Scale of Intelligence,



However, none of the studies reviewed, targeted the entire range of the different types of attention (i.e. focussed, sustained, selective, alternating and divided attention) in a single rehabilitation package. This is at odds with the availability of attention rehabilitation packages such as Sohlberg and Mateer's (1987) Attention Process Training (APT), which was designed for the remediation of attention and memory disorders with mild, moderate and severe brain injury. The APT is based on a theoretical model to facilitate attention as a comprehensive and multilevel functional process (Sohlberg & Mateer 1987) comprised of sustained, alternating, selective and divided attention and the APT programme provides the opportunity for addressing each of these particular aspects of attention. (See Table 1 Sohlberg and Mateer's Clinical Model of Attention). It has been evaluated within brain injured samples, including some post-stroke individuals.

It has been claimed that specific components of attention require specific training in order for improvement to occur (Sturm & Willmes, 1991; Sturm et al., 1997). Thus, APT treatment involves a group of hierarchical organised tasks that place increasing demands on the individual as they progress through the programme. Examples of exercises include auditory tapes such as listening to descending letter sequences, detecting target stimuli with the presence of distractor noise or complex semantic categorisation tasks requiring switching sets. A combination of both auditory and visual activities are utilised on a number of tasks. Progression through one module builds skills that are thought to be necessary for performing in subsequent modules (Sohlberg & Mateer, 2001). Sohlberg, Johnson, Paule, Raskin, and Mateer (2001) published the APT-II for

use with adults with attention and memory disorders and mild cognitive impairment.

Studies conducted to evaluate the efficacy of APT-I and APT-II are presented in Table 4. Of the 12 studies, two were RCTs, two CCTs and eight were Pre-Post Designs. The studies recruited participants who had experienced a range of neurological illness including four studies with TBI patients, two with patients with Cerebral Vascular Accident, one study with both TBI and CVA participants and one study with a patient with Attention Deficit Disorder. Injury or illness was greater than two months post-onset. The four remaining studies included participants with schizophrenia or aphasia. Thus a range of participants have been evaluated with this intervention. All participants were identified as having an attention deficit by neuropsychological testing or self-assessment. The amount of APT provided to subjects varied considerably across studies ranging from 15 sessions to 85 sessions with no apparent relationship between the number of sessions and participant's performance on measured outcomes. Eleven of the twelve studies concluded a positive outcome for the remediation of attention deficit post-APT treatment, however, four of those studies (Boman, Lindstedt, Hemmingsson, & Bartfai, 2004; Insalaco; 2009; Kurtz et al., 2001; Palmese & Raskin, 2000), included a combined treatment thus confounding the attribution for the agent of change.

There were a range of different outcome measures utilised across the studies. Of the four studies that used a Continuous Performance Test as an outcome measure (Butler & Copeland, 2002; Kurtz et al., 2001; Lopez-Luengo & Vaaquez, 2003; Sohlberg, McLaughlin, Pavese, Heidrich, & Posner, 2000), three found an improvement on that measure following APT intervention. A

neuropsychological measure common to five of the studies was the Paced Auditory Serial Addition Test (PASAT; Gronwall, 1977), with improvement on this measure post-APT achieved in all five studies. However, despite this commonality, two of the studies (Park et al., 1999; Sohlberg et al., 2000), included control groups who also demonstrated an improved score on the PASAT, suggesting that it was not APT that caused the change. Another outcome measure common to three studies was the Test of Everyday Attention (TEA). The participants in each of the studies suffered from mild aphasia and improvement on all three studies was recorded for four to seven subtests of the TEA.

Different outcomes were achieved by the participants with schizophrenia in the two respective studies. There was no significant improved attention for participants in the Lopez- Luengo and Vazquez (2003) study, however, in the Kurtz et al. study (2001) significant improvement was achieved for divided attention and sustained visual attention. In two studies (Coelho, 2005; Sinotte & Coelho, 2007) the participants with mild reading impairment showed an improvement of language proficiency without the benefit of language intervention suggesting that improved attention abilities may facilitate language processes in general.

To summarise, there is growing evidence that decreased attentional capacities which frequently present as a consequence of stroke, are amenable to intervention (Michel & Mateer, 2006). Specifically, APT has had some positive effect on improving attention, albeit primarily in small sample trials. In a world where the survival of stroke is on the increase, the necessity for effective cognitive rehabilitative techniques has become more imperative. The need for more rigorously designed trials with large samples is needed to build on current

research and provide more robust findings of APT. However, the RCT and CCT investigative framework provides difficulties when working in this field of health research and it is those issues that provide the theme for discussion in the following sections.

Table 4  
*Studies evaluating Attention Process Training*

Authors	Design and Sample	Interventions	Outcome Measures	Conclusions
Sohlberg and Mateer (1987)	Pre-Post N = 4 (2= CHI, 1= OHI, 1= with aneurysm)	4-8 weeks APT with 7-9 sessions	S&M hypothesised subjects would improve on the PASAT but not on a measure of spatial relations because the latter utilises other cognitive processes	All subjects showed an improvement on PASAT but not on a measure of spatial relations.
Park Proulx and Towers (1999)	CCT N = 46 (23=TBI, 23=Controls)	Experimental condition=40Hrs APT Control condition=No APT	PASAT, Consonant Trigrams, BDI	Exp group improved on both neuropsychological measures. Control group improved on PASAT only suggesting APT results in learning of new skills rather than improved processing. No change in BDI scores for both groups.
Palmese and Raskin (2000)	Pre-Post Multiple Baseline Design N = 3 MTBI	1hr APT per week for 10 weeks followed by 6-7 hrs educational and applications programme	CTT, PASAT.SDMT, Stroop, DV, R-APT	All individuals demonstrated improvement on CTT, PASAT and SDMT but the changes may not be specific to the APT-11 Programme
Sohlberg, McLaughlin, Pavese, Heidrich and Posner (2000)	RCT N = 14 TBI 2 x groups using A-B Crossover design	Experimental Condition = 24 hrs APT Control Condition = 10 hrs Brain Injury Education	TMT, PASAT, COWAT, CPT, Stroop, Gordon Diagnostic, Covert Orienting, Sternberg Questionnaires, Structured Interviews	Overall, there was improvement in performances on neuropsychological measures for both conditions. Specific improvement for PASAT, Stroop, Trails, and Memory for locations was greater for APT than BIE
Kurtz, Moberg, Mozley, Swanson, Gur and Gur (2001)	CCT N = 6 patients with schizophrenia	Experimental Condition = 1hr APT 2 x per week for ≤ 4 months plus 2 months of PROMPT Control Condition = No remediation treatment	Digit Span (F & B), Stroop, Cancellation Tests, CPT, ACT	2/3 patients in A condition improved significantly on tasks of divided attention (ACT) and 2/3 on sustained visual attention (CPT) tasks. The control group did not improve on any tests for which data was available (Digit Span, COT & cancellation tests)
Butler and Copeland ( 2002)	CCT N = 31 children with an attentional deficit following a treatment and/or cancer that was CNS related.	Experimental condition (CRP) =21 completed 50 hrs cognitive remediation programme included APT/Special education/CBT techniques over 6 months. Control condition (CS)=10	WISC-111 Digit Span subtest, WRAML Sentence Memory subtest, CPT , WRAT-111 Arithmetic subtest	CRP group significantly improved on Digit Span, Sentence Memory and the CPT. No significant changes made by the control group.

Lopez-Luengo and Vaaquez (2003)	RCT N = 24 Schizophrenia patients	Experimental Condition=13 patients received APT Control condition = 11 patients received standard care	CPT, PASAT, CVLT, WCST, TMT A&B, a dichotic listening task , a dual dichotic listening and a cancellation task. Everyday Attention Questionnaire	No significant improvement of attention found as a result of APT although treatment group demonstrated improvement in executive functions as measured by WCST.
Boman, Lindstet, Hermingsson and Barfai (2004)	Pre-Post follow-up Design N = 10 (5= stroke, 2=SH, 2= Encephalitis, 1= TBI All >9 months post-injury	9hrs APT per week for 3 weeks plus generalisation for training and teaching of compensatory strategies for self-selected tasks	Post performance on APT, Digit Span, RBMT, Claeson-Dahl Memory test, AMPS, EBIQ	Improvement on more complex tasks of attention. No change on Digit Span score.
Murray, Keeton and Karcher (2006)	Single subject multiple baseline ABA design N = 1 patient with mild conduction aphasia	17hrs APT-II and 20hrs of APT-11 home practice	ADP, TLC-E, CETI, Logical memory and Digit Span, TEA, APT-11 questionnaire	Higher raw scores achieved on Digit Span but not significant. Higher scaled score achieved on seven TEA subtests with significant change on two subtests measuring sustained selective and divided attention. Minimal changes on APT questionnaire
Pero, Incoccia, Caracciolo, Zoccolotti and Formisano (2006)	Pre-Post N = 2 patients with severe TBI >1 yr post-trauma	85 sessions APT	TAP, TEA	Both patients improved on measures of selective attention. The patient with pre-treatment vigilance and divided attention deficits demonstrated improvement in both these areas.
Sinotte and Coelho (2007)	Pre-Post N = 2 (1 with CVA 6 months post-onset with mild reading impairment, 1 control with no neurological history)	16 sessions of APT-II over 5 weeks	WAB-AQ, GORT-4, TEA, APT-II questionnaire, Reading Rate	Improvement on WAB-AQ and GORT-4 although not clinically significant. Significant improvement on seven subtests of TEA. Improved rating on APT-II questionnaire
Insalaco (2009)	Pre-Post follow up multiple Baseline Design N = 6 with ADHD and ED	13 weeks APT-II and GPDR training	WMS-111, CTT, DVT, BRIEF-A, Attention Questionnaire from APT-11	3/6 reduced the APT questionnaire raw score. APT with GPDR appears helpful in reducing attention problems in adults with ADHD and ED

ACT=Auditory Consonant Trigrams, ADHD=Attention Deficit Hyperactive Disorder, ADP=Aphasia Diagnostic Profiles, AMPS=The Assessment of Motor and Processing Skills, APT=Attention Process Training, BDI=Beck Depression Inventory, BIE=Brain Injury Education, CCT=Clinical Control Trial, CETI=The communicative effectiveness index, CHI=Closed Head Injury, COWAT=Controlled Oral Word Association Test, CPT=Continued Performance Test, CRP=Cognitive Remediation Programme, CS=Comparison subjects, CTT=Consonant Trigrams, CVA=Cerebrovascular Accident, CVLT=California Verbal Learning Test, DV=Digit Vigilance, EBIQ=The European Brain Injury Questionnaire, ED=Executive Dysfunction, GORT-4=Gray Oral Reading Tests-4, GPDR=Goal, Plan, Do, Review, MTBI=Mild Traumatic Brain Injury, OHI=Open head Injury, TBI= Traumatic Brain Injury, PASAT=Paced Auditory Serial Attention Task, PROMPT=Prospective Memory Training, R-APT=Revised Auditory Processing Test, RBMT=Rivermead Behavioural Memory Test, RCT=Randomised Control Trial, SDMT=Symbol Digit Modalities Test, SH=Subarachnoid Haemorrhage, SSD=Single Subject Design, TAP=Test for Attentional Performance, TBI=Traumatic Brain Injury, TEA=Test of Everyday Attention, TLC-E=Test of Language Competence-Expanded Version, TMT=Trail Making Test, WAB-AQ=Western Aphasia Battery-Aphasia Quotient, WAIS-R=Wechsler Adult Intelligence Test-Revised, WCST=Wisconsin Card Sorting Test, WISC 111=Wechsler Intelligence Scale for Children-Third Edition, WMS-111=Wechsler Memory Scale-Third Edition, WRAML=Wide Range Assessment of Memory and Learning, WRAT=Wide Range Achievement Test-Third Revision.

## **Barriers to Trials in Cognitive Rehabilitation**

Non-medical research (i.e. non-pharmaceutical and non-surgical research) endeavours to conduct first class research, however, the RCT design has several limitations and inherent problems when applied outside this context. Some behaviour researchers criticise the RCT as being too concerned with theory and the remediation of impairment with little focus on reducing disability and restoring social functioning (Bottomley, 1997; Hart, Fann, & Novack, 2008; Pringle & Churchill, 1995; Roth & Fonagy, 2005; Stephenson & Imrie, 1998). Unlike pharmaceutical or surgical trials, standardization of the content and delivery of behavioural interventions, such as cognitive rehabilitation, is more complex and therefore presents major challenges to investigators. The treatment under investigation is more likely to be influenced by unknown factors which may affect the scientific reliability of measured outcomes (Hart et al., 2008). 'Extraneous variable' is a general term sometimes used to refer to any variable other than the treatment which might have an effect on the outcomes of interest (Bordens & Abbott, 2002; Isaac & Michael, 1981). Non-specific features of the treatment such as time spent with participants, number of contacts, timing of intervention, or length of follow-up, are examples of extraneous variables and need to be comparable in all conditions so that the mechanistic effects of the experimental intervention, rather than instructor or research-team attention and social support, will be credited for differences in outcomes (Bordens & Abbott, 2002). Non-medical research design requires rigorous procedures to eliminate the influence of such variables. It is argued that rigorous systemised randomising should evenly balance out such factors (Togerson & Togerson, 2001), however, this assumption is only true when trials are large enough for this balancing to take place (Roth, Fonagy, Parry, Target, & Woods, 2005).



An additional difficulty lies in defining the characteristics of the sample and achieving adequate sample sizes. While large samples are required in order to have sufficient power to detect treatment effects (Aveline, Shapiro, Parry, & Freeman, 1995) in stroke populations, it is often the case that the larger the sample the more heterogeneous its characteristics. Stroke patients present with different types of stroke (e.g. ischaemic stroke, haemorrhagic stroke and subarachnoid haemorrhage) with an extensive array of corresponding problems. This makes it less likely that everyone receiving the treatment will benefit and will therefore reduce the likelihood of finding significant treatment effects. Thus, there is a need to define samples a priori in such a way as to ensure confidence in the conclusions drawn from the data, but not be as restrictive as to reduce your likelihood of obtaining your required sample size (Schulz & Grimes, 2005). This must include the need to define time post-injury and exclude or balance for confounds such as stroke severity, while ensuring a sample that is diverse enough to be representative but with reasonable expectation that treatment will be beneficial. This should also include a definition of how those individuals who are likely to benefit from the treatment will be defined with regard to the problem being treated. For example, in defining the minimal level of deficit on an assessment of attention that would be considered sufficient to require treatment, one must consider not only the psychometric properties of the assessment tool and its appropriateness to the population of interest, but also the ease with which the assessment could be integrated into existing clinical settings (Meinert & Tonascia, 1986).

It is commonly agreed that non-medical research is expensive to conduct because recruitment usually requires large samples in order to ensure any observed differences are due to the intervention. In addition, those administering the

intervention are either professionals with expertise in that particular field of investigation or are researchers who require extensive training and monitoring, both of which can be expensive (Bottomley, 1997). Reimbursement provided to physicians for their role in clinical trials often falls far short of their costs, which may include the hiring of additional nursing and data management staff to ensure that patients fully understand the risks and benefits of participation, to track participating patients and collect and report the necessary data.

An additional problem with non-medical research, such as is the case for cognitive rehabilitation, is that double blinding is not possible and effective blinding of the participant is only rarely achievable (Rains & Penzien, 2005). Therefore, there is a need to put greater emphasis on ensuring systems are in place to guarantee blinding of the individual conducting baseline and outcome assessments. Extensive effort by the researchers is required to ensure appropriate recruitment and randomisation so as to reduce the risk of bias. However, randomisation in non-medical studies, where it is often difficult to blind participants to group allocation, can affect levels of motivation in potential participants. Excluding choice by allocating patients randomly to one or other treatment is seen as a great strength in clinical trials, yet in behavioural trials this has brought criticism (Bottomley, 1997; Pringle & Churchill, 1995). Choosing your preferred intervention, it is argued, increases motivation. If this is so, then it must be established if those motivational factors affect the efficacy of an intervention and if so, how motivation can best be measured.

A final challenge in conducting non-medical research relates to its potential to interfere with existing practises. Health professionals already have demanding workloads, and can become de-motivated toward the research when established

schedules are disrupted, particularly if immediate patient benefit is not obvious. Research methodology should adapt to existing routines as much as practicable, not only to maintain effective relationships with hospital staff but also to ensure continuity in the delivery of health care (Bottomley, 1997; Pringle & Churchill, 1995).

### **Appropriateness of Control Conditions**

The control group is not exposed to the experimental treatment and provides a baseline measure which gives investigators important clues to the effectiveness of the treatment, its side effects, and the parameters that modify those effects. The absence of a control group raises concerns that any observed improvement post-training arises as a function of practice on the outcome measures or due to some extraneous factor. When conducting research for neuropsychological rehabilitation different control conditions present complex risk-benefit ratios that need close consideration before decisions are made for the design of the research (Saks, Jeste, Granholm, Palmer, & Schneiderman, 2002).

The no-treatment control group is deemed to be the easiest and most cost effective to run. This condition provides control for the effects of spontaneous recovery, regression to the mean and the effects of repeated testing on outcome measures which is particularly crucial in studies of cognitive remediation (Park & Ingles, 2001). However, considerable ethical objections have been raised about denying interventions that are believed or assumed to be beneficial, to a group who is defined as having a particular problem (Saks et al., 2002).

Another disadvantage is that high drop-out rates may more likely be under a no-treatment group condition. In response to the ethical concerns, many clinical studies do not include a non-treatment group but rather, when available, utilise existing

standard-care therapy thereby assuring participants do not go untreated for their condition. As well as overcoming the problem of denying treatment, standard care often provides a simple, clean, and cheap control condition. The disadvantages of standard care include possible high variability in care, the lack of effectiveness if standard care is minimal or not accessible and the potential for spill-over of the intervention into the standard care groups.

Wait-list control groups receive the treatment after the treatment period for the experimental group and the waiting time is typically the same duration as the treatment period. The wait-list design condition controls for the effects of time and regression to the mean (Hart et al., 2008). However, there are disadvantages to using this form of control group design. Wait-list control groups are not appropriate for protracted time periods as it raises the ethical dilemma of withholding treatment. This design is not suitable for those patients who are in the acute stages of their illness as the wait period introduces possible extraneous variables, such as spontaneous recovery, that may not be measureable but may impact on outcomes. The waiting period also increases the probability of higher attrition and is likely to worsen over time (Byrne, Fursland, Allen, & Watson, 2011). Furthermore, the wait-list group must undergo two rounds of pre-assessment, thus introducing practise effects as a possible confounding factor in the performance of participants on those measures.

The true placebo condition, as employed in pharmaceutical trials, is virtually impossible to achieve in non-medical research given that double blinding is impossible. Single blinding is also elusive since most participants are able to discern whether they are in the “more active” or “less active” treatment group, so reducing their expectation of improvement and thus the integrity of the placebo effect

(Whitehead, 2004). Alternative care or sham treatments can be defined as one performed on a control group participant to ensure that he or she experiences the same incidental effects as the experimental group. In non-medical research this control condition can be problematic if both groups improve, as the ability to determine real treatment effects is less discernible. Again ethical opposition to alternative treatment groups include the withholding of effective treatment, providing irrelevant treatment, possible risk to the participant and the inherent nature of misleading the participant assigned to the alternative treatment (Rothman & Michels, 1994).

A further concern that can arise with randomisation is that some individuals may consent to participation in the study, however, with the steadfast hope that they will be randomised into the treatment group particularly if they perceive the treatment on offer as beneficial to their recovery. If this fails to eventuate, dropout rates may increase due to disappointment and frustration (Karlavish & Whitehouse, 1998). A well-conceived control condition can include an alternative activity of value that is unrelated to the desired outcomes of the trial and that is attractive to prospective participants. Burdening participants with time-consuming and worthless tasks is not appropriate.

The problems associated with RCTs for studying the efficacy of cognitive interventions must be balanced against the benefits that this type of design provides, (i.e., the most rigorous and robust means of identifying cause and effect). Some of the difficulties identified could be resolved if sufficient funding was available, so in line with current knowledge and demands, government and private funding agencies need to prioritise support for this type of research. Multi-centre approaches may be one solution to reducing the high costs of conducting non-medical research. Not only would financial and staff costs be distributed across settings, the problem of having to

recruit large sample sizes would also be divided between sites. Data from each site would be collated for analysis.

In summary, the world-wide trend for the incidence of stroke is on the increase, as is the number of people surviving stroke. A significant number of stroke survivors have been identified as experiencing post-stroke cognitive deficits with impairment of attention being a common factor. To date, there have been very few studies investigating neuropsychological rehabilitation for people with attention deficits post-stroke. Reasons behind the shortage of such research may lie in the difficulties inherent with randomised control trials when conducting non-medical research such as ethical considerations and available resources. The increasing demand for cognitive rehabilitation for this population necessitates further well-designed research in this area with outcomes measured on both tests of attention and functional abilities.

The purpose of the current study is to add to a developing and evolving knowledge base on cognitive rehabilitation post-stroke. In order to obtain the highest level of evidence the study design is that of a randomised control trial. Those problems previously discussed in this chapter that may arise when a RCT design is used for behavioural interventions were addressed and either minimised or eliminated altogether. For example, sample size, the make-up of the sample, the inclusion of rigorous systemised randomising, the inclusion of health-related quality of life measures as well as the use of conventional cognitive measures, and ensuring stringent standardisation of the content and delivery of the intervention, were all measures taken to ensure confidence in the findings.

The primary aim of this study was to investigate the impact of Attention Process Training on attention deficit in patients at the post-acute stage of stroke (between 5 to 9 weeks after the occurrence of the stroke). The impact, if any, of APT

on health-related quality of life was also of interest as was investigating whether the neuropsychological profile of stroke patients had any impact on attention process training. Finally, the study attempted to look at any change in the clinical profile of stroke patients that might occur as a result of APT.

## **Chapter 4: Methods**

The Stroke Attention Rehabilitation Trial (START) was a Health Research Council of New Zealand funded project. This PhD thesis focuses on an aspect of the START project (i.e. the impact of APT on attention in stroke survivors at five to nine weeks post-stroke). I was a member of the operations committee which was responsible for the execution and management of the study. Initially, the committee convened weekly, then fortnightly for the six months leading up to the commencement of data collection. I was also employed full-time on the study for two years and responsible for the recruitment of the participants and the administration of the APT programme.

### **Ethics**

Approval for the conduct of this study was obtained from the Northern X Regional Health and Disability Ethics Committees Ref (NTX/06/10/124). This study is registered with the Australian Clinical Trials Register (CTRN12607000045415). The research was run according to New Zealand Good Clinical Research Practice Guidelines. Ethics for this study was also granted by the Department of Psychology Research and Ethics Committee at the University of Waikato (No. 08:07).

### **Participants**

Participants included all survivors of ischemic stroke or primary intracerebral haemorrhage consecutively admitted to inpatient neurological rehabilitation units of Middlemore and North Shore Hospitals over an 18 month period. Stroke diagnosis was according to the standard WHO criteria which describe a stroke as an interruption of the blood supply to the brain causing damage to the brain tissue (Aho et al., 1980).



Patients were required to meet certain eligibility criteria for inclusion in the study. The participant must have had a first-ever confirmed stroke which must have occurred in the previous 30 days. The participant must also have given informed consent, and must have been comfortable having a conversation in English (as standardised administration of tests requires English fluency). The participant must have obtained a score  $\geq 20$  on the Mini Mental State Exam and finally, the participant must have been signed off by their medical officer as being medically stable to complete APT training.

Potential participants were excluded from the study if they had a psychiatric illness and/or were on psychotropic medications which in the opinion of a clinician would have had a significant effect on attention processing. Exclusion also applied if the participant had been informed that the amount of alcohol they consumed was a problem or there was a record in the medical notes of a person having alcoholism. Other exclusion criteria included the participant being involved in another study that, in the opinion of the investigator, may have affected cognitive performance or added significantly to participant burden or if the participant had a contraindication to Attention Process Training such as blindness, deafness, or an inability to talk.

If the patient passed the initial screening, they were administered the attention tests to ascertain the presence of an attention deficit. This was determined by a score of one standard deviation below the normative mean scores on any one test reflective of attention (i.e., Integrated Visual and Auditory Continuous Performance Test [IVA-CPT] Attention Quotient score, Trail Making Test Part A and B, the Bells Cancellation Test, and the Paced Auditory Serial Addition Test). These measures were selected because they provide a measure of the four components of attention

that are addressed by APT and because they provide measures for both auditory and visual modalities of attention.

Of the 334 patients initially approached, 107 gave informed consent however only 95 patients were found to meet the eligibility criteria. (Refer to Figure 5 for the number of participants at each stage of the study). Two the 12 who did not meet the eligibility criteria did not have confirmed stroke diagnoses, three had a MMSE <20, three were not medically stable, one was less than four weeks post-stroke, one was in a competing study and two withdrew their interest.

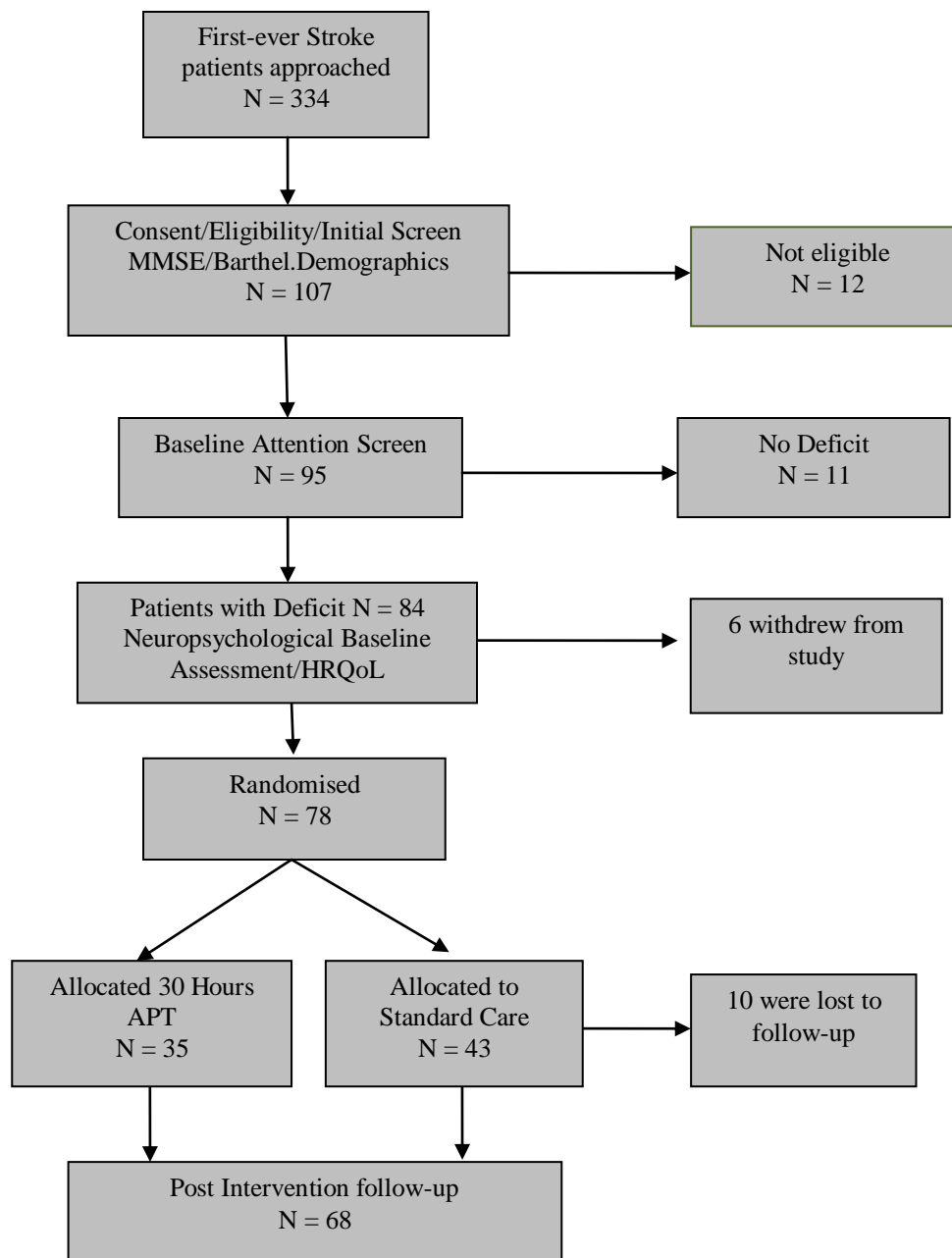


Figure 5. Study Design and recruitment

Eighty four participants were then found to have an attention deficit while 11 did not. Five patients then withdrew from the study and one other moved away from the area leaving 78 patients to be randomised. Thirty five patients were randomised into the APT group and 43 were randomised into the Standard Care group. (See Figure 5).

The ethnic composition of the APT intervention group was; 80% European, 5.7% Maori, 11.4% Pacific Islander, and 2.9% Indian. Sixty percent of the APT group were male, 40% were female and the range of ages was from 24-94 years with a mean age of 69.54 years. The Standard Care group was made up of 76.7% European, 16.3% Maori, and 7% Pacific Islander and there were 60.5% male and 39.5% female. The ages ranged from 38 to 85 years with a mean age was 68.51 years.

## **Design**

### **Study Overview.**

The staff of the inpatient Acute Treatment and Rehabilitation wards and the acute stroke wards of Middlemore Hospital and North Shore Hospital were informed of the study by means of a presentation at their weekly in-service meeting. Senior stroke consultants, some of whom were part of the steering committee for the START project, ward clerks, stroke nurse specialists and staff of a community based rehabilitation team were key referrers of potential participants. Those patients who consented to participate in the study and met the eligibility criteria were assessed to determine whether or not they had an attention deficit. If they did have an attention deficit, they were administered the Barthel Index as well as all other neuropsychological measures and all health related quality of life measures. The data from these measures were used as baseline measures however for the purposes of this

study, only the attention measures and the SF-36 were re-administered at four-five weeks post-randomisation. All other measures were re-administered at six months post stroke as part of the START project (Barker-Collo, et al., 2009). (Refer to Table 5 for Schedule of assessments).

Table 5  
*Schedule of Assessments*

	Measure	Screening	Baseline	5 Weeks	6 months
Inclusion	MMSE	√			
Attention Screen	IVA-CPT	√		√	√
	Bells	√		√	√
	Trails A/B	√		√	√
	PASAT	√		√	√
Randomisation	Barthel Index	√			
Additional Neuropsychological Tests	Stroop		√		√
	CVLT-11		√		√
	LM		√		√
	VPA		√		√
	ROCF		√		√
	BNT		√		√
	COWAT		√		√
HRQoL	SF-36		√	√	√
	CFQ		√		√
	mRS		√		√
	GHQ		√		√

BNT = Boston Naming Test, CFQ, Cognitive Failures Questionnaire, COWAT = Controlled Oral Word Association Test, CVLT = California Verbal learning Test, CHQ = General Health Questionnaire, HRQoL = Health Related Quality of Life, IVA-CPT = Integrated Visual and Auditory Continuous Performance Test, LM = Logical Memory, MMSE = Mini Mental State Exam, mRS = Modified Rankin Scale, PASAT = Paced Auditory Serial Addition Test, ROCF = Rey Osterreith Complex Figure, SF-36 = Short Form 36, VPA = Visual Paired Associates.

## Apparatus and Measures

### Eligibility Measure.

#### *Mini Mental State Exam (MMSE) Folstein, Folstein & McHugh, (1975).*

The MMSE is a brief objective assessment (administration time is approximately 10 minutes), that can be used to indicate the presence of cognitive impairment. It is employed extensively in clinical studies, community surveys and epidemiological studies (Tombaugh, McDowell, Kristjansson, & Hubley, 1996). It

was used in this study to screen for eligible participants who must have obtained  $\geq 20$  in order to qualify for inclusion.

Reliability coefficients for the MMSE are moderate to high and there is high sensitivity for cognitive decline in dementia and head injury. Test-retest reliability was .89 and a combination of test/retest and inter-rater reliability was .82 (Folstein, Folstein, & McHugh, 1975). A correlation coefficient of .78 has been found with the Wechsler Adult Intelligence Scale for Verbal Intelligence Quotient and .66 for Performance Intelligence Quotient (Burns, Lawlor, & Craig, 2004).

The MMSE is an 11 question measure that tests five areas of cognitive function including orientation, registration (immediate memory), attention and calculation, recall, and language. The maximum score that can be achieved on this measure is 30

#### **Baseline Measures.**

##### ***Barthel Index (Mahoney & Barthel, 1965).***

The Barthel Index has become one of the most widely used tools to measure a person's ability in activities of daily living. It was developed to assess the severity of disability or independence in personal care and mobility in stroke patients. It is easily administered requiring 2 to 10 minutes to complete. Shah, Vanclay and Cooper (1989) report an alpha internal consistency coefficient of 0.87 to 0.92. The scale consists of 10 variables that are related to activities of self-care (feeding, grooming, bathing, dressing, bowel and bladder care, and toilet use) and mobility (ambulation, transfers, and stair climbing). Scoring of this measure is also easy with scores of 0-20 indicating total dependence; 21-60 severe dependence; 61-90 moderate dependence and 91-99 slight dependence (Granger, Sherwood, & Greer, 1977; Granger, Albrecht, & Hamilton, 1979; Sulter, Steen, & De Keyser, 1999). For the

purposes of randomisation participants were grouped into two groups, those who obtained scores of  $\geq 18$  or  $<18$ .

### **Neuropsychological Measures.**

The neuropsychological tests used in this study are well validated and commonly used in stroke samples (Spreeen & Strauss, 1998). Procedure for administration of all neuropsychological tests was conducted in accordance with respective manuals or published standard procedures. Except for the Bells Cancellation Test, raw scores on all tests were converted to z-scores and compared to normative data. Normative data for The Trail Making Test Part A & B, the Paced Auditory Serial Addition Task, The Victorian Stroop Test and the Controlled Oral Word Association Test were obtained from A Compendium of Neuropsychological Tests (Spreeen & Strauss, 1998). Normative data for the IVA-CPT, the Wechsler Adult Intelligence Scale-III, the Wechsler Memory Scale-III, the California Verbal Learning Test and the Boston Naming Test were obtained from the respective test manuals. If normative data was not available for older adults, data from the Mayo's Older Americans Normative Studies (MOANS) was used (Steinberg, Bieliauskas, Smith, & Ivnik, 2005; Steinberg, Bieliauskas, Smith, Langellotti, & Ivnik, 2005; Steinberg, Bieliauskas, Smith, Ivnik, & Malec, 2005).

### **Attention.**

The attention measures that were selected for this study addressed all four components of attention targeted in APT. The IVA-CPT was used to obtain a measure of sustained and selective attention, the Trail Making Test was used to measure sustained and alternating attention, the PASAT was used to measure sustained and divided attention and the Bells Test was used to measure sustained and selective attention in the visual modality. Initially, the scores obtained on measures of attention

were used as a screening tool to identify an attention deficit. An attention deficit was identified as present if participants scored below one standard deviation below the normative mean on any one of the following measures; the auditory quotient or visual quotient of the IVA-CPT, the PASAT or either trial of the Trail Making Test, or if they made > 3 errors on the left or right side of The Bells Test. The scores obtained on all attention measures for those participants who were identified as having an attention deficit, were also used as the baseline measures.

***The Integrated Visual Auditory- Continuous Performance Task (IVA-CPT)***  
***(Sandford & Turner, 2000).***

The IVA-CPT is an easy to administer computerised continuous performance test that is designed to assess two major factors. The Full Scale Attention Quotient (FSAQ) is a measure of problems of inattention, loss of focus, and slow processing speed. The Full Scale Response Quotient is a global composite score reflecting problems of response inhibition (i.e. impulsivity), sustaining effort, and making consistent responses. Seckler et al. (1995) reported 1 to 4 week test-retest reliability coefficients ranging from .37 to .75 which they concluded were very small practise effects. It was concluded that “any observed effects of 15 quotient points (i.e. one standard deviation) or more are interpreted as not likely to be due to random fluctuations” (Sandford & Turner, 2000, p.19; Thickpenny-Davis, Barker-Collo, & Caplan, 2007, p. 306). Of the 22 scale raw scores produced on this test, 20 scales had significant positive relationships with 18 of the 20 showing a moderately strong to very strong relationship (.46 to .88; Seckler, Burns, Montgomery, & Sandford, 1995). The following are guidelines for the description of observed changes in quotients:  
<15 = No Significant Change; 15 to 22 = Mild Change; 23 to 29 = Mild to Moderate

Change; 30 to 37 = Moderate Change; 38 to 44 = Moderate to Marked Change; and 45+ = Marked Change (Sandford & Turner, 2000).

*Administration.*

Test instructions are presented visually on the computer screen using a clear female voice. Like all good continuous performance tasks the IVA-CPT is designed to be mildly boring and therefore demanding of sustained attention, with a poor performance producing errors of inattention (omission) and impulsivity (commission). It also provides an objective measure of fine motor regulation and speed. On this task the individual was required to press a mouse button in response to an auditory or visual target stimulus (number 1) that appeared or was heard via a computer and to refrain from pressing the mouse button when a non-target stimulus (number 2) was presented. A one and a half minute warm-up session for both the auditory and visual stimuli was given, with only the number 1 presented as stimuli, which provided an opportunity for those participants who had not worked on a computer before, to become familiar with the use of the mouse. This was followed by a 32 item practice session using both numbers 1 and 2. The computer provided corrective feedback to the participant when errors were made. As well as allowing for minimising of practise effects, the practise session also provided the examiner with the opportunity to determine whether the participant understood the task. If the participant appeared uncertain of what was required on this task further instruction was provided until full comprehension was achieved. The test itself took about 13 minutes and involved the participant responding to or inhibiting a response over five sets of 100 stimulus presentations. The visual “1”s and ‘2’s were presented for 167 milliseconds, and the verbal “1”s and “2”s were presented for 500 milliseconds. The main test collected a measure of impulsivity by creating a response set of not



responding; 84% of the stimuli or 42 out of the first 50 ‘frequent’ block of trials were “1”s intermixed with eight “2”s. During the second ‘rare’ block of 50 trials, many “2”s (84% of stimuli) were presented and few “1”s, and the examinee was forced to wait to make his or her responses every six to nine seconds when a “1” was heard or seen. This second block of trials “pulls” for inattention, and creates a response set of responding. An equal number of auditory and visual stimuli were presented in a fixed pattern in each block, with the patterns of “1”s and “2”s during the frequent and rare blocks being mirror images of each other. The computer automatically saved the performance scores for later analysis.

*Scoring.*

The primary diagnostic scales are the Full Scale Attention Quotient and the Full Scale Response Control Quotient.

The global Full Scale Attention Quotient consists of three separate Auditory and Visual Attention quotients made up of three scales. They are:

1. Vigilance: measures errors of omission, providing an indication of problems related to inattention. Vigilance is related to failure to respond to a target during rare blocks. A low Vigilance score may indicate problems with staying on task, being alert, negligence or indifference. A high score may indicate attentive and alert responding.
2. Focus: “reflects the total variability of the speed of mental processing for all correct responses and thus, is designed to be sensitive to an unusual number of occurrences of slow reaction times” (Sandford & Turner, 2000, p. 2).
3. Speed: “the reaction time of all correct responses throughout the test and helps identify attention processing problems related to slow mental processing” (Sandford & Turner, 2000, p. 2).

The Full Scale Response Quotient consists of three response control primary scales for both visual and auditory modalities. They are:

1. Prudence: measures errors of commission - reflecting poor response inhibition problems and impulsivity. It is a measure of “the ability to stop, think and not automatically react to a foil” (Sandford & Turner, 2000, p. 8). Low prudence scores indicate thoughtlessness, carelessness or over-reactivity. High scores indicate mindful, cautious, careful and circumspective responding. For the purposes of this study, the data from the Prudence (auditory and visual) scale were presented as quotients to provide information on whether APT improved impulsivity.
2. Consistency: the variability and reliability of response times and “is used to help measure the ability to stay on task” (Sandford & Turner, 2000, p. 2).
3. Stamina: identifies any difficulty with sustained attention and effort over time, and with maintaining speed of mental processing. This score is calculated through comparing the mean reaction times of correct responses during the first 200 trials to those of the last 200 trials.

In addition, the IVA-CPT has three validity scales (Sandford & Turner, 2000). First, the Persistence scale compares simple reaction times before and after the test. Reductions in reaction time may be indicative of: a lack of motivation when the examinee is asked to do ‘one more thing’; an oppositional attitude; or a reflection of mental or motor fatigue (Sandford & Turner, 2000). Second is the Sensory/Motor scale which is used to “rule out possible unusual neurological, psychological or learning problems evidenced by slow simple reaction time when only the target “1” is presented” (Sandford, 2000, p. 7). Finally, the Comprehension scale indicates the likelihood of random responding (Sandford & Turner, 2000).

All IVA-CPT scores are presented both as raw scores and quotient scores, with the quotient scores having a mean of 100 and a standard deviation of 15. The normative database (N=781) for the IVA-CPT is broken into the following age groups: 5-6, 7-8, 9-10, 11-13, 14-17, 18-24, 25-34, 35-44, 45-54, 55+ years; and is also divided by gender; with each gender/age group being sufficiently large for valid clinical interpretation (Sandford & Turner 2000). All of the individuals in the database reported having no learning, attention, neurological, or psychological problems. For each of the IVA-CPT global scales, attention scales, response control scales and the Sensory/Motor scale, it is proposed that an individual score of less than 90 can be labelled Mildly Impaired; less than 80 is Moderately Impaired; less than 70, Severely Impaired; and less than 60, Extremely Impaired.

In addition, the IVA-CPT has three validity scales (Sandford & Turner, 2000). First, the Persistence scale provides a comparison of simple reaction times before and after the test. Reductions in reaction time may be indicative of: a lack of motivation when the examinee is asked to do ‘one more thing’; an oppositional attitude; or a reflection of mental or motor fatigue (Sandford & Turner, 2000). Second is the Sensory/Motor scale which is used to “rule out possible unusual neurological, psychological or learning problems evidenced by slow simple reaction time when only the target “1” is presented” (Sandford & Turner, 2000, p. 7). Finally, the Comprehension scale indicates the likelihood of random responding (Sandford & Turner, 2000).

However, for the purposes of this study only the measures of interest were used: The Full Scale Attention Quotient, the Auditory Attention Quotient, the Visual Attention Quotient, the Full Scale Response Quotient, the Auditory Prudence Quotient and the Visual Prudence Quotient.

***Paced Auditory Serial Addition Test (PASAT) (Gronwall, 1977).***

The PASAT is a serial addition task used to assess rate of information processing (Gronwall & Wrightson, 1981), sustained attention (Cohen, Sparling-Cohen, & O'Donnell, 1993), and divided attention (Lezak, 1995; Lezak et al., 2004; Ponsford & Kinsella 1992; van Zomeren & Brouwer, 1994). It is one of the most frequently used measures of attention in patients with mild traumatic brain injury (Cicerone, 1997; Gordon & Zilmer, 1997; O'Jile et al., 2006; Sohlberg & Mateer, 1989; Tombaugh, 2006; Vanderploeg, Curtiss, & Belanger, 2005).

The PASAT has been a primary outcome measure in a number of efficacy studies of APT (Palmese & Raskin, 2000; Park et al., 1999; Sohlberg & Mateer, 1987; Sohlberg et al., 2000). (See Table 4 Studies evaluating Attention Process Training). It is also used as a neuropsychological measure in clinical settings with patients suffering from a wide variety of neuropsychological syndromes including degenerative disorders, Parkinson's Disease (Dujardin et al., 2007), Huntington's Disease, Vascular Cognitive Impairment, Korsakoff's Syndrome, and Multiple Sclerosis (Lezak, 1995). Indeed, the PASAT is included as a core measure in the Multiple Sclerosis Functional Composite (MSFC), a quality-of-life outcome measure in Multiple Sclerosis-related clinical trials (Barker-Collo, 2005; Nagels et al., 2005; Rudick et al., 1997).

Egan (1988) and Johnson, Roethig-Johnson, and Middleton (1988), found split-half reliability for the PASAT to be high (.9) and MacLeod and Prior (1996), found performance across different paces was also highly correlated. Test-retest correlations of this test after 7 to 10 days is high (>.9) (McCaffrey et al., 1995) however practice effects on this task are significant although minimal after the second presentation (Gronwall, 1977; Tombaugh, 2006).

The PASAT was selected for use in this study because although it can be a difficult and stressful test, it is nevertheless useful for the detection of subtle attention deficits (Lezak et al., 2004). Furthermore, it provides another index of auditory attention beside the IVA-CPT.

*Administration.*

For this task, sixty one numbers ranging from 1 to 9 were presented to the participant with the use of a tape recorder. The participant was required to add each number to the number that immediately preceded it. For example if the digit 6, 3, and 2 were presented, the participant would respond with the correct answers 9 and 5. The pace at which the numbers are presented can vary at 1.2, 1.6, 2.0 or 2.4 seconds apart. In this study only the two slowest trials (2.0 and 2.4 seconds) were administered as they were considered to be appropriate paces for stroke patients in the acute stage. The participant's response was required prior to the presentation of the next digit for a response to be scored as correct. In this study, the participant was initially presented with a practice trial followed by the two test trials.

*Scoring.*

Scoring of the task was the total number of correct responses made with a maximum score of 60 per trial.

***The Trail Making Test A and B (TMT A & B) (Partington & Leiter, 1949).***

The TMT is a paper and pencil test that requires the connection of encircled numbers (Part A) and numbers and letters (Part B) in the proper order. It is a well-established test that is available in written and oral forms, the latter omitting the visual-motor component of the task. The TMT is a test frequently used to assess attention, visual search, scanning, speed of processing, mental flexibility and executive functions (Spreeen & Strauss 1998; Tombaugh, 2004). Part B of the TMT is

highly sensitive to the effects of brain injury (Lezak, 1995). The TMT has also been reported as a useful tool in identifying cognitive decline in dementia (Kowalczyk, McDonald, Cranney, & McMahan, 2001). It is correlated highly with driving ability and along with other measures is used for determining a person's driving "readiness" following stroke (Lundqvist, Gerdle, & Ronnberg, 2000; Mazer, Korner-Bitensky, & Sofer, 1998). The TMT has however been criticised for its use with patients who may experience physical constraints (Lezak, 1995; Waldstein, et al., 2003).

In terms of construct validity, Part A and Part B correlate only .49 with each other. The results of studies conducted by Gaudino, Geiser, and Squires (1995) and Woodruff, Mendoza, Dickson, Blanchard, and Christenberry (1995), suggest that Part B is a more difficult cognitive task than Part A as the participant is required to switch between letters and numbers. As such Part B is a measure of alternating attention and it is for this reason and its use as a primary outcome measure in previous studies evaluating APT (Lopez-Luengo & Vaaquez, 2003; Sohlberg et al., 2000) that the TMT was selected for use in this study.

Performance on the TMT is affected by age and education but not gender nor culture (Arnold, Montgomery, Castaneda, & Longoria, 1994; Heaton, Grant, & Matthews, 1986, as cited in Spreen & Strauss, 1998; Tombaugh, 2004). Inter-rater reliability is excellent with coefficients of .94 for Part A and .90 for Part B having been reported (Fals-Stewart, 1991, cited in Spreen & Strauss, 1998). Other reliability coefficients have been reported for this test with most having been above .60, several above .90 and most above .80 (Spreen & Strauss, 1998). The TMT has shown high concurrent validity with the Arithmetic, Digit Span and Digit Symbol subtests of the Wechsler-Bellevue Scale.

*Administration.*

On Part A the participant, using a pencil, was required to join a series of circled numbers (without lifting the pencil) in ascending order from 1 to 25, strategically displayed on an A4 sheet, in as little time as possible. This provided a baseline measure of processing speed. On Part B, a more complex task, the sheet contained circled numbers (1 to 13) and letters (A to L) and the participant was required to join the circles alternating between numbers and letters in ascending order (i.e., 1-A-2-B-3-C etc). The participant was instructed to connect the circles as quickly as possible, without lifting the pencil from the paper. If the participant made an error on either Part A or Part B, it was immediately pointed out to the participant who was then instructed to return to the last circle completed correctly in the sequence. Errors affected the participants score in that the correction of the error was included in the time to complete the task.

The test took on average 5 to 10 minutes to administer. Participants were urged to perform the task as quickly as they could and performance was measured as the time in seconds it took to complete each task. It was important to ensure that the participant understood the instructions fully before the task began because timing commenced immediately the pencil touched the paper.

*Scoring.*

Raw scores were converted to standardised scores and compared to available normative data. (Strauss, Sherman, & Spreen, 2006).

***The Bells Cancellation Test (Gauthier, De haut & Joanne, 1989).***

The Bells Cancellation Test is widely used to detect visual inattention or unilateral neglect and is appropriate for use with teenagers and adults (Azouvi et al., 2006; Beis et al., 2004; Tant, Kuks, Kooijman, Cornelissen, & Brouwer, 2002). It has

also been used as part of a battery of tests determining the impact of perceptual deficits on functional autonomy in elderly patients post-stroke (Mercier, Desrosiers, Herbert, Rochette, & Dubois, 2001). They examined the Bells Test in 59 subjects, of which 20 were controls, 19 had right cerebral lesions and 20 had left cerebral lesions. A statistically significant difference in mean scores between the group with right cerebral lesions and the group with left cerebral lesions was observed. They reported test-retest reliability as being marginal ( $r = .69$ ) however Vanier et al. (1990) pointed out that hemi-neglect is a fluctuating phenomenon and therefore comparison of performance on the two tests should not be expected. When establishing concurrent validity, this test was found to identify a much higher percentage of stroke patients with visual inattention than the Albert's Test of Visual Neglect (Vanier et al., 1990) and the Diller Test (Mercier, Audet, Herbert, Rochette, & Dubois, (2001). In another study, the authors found that the distractor items on the Bells test tended to detect mild and moderate neglect more readily (Marsh & Kersel, 1993).

The test consists of a 21.5 x 28 cm sheet of paper on which seven vertical sections each containing 35 distractor figures (e.g. bird, key, apple, mushroom, car) and five target figures (bells) are presented. All figures are presented as solid black silhouettes. The target figures are arranged so that five each appear in seven equal columns on the page. The number of distractor figures also remains constant.

#### *Administration.*

The examinee was first presented with a demonstration sheet that included the target figure in the centre surrounded by all the distractor figures. The participant was asked to identify each figure, thus ensuring correct recognition for each object. The test copy was then presented and the participant was required to identify and



draw a circle around all the bells on the sheet. Performance on the Bells Cancellation Test is not timed.

*Scoring.*

Scoring consisted of the number of bells correctly circled and the time for completion of the task. The total number of omissions in the three left segments versus the centre and the three right segments was then compared to normative data for non-neurologically impaired adults and to norms for those with left or right CVA (Gauthier et al., 1989).

**Other Neuropsychological Baseline Measures.**

The following selection of neuropsychological tests were administered at baseline in order to obtain measures of cognitive domains other than attention. Those domains included executive functioning, language and verbal and visual memory. These neuropsychological measures were administered again at six months post-stroke for data pertaining to the START project (Barker-Collo et al., 2009)

**Executive Functions.**

***The Stroop Test: Victorian Version (Regard, 1981).***

The Victorian Stroop Test is a shorter version of the original Stroop Test (Stroop, 1935) taking approximately five minutes to administer. It is used to determine the ease with which a person can maintain a goal in mind and suppress a habitual response in favour of an unusual one (Spreen & Strauss, 1998). This task requires the examinee to correctly name the colour of the ink used for written words which are the names of colours and incongruent to the colour of the print used. This measure of selective attention and cognitive flexibility is routinely used when screening for brain dysfunction, and is used in a wide variety of other applications including testing for Attention Deficit Hyperactivity Disorder (Assef, Gotuzo, &

Capovilla, 2007; King, Colla, Brass, Heuser, & von Cramon, 2007; Lavoie & Charlebois, 1994; Wodka et al., 2008), Posttraumatic Stress Disorder (Beers & De Bellis, 2002; Constans, 2005) and Schizophrenia (Grapperon & Delage, 1999; Henik & Salo, 2004). The Stroop Test also appears to be sensitive to severity of dementia (Koss et al., 1984, as cited in Spreen and Strauss, 1998) and has been a primary measure in studies on the efficacy of APT (Kurtz et al., 2001).

The Stroop Test is negatively impacted by reading ability, and slower with advancing age has also been consistently documented (Boone, Victor, Wen, Razani, & Ponton, 2007; Obler, Fein, Nicholas, & Albert, 1991; Spreen & Strauss, 1998, as cited in Lezak et al., 2004). Performance is also affected by education and culture, to a lesser degree, but not by gender (Macleod, 1991; Protopapas, Archonti, & Skaloumbakas, 2007). Test-retest reliability coefficients were high (.90, .83, and .91) for the three parts of the test, when university students were tested with a one-month interval between tests (Bullock et al., 1996, as cited in Strauss, Sherman, & Spreen 2006). However practise effects have been found to impact on performance (Spreen & Strauss, 1998). In a factor analysis the Stroop was found to draw on speed of processing skills and conceptual abilities, and related to the Block Design, Digit Symbol and Digit Span subtests (Graf et al., 1995, as cited in Spreen & Strauss, 1998). Other studies have found a moderate relation with the PASAT (MacLeod & Prior as cited in Spreen & Strauss 1998) and the Tower of London (Hanes et al., 1996, as cited in Spreen & Strauss, 1998).

#### *Administration.*

The task involved the presentation of three white cards 21.5 x 14cm, each containing six rows of four items printed in green, blue, yellow, or red ink. The four colours were presented in pseudo-random order on the card although each colour

appeared once in each row. The stimuli on the first card (Card 1) were dots. When presented with the first card (Card 1), the participant was required to read as quickly as possible the colour of each of the 24 dots printed on the card. The stimuli on Card 2 were the words 'when' 'hard' and 'over'. When presented with the second card, the participant was required to state the colour of the print of each of the 24 words printed on the card. When presented with the final card (Card 3), the task was the same as that for Card 2, only the stimuli words presented were the colour names 'red', 'yellow', 'blue', and 'green'. The colours of the print of the words on the Card 3 did not correspond to the content of the words.

#### *Scoring.*

Scoring of the three cards included the time it took (in seconds) to complete each card and the number of errors made on each card. Any spontaneous corrections made by the examinee were scored as correct. A discrepancy score was obtained from the difference in time taken to finish the coloured colour names trial (Card 3), compared with the baseline dots condition (Card 1). These results were compared to the normative data available for the Victorian version of the Stroop for 17 to 90 + year olds (Bullock, Brulot, & Strauss, cited in Spreen & Strauss, 1996).

#### **Memory.**

*California Verbal Learning Test-II (CVLT-II; Delis, Kramer, Kaplan, & Ober, 1987).*

The CVLT-II is a multiple-trial list learning task and a widely used neuropsychological test used for the assessment of the processes and effectiveness of the strategies (semantic associations) involved in memory and learning verbal material. The CVLT-II has normative data for individuals aged 16-89 years. Lezak,

(1995) considers the CVLT-II to be more ecologically valid than other similar assessments of word list learning.

Reliability studies conducted by the authors of the CVLT-II show high internal consistency with split-half reliability correlation coefficients from Total Trials 1-5, ranging from .87 to .89 (Delis, Kaplan, Kramer, & Ober, 2000, cited in Lezak et al., 2004). Test-retest (21 days later) reliability was also found to be high for Total Trials with .82 however reliability was much less for other variables such as Total Learning Slope (.27) and Total Repetitions (.30). A number of factor analyses have consistently shown a general verbal learning factor with small effects of response discrimination, learning strategy, proactive interference, and serial position (Lezak et al., 2004). A four factor model incorporating attention span, learning efficiency, delayed recall, and inaccurate recall was proposed by Wiegner and Donders, (1999). Performance on the CVLT-11 is affected by age, gender and education accounting for 29%, 5.1% and 4.5% of the variance respectively (Delis, Kramer, Kaplan, & Ober, 1987).

#### *Administration.*

The examinee was presented with 2 verbal lists (List A & B) of 16 words containing items that were grouped into four different semantic categories (i.e., types of furniture, vegetables, ways of travelling and animals). Words from the same semantic grouping are never presented consecutively. The words were presented at a rate slower than 1 per second. List A was presented over five trials and immediately after each trial, the participant was required to recall as many of those words as possible. After the fifth trial a 16-word interference list (List B) containing two extra categories was presented followed by a short delay free-recall and short delay cued-recall of List A. After a 20-minute delay, long-delay free and long-delay cued recall followed by a yes/no recognition trial of List A was administered.

*Scoring.*

Raw scores on each trial were converted to standard scores (z-scores) based on age and gender appropriate norms. The z-scores were rounded to whole numbers. Data from the following recall measures obtained for this study were obtained: List A Trial 1; Short-Delay Free Recall; Long-Delay Free Recall; Recognition Hits; and False Positives.

***Wechsler Memory Scale-III (WMS-III; Wechsler, 1997) Logical Memory subtest (LM).***

LM is a measure of verbal learning and memory for conceptual material presented in the auditory modality and is reflective of memory for everyday conversation (Spren & Strauss, 1991). As part of the WMS-III, the LM is used widely in the detection of memory impairment for patients with TBI. It is administered as part of the Iowa Screening Battery for Mental Decline and is therefore a frequently used neuropsychological tool for dementia. Other populations upon which LM is administered, include patients with Huntington's Disease, Parkinson's Disease, Multiple Sclerosis, Epilepsy, and patients with carotid artery disease and cerebrovascular disease (Diamond et al., 1992; Johnson, Storandt, & Balota, 2003; Lezak, 1995; McKinlay, Grace, Dalrymple-Alford, & Roger, 2010; Pelosi, Geesken, Holly, Hayward, & Blumhardt, 1997; Romero et al., 2009; Schneider, Boyle, Arvanitakis, Bienias, & Bennett, 2007; Waldstein & Katzel, 2005; Wechsler, 1997).

Split-half reliability estimates for LM immediate recall range from .67 to .80 with an average of .74; and for LM delayed recall range from .55 to .85 with an average of .75 (Wechsler, 1987). Another study reported a reliability coefficient of .71 (Mittenberg, Burton, Darrow, & Thompson, 1992). Wechsler found inter-scorer

reliability for LM was very high at .99, a similar finding to that of McGurie and Batchelor (1998) whose sample consisted of neurosurgery patients. Practise effects were demonstrated in average gains of between one and a half and two raw score points on immediate recall and two and a half to almost three raw score points on delayed recall (Wechsler, 1987). Factor analysis yielded two factors; a general memory and learning factor and an attention and concentration factor (Elwood, 1991).

*Administration.*

LM examines the immediate and delayed recall of two orally presented short prose passages (Story A and B), each of which contains 25 ideas or units of information. On this task, Story A and Story B were read aloud to the participant, after which immediate free recall of that passage was required. A second recall of Story B was then required. The delayed recall condition involves recall of each passage (without prior warning) approximately 30 minutes after the administration of the immediate recall condition. The story was not read out aloud on the delayed recall condition. On all conditions the participant was asked to recall the story verbatim.

*Scoring.*

A point was scored for each correct idea recalled from the passages, with a maximum score of 50. The total score for both immediate and delayed conditions is the total number of ideas recalled on both stories for each condition. The total raw scores for immediate and delayed memory are converted to scaled scores using age standardised norms.

*Wechsler Memory Scale-Revised (WMS-R; Wechsler, 1987).*

*Visual Paired Associates (VPA).*

VPA is among the most widely used instruments for assessing visual memory. This test pairs abstract line drawings with colours, and a colour pointing response is required on immediate and delayed conditions. The test-retest reliability coefficient ranged from .31 to .68. In factor analytic studies, VPA loaded onto a nonverbal memory factor or a visual concentration/visual memory factor (Bornstein & Chelune, 1988; Leonberger et al., 1991, as cited in Moye, 1997). As a subtest of the WMS-R, the VPA has been used extensively with neurologically impaired patients for the detection of memory deficits and is also utilised in research studies such as assessing cognitive effects of anti-hypersensitive drugs in the elderly (Louis, Mander, Dawson, O'Callaghan, and Conway (1999), and memory impairment in psychosis (Brewer et al., 2005) and post-cardiac surgery (Jonsson et al., 1999).

*Administration.*

The subject was shown six nonsense line drawings, each quite different, and each paired with a square of a different colour. As they looked at the figures they were instructed to remember the colour that goes with each figure. After they were shown the figures with their respective colours, they were then shown the figures in a different order, without their colours, and were asked to indicate the appropriate colour (from an array of six) that was associated with each figure. If they answered incorrectly, the correct colour was pointed out to them. The set of six figures were presented three times followed by a recall trial. If the examinee answered all six items correctly on or before the third trial, the subtest was discontinued after three sets. If any of the items on the third set were incorrect, a fourth, fifth and sixth set were administered if necessary. The examiner continued to correct any incorrect answers.

The subtest was discontinued when the participant answered all six items of the third or any subsequent set correctly, or after the sixth set, whichever came first. For each correct response one point was scored. A delayed recall trial was administered approximately 30 minutes later, in which the participant was shown the six figures and asked to indicate which colour went with which figure. The correct figure-colour pairings were not present prior to the delayed recall trial. For each correct response one point was scored. Corrective feedback was not given during the delayed recall trial.

*Scoring.*

The total score for the immediate recall trial was the sum of correct responses across the first three sets only. The total score for delayed recall was the total number of items correctly recalled with a maximum score of six. The raw scores of the subtest were converted to z scores using the means and standard deviations of raw scores on subtests by age for the standardisation sample of the WMS-R (Wechsler, 1987).

***Rey-Osterrieth Complex Figure (ROCF) (Rey, 1941; Osterrieth, 1944).***

The ROCF is a complex diagram that is widely used for the assessment of visuospatial constructional ability organisation and visual memory. It draws on such cognitive domains as planning and organisation, problem-solving strategies as well as perceptual and motor functions. The ROCF is a task that requires the examinee to copy a complex figure and then replicate that figure at later stages from memory.

A number of studies have found high ( $r = .91$ ) inter-rater reliability for this test (Berry, Allen & Schmitt, 1991; Delaney, Prevey, Cramer, & Mattson, 1988).

Repeated administration of the same version of this test resulted in significant practise effects in normal adults (Spreeen & Strauss, 1998). There are a number of



alternate versions of the ROCF, which help reduce practise effects (Hamby, Wilkins, & Barry, 1993; Yasugi & Yamashita, 2010). Reliability coefficients between the ROCF and Taylor Figure suggest the two measures are comparable (Hubley, 2010). Performance on the ROCF is impacted by age (Spreeen & Strauss, 1998) and education however, there is conflicting evidence regarding the influence of gender (Freides & Avery 1991; Lezak, 1995). In a sample of patients with neurological disorders the four trials of ROCF were found to have high concurrent validity with other commonly used neuropsychological tests including the Benton Visual Retention Test, the Rey Auditory Verbal Learning Test, Form Discrimination, Hooper, Trails B and the Token Test (Spreeen & Strauss, 1998). The ROCF is also used extensively in studies of stroke patients (Blake et al., 2002; Phillips & Mate-Kole, 1997; Rapport, Dutra, Webster, Charter, & Morrill, 1995; Szabo et al., 2009). Indeed, the ROCF has been included in the National Institute of Neurological Disorders and Stroke-Canadian Stroke Network's (NINDS-CSN) protocol for cognitive testing (Greenberg, 2009).

The manner in which the two recall trials are drawn can provide valuable information for the examiner. Patients whose errors are based on poor recall of the details of the structure tend to have left hemispheric lesions whereas people with right hemispheric lesions often have difficulty recalling the overall larger structures. The ROCF is sensitive to the detection of traumatic brain injury with significant deficits produced by patients with mild head injuries. Behaviours of executive dysfunction such as perseveration, confabulation, personalisation or other distortion of the design tend to be exhibited on long-delayed trials.

*Administration.*

For this study four conditions of the ROCF were used; the copy trial, the immediate recall trial, the delayed recall trial and the recognition trial. On the copy condition, the participant was required to produce a copy of the complex figure on a separate blank A4 page. After three minutes the immediate recall condition, was administered. This time the participant was required to produce the figure from memory, without prior warning. The delayed recall condition was administered approximately 30 minutes later and again the participant was required to produce the figure from memory. A recognition trial was administered immediately after the delayed recall trial. On the recognition trial the participant was shown a booklet containing 24 details, 12 of which were part of the original figure and 12 that were not. The participant was asked to circle those details that belonged in the original figure.

*Scoring.*

The figure is divided into 18 scorable details with points awarded to each detail depending on accuracy, distortion and location of its reproduction. Two points are awarded for each detail that is accurate and properly placed. One point is awarded for an accurate copy that is poorly placed. One point is awarded if a detail is distorted or incomplete but recognizable and placed properly and a half point is awarded if a detail is distorted or incomplete but recognisable and placed poorly. No points are awarded if the detail is absent or not recognisable. The maximum points scored, is 36 for each trial i.e., the copy, immediate and delayed trial. Two points are awarded for each correctly identified figure on the Recognition trial giving a maximum of 24 points.

**Language.**

*Boston Naming Test (BNT) – 60 item version (Kaplan, Goodglass, & Weintraub, 1983).*

The BNT is a widely used confrontation naming test in English-speaking countries (Barker-Collo, 2007). Utilised for the purpose of assessing the ability to name pictured objects, it is sensitive to subtle word-finding difficulties as well as to subcortical brain disease or damage (Lezak et al., cited in Sbordone, Saul & Arnold, 2007).

Sawrie, Chelune, Naugle, and Luders (1996) reported that test-retest reliability after 8 months was high (.94) in 51 participants with intractable epilepsy. Concurrent validity with other tests of language tests has also been found to be high (Spreen & Strauss, 1998). Split-half correlations for the original version were in the .71 to .82 range for a small control group of normal elderly people and at .97 for a group of Alzheimer patients with the latter group producing scores significantly below the control group (Huff, Collins, Corkin, & Rosen, cited in Lezak, 1995). The original version of the BNT contains 85 items however, a revised 60-item version with well standardised data across ages, from 5 years through to 97 years. This revised version is used in virtually all cases (Kaplan, Goodglass, & Weintraub, 1983) and was used in this study.

*Administration.*

The participant was presented with 60 cards, each with an ink line drawing of objects representing a range of simple high-frequency words (tree) to rare words (abacus). Each card was presented one at a time and the participant was asked to name the object. If the participant did not provide an answer, or provided an incorrect answer, within 20 seconds, a stimulus or phonemic cue was provided. The

standard discontinuation criterion of failure to correctly name objects on six consecutive trials was used. Administration of this test was approximately 10 – 20 minutes.

*Scoring.*

Each correct answer received a score of 1 if the answer was spontaneous or followed a stimulus cue. A correct answer following a phonemic cue received a score of 0. Administration of the test began with item 30 (harmonica) and full credit (30 points) was given for items 1 to 30 if the participants gave the correct answer. If either item 30 or 31 did not receive a correct answer (one point), then items were administered in reverse order until a total of eight consecutive preceding items were passed. Administration in a forward direction was then resumed. The total maximum score may range up to 60 and raw scores were converted to standard scores using age related norms.

***Controlled Oral Word Association (COWA) (Benton & Hamsher, 1989).***

The COWA is widely used in clinical neuropsychology as a measure of verbal fluency (Iverson, Franzen & Lovell, 1999). The object of the COWA is to say as many words as possible that begin with specified letters. There are two commonly used versions of this test; one uses the letters F, A, S and the other uses C, F, L although evidence suggests the CFL version is harder than the FAS version (Barry, Bates & Labouvie, 2008). In this study, the FAS version was used.

The purpose of this easily administered test is to assess verbal fluency and the spontaneous production of words beginning with a specific letter, within a given time frame. It is thought to determine whether an individual can access a strategy to guide their search for words rather than lexicon definition, and is associated with frontal

lobe function (Penndleton, Heaton, Lehman, & Hulihan, 1982; Phelps, Hyder, Blamire, & Shulman, 1997). It is widely used in the detection of brain injury.

The COWA is included in the Neurosensory Center Comprehensive Examination for Aphasia (NCCEA) (Spreen & Benson as cited in Spreen & Strauss, 1998). It is also frequently used as one of the measures of dementia, although the underlying defects across disorders, differs. For example in patients with Parkinson's Disease, the reduced capacity to generate words, lies in the mental inflexibility of these patients, however in patients with Alzheimer's Disease the underlying problem lies reduced semantic processing and recall (Lezak, 1995). The COWA is also used in research with many other populations including patients with HIV (Dolan et al., 2003), Korsakoff's disease, (Dirksen, Howard, Cronin-Golomb, & Oscar-Berman, 2006), Huntington's disease (Backman, Robins-Wahlin, Lundin, Ginovart, & Farde, 1997), and stroke (Blake et al., 2002; Suhr, Grace, Allen, Nadler, & McKenna, 1998).

Re-test reliability for this test has ranged from .88 after 19-42 days (des Rosiers and Kavanagh, as cited in Spreen & Strauss, 1988), to .65 after eight months for patients with intractable epilepsy, 70 after one year (Sawrie et al. cited in Sbordone et al., 2007) and .74 after an interval of five years (Tombaugh, Kovak, & Rees, 1999). This test has also been found to have high concurrent validity with other language tests and appears to be sensitive to word-finding difficulties as well as subcortical disease and brain damage (Lezak et al., cited in Sbordone et al., 2007). In their presentation of normative data for the FAS measure of verbal fluency, Tombaugh, Kozak, and Rees (1999) found that education accounted for more of the variance in performance than age (education = 21.7% vs age = 11.8%). However, it is the ability to initiate and maintain effort and organise information for retrieval, abilities which

are sensitive to the aging process that is required to perform well on this task, (Barry et al., 2008).

*Administration.*

On this version of the COWA, trials using the letters F, A & S were used. The frequency of use of these letters in the English language ranges from high for the first letter (F) to a lower frequency for the second letter (A) and a further lower frequency for the third letter (S). On the first trial the participant was asked to say as many words as they could think of that began with the letter F. They were instructed to exclude proper nouns, numbers and the same word with a different suffix. The same procedure was conducted for the second trial except the specified letter was A. The third trial was then administered and this time the specified letter was S. The participant was given 1 minute for each trial.

*Scoring.*

All answers were written down verbatim. The score was the sum of all admissible words across the three letter trials. All non-words, repetitions and proper nouns were excluded. Norms for both males and females according to age and educational level are available. Raw scores were converted to standardised scores and compared to available normative data (Strauss, Sherman, & Spreen, 2006).

**Health-Related Quality of Life (HRQoL).**

One of the often cited problems with cognitive research is that it is difficult to translate the findings into real-life situations. It was therefore decided to utilise health related quality of life measures in an attempt to determine if improving attention deficit has wider benefits to the individual other than possible cognitive improvement. Thus, the Mental Component Summary (MCS) score of the Medical Outcomes Study 36-item Short Form questionnaire (SF-36) was used as the

secondary outcome measure and was administered at baseline and at post-intervention. All other health related quality of life measures (CFQ, mRS, & GHQ) were administered at baseline and at six months post-stroke.

### ***SF-36.***

The SF-36 is a widely used generic instrument for measuring quality of life designed for use in clinical practice and research for many diseases and conditions including stroke patients (de Haan, 2002; Dorman, Slattery, Farrell, Dennis, & Sandercock, 1998; Hackett et al., 2000; Williams, Weinberger, Harris, & Biller, 1999). The SF-36 has been tested for validity and reliability across various populations (Fukuhara, Bito, Green, Hsiao, & Kurokawa 1998; Sanson-Fisher & Perkins, 1998) including Maori, Pacific and New Zealand European ethnic groups (Scott, Sarfati, Tobias & Haslett, 2000; Scott, Tobias, Sarfati, & Haslett, 1999).

It comprises 36 self-rated items organised into eight scales; 1) Physical Functioning, 2) Role limitations because of physical health problems, 3) Bodily pain, 4) Social functioning, 5) General mental health (psychological distress and psychological well-being), 6) Role limitations because of emotional problems, 7) Vitality (energy/fatigue), and 8) General health perceptions, with each scale scored out of 100 points. Each scale has been standardised to have a mean of 50 and standard deviation of 10. Higher scores are associated with better HRQoL (Ware et al., 1994). The Mental Component Score (MCS) is comprised of four of those scales; Vitality, Social Functioning, Role Emotional and Mental Health, with each scale scored out of 100 points. The MCS score was used for this study.

### ***modified Rankin Scale (mRS) (Rankin, 1957).***

The Modified Rankin Scale is a commonly used scale for measuring the degree of disability or dependency in the daily activities of people who have suffered a

stroke. It is also a widely used measure in stroke clinical trials (Banks & Marotta, 2007; Sulter et al., 1999). The level of disability or independence is defined on 6 levels (grade 0 to level 5) with Level 0 reflecting no disability and each subsequent level from 1 to 5 indicative of more severe disability with 6, denoting death. The MRS has high correlation with other post-stroke disability indexes including the Barthel Index (BI) and the motor component of the Functional Independence Measure (M-FIM) (Kwon, Hartzema, Duncan, & Min-Lai, 2004). However, unlike the BI and the M-FIM, the MRS is heavily weighted toward global disability and as such allows for consideration of non-physical attributes, such as cognition and language that may contribute to disability (Banks & Marotta, 2007). Inter-rater reliability has been found to be moderate to nearly perfect and can be improved with the addition of structured interviews (Banks & Marotta, 2007). Patients with a score  $\leq 2$  on this scale, by definition are independent (Uyttenboogaart, Luijkx, Vroomen, Stewart, & De Keyser, 2007).

***Cognitive Failures Questionnaire (CFQ). (Broadbent, Cooper, FitzGerald & Parkes, 1982).***

The CFQ was developed to explicitly investigate a person's propensity for committing a cognitive failure and are the reported difficulties people experience in typical everyday situations (e.g., forgetting names or misinterpreting directions) that are linked to lapses in controlled processes, such as focus of attention and working memory (Austin, Mitchell, & Goodwin, 2001; Wallace, 2004). The CFQ is a 25 item self-report assessment that measures the frequency of everyday cognitive failures or lapses (e.g. Q.2 Do you find you forget why you went from one part of the house to the other? Q. 20. Do you find you forget people's names? ) that have occurred in the previous six months. Participants were asked to respond to items using a 5 point



Likert scale where 4= very often, 3 = quite often, 2 = occasionally, 1 = very rarely and 0 = never. Scores therefore range from 0 to 100.

***General Health Questionnaire (GHQ-28). (Goldberg & Williams, 1988).***

The GHQ-28 is a shortened version of the original 60-item General Health Questionnaire and is used for the detection of psychiatric distress related to general medical illness (Lykouras et al., 1996). The GHQ has four subscales (each with 7 items) representing dimensions of symptomology; somatic symptoms, anxiety and insomnia, social dysfunction and severe depression. It is not a diagnostic tool but rather one that may indicate the need for a formal psychiatric interview. The participant's responses to the self-report questionnaire are based on their health state over the previous two weeks. The questionnaire takes approximately 15 minutes to administer and score. Scores range from 0 to 28 with higher scores indicating a greater probability of psychiatric distress (Goldberg & Williams, 1988). The strength of the General Health Questionnaire lies in its acceptability across a wide range of clinical settings and cultures, and its appropriateness with all age ranges from adolescents through to adults (Goldberg et al., 1997; Rush, First, Blacker, & APA., 2008).

**Primary Outcome Measure.**

The Full Scale Attention Quotient of the IVA-CPT was the primary outcome measure used to gauge improvements in attention. A clinically significant change on this test was defined as one standard deviation.

**Secondary Outcome Measure.**

The Mental Component Summary (MCS) score of the Medical Outcomes Study 36-item short Form questionnaire (SF-36) was used as the secondary outcome measure.

**Intervention - Attention Process Training-I (APT-1), (Sohlberg & Mateer, 1987).**

APT-I is a theoretically based, hierarchical, multilevel cognitive therapy programme that is used by neuropsychologists, occupational therapists, speech language therapists and other qualified rehabilitation therapists for the purpose of remediating attention deficit. This programme has been designed to provide activities that target the four components of attention i.e., sustained, selective, alternating and divided attention. The APT-1 programme consists of a series of auditory and visual tasks that become progressively more difficult. The APT manual provides a hierarchy for the administration of each task however the specific order of each exercise was defined prior to commencement of treatment, by the researcher who conducted a trial run of the APT on academic staff. The authors (Sohlberg and Mateer) of APT were advised via email of the order and responded by endorsing the proposed order. (See Table 6 showing the order in which APT tasks were administered).

*Administration of APT.*

Each auditory exercise was presented at a slow (A) or fast (B) pace thus allowing for targeting of speed of information processing. Directions were delivered at the beginning of each exercise by a male voice heard on a compact disc (CD) and speakers connected to a laptop computer. The treating neuropsychologist confirmed with the participant that they understood the directions.

All participants began the programme with exercise no. 1 and proceeded through the programme as each exercise was “mastered”, which was defined as an 85% or greater success rate and no less than a 35% decrease in time (for timed activities) from baseline trials. As well as target responses, non-target responses were also recorded thereby providing information for impulsivity. Table 6 outlines the

sequence in which the exercises were administered, the number of exercises for each type of task, the mode of delivery and which component of attention was being targeted. Altogether the programme consisted of 116 separate exercises.

Every participant received feedback after each activity and open discussion on the participant's experience was encouraged. If a participant was frustrated and became discouraged by non-mastery of an activity, they were presented with another task and returned to the previous task at a later stage. On some occasions, the participant was unable to master the fast trial of a particular activity but was able to master the slow trial of the activity next up in the hierarchy.

Table 6

The order in which APT tasks were administered (the number in brackets depicts the order in which the tasks were administered)

Type of Attention		Auditory	Activity	Visual
<b>Sustained</b>	1	(1) Attention CD 1 ( <i>exercises 1-24</i> )	1	(2) Shape Cancellation ( <i>exercises 25-30</i> )
	2	(4) Attention CD 2 ( <i>exercises 34-42</i> )	2	(3) Number Cancellation ( <i>exercises 31-33</i> )
	3	(5) Serial Numbers ( <i>exercises 43-48</i> )		
	4	(6) Attention CD 4 (quiet) ( <i>exercises 49-58</i> )		
<b>Selective</b>	5	(7) Attention CD 2 (noise) ( <i>exercises 59-66</i> )	3	(10) Shape Cancellation with Overlay ( <i>exercises 93-98</i> )
	6	(8) Attention CD 3 (noise) ( <i>exercises 67-86</i> )	4	(11) Number Cancellation with Overlay ( <i>exercises 99-101</i> )
	7	(9) Attention CD 4 (noise) ( <i>exercises 87-92</i> )		
<b>Alternating</b>			5	(12) Flexible Shape Cancellation ( <i>exercises 102-107</i> )
			6	(13) Flexible Number Cancellation ( <i>exercises 108-110</i> )
			7	(14) Odd & Even number identification ( <i>exercise 111</i> )
			8	(15) Addition/Subtraction Flexibility ( <i>exercise 112</i> )
			9	(16) Set Dependent Activities I and II ( <i>exercises 113-114</i> )
<b>Divided</b>	8	(17) Dual Task: Combine Attention CD and Cancellation Tasks ( <i>exercise 115</i> )	10	(18) Card Sorting ( <i>exercise 116</i> )

## **Sustained Attention Tasks.**

### *Auditory Tasks*

There were two types of auditory tasks involving different formats. On the first set of tasks, participants were required to listen to a male voice on CDs 1, 2 & 4 and press the buzzer each time a target stimulus was identified. For example, an early task (Exercise 2) required the participant to identify (by pressing a buzzer) a single number (e.g. 1) amongst a list of random numbers being read out. In total there were 41 exercises designed to target sustained attention, with the target stimuli becoming more complex on each consecutive exercise. For example, on Exercise 14 the participant was required to press the buzzer to identify 2 consecutive numbers (e.g. 6, 7) and on Exercise 34, the participant was required to press the buzzer to identify 2 months that had been read out in the correct descending order (e.g. May, April). More complex exercises such as solving math problems required the participant to give a verbal response (Ex 49-Ex 58).

The second group of auditory tasks (*Serial Numbers*) is a task of mental subtraction and requires the participant to count backwards by a given number (e.g. 1) beginning at a defined number (usually 100). On subsequent trials, the number to count backwards, were 3, 4, 6 and 7. On the final trial the participant beginning with the number 100, was required to count backwards by mentally subtracting 6, then adding 1. The time it took to complete the task plus the number of errors made were recorded on the appropriate score sheet. If the participant completed the task fluently and accurately at baseline they would automatically proceed to the next exercise.

### ***Visual Tasks***

The visual tasks were presented as shape or number cancellation tasks.

On *shape cancellation* tasks the stimulus sheets contained rows of random shapes and shape features and the participant was instructed to cross out with a pencil, target shapes or shape features. Each stimulus sheet contained many different shapes with subsequent sheets containing more complex shapes. Complexity was further increased by instructing the participant to simultaneously cross out two target shapes on the stimulus sheet, thereby providing 12 exercises for this type of task. Performance, including time and number of errors, was noted on the scoresheet provided in the APT package.

On *number cancellation* tasks the participants were given a sheet containing random numbers and instructed to cross out target numbers. The participant was instructed to work from left to right, line by line, down the page as quickly and accurately as possible. There are nine trials of this type of exercise, with a different target number for each trial. After each block of three different target numbers, consecutive stimulus sheets contained more numbers due to the increasingly smaller print, therefore each block was deemed to be more difficult than the previous block. Timing commenced when the command “go” was given and the number of errors and time was recorded in the appropriate scoresheet.

### **Selective Attention Tasks.**

#### ***Auditory Tasks.***

These tasks were identical to the auditory tasks for sustained attention with the addition of background noise. The participant was required to listen to CDs 2, 3, and 4 select and respond to the target stimulus while simultaneously inhibiting a response to the extra stimulus. On all auditory tasks, the CDs were arranged in

hierarchical fashion from easier to more difficult and were presented at a slow or fast pace. Complexity of the task was increased by instructing the participant to press the buzzer after identifying 2 target stimuli. Scoring on these tasks included the number of errors made and the number of false alarms.

### ***Visual Tasks.***

These tasks were identical to the shape and number cancellation tasks with the addition of a distractor overlay that introduced “visual noise”. The participant was required to cross out the target stimulus while simultaneously inhibiting a response to the additional design. Complexity of all visual cancellation tasks was increased by instructing the participant to cross out either two numbers or target shapes or shape features.

### **Alternating Attention Task**

#### ***Flexible Number & Shape Cancellation.***

This component of attention was presented in the visual mode only and was similar to the shape and number cancellation tasks. Participants were required to alternate between identifying two different numbers or target shapes or shape features, on the stimulus sheet in response to the command “change”. For example, the participant began by crossing out all of the numbers “3” and upon the command “change” would then draw a slash mark and begin crossing out all of the numbers “6”. When the stimulus sheet contained shapes, the target shapes or shape features were identified prior to commencing the task and again the participant would alternate between those stimuli on the command “change”.

*Odd & Even number identification.*

The participant was presented with a sheet containing random numbers and the participant was required to alternate between circling an odd or even number every 15 seconds, on the command “change”.

*Addition/Subtraction Flexibility.*

The participant was presented with a sheet consisting of pairs of numbers. On the command “change” which occurred every 15 seconds, the participant was required to alternate between adding and subtracting the pairs of numbers.

*Set Dependent Activities I.*

The participant was presented with a sheet containing the words “big” and “little” printed in incongruous sizes. The participant was required to read the actual words as they appear and on the command “change” after every 15 seconds, the participant was required to switch to naming the size of the type.

*Set Dependent Activities II.*

The participant was presented with a sheet containing the words “high”, “mid” and “low” printed in incongruous line positions. The participant was required to read the words as they actually appear and on the command “change” after every 15 seconds, switched to reading the line position of the words, ignoring the words themselves.

**Divided Attention Tasks***Auditory Tasks.*

This activity required the participant to respond to the auditory stimuli on the attention CD while performing a concurrent visuo-motor task with the shape or number cancellation tasks.



### ***Visual Tasks.***

The participant was required to sort a deck of cards by suit while simultaneously turning face down any cards that contain a certain target letter in the spelling of their name (e.g. 'e' appears in the names ten, queen etc.).

### **Design Overview**

The apparatus and measures described above were administered at four stages throughout this study. First, the MMSE was administered to establish inclusion then the attention measures were administered to establish an attention deficit. This was followed by administration of the Barthel Index and all other neuropsychological measures. Finally all health related quality of life measures were administered. At the follow-up stage, all the attention measures and the SF-36 were re-administered. (See Table 5 for schedule of assessments).

### **Procedure**

Inpatients of neurological wards of two major New Zealand hospitals Middlemore Hospital and North Shore Hospital were approached to take part in the study. Participants were identified at the daily triage of the rehabilitation wards, at the twice weekly multi-disciplinary team meetings held in the acute stroke ward of Middlemore Hospital or by referral from key hospital staff at both hospitals. Recruitment of participants took place over an 18 month time period.

Only those stroke survivors who met the eligibility criteria were invited to participate in the study. All participants were provided with a patient information sheet (See Appendix C) describing the study and a Consent Form (See Appendix D), and given the opportunity to discuss the procedures with research staff before consenting to take part. The consent forms were signed and dated by the

participant. All participant data was secured in a locked filing cabinet onsite and transferred to the University of Auckland every week where it was again stored in a locked cabinet in a secured room where it will remain stored for 16 years.

After obtaining medical officer consent, patients with first-ever stroke were approached within 4 weeks after stroke ( $M = 18.6$  days post-stroke;  $SD = 7.6$ ), and provided with a written and verbal description of the study in which they were then invited to take part. Patients who had already been discharged from hospital were contacted by phone and provided with a description of the study. Data including age, education, marital status, gender, days since stroke, side of lesion, and stroke subtype according to the Oxford classification was obtained from the patient's medical records or from the participants themselves. They were also administered the MMSE and were required to score  $\geq 20$  to be eligible for inclusion into the study. All participants were then scheduled to meet with the research associate (usually within 24 hours) for the purpose of administering the IVA-CPT, PASAT, TMT, and Bells Cancellation Test to determine the presence of an attention deficit. Those participants who did not have an attention deficit as per our criteria were advised and thanked for their participation. The 84 patients identified as having an attention deficit were administered the Barthel Index and underwent further neuropsychological testing (details of the measures given above) to obtain baseline data for memory, spatial functions and language, health related quality of life measures plus other demographic data including other therapies and health services being received. (See Table 4 for Schedule of Assessments).

The participants were then randomised into either the standard care group or the treatment and standard care group. Randomisation was concealed using an

internet on-line service based at the Clinical Trials Research Unit of the University of Auckland. Randomisation by minimisation was used to ensure the balance for possible prognostic factors, namely gender, age (<70yrs, >70yrs), ethnicity, Barthel Index Score ((18 and above (high) or below 18 (very high), and hospital site. The participant was then informed whether they had been randomised into the standard care group or the treatment plus standard care group. The participants in the standard care group received ad hoc individual therapy provided to address specific neuropsychological deficits, most notably visual neglect/inattention which typically involved Occupational Therapy. Speech Language Therapy, Physiotherapy and other rehabilitation programmes (such as Stroke Recovery Education) were also part of standard care as required. Those participants randomised to the APT group received APT in addition to standard care.

In order to reduce measurement bias, assessments were carried out by a research associate, blind to the treatment status of the participant, while treatment was conducted by a neuropsychologist (the author). Participants were also cautioned not to reveal to the research associate, their treatment status. Participants who were randomised into the treatment group were scheduled to start treatment as soon as practicable, which was usually the next day. Follow-up data was obtained by the research associate during the 5th week after randomisation and assessment sessions took place either in hospital, at a rehabilitation in-patient facility, or at the participant's home if they had been discharged from hospital.

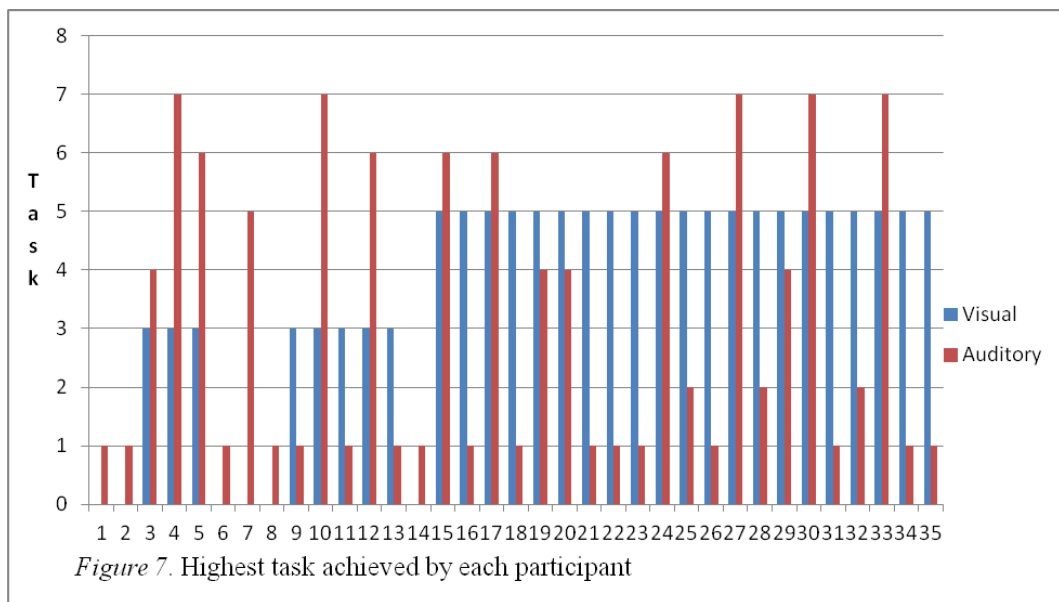
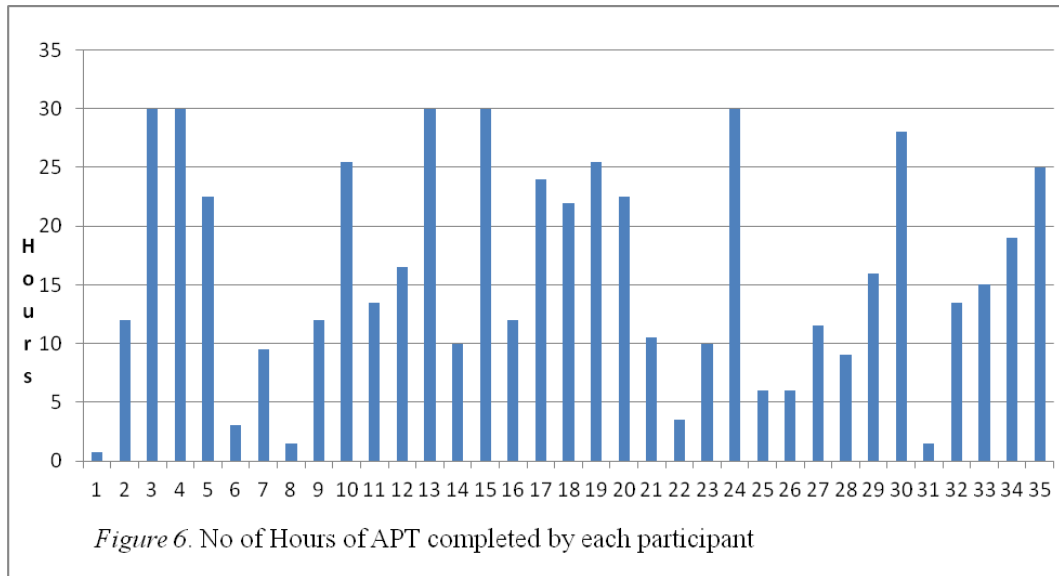
### **Therapy Regime.**

The participants who were randomised into the APT treatment group received up to 30 hours of individual APT. Thirty hours of treatment was chosen as this falls within the range of APT treatment lengths reported in evaluations within the existing literature and is within the usual length of stay in inpatient neurological rehabilitation units. Participants were scheduled each weekday for 1.5 hours treatment sessions, for four weeks. This treatment schedule was consistent with other rehabilitation services provided in in-patient neurological rehabilitation settings. When possible each session was conducted in two forty minute blocks with a 10 minute break between blocks. Participants began treatment on the next available day which was normally the day after randomisation. Treatment took place either at bedside if the participant was in a single room, in a pre-booked room within the ward, or at the participant's place of residence if they had been discharged. The amount of APT each patient received varied greatly and was largely determined by the participant's tolerance of the therapy on any particular day.

Compliance was monitored and recorded by the treating neuropsychologist and included data on the number of hours of total APT training (See Figure 6) and the tasks that were completed (See Figure 7). Information for when treatment commenced and ended as well as the reasons for the participant completing less than 30 hours, was recorded. As can be seen in Figure 7 many more people progressed through the auditory tasks compared to the visual tasks. The highest auditory task that was reached was Task 7 however nobody reached the task (Task 8) at the highest level in this modality. Although two participants did reach the

highest visual task (Task 10), they were also the only two who progressed beyond Task 3 which was the second level of difficulty in the visual modality.

The clinical data relating to the performance of participants on APT will be presented in the following chapter as well as the data from statistical analyses. A discussion on both sets of data will then be presented in the final chapter.



## Chapter 5: Results

In the current study the analyses were conducted in four parts. The focus of Part 1 of this chapter is the analyses of baseline data. Descriptive data regarding the demographic characteristics of the participants are presented and statistical analyses are conducted to compare differences in characteristics between the Attention Process Training (APT) and standard care groups. Following this, data regarding the participants' performance on the baseline neuropsychological measures are presented and the findings from statistical analyses comparing baseline performances between the APT and standard care groups are summarised. The last part of this section will present data from a series of correlation analyses conducted to investigate the relationships between baseline attention measures with demographic variables, functional variables and other neuropsychological measures.

In Part two of this chapter, the main hypothesis of this study, (i.e., examination of the effect of APT on attention deficit in acute stroke patients), was investigated. A series of 2 x 2, analyses of variance (ANOVAs) were conducted with APT and standard care as the between-group factor and time at baseline and post-treatment as the within subjects factor with each of the attention measures as the dependent variable. The statistical analysis regarding the effect of APT on the secondary outcome measure, the SF-36, is also reported.

Part three focuses on the qualitative data obtained from the study. All baseline neuropsychological measures for both APT and standard care groups were placed into qualitative descriptive categories (e.g., average range, below average range, etc.) and an examination of the participant's changes across qualitative descriptive categories from baseline to post-treatment was conducted.

Finally, Part four provides data for those factors that may have influenced how the participants engaged in the intervention process. Factors investigated included the relationships between demographics and performance on neuropsychological measures with how far they reached on both auditory and visual tasks as well as the number of completed hours of APT.

### **Part 1: Analyses of Baseline Measures**

In order to determine if there were any significant differences between the demographic characteristics of the APT and standard care groups at baseline, a series of independent t-tests for continuous variables, a chi square analysis for gender and Fisher's exact test for categorical variables with fewer than 5 frequencies were conducted, the results of which can be seen in Table 7. The results of the analyses show that the average age for the two groups and their levels of education were similar. There were a greater number of Maori participants in the standard care group compared to the APT group but this was not a significant difference and overall ethnicity was distributed evenly across both groups. There was also a larger group of widowed participants in the standard care group compared to the APT group; however, overall marital status did not differ significantly between the two groups. The majority of participants in both groups experienced ischaemic strokes and although there were a higher number of lacunar strokes in the standard care group compared to the APT group, distribution of the type of stroke was evenly distributed across the two groups. Left hemispheric strokes were also more predominant in the standard care group but overall there was no statistical difference between the groups for side of lesion. The length of time since their stroke had occurred was approximately the same for both groups. MMSE and Barthel Index scores were similar across groups

revealing two groups of similar broad cognitive and functional levels. The latter was somewhat expected given that the Barthel Index was one of the factors of randomisation. Although there were more males than females in both groups, ‘gender’ was balanced across both groups. Overall the results of these analyses show that the two groups were well-balanced for all demographic variables.

Table 7  
*Demographics of participants randomised to APT group and standard care group*

Demographic	APT Treatment Group <i>n</i> =35	Standard Care Group <i>n</i> =43	Significance of difference (X <sup>2</sup> FET, or t, p)
Age			
Mean (SD)	69.54 (16.08)	68.51 (15.31)	t(76) = 0.29, p = .77
Gender, N (%)			
Male	21 (60)	26 (60.5)	X <sup>2</sup> (1) = 0.002 p >.05
Female	14 (40)	17 (39.5)	
Ethnicity, N (%)			
European	28 (80)	33 (76.7)	FET = 3.42, p >.05
Maori	2 (5.70)	7 (16.3)	
Pacific Island	4 (11.40)	3 (7)	
Indian	1 (2.90)		
Education N (%)			
Primary	2 (5.70)	5 (11.6)	FET = 2.67, p >.05
Secondary	22 (62.90)	28 (65.1)	
Polytechnic	5 (14.30)	2 (4.7)	
University	6 (17.10)	8 (18.6)	
Marital Status N (%)			
Married/De Facto	24 (68.60)	24 (55.8)	FET = 3.71, p >.05
Widowed	4 (11.40)	12 (27.9)	
Never Married	2 (5.70)	1 (2.3)	
Separated/Divorced	5 (14.30)	6 (14)	
Stroke Type N (%)			
Ischaemic	28 (80)	40 (93)	FET = 6.97, p >.05
TACS	3 (8.6)	3 (7.0)	
PACS	12 (34.3)	15 (34.9)	
LACS	1 (2.9)	7 (16.3)	
POCS	2 (5.7)	3 (7.0)	
Uncertain	10	12	
Intracerebral	2 (5.70)	1 (2.3)	
Subarachnoid	3 (8.60)	0	
Unknown	2 (5.70)	2 (4.7)	
Hemisphere of lesion N (%)			
Left	14 (43.8)	25(58.1)	FET = 4.38, p >.05
Right	15 (46.9)	18(41.9)	
Unknown	3 (9.4)		
Time since stroke - Days			
Mean (SD)	-18.48 (11.95)	-18.58 (7.61)	t(76) = 0.04, p = .97
Barthel Index			
Mean (SD)	14.60 (5.38)	14.33 (5.81)	t(76) = 0.21, p = .83

APT = Attention process Training, LAC = Lacunar Stroke, MMSE = Mini Mental State Examination, N = Number, PACS = Partial Anterior Circulation Stroke, POCS = Posterior Circulation Stroke, SD = Standard Deviation, TACS= Total Anterior Circulation Stroke.



All raw scores from the neuropsychological tests were standardised by converting them to z-scores using normative data obtained in respective test manuals or from a compendium of neuropsychological tests (as noted on page 80 of the Methods Chapter). Independent-sample t-tests were conducted to determine if there were significant differences between the APT group and the standard care group's performance/scores on any of the neuropsychological baseline measures and health related quality of life measures. For clarity, t-test results are summarised in two separate tables. Table 8 provides the results for all the attention measures and the SF-36 which were the measures that were administered at baseline and again at post-intervention. Table 9 provides the results of t-tests for those other measures that were administered at baseline and at 6 months post-stroke and include all other neuropsychological measures, plus all other health related quality of life measures including the mRS, the CFQ and the GHQ. However, as can be seen in Tables 8 and 9 completion rates varied considerably across measures. The factor that most influenced non-completion of the tasks was heightened fatigue where participants were just too tired to do the task. Other participants were unable to complete certain measures because of their aphasia, hemiplegia or problems with vision. A number of participants were overwhelmed by some tasks (particularly the PASAT) and declined to engage in the task at all.

As can be seen in Table 8 there was a significant difference between the two groups on the Full Attention Score of the IVA-CPT with the standard care group obtaining a significantly higher score. Ideally, an

analysis of co-variance (ANCOVA) controlling for this difference would have been conducted, however, this was not possible because the covariate (the Full Scale Attention score) is not independent from the experimental effect (the dependent variable) (Field, 2009).

Table 8

*Performance of the APT and SC Groups on baseline measures of attention and the SF-36. (Data represented as z-scores apart from the Bells Test which is the raw score)*

Measure	<i>n</i>	APT <i>M(SD)</i>	<i>n</i>	SC <i>M(SD)</i>	<i>t-test</i>
IVA-CPT					
FSAQ	34	-5.14 (3.40)	40	-3.41 (2.98)	<i>t</i> (72) = -2.33, <i>p</i> = .023*
AAQ	34	-4.16 (3.16)	40	-3.25 (2.79)	<i>t</i> (72) = -1.32, <i>p</i> = .192
VAQ	34	-4.63 (3.79)	40	-3.33 (3.40)	<i>t</i> (72) = -1.56, <i>p</i> = .124
FSRQ	34	-2.79 (3.96)	40	-1.28 (2.79)	<i>t</i> (72) = -1.93, <i>p</i> = .058
APQ	34	-1.54 (3.80)	40	-1.03 (2.95)	<i>t</i> (72) = -0.66, <i>p</i> = .515
VPQ	34	-2.05 (4.05)	40	0.95 (2.80)	<i>t</i> (72) = -1.37, <i>p</i> = .175
TMT					
A	28	-2.82 (3.97)	38	-3.70 (5.40)	<i>t</i> (64) = 0.72, <i>p</i> = .472
B	22	-2.24 (2.90)	33	-3.07 (3.70)	<i>t</i> (53) = 0.89, <i>p</i> = .379
PASAT					
2.4	19	-1.65 (0.95)	20	-1.57 (0.53)	<i>t</i> (37) = -0.35, <i>p</i> = .731
2.0	19	-1.27 (0.83)	20	-1.26 (1.00)	<i>t</i> (37) = -0.04, <i>p</i> = .971
Bells-Raw scores					
Left	32	11.69 (4.92)	43	12.51 (4.66)	<i>t</i> (73) = -0.74, <i>p</i> = .461
Centre	32	4.28 (1.14)	43	4.33 (1.44)	<i>t</i> (73) = -0.14, <i>p</i> = .886
Right	32	13.41 (4.28)	43	13.37 (2.74)	<i>t</i> (73) = -0.05, <i>p</i> = .959
SF-36					
PCS	35	31.35 (9.91)	43	34.27 (10.66)	<i>t</i> (76) = 1.24, <i>p</i> = .218
MCS	35	46.17 (11.34)	43	42.79 (11.35)	<i>t</i> (76) = 1.31, <i>p</i> = .195

\* *p* < .05

APT = Attention Process Training, AAQ = Auditory Attention Quotient, APQ = Auditory Prudence Quotient, FSAQ = Full Attention Quotient, FSRQ = Full Scale Response Quotient, IVA-CPT = Integrated Visual and Auditory Continuous Performance Test, N = Number, PASAT = Paced Auditory Serial Addition Test, SC = Standard Care, SD = Standard Deviation, TMT = Trail Making Test, VAQ = Visual Attention Quotient, VPQ = Visual Prudence Quotient.

In Table 9 it can be seen that the only significant difference between the groups was on the CVLT Recognition trial with the standard care group obtaining significantly lower scores than the APT group at baseline. To summarise, it was found that the performance of the two groups differed significantly on two neuropsychological measures, i.e. the FSAQ of the IVA-CPT and the Recognition trial of the CVLT. The difference on the FSAQ has implications for the study given that it is the

primary outcome measure. However, the results of this analysis show that overall the two groups were similar on all other measures of attention other baseline neuropsychological measures and health related quality of life measures.

Table 9.

*Performance of the APT and standard care groups on baseline neuropsychological measures of executive functions, memory and language and remaining health related quality of life measures (neuropsychological data are presented as z-scores)*

Measure	APT		sc		<i>t</i> -test
	<i>n</i>	<i>M</i> ( <i>SD</i> )	<i>n</i>	<i>M</i> ( <i>SD</i> )	
Stroop					
Dot	27	-2.01 (2.80)	34	-2.80 (5.54)	<i>t</i> (59) = 0.67, <i>p</i> = .51
Word	27	-2.04 (2.84)	34	-2.49 (3.19)	<i>t</i> (59) = 0.58, <i>p</i> = .57
Colour	27	-0.82 (1.89)	34	-1.18 (3.52)	<i>t</i> (59) = 0.48, <i>p</i> = .63
ROCF					
Copy	24	-2.75 (3.91)	33	-3.16 (4.31)	<i>t</i> (55) = 0.37, <i>p</i> = .71
ShD	24	-0.72 (1.67)	32	-0.45 (1.65)	<i>t</i> (54) = -0.61, <i>p</i> = .55
LD	24	-0.91 (1.87)	31	-0.49 (1.77)	<i>t</i> (53) = -0.85, <i>p</i> = .40
Recognition	24	-2.05 (2.80)	31	-1.73 (2.76)	<i>t</i> (53) = -0.43, <i>p</i> = .67
VPA					
Learning	30	-0.18 (0.91)	37	-0.55 (1.14)	<i>t</i> (65) = 1.40, <i>p</i> = .16
Delayed	30	-0.25 (0.86)	35	-0.21 (1.09)	<i>t</i> (63) = -0.18, <i>p</i> = .86
LM1	32	0.10 (1.07)	34	-0.09 (1.16)	<i>t</i> (64) = 0.70, <i>p</i> = .49
LM11	32	0.19 (1.07)	34	0.22 (1.12)	<i>t</i> (64) = -0.10, <i>p</i> = .92
CVLT					
Trial 1	33	-0.85 (1.28)	40	-0.55 (1.60)	<i>t</i> (71) = -0.87, <i>p</i> = .39
ShD Free	33	-0.65 (1.24)	38	-0.76 (1.33)	<i>t</i> (69) = -0.36, <i>p</i> = .72
LD Free	33	-0.68 (1.19)	40	-1.03 (1.45)	<i>t</i> (71) = 1.09, <i>p</i> = .28
Recognition	33	-1.48 (1.62)	40	-0.65 (1.30)	<b><i>t</i>(71) = -2.44, <i>p</i> = .02*</b>
BNT	32	-1.11 (2.12)	36	-0.91 (2.36)	<i>t</i> (66) = -0.37, <i>p</i> = .72
COWA					
Word	31	-0.95 (1.01)	38	-0.91 (1.11)	<i>t</i> (67) = -0.14, <i>p</i> = .89
mRScale	35	2.66 (1.26)	43	2.49 (1.32)	<i>t</i> (76) = 0.57, <i>p</i> = .57
CFQ	35	24.26 (12.72)	43	27.07 (11.56)	<i>t</i> (76) = -1.02, <i>p</i> = .31
GHQ	35	6.66 (4.62)	43	7.74 (4.90)	<i>t</i> (76) = -1.00, <i>p</i> = .32

**\**p* < 0.05 level (2.tailed)**

APT = Attention Process Training, BNT = Boston Naming Test, CFQ = Cognitive Failures Questionnaire, COWA = Controlled Oral Word Association, CVLT = California Verbal Learning Test, GHQ = General health Questionnaire, LD = Long Delay, LD Free = Long Delay Free Recall, LM1 = Logical Memory 1, LM11 = Logical Memory 11, MCS = Mental Component Score, mRS = Modified Rankin Scale, N = number, PCS = Physical Component Score, ROCF = Rey Osterreith Complex Figure, sc = standard care, SD = Standard Deviation, ShD = Short Delay, SD Free = Short Delay Free recall, SF-36 = Short Form Health Survey-36, VPA = Visual Paired Associates,

Pearson's bi-variate correlations were used to examine the relationships between attention and all continuous variables (age, time since stroke, education, Barthel Index and Mini Mental State Exam scores). Spearman's rank order

correlation was used to determine the relationship between attention and gender. The correlations are presented in Table 10.

As can be seen in Table 10, there was no significant relationship between age and any attention measure, however, time since stroke was significantly related to all measures of the IVA-CPT except the Auditory Prudence score, and both trials of the PASAT. Overall, the results show that the longer the time that had elapsed since the stroke occurred, the better the performance was on attention measures. There was a significant negative relationship between gender and the 2.0 second trial of the PASAT with females performing worse than males. On all other measures gender was not a significant factor. There was also a significant relationship between education and the Full Response and Auditory Prudence scores of the IVA-CPT with higher levels of education relating to better scores on those measures.

Functional independence, as measured by the Barthel Index, was significantly related to increased attention as measured by the IVA-CPT Full Attention Scale, the Visual Attention Scale and Bells Centre. An even stronger relationship between the Barthel Index and Part A of the Trail Making Test, and Bells Left and Right was revealed. These were all positive relationships with the implication that higher scores on those measures of attention equates to a greater level of functional independence. Scores on the MMSE were highly correlated with four indices of the IVA-CPT as well as Bells Left and Right and TMT A. That is, less cognitive impairment related to higher scores on those measures of attention.

Given that multiple correlations were conducted a Bonferroni correction was considered. The Bonferroni correction adjusts the alpha level by dividing .05

by the number of tests being conducted thereby minimising the likelihood of finding a significant finding by chance. Therefore, the advantage of the Bonferroni correction reduces the risk of Type 1 errors, i.e., erroneously concluding the presence of a significant correlation; however, the disadvantage is an increase in the risk of Type 2 errors. i.e., concluding the presence of a non-significant correlation. This disadvantage is a point of contention in the debate of the use of the Bonferroni correction (Nakagawa, 2004). Therefore, given that the correlations being looked at in this study were not primary outcomes and the relationships were only points of interest, it was decided not to use the Bonferroni correction.

Table 10

*Correlations of demographic and functional variables with baseline measures of attention*

	Age <i>n</i> = 87	Time Since Stroke <i>n</i> = 79	Gender <i>n</i> = 87	Education <i>n</i> = 87	Barthel <i>n</i> = 87	MMSE <i>n</i> = 87
<b>IVA -CPT</b>						
FSAQ	-.10	<b>0.28*</b>	.14	.13	<b>0.23*</b>	<b>0.38**</b>
AAQ	-.09	<b>0.25*</b>	.17	.13	.17	<b>0.40**</b>
VAQ	-.10	<b>0.28*</b>	.12	.12	<b>0.22*</b>	<b>0.40**</b>
FSRQ	-.01	<b>0.25*</b>	-.00	<b>0.23*</b>	.09	<b>0.29**</b>
VPQ	.13	<b>0.26*</b>	.02	.19	.09	0.17
APQ	.06	.21	-.05	<b>0.26*</b>	-.01	0.14
<b>TMT</b>						
A	-.01	<b>0.32**</b>	.06	-.02	<b>0.28**</b>	<b>0.23*</b>
B	<b>.27*</b>	<b>0.34**</b>	-.03	-.07	.18	<b>0.24*</b>
<b>PASAT</b>						
2.4	.01	-.05	-.21	.07	.15	.03
2.0	.15	.19	<b>-0.32*</b>	.17	.17	.18
<b>Bells</b>						
Left	-.06	<b>0.34**</b>	.09	-.06	<b>0.36**</b>	<b>2.88**</b>
Centre	.08	<b>0.34**</b>	.18	.01	<b>0.26*</b>	.20
Right	.01	<b>0.30**</b>	.02	-.01	<b>0.41**</b>	<b>0.37**</b>

**\*\*p < 0.01 level (2-tailed)**

**\*p < 0.05 level (2-tailed)**

AAQ = Auditory Attention Quotient, APQ = Auditory Prudence Quotient, FSAQ = Full Scale Attention Quotient FSRQ = Full Scale Response Quotient, IVA-CPT = Integrated Visual and Auditory Continuous Performance Test, N = Number, PASAT = Paced Auditory Serial Addition Test, SC = Standard Care, SD = Standard Deviation, TMT = Trail Making Test, VAQ = Visual Attention Quotient, VPQ = Visual Prudence Quotient.

As points of interest, further information for each ethnic group was obtained. Unfortunately, statistical comparisons across the ethnic groups could

not be conducted to the widely varying sample sizes. In addition, analyses examining ethnicity and stroke type could not be done due to low participant numbers. However, to obtain an estimation of the severity of the stroke according to ethnic group, the means and standard deviations each ethnic group produced on the Mini Mental State Exam and the Barthel Index were generated, and these can be seen in Table 11. These scores suggest that severity of stroke across each ethnic group was similar. Also of interest was the performance of each ethnic group on baseline measures of attention. The means and standard deviations for those measures were obtained and are also included in Table 11. It can be seen that overall the Pacific Island group performed worse on the IVA-CPT and TMT compared to the other groups. This was followed by Pakeha then Maori and finally Indian in that order. Performance on the fast trial of the PASAT was uniform across groups although on the slower trial Maori and Pacific Island did considerably worse than Pakeha and Indian who were relatively similar. Overall, on the Bells Cancellation Test the Pakeha, Maori, and Pacific Island groups performed similarly and the Indian group performed slightly better. However, these observations were points of interest only and the causes for differences between the ethnic groups may be the result of a number of factors such as stroke location, severity of stroke, low SES, time since stroke, age or years of education to name a few.

Table 11  
*Means and standard deviations of MMSE and BI measures and baseline attention measures according to ethnicity.*

Measure	Pakeha		Maori		Pacific Island		Indian	
	<i>n</i>	<i>M (SD)</i>	<i>N</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
MMSE	72	26.65 (2.71)	11	28.09 (1.51)	8	26.50 (2.67)	3	28.00 (1.00)
BI	45	14.67 (5.69)	11	13.18 (6.72)	8	15.50 (4.31)	3	16.33 (6.35)
IVA-CPT								
FSAQ	65	-3.64 (3.31)	11	-2.75 (3.01)	8	-5.86 (3.59)	3	-1.47 (1.76)
AAQ	65	-3.17 (3.04)	11	-2.61 (2.58)	8	-4.44 (3.68)	3	-1.67 (1.59)
VAQ	65	-3.43 (3.54)	11	-2.65 (3.34)	8	-5.91 (4.29)	3	-1.50 (1.21)
FSRQ	65	-1.65 (3.32)	11	-0.69 (1.76)	8	-3.69 (4.86)	3	-0.60 (1.91)
APQ	65	-0.83 (2.93)	11	-0.06 (1.35)	8	-4.85 (5.29)	3	-1.67 (0.96)
VPQ	65	-0.90 (3.25)	11	-0.94 (2.27)	8	-5.31 (4.59)	3	-0.13 (1.21)
TMT								
A	65	-2.58 (4.57)	9	-2.97 (4.64)	6	-4.89 (4.90)	2	0.90 (0.32)
B	54	-1.74 (2.45)	9	-4.29 (4.67)	6	-5.70 (3.63)	2	0.44 (0.32)
PASAT								
2.4secs	37	-1.31(0.87)	8	-1.78 (0.53)	3	-1.61 (0.98)	3	-1.32 (0.88)
2.0secs	37	-0.90 (0.86)	8	-1.71 (1.22)	3	-1.85 (0.59)	3	-0.85 (0.70)
Bells								
Left	69	12.67 (4.10)	11	11.73 (5.41)	8	11.13 (6.88)	3	14.67 (0.58)
Cntr	69	4.45 (1.13)	11	4.36 (1.50)	8	4.00 (1.77)		5.00 (.00)
Right	69	13.46 (2.58)	11	14.09 (2.70)	8	13.38 (3.46)	3	14.67 (0.58)

APT =Attention Process Training, AAQ = Auditory Attention Quotient, APQ = Auditory Prudence Quotient, BI = Barthel Index, FSAQ = Full Scale Attention Quotient FSRQ = Full Scale Response Quotient, IVA-CPT = Integrated Visual and Auditory Continuous Performance Test, MMSE = Mini Mental State Exam, PASAT = Paced Auditory Serial Addition Test, SC = Standard Care, SD = Standard Deviation, TMT = Trail Making Test, VAQ = Visual Attention Quotient, VPQ= Visual Prudence Quotient.

Another point of interest was the relationship between type of stroke and baseline attention measures. As can be seen in Table 12 participants with POCS performed the best on the FSAQ, the AAQ and the VAQ of the IVA-CPT followed by participants with LACs. Participants with TACs, PACs and haemorrhagic strokes performed the worst on those measures. On the FSRQ, the APQ and the VPQ the POCs scores were a lot more uniform across all groups although overall the participants in the POCs group again performed the best.

On the TMT A the TACs and PACs groups performed worse than the LACs and POCs groups although the haemorrhagic group performed considerably worse than all other groups. TMT B the LACs and POCs groups performed better than the other groups. On both trials of the PASAT all groups were relatively similar as was their performance on the three trials of the Bells Cancellation Test although the LACs group did perform slightly better than the other four groups.

In summary, correlation analyses showed that the most number of significant relationships with baseline attention measures was with time since stroke followed by the MMSE, then the BI score, then education with finally just one significant correlation with gender and age. Stroke severity seems similar across the different ethnic groups. The means and standard deviations generated for ethnicity and stroke type indicated differences in performance on attention measure



Table 12  
*Ms and SDs of baseline attention measures for stroke type*

Measure	TACs		PACs		LACs		POCs		Haemorrhagic	
	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
IVA-CPT										
FSAQ	8	-3.83 (3.36)	30	-3.78 (3.29)	10	-2.49 (3.88)	7	-1.84 (2.07)	7	-3.56 (4.18)
AAQ	8	-3.60 (3.08)	30	-3.04 (2.85)	10	-2.56 (3.68)	7	-2.19 (2.63)	7	-2.97 (3.70)
VAQ	8	-3.70 (3.73)	30	-3.84 (3.55)	10	-2.34 (3.82)	7	-1.21 (1.42)	7	-3.11 (4.80)
FSRQ	8	-1.50 (2.93)	30	-1.69 (3.07)	10	-1.86 (3.61)	7	-0.51 (1.28)	7	-1.17 (4.60)
APQ	8	-4.38 (2.03)	30	-0.55 (2.67)	10	-2.24 (3.95)	7	0.56 (1.01)	7	-1.39 (4.12)
VPQ	8	-1.14 (2.81)	30	-1.51 (3.39)	10	-1.90 (3.46)	7	-0.17 (1.09)	7	-0.70 (4.19)
TMT										
A	7	-2.67 (3.40)	31	-2.68 (5.26)	10	-1.13 (2.50)	5	-1.51 (1.58)	7	-4.57 (7.06)
B	5	-2.44 (2.45)	28	-2.07 (2.70)	10	-1.16 (1.44)	5	-1.54 (0.91)	6	-2.22 (4.08)
PASAT										
2.4secs	4	-1.16 (1.01)	18	-1.09 (0.91)	7	-1.18 (0.46)	5	-1.47 (0.36)	5	-1.39 (0.91)
2.0secs	4	-0.74 (1.15)	18	-1.01 (1.17)	7	-0.79 (0.80)	5	-0.89 (0.49)	5	-1.17 (0.95)
Bells										
Left	8	12.63 (3.20)	33	12.24 (4.63)	11	14.55 (0.52)	7	13.71 (2.98)	7	12.71 (5.62)
Centre	8	4.50 (1.41)	33	4.24 (1.48)	11	4.82 (0.41)	7	4.43 (0.79)	7	4.57 (0.79)
Right	8	10.75 (5.50)	33	14.00 (1.79)	11	14.00 (1.00)	7	13.86 (2.61)	7	13.29 (3.73)

APT = Attention Process Training, AAQ = Auditory Attention Quotient, APQ = Auditory Prudence Quotient, FSAQ = Full Scale Attention Quotient, FSRQ = Full Scale Response Quotient, IVA-CPT = Integrated Visual and Auditory Continuous Performance Test, LACS = Lacunar Stroke, PACS = Partial Anterior Circulation Stroke, PASAT = Paced Auditory Serial Addition Test, POCS = Posterior Circulation Stroke, SC = Standard Care, TACS = Total Anterior Circulation Strokes, TMT = Trail Making Test, VAQ = Visual Attention Quotient, VPQ = Visual Prudence Quotient.

Correlations were also generated to investigate relationships between baseline measures of attention and other neuropsychological measures. The significant correlations presented in Table 13, all represent a positive direction and signify that a higher score on the attention measure is indicative of a better performance on the neuropsychological measure.

The majority of attention scores were significantly correlated to scores on most trials of the Rey Osterreith Complex Figure, although for Bells (Left, Centre and Right), the only other significant correlation was with the BNT. A large number of the IVA-CPT scores correlated strongly with scores on Verbal Paired Associates, particularly the Delayed Recall trial and also with a number of scores on the CVLT. Scores on TMT B correlated strongly with almost all neuropsychological measures except for Logical Memory (1 and 2) and COWA. The 2.4 second trial of the PASAT had a strong positive correlation with all trials of the Stroop while the 2.0 second trial of the PASAT, correlated positively with the Word and Colour trials of the Stroop and all three trials of the CVLT. Of all the other neuropsychological measures, only Logical Memory (both I and II) had no relationship to any attention measure.

Table 13  
Correlations between baseline measures of attention and other neuropsychological measures

Measure	Stroop			ROCF			VPA		BNT	LM		COWA	CVLT			
	Dot	Word	Colour	Copy	Short Delay	Long Delay	Recognition	Learning	Delay Recall	Short Delay	Long Delay	Short Delay	Long Delay	Recognition		
	<i>n</i> = 58	<i>n</i> = 58	<i>n</i> = 58	<i>n</i> = 53	<i>n</i> = 52	<i>n</i> = 52	<i>n</i> = 52	<i>n</i> = 64	<i>n</i> = 63	<i>n</i> = 65	<i>n</i> = 64	<i>n</i> = 64	<i>n</i> = 66	<i>n</i> = 68	<i>n</i> = 70	<i>n</i> = 70
<b>IVA-CPT</b>																
FSAQ	.18	.17	.09	.27	<b>.28*</b>	.27	.27	<b>.25*</b>	<b>.43*</b>	<b>0.26*</b>	.09	.10	.10	.21	.23	<b>.24*</b>
AAQ	-.01	.10	-.05	.14	.11	.07	-.01	.24	<b>.44*</b>	.15	.19	.18	.03	.20	.20	.22
VAQ	<b>.32*</b>	.20	.18	<b>.35*</b>	<b>.40*</b>	<b>.40**</b>	<b>.34*</b>	.24	<b>.39**</b>	<b>.34**</b>	.15	.21	.11	<b>.30*</b>	<b>.31*</b>	<b>.29*</b>
FSRQ	-.03	-.01	-.07	<b>.28*</b>	.26	<b>.28*</b>	<b>.35*</b>	.18	<b>.31*</b>	<b>.27*</b>	-.04	.01	.11	.20	.19	.05
APQ	-.01	.03	.04	.15	.18	.19	.01	.24	<b>.31*</b>	.23	.16	.16	.06	<b>.31**</b>	<b>.30*</b>	.09
VPQ	.11	.22	.15	<b>.30*</b>	<b>.42**</b>	<b>.46**</b>	<b>.40</b>	.22	<b>.28</b>	<b>.27*</b>	.12	.10	.10	.23	<b>.26*</b>	.05
<b>TMT</b>																
A	.25	.23	.17	<b>.40**</b>	<b>.39**</b>	<b>.48**</b>	<b>.48**</b>	.24	.19	<b>.34**</b>	.19	.15	.03	<b>.30*</b>	<b>.30*</b>	<b>.42**</b>
B	<b>.36*</b>	<b>.36*</b>	<b>.45**</b>	<b>.52**</b>	<b>.43**</b>	<b>.43**</b>	.25	<b>.54**</b>	<b>.39**</b>	<b>.51**</b>	.22	.16	.22	<b>.30*</b>	<b>.38**</b>	.24
<b>PASAT</b>																
2.4	<b>.41*</b>	<b>.46**</b>	<b>.58*</b>	.36	.24	.17	.11	<b>.49**</b>	.30	.18	.05	-.04	<b>.33*</b>	.27	.21	<b>.41*</b>
2.0	.31	<b>.45**</b>	<b>.68**</b>	.32	<b>.44*</b>	.37	.17	<b>.49**</b>	.26	.27	.27	.14	.29	<b>.43**</b>	<b>.41*</b>	<b>.34*</b>
<b>Bells</b>																
Left	.16	.16	-.04	<b>.46*</b>	<b>.31*</b>	<b>.46*</b>	<b>.37**</b>	-.04	-.03	<b>.26*</b>	.02	.02	-.03	.11	.10	.16
Centre	.23	.13	-.09	<b>.34*</b>	ns	<b>.35**</b>	<b>.28*</b>	-.11	-.09	<b>.31*</b>	.09	.08	-.01	.07	.08	.13
Right	.21	.19	-.03	<b>.42*</b>	<b>.34*</b>	<b>.39**</b>	.16	-.08	.01	<b>.37**</b>	.10	.11	-.01	.14	.11	.04

\* $p < .05$

\*\*  $p < .01$

AAQ = Auditory Attention Quotient, APQ = Auditory Prudence Quotient, BNT = Boston Naming Test, COWAT = Controlled Oral Word Association Test, CVLT = California Verbal Learning Test, FSAQ = Full Scale Attention Quotient, FSRQ = Full Scale Response Quotient, IVA-CPT = Integrated Visual and Auditory Continuous Performance Test, LM = Logical Memory, N = Number, PASAT = Paced Auditory Serial Addition Test, SC = Standard Care, SD = Standard Deviation, TMT = Trail Making Test, VAQ = Visual Attention Quotient, VPQ = Visual Prudence Quotient.

## **Part 2: Analyses of Primary and Secondary Outcomes**

The focus of Part 2 was to test the main study hypothesis (i.e. does APT improve attention in acute stroke patients with attention deficit?). To examine the effect of APT on attention deficit, a series of mixed 2 x 2 ANOVAs were conducted, with a within subjects factor of time (pre- and post-intervention), and a between subjects factor of treatment type (APT or standard care). Means and standard deviations for attention measures and the SF36 for both the APT group and standard care group at baseline and 5 weeks, are presented in Table 14 including the results of the ANOVAs.

The results show that irrespective of the treatment provided between baseline and 5 weeks, performance on the attention scores improved significantly as an effect of time on the IVA-CPT, Full Scale Attention Quotient, Auditory Attention Quotient, Visual Attention Quotient as well as the Full Scale Response Quotient. Significant improvement over time was also demonstrated on other measures of attention including both trials of the Trail Making Test, the 2.0 second interval trial of the PASAT and the Bells left trial. There were no effects on any measures for group type alone. There were, however, significant Group x Time interactions on the IVA-CPT Full Scale Attention Score, Auditory Attention score and the Full Scale Response score. (See Figure 8).

Repeated measures t-tests were used for post hoc analysis to look at changes over time in the APT and standard care group separately for the Full Scale Attention Score, Auditory Attention score and Full Scale Response Score. In these analyses, the Bonferroni correction was used to adjust for multiple comparisons. Results of these analyses showed that significant improvement for the APT group alone

occurred on all three measures. On the FSAQ the APT groups showed a significant improvement from pre to post ( $t(27) = 6.14, p < .01$ ). The APT group also improved significantly on the AAQ ( $t(27) = -4.54, p < .01$ ) and on the FSRQ ( $t(27) = -3.69, p < .01$ ).

There was no significant difference between the baseline and post-treatment scores for the standard care group on any of these three measures. Figure 8 shows the main findings for both the APT and the standard care group.

T-tests were then carried out to investigate whether or not there were any significant differences between the means of the two groups for the IVA-CPT Full Scale Attention score, the Auditory Attention score and the Full Scale Response score at post-intervention. These analyses revealed there were no significant differences between the groups on any of the three measures. In terms of the IVA-CPT Full Scale score the results of the t-test suggest that the APT group made greater improvement than their standard care group counterparts given that the APT group was found to be significantly worse than the standard care group at baseline for this measure.

There were no significant differences between baseline and post-intervention measures on the secondary measure, the SF-36, as a result of time nor did time interact with group. In summary, the analyses show an improvement on eight measures of attention as an effect of time and significant improvement on three quotients of the IVA-CPT as a result of Group x Time interactions although no differences were detected on the secondary measure, the SF-36.

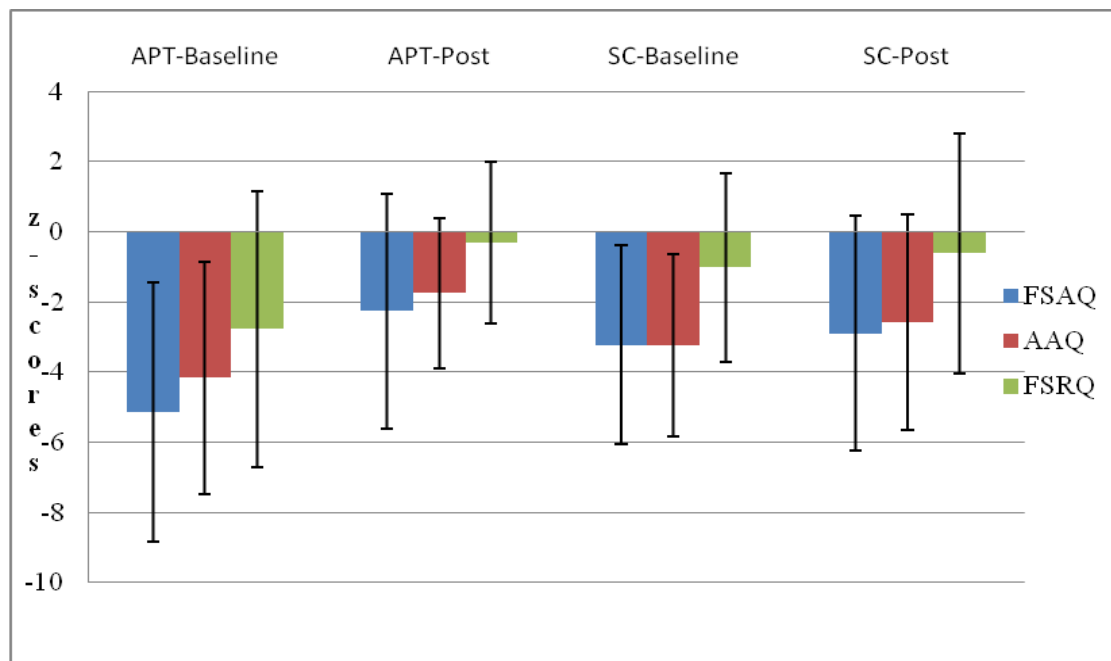
Table 14  
Descriptive and inferential statistics showing significant results in bold

Measures of Attention	Baseline			Post APT			ANOVA Results Main Effect Time	Main effect Group	Interaction (Time x Group)
	APT	SC	Average	APT	SC	Average			
	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>			
IVA-CPT									
FSAQ	-5.14 (3.69)	-3.23 (2.83)	-4.13 (3.38)	-2.26 (3.35)	-2.90 (3.35)	-2.59 (3.34)	<b>F(1,57)=20.94**</b>	F(1,57)=0.65	<b>F(1,57)=13.24**</b>
AAQ	-4.17 (3.30)	-3.23 (2.60)	-3.68 (2.96)	-1.75 (2.15)	-2.58 (3.07)	-2.19 (2.69)	<b>F(1,57)=17.88**</b>	F(1,57)=0.01	<b>F(1,57)=5.97*</b>
VAQ	-4.44 (4.07)	-3.01 (3.24)	-3.69 (3.70)	-2.75 (3.91)	-2.66 (3.39)	-2.71 (3.61)	<b>F(1,57)=7.15**</b>	F(1,57)=0.76	F(1,57)=3.13
FSRQ	-2.78(3.93)	-1.02(2.69)	-1.85(3.42)	-0.31(2.30)	-0.62(3.42)	-0.47(2.92)	<b>F(1,57)=8.84**</b>	F(1,57)=1.21	<b>F(1,57)=4.60*</b>
APQ	-1.53(3.74)	-0.80(2.69)	-1.14(3.22)	-0.54(2.17)	-0.80(3.22)	-0.45(2.77)	F(1,57)=2.21	F(1,57)=0.01	F(1,57)=2.23
VPQ	-1.87(4.05)	-0.83(2.60)	-1.32(3.38)	-0.78(3.38)	0.92(3.38)	-0.85(3.35)	F(1,57)=0.88	F(1,57)=0.42	F(1,57)=1.23
TMT									
A	-2.91 (4.10)	-3.10 (4.80)	-3.01 (4.44)	-1.62 (3.60)	-1.67 (2.96)	-1.65 (3.25)	<b>F(1,53)=11.04**</b>	F(1,53)=0.01	F(1,53)=0.03
B	-2.28 (3.04)	-3.24 (4.01)	-2.81 (3.61)	-1.10 (3.10)	-1.68 (2.26)	-1.42 (2.65)	<b>F(1,43)=12.35**</b>	F(1,43)=0.79	F(1,43)=0.23
PASAT									
2.4 secs	-1.49 (0.95)	-1.68 (0.55)	-1.58 (0.78)	-1.11 (1.01)	-1.73 (0.75)	-0.82 (1.02)	F(1,26)=1.66	F(1,26)=1.88	F(1,26)=2.80
2.0 secs	-1.11 (0.83)	-1.51 (1.15)	-1.30 (1.00)	-0.39 (0.96)	-1.31 (0.89)	-0.82 (1.02)	<b>F(1,26)=8.54**</b>	F(1,26)=4.13	F(1,26)=2.67
Bells									
Left	11.62 (5.02)	12.49 (4.62)	12.09 (4.78)	12.79 (2.70)	12.74 (4.05)	12.77 (3.48)	<b>F(1,62)=4.87*</b>	F(1,62)=0.16	F(1,62)=2.00
Centre	4.34 (1.17)	4.34 (1.39)	4.34 (1.29)	4.48 (0.78)	4.66 (0.87)	4.58 (0.83)	F(1,62)=2.42	F(1,62)=0.14	F(1,62)=0.37
Right	13.55 (2.96)	13.40 (2.91)	13.47 (2.91)	13.76 (1.70)	14.14 (1.81)	13.97 (1.76)	F(1,62)=2.18	F(1,62)=0.05	F(1,62)=0.69
SF-36									
MCS	45.56 (11.47)	43.11(11.34)	44.28(11.39)	45.58(11.47)	46.30(10.23)	45.95 (9.47)	F(1,65)=1.37	F(1,65)=0.16	F(1,65)=1.34

\*\*Significant at the 0.01 level (2-tailed)

\*Significant at the 0.05 level (2-tailed)

AAQ = Auditory Attention Quotient, ANOVA = analysis of variance, APQ = Auditory Prudence Quotient, APT = Attention Process Training, FSAQ = Full Scale Attention Quotient, FSRQ = Full Scale Response Quotient, IVA-CPT = Integrated Visual and Auditory Continuous Performance Test, MCS= Mental Component Score, PASAT= Paced Auditory Serial Addition Task, PCS= Physical Component Score, SC = Standard Care, SF-36 = Short Form Health Survey-36, TMT= Trail Making Test, VAQ = Visual Attention Quotient, VPQ = Visual Prudence Quotient



*Figure 8.* The effects of APT on IVA-CPT measures. Data presented as means and standard deviations.

### Part 3: Analysis of qualitative data

Although the means of baseline measures were similar across the two groups it was also of interest to look at the distribution of participants from each group into qualitative descriptive categories. Qualitative descriptive terms are commonly used in clinical practice (Lezak, 2004) to describe performance, providing further clarity and understanding of an obtained numerical score. In this study the range of z-scores were classified into seven distinct qualitative groups according to the number of standard deviations from the mean as follows:  $<-3$  SDs = Very impaired,  $\geq-3$  to  $<-2$ = Impaired,  $>-2$  to  $<-1$ =Below Average,  $>-1$  to  $<1$ =Average,  $>1$  to  $<2$ = Above Average,  $>2$  to  $<3$ =Superior and  $>3$ =Very Superior (Spreen & Strauss, 1998; Strauss, Sherman & Spreen, 2006). However, scores on the Bells test were not converted to z-scores therefore participants were

grouped according to the actual number of bells (0, 1, 2, 3, or >3), missed on each trial. The number of participants in each group is presented in percentages.

Table 15 provides a summary of the proportion of individuals in each group (APT and SC) whose z-scores fell into each qualitative category for each measure of attention. Table 16 provides a summary of the proportion of individuals in each group (APT and standard care) whose z-scores fell into each qualitative category on the other neuropsychological measures of executive functioning, memory and language.

As seen in Table 15, a large proportion of participants in both the APT and standard care groups had poor attention abilities as evidenced by the number of participants who obtained scores in the impaired ranges on the Full Attention, Auditory Attention and Visual Attention scores of the IVA CPT, with the largest percentage of these scores falling in the very impaired range. Only one participant obtained a score that placed him/her above the average range. Scores on the IVA-CPT Full Response were relatively evenly distributed across ranges although there were more scores in the very impaired range for the APT group than the standard care group. Scores on the IVA-CPT Auditory Prudence were also more evenly distributed with most scores falling in the average range. Both groups were similar in their distribution of scores. On IVA-CPT Visual Prudence, while a large percentage of scores fell in the impaired range, there were also more scores falling in the superior range. However, again the distribution of scores in both groups was similar. On the Trail Making Test, Part A and B, all scores on this task fell within the very impaired to average range with no scores reaching the above average range. On Part B of the TMT there were more participants from the standard care group in the very impaired range compared to the APT group.



On the PASAT, there were fewer scores at either end of the range and is likely attributable to fewer participants completing this task. Only 28 participants were able to complete the 2.0 sec trial and 38 participants completed the 2.4 sec trial.

On the Bells test the majority of participants from both groups circled all the bells.

Table 15  
Proportion of participants in each qualitative category on measures of attention

Measures of Attention	z-score ranges																		
	< -3		≥ -3 to < -2		≥ -2 to < -1		≥ -1 to < 1		≥ 1 to < 2		≥ 2 to < 3								
	Very impaired		Impaired		Below Average		Average		Above Average		Superior								
	APT	SC	APT	SC	APT	SC	APT	SC	APT	SC	APT	SC							
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)							
IVA-CPT																			
FSAQ	24 (68.6)	19 (44.2)	3 (8.6)	5 (11.6)	4 (11.4)	7 (16.3)	3 (8.6)	9 (20.9)	0	0	0	0							
AAQ	19 (54.3)	19 (44.2)	4 (11.4)	3 (7.0)	6 (17.1)	11 (25.6)	4 (11.4)	7 (16.3)	1 (2.9)	0	0	0							
VAQ	19 (54.3)	13 (30.2)	4 (11.4)	11 (25.6)	3 (8.6)	7 (16.3)	8 (22.9)	9 (20.9)	0	0	0	0							
FSRQ	13 (37.1)	6 (14.0)	4 (11.4)	8 (18.6)	3 (8.6)	5 (11.6)	10 (28.6)	15 (34.9)	3 (8.6)	4 (9.3)	1 (2.9)	2 (4.7)							
APQ	7 (20.0)	7 (16.3)	3 (8.6)	2 (4.7)	1 (2.9)	4 (9.3)	16 (45.7)	19 (44.2)	7 (20.0)	7 (16.3)	0	1 (2.3)							
VPQ	10 (28.6)	7 (16.3)	1 (2.9)	3 (7.0)	4 (11.4)	4 (9.3)	14 (40.0)	16 (37.2)	3 (8.6)	8 (18.6)	2 (5.7)	2 (4.7)							
TMT																			
A	8 (22.9)	13 (30.2)	5 (14.3)	4 (9.3)	4 (11.4)	5 (11.6)	11 (31.4)	16 (37.2)	0	0	0	0							
B	5 (14.3)	12 (27.9)	4 (11.4)	4 (9.3)	4 (11.4)	6 (14.0)	9 (25.7)	11 (25.6)	0	0	0	0							
PASAT																			
24	0	0	6 (17.1)	5 (11.6)	10 (28.6)	13 (30.2)	2 (5.7)	2 (4.7)	1 (2.9)	0	0	0							
20	0	1 (2.3)	4 (11.4)	4 (9.3)	8 (22.9)	4 (9.3)	7 (20.0)	11 (25.6)	0	0	0	0							
					% with specified number of Bells missed														
Bells-raw score*		>3		3		2		1		0									
Left	28.8		13.9		5.7		7.0		2.9		9.3		14.3		20.9		40.0		48.8
Centre	2.9		9.3		2.9		2.3		11.4		0		20.0		16.3		54.3		72.1
Right	11.6		13.9		2.9		4.7		5.7		11.6		22.9		23.3		48.6		46.5

APT = Attention Process Training, AAQ = Auditory Attention Quotient, APQ = Auditory Prudence Quotient, FSAQ = Full Scale Attention Quotient, FSRQ = Full Scale Response Quotient, IVA-CPT = Integrated Visual and Auditory Continuous Performance Test, PASAT = Paced Auditory Serial Addition Test, SC = Standard Care, SD = Standard Deviation, TMT = Trail Making Test, VAQ = Visual Attention Quotient, VPQ = Visual Prudence Quotient

\*Bell scores are not converted to z-scores

Table 16  
*Proportion of participants in each qualitative category on measures of executive functioning, language and memory*

Measure	z-score ranges											
	< -3		≥-3 to <-2		≥-2 to <-1		≥-1 to <1		≥ 1 to <2		≥2 to <3	
	Very impaired		Impaired		Below Average		Average		Above Average		Superior	
	APT n (%)*	SC n (%)	APT n (%)	SC n (%)	APT n (%)	SC n (%)	APT n (%)	SC n (%)	APT n (%)	SC n (%)	APT n (%)	SC n (%)
Stroop												
Dot	8 (22.9)	10 (23.3)	3 (8.6)	1 (2.3)	1 (2.9)	6 (14.0)	14 (40.0)	16 (37.2)	1 (2.9)	1 (2.3)	0	0
Word	8 (22.9)	11 (25.6)	1 (2.9)	6 (14.0)	4 (11.4)	3 (7.0)	14 (40.0)	13 (30.2)	0	1 (2.3)	0	0
Colour	3 (8.6)	4 (9.3)	3 (8.6)	2 (4.7)	3 (8.6)	5 (11.6)	16 (45.7)	20 (46.5)	2 (5.7)	3 (7.0)	0	0
ROCF												
Copy	8 (22.9)	12 (27.9)	2 (5.7)	4 (9.3)	2 (5.7)	3 (7.0)	12 (34.3)	12 (27.9)	0	2 (4.7)	0	0
ShD	3 (8.6)	1 (2.3)	1 (2.9)	5 (11.6)	6 (17.1)	10 (23.3)	11 (31.4)	7 (16.3)	2(5.7)	7 (16.3)	1 (2.9)	2 (4.7)
LD	4 (11.4)	3 (7.0)	4 (11.4)	3 (7.0)	2 (5.7)	9 (20.9)	9 (25.7)	7 (16.3)	3 (8.6)	7 (16.3)	1 (2.9)	2 (4.7)
Rec	4 (11.4)	5 (11.6)	6 (17.1)	8 (18.6)	5 (14.3)	5 (11.6)	7 (20.0)	10 (23.3)	2 (5.7)	1 (2.3)	0	2 (4.7)
VPA												
1	0	0	0	4 (9.3)	5 (14.3)	10 (23.3)	21 (60.0)	18 (41.9)	4 (11.4)	4(9.3)	0	0
2	0	0	0	1 (2.3)	9 (25.7)	11 (25.6)	21 (60.0)	15 (34.9)	0	8 (18.6)	0	0
BNT	5 (14.3)	3 (7.0)	5 (14.3)	5 (11.6)	4 (11.4)	4 (9.3)	15 (42.9)	20 (46.5)	3 (8.6)	4 (9.3)	0	0
LM 1	0	0	0	1 (2.3)	3 (8.6)	5 (11.6)	22 (62.9)	21 (48.8)	5 (14.3)	5 (11.6)	2 (5.7)	2 (4.7)
LM 2	0	0	1 (2.9)	0	2 (5.7)	4 (9.3)	21 (60.0)	18 (41.9)	6 (17.1)	10 (23.3)	2 (5.7)	2 (4.7)
COWA	0	1 (2.3)	5 (14.3)	6 (14.0)	13 (37.1)	10 (23.3)	13 (37.1)	19 (44.2)	0	2 (4.7)	0	0
CVLT												
ShD Free	1 (2.9)	2 (4.7)	3 (8.6)	6 (14.0)	7 (20.0)	4 (9.3)	17(48.6)	22 (51.2)	5 (14.3)	4 (9.3)	0	0
ShD Cued	1 (2.9)	4 (9.3)	6 (17.1)	3 (7.0)	5 (14.3)	10 (23.3)	18 (51.4)	18 (41.9)	2 (5.7)	5 (11.6)	1(2.9)	0
LD Free	1 (2.9)	2 (4.7)	2 (5.7)	7 (16.3)	8 (22.9)	7 (16.3)	19 (54.3)	21 (48.8)	3 (8.6)	2 (4.7)	0	1 (2.3)
LD Cued	1 (2.9)	4 (9.3)	4 (11.4)	4 (9.3)	9 (25.7)	5 (11.6)	17 (48.6)	24 (55.8)	1 (2.9)	3 (7.0)	1(2.9)	0
Recognition	5 (14.3)	1 (2.3)	5 (14.3)	4 (9.3)	7 (20.0)	5 (11.6)	15 (42.9)	24 (55.8)	1 (2.9)	6 (14.0)	0	0

APT = Attention Process Training, BNT, Boston Naming Test, COWA = Controlled Oral Word Association, CVLT = California Verbal learning Test, LD Cued = Long Delay Cued, LD Free = Long Delay Free, LM = Logical Memory, ROCF = Rey Osterreith Complex Figure, SC = Standard Care, VPA = Visual Paired Associates

As can be seen in Table 16, generally, the greater proportion of scores for each neuropsychological measure fell within the average range. Those which were not in the average range tended to fall below the average range while very few were above the average range. On the Stroop Test, most performances were spread across all ranges although no scores reached the superior range. The largest proportion of participants on this task produced scores that fell within the average range with the next largest group performing in the very impaired range. The APT and standard care groups had similar distributions across most ranges for each trial although the APT group had more participants in the impaired range and the below average range for Stroop Word and Stroop Dot respectively, compared to the standard care group.

On some measures a small number of participants scored in the superior range. This was noted on the short delay and long delay trial of the ROCF, Logical Memory 1 and 2, the short and long delay cued recall and long delay free recall of the CVLT. No participants fell in the very impaired range on both Verbal Paired Associates 1 and 2 or the Logical Memory test 1 and 2 and only one participant from both groups obtained a score that fell in the impaired range for both Logical Memory 1 and 2. However, an equal number of participants from both groups reached the superior range for Logical Memory 1 and 2. These results indicate that the two groups were quite similar on these two measures. Indeed, an overview of all the scores of neuropsychological measures, indicate that overall, the APT and standard care groups were comprised of participants with similar distribution of abilities. This finding is consistent with the results of the earlier analysis when t-tests were conducted to compare performance of baseline neuropsychological measures between the two groups (See Table 9).

### **Changes in qualitative ranges from pre to post-intervention**

In order to track movement across qualitative categories, Table 17 shows the proportion of people in each qualitative category for attention measures at baseline and post-intervention for the APT group. It can be seen that there has been considerable changes in the number of participants falling into each category on most measures. On all indices of the IVA-CPT, there was a considerable reduction in the number of participants in the very impaired range at the post-intervention measure compared to baseline. As a consequence of this change an increase in the number of participants falling in the average, above average, and superior ranges at post-intervention, was observed. These changes reflect the main effect of improvement over time as shown by the results of the ANOVAs in Part 2 of this chapter.

On TMT A, there was a general improvement across all categories as seen by the reduction in the number of participants falling in the below average, impaired and very impaired ranges and an increase in those numbers falling in the average and above average range. On both TMT A and B there was a notable increase in the number of participants in the average range at post-APT. On both trials of the PASAT, there were fewer participants in the below average and impaired range at post-APT and there was an increase in the number of participants in the average range and above average range.

On Bells Left and Bells Centre, there was a reduction in the number of participants who missed more than 3 bells over time and a significant increase in the number of participants who only missed 2 bells on Bells Left. On Bells Right there was also a significant increase in the number of participants who only missed 2 bells.

Table 17

Changes across qualitative categories by the APT group from baseline to post-intervention. Data presented as percent of participants who fell within each category at baseline and post-intervention.

Measure	z-score range											
	< -3		≥ -3 to < -2		≥ -2 to < -1		≥ -1 to < 1		≥ 1 to < 2		≥ 2 to < 3	
	Very impaired		Impaired		Below Average		Average		Above Average		Superior	
	Baseline n (%)	Post n (%)	Baseline n (%)	Post n (%)	Baseline n (%)	Post n (%)	Baseline n (%)	Post n (%)	Baseline n (%)	Post n (%)	Baseline n (%)	Post n (%)
IVA-CPT												
FSAQ	24 (68.6)	10 (28.6)	3 (8.6)	4 (11.4)	4 (11.4)	4 (11.4)	3 (8.6)	7 (20.0)	0	4 (11.4)	0	0
AAQ	19 (54.3)	7 (20.0)	4 (11.4)	7 (20.0)	6 (17.1)	5 (14.3)	4 (11.4)	7 (20.0)	1 (2.9)	2 (5.7)	0	1 (2.9)
VAQ	19 (54.3)	9 (25.7)	4 (11.4)	3 (8.6)	3 (8.6)	5 (14.3)	8 (22.9)	9 (25.7)	0	3 (8.6)	0	0
FSRQ	13 (37.1)	2 (5.7)	4 (11.4)	1 (2.9)	3 (8.6)	4 (11.4)	10 (28.6)	15 (42.9)	3 (8.6)	6 (17.1)	1 (2.9)	1 (2.9)
APQ	7 (20.0)	1 (2.9)	3 (8.6)	0	1 (2.9)	4 (11.4)	16 (45.7)	17 (48.6)	7 (20.0)	7 (20.0)	0	0
VPQ	10 (28.6)	3 (8.6)	1 (2.9)	0	4 (11.4)	4 (11.4)	14 (40.0)	16 (45.7)	3 (8.6)	6 (17.1)	2 (5.7)	0
Trails												
A	8 (22.9)	7 (20.0)	5 (14.3)	1 (2.9)	4 (11.4)	3 (8.6)	11 (31.4)	19 (54.3)	0	1 (2.9)	0	0
B	5 (14.3)	6 (17.1)	4 (11.4)	2 (5.7)	4 (11.4)	4 (11.4)	9 (25.7)	16 (45.7)	0	0	0	0
PASAT												
24	0	0	6(17.1)	3(8.6)	10(28.6)	8(22.9)	2(5.7)	7(20.0)	1(2.9)	1(2.9)	0	0
20	0	0	4 (11.4)	1 (2.9)	8 (22.9)	2 (5.7)	7 (20.0)	14 (40.0)	0	2 (5.7)	0	0
	% with specified number of Bells missed											
	>3		3		2		1		0		0	
Bells-raw score *	Baseline	Post	Baseline	Post	Baseline	Post	Baseline	Post	Baseline	Post	Baseline	Post
Left	28.8	22.6	5.7	5.7	2.9	20.0	14.3	25.7	40.0	17.1		
Centre	2.9	0	2.9	2.9	11.4	8.6	20.0	25.7	54.3	51.4		
Right	11.6	14.4	2.9	2.9	5.7	14.3	22.9	20.0	48.6	37.1		

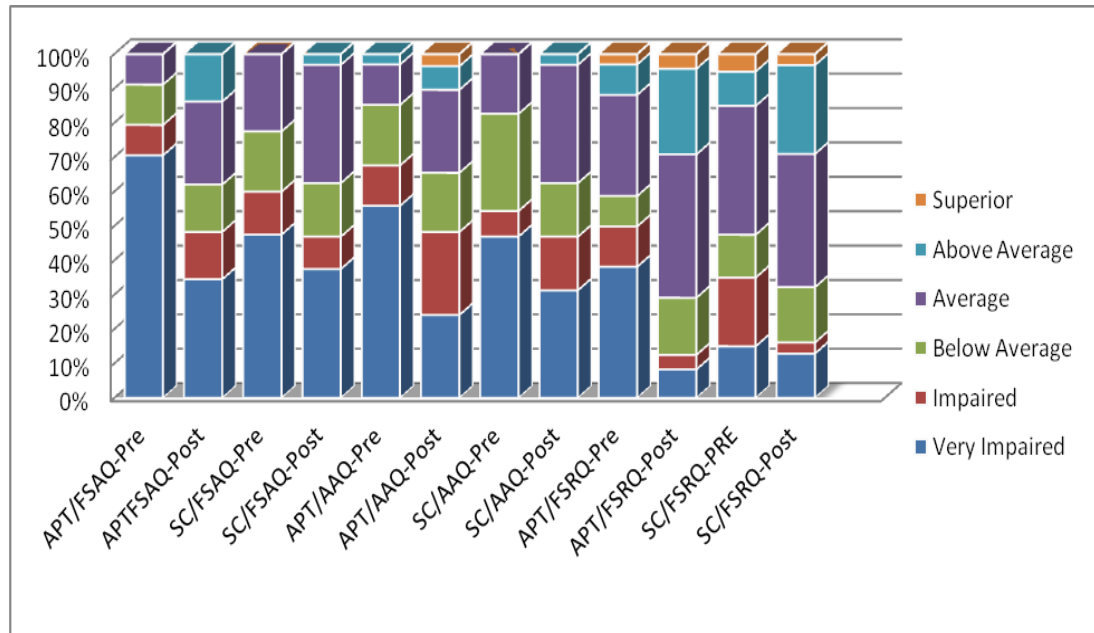
APT = Attention Process Training, AAQ = Auditory Attention Quotient, APQ = Auditory Prudence Quotient, FSAQ = Full Scale Attention Quotient, FSRQ = Full Scale Response Quotient, IVA-CPT = Integrated Visual and Auditory Continuous Performance Test, N = Number, PASAT= Paced Auditory Serial Addition Test, TMT= Trail Making Test, VAQ = Visual Attention Quotient, VPQ = Visual Prudence Quotient..

\*Bell scores are not converted to z-scores

Table 18 presents the qualitative categories for the z-scores obtained by the standard care group at baseline and at post-intervention. There was a reduction in the number of participants in the below average, impaired and very impaired range from pre to post-intervention on all but one index of the IVA-CPT. It can also be seen that on the Full Scale Attention, Auditory Attention and Visual Attention indices, there was an increase in the number of participants reaching the average range and above average range. On the Full Scale Response Index there was an increase in the number of participants in the above average range and one participant in the very superior range at the post-intervention stage. On both trials of the TMT there were fewer participants falling in the very impaired range at post-intervention compared to baseline. Also on both trials of the TMT, there was an increase in the number of participants in the above average range. On the Bells test there was a reduction in the number of participants who missed more than 3 bells on Bells Centre and Bells Right.

Figure 9 shows the changes in categories from baseline to post-treatment for both groups for the main findings. Figure 10, shows category changes from baseline to post-treatment for both groups on all scores of the IVA-CPT. It can be seen that although participants from both groups moved out of the very impaired range on all indices of the IVA-CPT, the percentage of participants that moved was considerably greater for the APT group. Overall, there was more movement into the higher ranges from the APT group although only one participant (from the standard care group) managed to reach the very superior range (Full Response) at post-intervention. It can be seen in Figure 11 that on both trials of the PASAT there were significant positive shifts for participants in the APT group compared to the standard care group where relatively little change over time

occurred. Interestingly, as seen in Figure 12 there was no change in either group for the percentage of participants who missed three bells on all three trials.



*Figure 9.* Bar graph showing the category changes from baseline to post-treatment for the main findings for both the APT and standard care group



Table 18

Changes across qualitative categories by the SC group from baseline to post-intervention. Data presented as percent of participants who fell within each category at baseline and post-intervention

Measure	z-score range													
	< -3		≥-3 to <-2		≥-2 to <-1		≥-1 to <1		≥ 1 to <2		≥2 to <3		>3	
	Very impaired		Impaired		Below Average		Average		Above Average		Superior		V Superior	
	Baseline	Post	Baseline	Post	Baseline	Post	Baseline	Post	Baseline	Post	Baseline	Post	Baseline	Post
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
IVA-CPT														
FSAQ	19 (44.2)	12 (27.9)	5(11.6)	3(7.0)	7 (16.3)	5 (11.6)	9 (20.9)	11 (25.6)	0	1 (2.3)	0	0	0	0
AAQ	19 (44.2)	10 (23.3)	3 (7.0)	5 (11.6)	11 (25.6)	5 (11.6)	7 (16.3)	11 (25.6)	0	1 (2.3)	0	0	0	0
VAQ	13 (30.2)	11 (25.6)	11 (25.6)	2 (4.7)	7 (16.3)	6 (14.0)	9 (20.9)	12 (27.9)	0	1 (2.3)	0	0	0	0
FSRQ	6 (14.0)	4 (9.3)	8 (18.6)	1 (2.3)	5 (11.6)	5 (11.6)	15 (34.9)	12 (27.9)	4 (9.3)	8 (18.6)	2 (4.7)	1 (2.3)	0	1 (2.3)
APQ	7 (16.3)	4 (9.3)	2 (4.7)	2 (4.7)	4 (9.3)	2 (4.7)	19 (44.2)	15 (34.9)	7 (16.3)	9 (20.9)	1 (2.3)	0	0	0
VPQ	7(16.3)	6 (14.0)	3 (7.0)	2(4.7)	4 (9.3)	2 (4.7)	16 (37.2)	14 (32.6)	8 (18.6)	8 (18.6)	2 (4.7)	0	0	0
TMT														
A	13 (30.2)	8 (18.6)	4 (9.3)	2 (4.7)	5 (11.6)	3 (7.0)	16 (37.2)	16 (37.2)	0	3 (7.0)	0	0	0	0
B	12 (27.9)	8 (18.6)	4 (9.3)	6 (14.0)	6 (14.0)	2 (4.7)	11 (25.6)	14 (32.6)	0	1 (2.3)	0	0	0	0
PASAT														
24	0	0	5 (11.6)	7 (16.3)	13 (30.2)	11 (25.6)	2 (4.7)	2 (4.7)	0	0	0	0	0	0
20	1 (2.3)	0	4 (9.3)	6 (14.0)	4 (9.3)	6 (14.0)	11(25.6)	8 (18.6)	0	0	0	0	0	0
	% with specified number of Bells missed													
	>3		3		2		1		0					
	Baseline	Post	Baseline	Post	Baseline	Post	Baseline	Post	Baseline	Post	Baseline	Post	Baseline	Post
Bells														
Left	13.9	13.9	7.0	7.0	9.3	7.0	20.9	23.3	48.8	34.9				
Centre	9.3	2.3	2.3	2.3	0	0	16.3	11.6	72.1	65.1				
Right	13.9	2.3	4.7	4.7	11.6	7.0	23.3	18.6	46.5	48.8				

APT = Attention Process Training, AAQ = Auditory Attention Quotient, APQ = Auditory Prudence Quotient, FSAQ = Full Scale Attention Quotient, FSRQ = Full Scale Response Quotient, IVA-CPT = Integrated Visual and Auditory Continuous Performance Test, N = Number, PASAT= Paced Auditory Serial Addition Task, TMT= Trail Making Test, VAQ = Visual Attention Quotient, VP = Visual Prudence.

\*Bell scores are not converted to z-scores

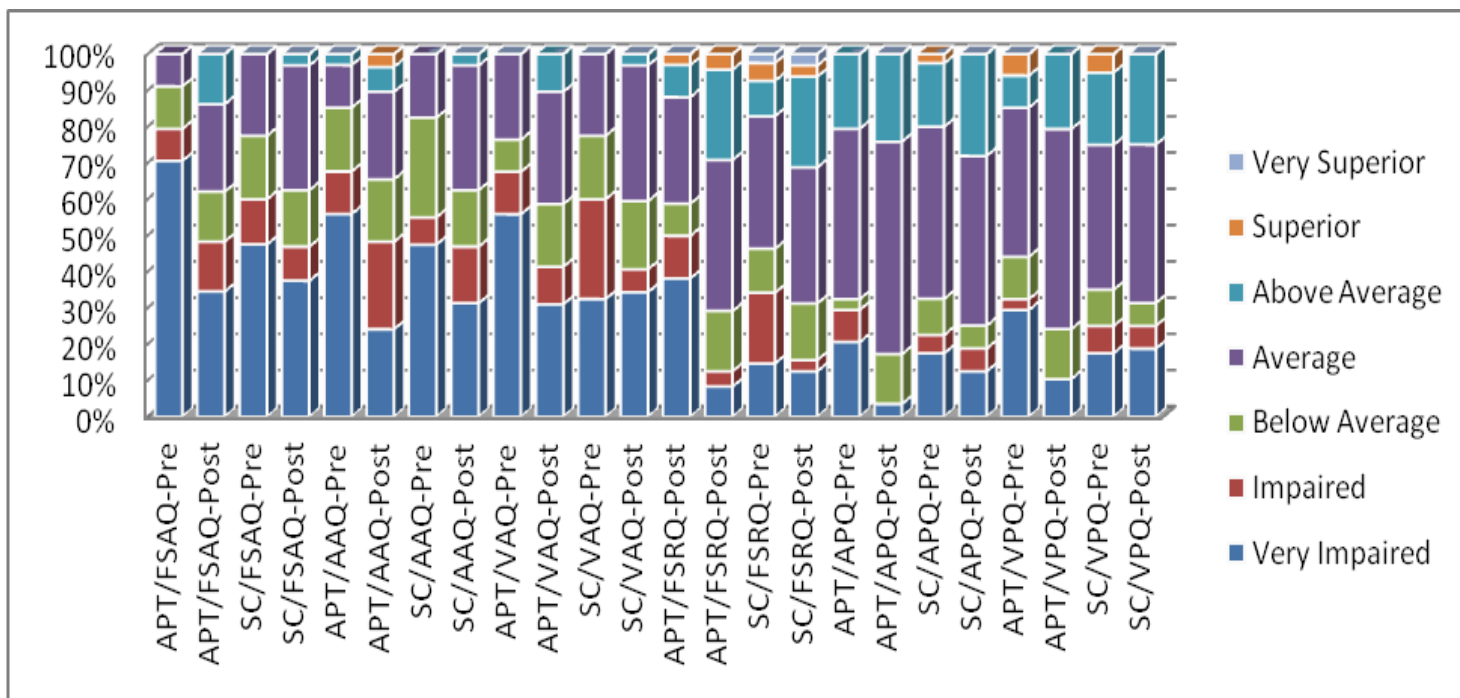
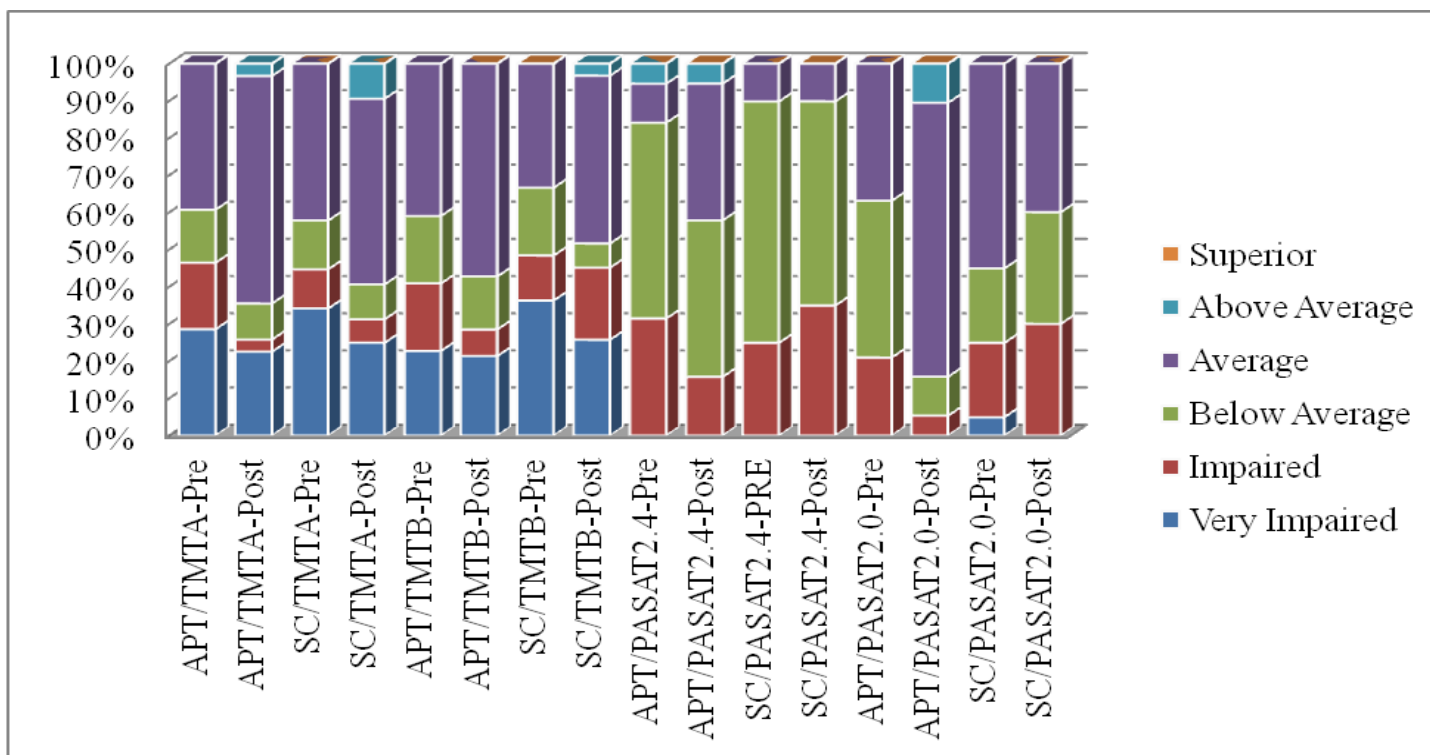


Figure 10. Comparison of the categories moved from pre to post-treatment for all scores of the IVA-CPT for both APT and Standard Care groups.



*Figure 11.* Comparison of the categories moved from pre to post-treatment for scores of the TMT Part A & B and PASAT 2.4 and 2.0 trials for both APT and Standard Care groups

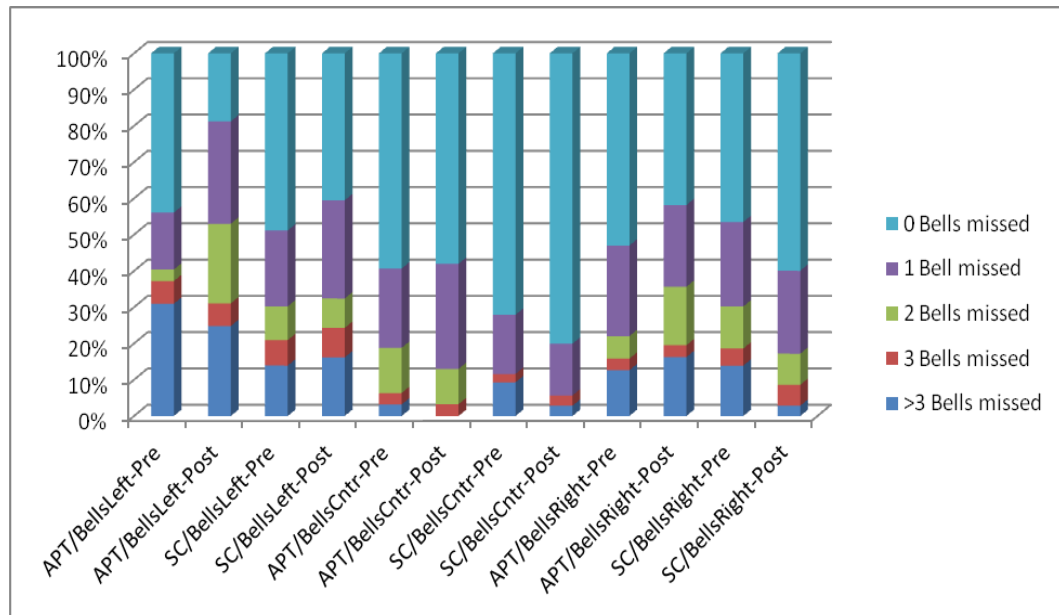


Figure 12. Comparison of the categories moved from pre to post-treatment for scores on the Bells Cancellation Test for both APT and Standard Care groups.

Also of interest to this study were the actual number of categories moved from baseline to post-intervention for participants in both APT and standard care groups. This data provided a clinical perspective of how the participants had improved. The means and standard deviations of the number of categories each group had moved are presented in Table 19. T-tests were conducted on this data in order to determine if either group had moved significantly more than the other group on any measure. The data shows that the Full Scale Attention Quotient was the only measure for which a significant difference in performance between the two groups was found, with the improvement in category changes made by the APT group from baseline to post-intervention, being significantly greater than the standard care group. At baseline the APT group was significantly worse than the standard care group on this measure and as such there was a lot more recovery of function for the APT group to achieve before a ceiling affect was reached. This result is consistent with the significant improvement as revealed by the ANOVA in the previous section.

Table 19  
*Ms and SDs of number of categories of change from baseline to post-intervention for both APT and SC groups.*

Measure	APT <i>M (SD)</i>	SC <i>M (SD)</i>	<i>t-test</i>
IVA-CPT			
FSAQ	0.96 (1.17)	0.29 (0.97)	<b>t(57) = 2.41, p = 0.02*</b>
AAQ	0.82 (1.19)	0.52 (1.21)	t(57) = 0.98, p = 0.33
VAQ	0.54 (1.35)	0.32 (1.05)	t(57) = 0.68, p = 0.50
FSRQ	1.25 (1.51)	0.48 (1.75)	t(57) = 1.80, p = 0.08
APQ	0.64 (1.31)	0.16 (1.86)	t(57) = 1.14, p = 0.26
VPQ	1.57 (1.35)	1.23 (1.45)	t(57) = 0.95, p = 0.35
TMT			
A	0.69 (1.26)	0.41 (0.91)	t(53) = 0.95, p = 0.35
B	0.55 (1.36)	0.48 (1.05)	t(53) = 0.20, p = 0.85
PASAT			
2.4	0.27 (0.70)	0.00 (0.71)	t(26) = 1.00, p = 0.33
2.0	0.87 (0.64)	0.38 (0.77)	t(26) = 1.81, p = 0.08
Bells			
Left	-1.17 (3.48)	-0.26 (1.48)	t(62) = -1.41, p = 0.16
Centre	-0.17 (1.10)	-0.31 (1.18)	t(62) = 0.49, p = 0.62
Right	-0.21 (2.76)	-0.74 (2.39)	t(62) = 0.83, p = 0.41

**\*p = <.05**

AAQ = Auditory Attention Quotient, APQ = Auditory Prudence Quotient, FSAQ = Full Scale Attention Quotient, FSRQ = Full Scale Response Quotient, IVA-CPT = Integrated Visual and Auditory Continuous Performance Test, PASAT= Paced Auditory Serial Addition Task, TMT= Trail Making Test, VAQ = Visual Attention Quotient, VPQ = Visual Prudence Quotient.

#### **Part 4: Factors influencing progress through APT**

To complete the analyses, a series of correlations were conducted in order to investigate if any participant characteristics influenced performance on APT.

Baseline demographics, performance on the attention measures and other neuropsychological measures were correlated with progression through the APT tasks as well and the total number of hours each participant completed.

Correlations were used to determine whether participants' demographics of interest and their performances on all neuropsychological measures at baseline related to how far they had progressed on tasks of APT. Pearson's bi-variate correlations were used for continuous variables. The PASAT 2.4sec trial correlated with reaching a higher auditory task ( $r = .48$ ,  $p < 0.05$ ) as did the PASAT 2.0 sec trial ( $r = .57$ ,  $p < 0.05$ ). Bells Right also correlated with reaching a

higher visual task ( $r = .43, p < .05$ ). There were no significant relationships between any other measure and the highest auditory or visual task reached. Of the demographic information no variable correlated significantly with the highest auditory or visual task (See Appendix E for the table of correlations). Statistical comparisons for ethnicity and stroke type could not be done due to low participant numbers. Therefore, the means and standards for ethnicity and stroke type for the highest tasks that were reached in both modalities of APT were generated and are presented in Table 20. Stroke severity as measured by scores on the MMSE and BI did not appear to differ between ethnic groups however despite this, it can be seen that the Pakeha group progressed considerably further than the other three groups on the auditory tasks. They were followed by the Pacific Island and Indian Groups whose progression through the tasks was similar. The Maori group made the least progression on auditory tasks. On the visual tasks the Pakeha group again made the most progress followed by the Pacific Island group. The Maori and Indian participants did not complete a visual task.

Also in Table 20, it can be seen that of the known stroke types, the PACs group made the most progress on the auditory tasks followed by the TACs, POCs and Haemorrhagic groups which were similar. The LACs group made the least progress on the auditory tasks. However on the visual tasks the TACs group made considerably further progress than the other groups followed by the PACs group. The POCs and Haemorrhagic groups made considerably less progress on the visual tasks. The lacunar stroke participant did not complete any visual task.

Table 20  
*Ms and SDs of ethnicity and stroke type for total highest auditory and visual task reached*

	<i>n</i>	Highest auditory task reached		Highest visual task reached	
		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Ethnicity					
Pakeha	28	52.86	30.52	26.21	29.53
Maori	2	11.50	16.26	.00	.00
Pacific Island	4	24.00	25.78	8.25	16.5
Indian	1	24.00		.00	
Stroke Type					
TACS	4	42.33	32.62	49.67	59.77
PACS	12	58.00	32.77	26.16	32.71
LACS	1	20.00		.00	
POCS	2	31.00	9.90	16.50	23.33
Haemorrhagic	5	33.60	26.95	19.80	18.07
Unknown	10	43.60	26.09	16.50	17.40

APT = Attention Process Training, LACS = Lacunar Stroke, PACS = Partial Anterior Circulation Stroke, POCS = Posterior Circulation Stroke, TACS = Total Anterior Circulation Strokes.

Of the 35 participants randomised to the APT group, only 5 participants completed the full 30 hours; 17 participants completed 15 or more hours (i.e., half of the scheduled intervention), and on average each participant received 15.92 hours. Pearson's correlations looking at the relationships between baseline demographics, measures of attention and other neuropsychological measures and the total number of hours of APT completed, were also conducted. (See Appendix F for table of correlations). On measures of attention there was a significant positive relationship between the Auditory Prudence score and the total number of hours completed ( $r = .409$ ,  $p = .05$ ) meaning a better Auditory Prudence score was associated with more hours of APT being completed. There was also a positive significant relationship between the score on the MMSE and the total number of hours completed ( $r = .401$ ,  $p = .05$ ). This meant that a higher score on the MMSE was related to more hours of APT being completed. Statistical comparisons for ethnicity and stroke type could not be done because of the low participant numbers. As seen in Table 21 means and standards were generated for ethnicity and stroke type for the number of APT hours completed.

Table 21  
*Ms and SDs for ethnicity and stroke type for the mean number of hours of APT Completed.*

	Hours of APT		
	n	mean	sd
Ethnicity			
Pakeha	28	17.60	9.57
Maori	2	9.75	9.55
Pacific Islander	4	8.75	4.97
Indian	1	10.00	
Stroke Type			9.30
TACS	3	22.3	8.44
PACS	12	17.88	
LACS	1	1.50	2.83
POCS	2	8.00	8.5
Haemorrhagic	5	17.20	9.45
Unknown	10	14.15	

APT = Attention Process Training, LACS = Lacunar Stroke, PACS = Partial Anterior Circulation Stroke, POCS = Posterior Circulation Stroke, TACS = Total Anterior Circulation Strokes.

Finally, correlations were conducted between post-intervention attention scores and the highest tasks reached in both auditory and visual modalities (See Appendix E), and between post-intervention scores and the total number of hours of APT received. (See Appendix F). Neither set of correlations revealed any significant relationship between those variables.

In summary, there was no evidence that progression through APT in this study was influenced by participants' baseline demographics and only minimal evidence that participant's ability on baseline neuropsychological measures affected how far the participants progressed on APT. The extent to which the participants progressed on APT tasks in both modalities was not associated with outcome measures. A comparison between demographics, neuropsychological measures and the number of APT hours received, revealed a positive relationship between one baseline attention measure and the MMSE with more hours. However the amount of intervention received was not associated with outcome measures.



## Chapter 6: Discussion

The evidence for the effectiveness of cognitive rehabilitation following neurological damage has grown steadily in the last decade amidst an environment that has historically been and indeed remains dominated by a focus on functional rehabilitation (Giles, 2010; Mok et al., 2004). Although much of the evidence for cognitive rehabilitation focuses on patients who have suffered a TBI (Carney et al., 1999; Cicerone et al., 2000; Park & Ingles, 2001; Prigatano, 1986; Wilson & Moffat, 1992) there is also significant interest in adopting this approach with stroke patients, a large percentage of whom are known to experience similar impairment in cognitive domains such as attention, as a result of their condition (Doornhein & De Haan, 1998). It is within this setting that the current study was conducted and as such, adds to a small yet growing pool of knowledge demonstrating the efficacy of cognitive rehabilitation for attention deficit in stroke patients.

The primary aim of the current study was to investigate the efficacy of a commercially available attention remediation programme, the APT programme, on attention deficit in the initial weeks after stroke. A further aim was to determine if improved attention impacted on the patient's quality of life at this early stage of recovery and a wider endeavour was to investigate what characteristics might influence the ability to benefit from the APT programme.

### **Main Findings**

This study demonstrated that the APT programme led to significant improvement on the primary outcome, the Full Scale Attention Quotient (FSAQ) of the IVA-CPT, as well as the Auditory Attention Quotient (AAQ) and the Full

Scale Response Quotient (FSRQ) of the IVA-CPT. On the secondary outcome, the Mental Component Score of the SF-36, there was no significant improvement as a result of the APT programme.

However, on the FSAQ, it was found that the APT group had significantly lower scores on this measure than the standard care group at baseline. This means that the attribution for the cause of the improvement that occurred on this measure is unclear and cannot be solely attributed to the intervention. This was the only attention measure in this study where there were differences between the groups at baseline. At post-intervention both groups obtained similar scores suggesting a ceiling effect for improvement on this measure.

On the IVA-CPT, AAQ, the APT group improved significantly but the standard care group did not. This measure provides a measure of sustained and selective attention in the auditory modality.

The APT intervention also led to significant improvements in participant's performance on the FSRQ (impulsivity) which is a composite of prudence, consistency and stamina. As the improvements in prudence scores were similar in the APT and standard care groups it appears then that the intervention improved consistency and stamina in particular. This suggests that the APT group was more consistent in the ability to stay on task and more able to sustain effort compared to the standard care group.

Not only were *statistically* significant improvements observed in attention as a result of the APT intervention, there were also *clinically* significant improvements as shown by the movement of participants' performances across qualitative categories. Movement across qualitative ranges provides valuable clinical information and to certain parties (i.e. patients, rehabilitation specialists,

behavioural researchers), is perhaps more meaningful than the statistical findings. Overall, on the IVA-CPT, FSAQ, AAQ, and FSRQ, there was a considerable shift by people in the APT group out of the low qualitative ranges, resulting in an increase in the number of participants reaching higher qualitative ranges. The pattern of movement across qualitative ranges generally reflected the statistically significant improvements from baseline to post-intervention obtained by the APT group. In contrast, there was considerably less movement by people in the standard care group out of the very impaired range and as a consequence considerably fewer standard care participants reached the average range. For both groups movement into the above average and superior ranges was limited to a small number of participants.

In summary, this study found that performance for the APT group improved on three attention measures, the FSAQ, the AAQ and the FSRQ at the post-treatment stage of APT. However, of those three measures the FSAQ was confounded by the differences on that measure at baseline. A clinical profile of the changes the participants made from pre to post intervention closely resembled the statistical findings.

### **Similarities with other studies**

The outcomes of this study have some similarities to those reported in the literature. First, comparisons will be made with other studies that evaluated APT with a stroke sample. This included three studies; Boman et al. (2004), Sinotte and Coelho (2007), and Sohlberg and Mateer (1987). In their study, Boman et al. (2004) found improvements in attention, specifically in complex sustained and selective attention tasks after APT although no change was found on an auditory attention task in that study. Sohlberg and Mateer (1987) and Sinotte and Coelho

(2007) also found that the two stroke participants in their studies, improved on measures of attention after APT.

Other studies evaluating APT did not include stroke patients but did utilise the same outcome measures and as such are also somewhat comparable. For example, a continuous performance task was used as an outcome measure in four previous studies investigating the efficacy of APT (Butler & Copeland, 2002; Kurtz et al., 2001; Lopez-Luengo & Vaaquez, 2003; Sohlberg et al., 2000). The results of the IVA-CPT, obtained in the current study are similar with the findings in three of those previous studies (Butler & Copeland, 2002; Kurtz et al., 2001; Sohlberg et al., 2000) where scores also improved on a continuous performance task, the Conners Continuous Performance Test (CPT), (Conners, 1992), (a measure of sustained attention in the auditory modality), after APT had *been* administered.

On another measure, the Bells Cancellation Test the outcomes of the current study were similar to the findings in the Lopez-Luengo and Vaaquez (2003) study, in that the participants in both studies did not demonstrate any improvement on that measure. In the Lopez-Luengo and Vaaquez (2003) study, both the participants in the experimental condition and the control group did not improve. However, in the context of their performance at baseline this is perhaps not a surprising outcome. That is, the baseline performances for both those groups as with the APT group in the current study fell within the normal range, thus limiting the scope for improvement. That is, those participants without an attention deficit are less likely to benefit from APT due to ceiling effects.

The current study also failed to find a difference in the scores on either trial of the Trail Making Test as a consequence of APT. This is another finding

consistent with Lopez-Luengo and Vazquez, (2003) who also did not find a change on this measure as a result of the APT intervention.

### **Differences from previous studies**

It is perhaps surprising that there were a number of attention measures used in the current study that did not show an improvement after APT as they did in some of the previous studies. Some of this difference can be explained by the variation in the methodologies between this and other studies. For example, there has been wide diversity in the samples used in these studies including patients with stroke, TBI, cancer, as well as patients with neuropsychiatric disorders. Furthermore, participants have all been at various stages post-onset and assessments have occurred at different times. The measures used in the studies have also been very diverse. These are all common problems that threaten the validity of comparing across studies and thus highlight the need for standardised practises in methodology.

In the current study, the PASAT is one of the measures on which the participants' score did not improve after treatment in the current study. This result is in contrast to previous studies which utilised the PASAT when evaluating APT. Indeed, the authors of APT, Sohlberg and Mateer, (1987) base their claim for the general effectiveness of this programme on the results of their study when all four participants improved on the PASAT following completion of the programme. The other outcome measure used in that study was the Spatial Relations subtest (SR) of the Woodcock-Johnson Psycho-educational Battery (Woodcock, 1977) which did not result in a significant change. From these results, the authors claimed that change occurred on the PASAT scores because it drew on attention skills which had improved as a result of the intervention

whereas the SR task was facilitated by other cognitive skills which were not subject to training and therefore did not improve. They therefore concluded that APT improved the functioning of specific cognitive processes underlying attention, but did not improve general cognitive functioning.

Improvement on the PASAT was also found in three other studies (Palmese & Raskin, 2000; Park et al., 1999; Sohlberg et al., 2000) although in one study (Park et al, 1999), the control group also improved on the PASAT leading to some ambiguity in the findings. In that study, only the experimental group showed improvement on the other neuropsychological measure, Consonant Trigrams. The authors speculated that this improvement may have resulted as a consequence of the practice of skills performed throughout attention process training that were similar to those skills required to successfully complete Consonant Trigrams. That speculation fits with a learning specific skills approach for cognitive rehabilitation purported by Kolers and Roediger (1984), Morris, Bransford, and Franks (1977) and Roediger, Weldon and Challis (1989) rather than the *improved cognitive function* hypothesis as postulated by Sohlberg and Mateer (1987). The specific skills approach asserts that improvement is the consequence of learning a skill rather than an improvement in functioning of an underlying cognitive process and therefore will not tend to generalise (Park et al., 1999).

Another measure for which the treatment group in this study did not show an improvement after the intervention was the Bells Cancellation Test. This is in contrast to the findings of the Kurtz et al. (2001) study which found that two participants did significantly improve their performance on a letter cancellation task by 22% and an impressive 66% respectively, and the second participant's performance also improved on a symbol cancellation task by 51%. Although the

third participant's performance did not improve significantly on this type of task, there was nevertheless a reduction in the time it took to complete the task at the second measure. The fact that no significant improvement was made on the cancellation tasks by any of the three control participants led the researchers in that study to assert that sustained visual attention improved as a result of the cognitive rehabilitation procedures adopted in that study which was primarily APT; although a prospective training model (memory for upcoming events) was also included.

### **Health Related Quality of Life results**

As well as investigating changes on neuropsychological measures of attention it was important to determine if APT actually improved the subjective experience of the participant's daily life. The inclusion of such a measure was in response to the often raised criticism of cognitive rehabilitation that while patients' test scores may alter as a result of intervention, it does not address how those changes may impact on real life, family, social and vocational settings. Hence, the Mental Component Score of the SF-36 was selected for inclusion of study outcomes. However, on this measure no significant changes were found as a result of time or treatment. Previous studies of APT have not included a health related quality of life measure although two studies of neglect interventions (Antonucci et al., 1995; Wiart et al., 1997) did, with the improvements that resulted on the neglect measures in both studies, generalising to everyday life. However, the lack of significant change in this study was somewhat unexpected in light of increasing recognition in the literature that cognitive deficits and even mild cognitive impairment impact on functional and psychiatric outcomes of neurological disorders (Mitchell et al., 2010).

One explanation for this outcome may be because many of the participants were still in hospital and therefore not able to respond accurately to those questions that evaluated performance in their “normal” setting, i.e. home or work, such as in question 6; “During the past 4 weeks, to what extent has your physical health or emotional problems interfered with your normal social activities with family, friends, neighbours, or groups?”. Another reason may have been because the time between pre and post-measures (approximately five weeks) was not long enough for change to occur or were so subtle as to fall outside the sensitivity of the SF-36 such as in question 2; “Compared to one year ago, how would you rate your health in general now?” Unlike other quality of life measures such as the Cognitive Failures Questionnaire which assesses cognitive problems in daily life (Broadbent et al., 1982), the SF-36 does not factor so well on the cognitive domain of attention, the cognitive function that is targeted by the APT programme. Therefore it is possible that any change in attention abilities that may have occurred as a result of the intervention in this study is less likely to have been detected by the SF-36, particularly given that some participants were still hospitalised.

In summary, quality of life did not appear to alter as a result of the APT intervention however it is possible that a more cognitively demanding quality of life measure such as the Cognitive Failures Questionnaire (Broadbent et al., 1982) may have been more able to discern a difference given that it was a cognitive intervention that was being evaluated. The CFQ was administered at baseline and again at six months post-intervention for the START analysis, however no measure was obtained at 5 weeks.



### **Changes as an effect of time**

As well as improvements in some aspects of attention as a result of APT, there were also improvements that occurred in both the APT and standard care groups. Specifically, significant improvements were found on the IVA-CPT, Visual Attention Quotient, both trials of the TMT, the faster trial of the PASAT, and the Bells (Left) Cancellation Test as a function of time.

This improvement on neuropsychological measures as a function of time since stroke has been seen in a number of other studies where the improvement was not attributable to the treatment. For example, in the Park et al. (1999) study, the control group improved significantly on the PASAT and in the Lopez-Luengo and Vaaquez (2003) study, the control group improved significantly on a dichotic listening task. Trends towards improvement, although not reaching significance, have also been noted as in the Sohlberg et al. (2000) study, when after controlling for practise effects, times for both trials of the TMT were shorter for the control group when measured at three subsequent stages throughout the study. Similarly, in the Lopez-Luengo and Vazquez (2003) study, the control group improved their performances on both trials of the TMT. These findings are consistent with the literature explaining the phenomenon of spontaneous recovery of cognitive deficit (Christensen et al., 2008; Williams, Potter, & Ryland, 2010; Wilson, 2010) specifically for attention deficit, within the first three months post-stroke (Hochstenbach, Den Otter, & Mulder, 2003; Wade et al., 1988). In contrast, two previous studies evaluating APT, did not find any improvement of cognitive functioning by the control groups as measured by neuropsychological testing (Butler & Copeland, 2002; Kurtz et al., 2001).

Spontaneous recovery is a consequence of time and refers to the brain's ability to repair itself, or more accurately, it is the neuronal re-organisation that takes place the instant the injury occurs and is influenced by pre-injury and post-injury factors. While the variability of this recovery is substantial generally recovery reaches a plateau within six months after brain damage (Munoz-Cespedes, Rios-Lago, Paul & Maestu, 2005; Richardson & Richardson, 2002; Rohling et al., 2009) including stroke (Cramer, 2008; Kreisel, Bazner, & Hennerici, 2006; Witte, 1998). As spontaneous recovery can occur with or without rehabilitation, the presence of a control group that does not receive any intervention is one method of assessing this phenomenon. The effects of spontaneous recovery may also be seen in the current study where significant positive correlations between "Time since stroke" and attention were evident for 10 of the 13 baseline attention measures. That is, scores on those measures of attention improved the longer the interval since the stroke first occurred.

To conclude this aspect of the discussion it was found that improvement on a number of measures occurred as a function of time, a finding that is consistent with previous similar studies. Spontaneous recovery is largely responsible for this recovery although extraneous variables may contribute to the improvement.

### **Appropriateness of measures**

The lack of consistency on some measures between the current study and previous studies gave rise to the notion that perhaps those measures that have been used across studies may not be entirely appropriate for use with a stroke population and therefore have led to inconsistencies in outcomes. The lack of a positive outcome on the PASAT in this study warrants some scrutiny for the suitability of this measure. The number of participants who were unable or

unwilling to complete the PASAT was considerable and may have contributed to the insignificant finding at post-intervention. Some participants after being provided with instructions and presented with the PASAT practice list, considered the task too difficult and declined to continue, while others aborted the task soon after commencement. One explanation behind that resistance could be that the age of the sample, compared to previous studies, was much older and therefore not as able to cope with the demands of this task. This raises the question as to the suitability of the PASAT in studies of stroke patients, a population which is largely made up of elderly participants. Indeed, the PASAT is a highly demanding task of auditory working memory and has frequently been reported as being too challenging and stressful for some populations including the elderly, for whom working memory is generally adversely affected (Holdwick & Wingenfeld, 1999; Raz, 2000; West & Bowry, 2005), regardless of their cognitive status (Tombaugh, 2006).

Another reason for low participation rates could be that a number of the participants were experiencing heightened fatigue. Indeed, it has been found that patients who experience attention problems often experience difficulty with fatigue and/or maintaining concentration over an extended period of time (Sohlberg & Mateer, 2001). Difficulty undertaking the PASAT has been previously recorded. In their study determining cognitive dysfunction in patients with first-ever brain infarct Cohen, Salloway, & Sweet (2008) found that fatigue impacted on the ability to undertake the PASAT. Furthermore, the high demands of the PASAT can lead to anxiety and frustration and deter patients from further engagement in this task (Tombaugh, 2006).

In another study, patients performed significantly lower than the control group on the Modified PASAT and the authors pointed out that those structures requiring activation to perform this task (i.e., the prefrontal cortex and posterior parietal cortex) had been damaged by the stroke (Jaillard, Naegele, Trabucco-Miguel, LeBas, & Hommel, 2009). So it may be that in this study, age, fatigue, location and severity of stroke were all factors contributing to some unwillingness to complete the task which ultimately resulted in low participation rates for this measure. However, despite its drawbacks the PASAT is a highly reliable measure and very sensitive to subtle attention deficits which is why it was used in this study. It is also the most used primary outcome measure in previous studies of APT.

There has also been some concern amongst researchers regarding the suitability of the TMT as a neuropsychological measure for stroke patients. A major criticism of the use of this measure in a stroke population is that performance has frequently been compromised due to non cognitive factors such as motor dysfunction of the dominant hand or hemianopia (Lezak, 1995; Waldstein, et al., 2003). The diminished ability of stroke patients on this task was vividly demonstrated in a study of patients with sub-cortical ischemic vascular lesions who demonstrated pronounced impairment of speed on both trials of the TMT (Peters et al., 2005) compared to matched controls. In fact, the inappropriateness of the TMT in patients with physical restrictions has long been established and has subsequently led to the development of the oral paradigm of the TMT which was clinically validated on a stroke population (Ricker, Axelrod, & Houtler, 1996). It is perhaps because of this limitation of the TMT that no differences were found in this study. Participants with subtle physical limitations

were not identified and therefore may have influenced outcomes on this measure. Given the potential of the TMT to affect the performance of participants with physical limitations, it may be more appropriate for future studies with stroke survivors to utilise the oral TMT.

Another measure that may have influenced performance level with this sample is the Victoria Stroop Test. On this task the participant was required to distinguish between the colours blue and green in order to make a correct response. However, it has been found that some older people suffer from impaired visual acuity due to a range of problems and may include difficulty in discriminating the blue-green dyad. This condition is due to the yellowing caused by the presence of cataracts (Pachana, Thompson, Marcopulos, & Yoash-Gantz, 2004). In order to counterbalance this problem Pachana et al. (2004) developed an alternate form of the Stroop called the California Older Adults Stroop Test (COAST). This modified version uses larger type face, fewer items and the colours red, yellow and green, which are more easily distinguishable. Although, in hindsight utilisation of the COAST may have been more appropriate given that the average age of the sample was 69.03 years, when designing the study, it was considered more appropriate to use the Victoria Stroop Test which is less age restrictive and because of the potential for a younger sample. That is, the primary site for recruitment was south Auckland which has a large Maori and Pacific Island population, two demographic groups that suffer stroke at a much younger age than their European counterparts (Fink, 2006). Nonetheless, the COAST is an appropriate measure of selective attention in further studies of older people.

The SF-36 is another measure used in this study that has its proponents and critics (Hagell, Reimer, & Nyberg, 2009; Jenkinson et al., 2002; Ware, 2000).

Some research has evaluated the SF-36 with elderly patients (Hayes, Morris, Wolfe, & Myfawny, 1995; Loge, 1998) and more specifically with elderly stroke patients (Hobart, Williams, Moran, & Thompson, 2002), including those in New Zealand (Bonita et al., 1997; Hackett et al., 2000) with mixed conclusions. A study conducted in Sweden with 188 acute stroke survivors with a mean age of 74 years found the Swedish version of the SF-36 to be an effective measure of HRQoL when administered two to three weeks after discharge from hospital (Almborg & Berg, 2009). In their study Hagen, Bugge, and Alexander (2003) also found the SF-36 to be an adequate and reliable measure with their stroke sample revealing particular sensitivity to change between one and three months post-stroke although poor sensitivity between three and six months.

Critics of the SF-36 include Hackett et al. (2000) who suggest that the SF-36 “may not be sensitive or specific enough to detect the psychological domains of mental health that are relevant to patients with stroke” (p.446), although their sample group consisted of long-term rather than short-term survivors of stroke. Anderson, Laubscher, and Burns (1996) also found that the Australian version of the SF-36 did not characterise social functioning very well and suggested that it be supplemented with other measures in order to gain a comprehensive assessment of stroke outcome. Furthermore, a number of researchers have found floor and ceiling effects for the SF-36 (de Haan, 2002; Dorman, Dennis, & Sandercock, 1999; Hobart et al., 2002; O’Mahoney, Rodgers, Thomson, Dobson, & James, 1998) and therefore advise caution on this measure, although others have not (Anderson et al., 1996). Fortunately, no such effects occurred in this study. Finally, the inadequacy of completion rates of the SF-36 has been shown

in the elderly who were contacted by post (O'Mahoney et al., 1998; Parker, Peet, Jagger, Farhan, & Castleden, 1998).

However, there are many benefits for the use of the SF-36 that led to its inclusion in the current study. It has been consistently shown to have good validity, reliability and sensitivity across various populations (Fukuhara, Bito, Green, Hsiao, & Kurokawa 1998; Gladman, 1998; Sanson-Fisher & Perkins, 1998), including Maori, Pacific and New Zealand European ethnic groups (Scott, Sarfati, Tobias, & Haslett, 2000; Scott, Tobias, Sarfati, & Haslett, 1999). Almborg and Berg, (2009) found the SF-36 to be a valid measure of health-related quality of life in a study of 188 post-stroke patients, and Mead et al. (2011) found it to be a valid measure of post-stroke fatigue.

Conversely, the IVA-CPT was shown to be an entirely appropriate measure with this stroke sample. Of all the measures used in this study the IVA-CPT was the most consistent in detecting change. One reason for these positive findings may be due to the highly sensitive properties of the IVA-CPT and its ability to detect slight change, compared to other measures of attention. For example, the IVA-CPT is frequently used when evaluating children with attention deficit because it has been found to be the most sensitive measure for detecting the subtle impact of treatment regimens with this population (Harding, Judah, & Grant, 2003; Sandford, Fine, & Goldman, 1995). Furthermore, it is distinct from other attention measures in that it combines scores from both visual and auditory modalities and therefore is able to detect change in more than one modality. The versatility and sensitivity of this measure renders it a highly appropriate measure of attention deficit in studies of stroke patients.

Although the measures used in this study are generally well supported for use in a stroke population the previous paragraphs have identified possible problems of some of the measures as well as the strengths of other measures. Further research of post-stroke cognitive functioning will increase confidence in which measures are most appropriate and scientifically valid. The ultimate goal is an understanding by researchers of standard measures for use with specific study designs and specific stroke sub-groups.

### **Factors that may have influenced outcomes**

The subjective experience of APT is also of interest to rehabilitative researchers, therapists and perhaps most of all, to the survivors of stroke who present with attention deficit. Observations and experiences from both the researchers and the participants who received APT will now be discussed.

The majority of participants in this study did not complete the full 30 hours of intervention and the reasons for this trend were varied although most commonly, participants did not offer a reason for their decision to cease participation in the intervention. However, the most frequent reason that was given, was that participants experienced heightened fatigue and they found that participation in the study was too taxing and/or challenging. Other participants, particularly those engaged in outpatient services found that it became too difficult to accommodate the study because so much of their time was being taken up with other appointments. Other reasons for reduced participation in the study included, participants returning to work, participants going on holiday, or general loss of motivation to take part in the study. One patient completed all APT tasks in less than the 30 hour maximum time period and another participant moved away from the area. Another patient died soon after the study began. In essence, the course



of some of the participants' lives after the stroke became somewhat unpredictable, less routine and more demanding and the consequence was that compliance to the study was affected.

In light of the evidence that intervention dose (i.e., amount of training) impacts on outcomes (Sohlberg & Mateer, 2001; Velligan, Kern, & Gold, 2006) it was speculated that those participants who completed more APT may have produced better post-treatment attention scores. There was considerable range in the number of APT hours completed by the participants, however, despite expectations there were no statistically significant relationships between the number of APT hours completed and performance on the post-intervention attention measures. (See Appendix F for the correlations between post-intervention measures of attention and total hours of treatment). Comparison of these relationships with other studies of APT is not possible as previous data is not available.

Second, what was also noted in this study was that the participants progressed considerably further on auditory tasks than they did on visual tasks. This pattern was possibly a consequence of the order in which the tasks were administered. All participants were administered the APT in a pre-determined order. Of the first nine tasks administered, seven were in the auditory modality. (See Table 6, p 109, in the Methods Section). Only two participants in the APT group progressed beyond task nine and therefore most participants did not engage in tasks presented in the visual modality. This order of administration gave rise to speculation that the significant result obtained on the Auditory Attention Quotient of the IVA-CPT may be related to the greater amount of training received in the auditory modality of the APT programme. However, as previously mentioned,

analyses did not reveal any such significant association between how far the participants reached on either auditory or visual tasks with post-treatment attention measures.

### **Factors that may have influenced how the participants engaged in APT**

There were other behaviours exhibited by the participants that may have influenced how they engaged in the intervention. For example, it was observed that tolerance levels of the participants for the APT programme varied considerably and was believed to have been largely influenced by levels of fatigue. The range in the number of total hours of APT achieved by the participants was extremely varied ranging from three quarters of an hour to the maximum of 30 hours (See Table 7, p 119). Of those participants who ceased involvement in the study before completion of the scheduled 30 hours, heightened fatigue was the most quoted reason for their withdrawal. It is widely acknowledged that fatigue compromises cognitive abilities (Barker-Collo et al., 2007; Englander, Bushnik, Oggins, & Katznelson, 2010; Fry, Greenop, & Schutte, 2010; Gehring et al., 2009; Roth, Geisser, Theisen-Goodvich, & Dixon, 2005). In their study of patients with attention problems, Sohlberg and Mateer (2001), found that fatigue was a recurring factor amongst those patients who completed fewer hours of APT. Boman et al. (2004) were aware of this problem also and acted to minimise the effects of fatigue in their evaluation of APT with neurologically compromised patients, by ensuring that the intervention was administered in the morning. Unfortunately, in the present study scheduling of the intervention for the hospital-based participants was typically out of the control of the researchers and therefore occurred whenever it was convenient for participants and hospital staff. Usually standard care took place during the

morning hours leaving mid to late afternoon to conduct the APT intervention. By that time, participants were already experiencing heightened fatigue and were less tolerant of the APT intervention. Whenever it was possible participants who had been discharged home were scheduled to receive the APT in the morning and it appeared that those participants performed better, however, this is the observation of the researcher only as formal data for these variables was not obtained. This observation is however consistent with the literature for traumatic brain injury that cognitive fatigue increases throughout the day (Brain Injury Association of Canada; Claros-Salinas et al., 2010). Therefore it is possible that the morning hours appear to be the ideal time to administer APT for participants to gain optimal benefit from the programme.

Another behaviour that appeared to impact on participants' engagement in the intervention, was their level of motivation. Although again not formally assessed, it became evident to the researcher throughout the course of the treatment that there was considerable variability in the willingness of the participants to engage in the intervention. Despite having consented to participating in the study some participants were reluctant to actually engage in the intervention. In contrast other participants were compliant and willing throughout. The researchers felt that this factor may have influenced performance on the APT.

Motivation has been identified as having an influence on the performance of individuals who participate in research studies and is linked to the reasons why they agree to being involved in research. That is, intrinsic factors such as 'a sense of the importance of scientifically controlled studies' or 'to gain more knowledge about their own condition' are usually more influential (Bell et al., 2008), than

extrinsic factors such as monetary rewards (Patel, Doku, & Tennakoon, 2003). It is possible that generally, those participants who considered the intervention to be of value to their own recovery maintained a higher level of enthusiasm and engagement throughout the intervention process compared to those whose reason for participating in the study was prompted by a “social conscience”. The least motivated as demonstrated by their decreased willingness to engage in the intervention, appeared to be those individuals whose participation in the study was primarily due to persuasion by family members. Some patients in this latter group also expressed feelings of depression and not only lacked motivation to engage in this study but demonstrated similar ambivalence to any form of rehabilitation.

Providing feedback after each task appeared integral to the success of the intervention. When the feedback was positive such as when the participant committed fewer errors or false alarms, increased motivation was apparent as noted by behavioural responses. When the participant’s performance was not improving or even sometimes if it was regressing, care was taken to express the feedback in a way designed to avoid de-motivation.

The current study recruited participants who were in the very early stages of recovery from their stroke and although no formal measures of depression or anxiety were implemented, there were indications that some participants were suffering from the emotional impact of stroke. The presence of heightened emotion is consistent with the literature identifying depression as a relatively common stroke outcome (Barker-Collo, 2007; Hackett et al., 2005; Paolucci, 2008). This generated some concern as to whether the participant’s emotional status was impacting on their performance on the APT. Certainly, this concern was not without foundation as there exists a significant amount of literature

linking Post Stroke Depression (PSD) with cognitive impairment (Murata et al., 2000; Narushima et al., 2003; Robinson et al., 1986; Saxena, 2006; Talelli et al., 2004; Yoo et al., 2009). Given the frequency of PSD and its possible confounding effects, it may have been prudent for a specific measure of depression to have been included in this study. In their study, Park et al. (1999) used the Beck Depression Inventory (BDI), to assess the impact of the APT programme on mood, although on that occasion there was no significant change detected on the BDI scores. However, there are other depression scales that may be more suited to a stroke in-patient population including the Poststroke Depression Rating Scale, the Hospital Anxiety and Depression Scale, the Hamilton Rating Scale for Depression and the Geriatric Depression Scale all of which have been validated with this population (Aben et al, 2002; Tilanus & Timmerman, 2005).

The preceding paragraphs provide some observations made by the study researchers, of the behavioural factors that may have influenced how the participants engaged with the APT programme. In summing up, this study demonstrated that administration protocols as well as physical, psychological and emotional functioning of the stroke patient in the early stages of recovery can affect the extent to which the patient engages in APT.

### **Which patients might benefit most from APT?**

It was considered important to collect data on the characteristics of those individuals who might benefit more from APT. In order to do this, relationships between participants' demographics and baseline performances on neuropsychological tests and progression on APT were investigated. Five positive associations were identified. First, those participants who obtained a

better MMSE score had a positive association with the number of hours of APT that were completed as did those participants who had a better performance on the baseline Auditory Prudence score. One possibility for this outcome is that those participants with higher MMSE and Auditory Prudence scores found the APT tasks easier and therefore were more likely to progress through the programme. However, although participants with better cognitive functioning might do better on cognitive rehabilitation tasks, they are more susceptible to the ceiling effects of rehabilitation tasks but are also less likely to require rehabilitation.

Three further significant relationships emerged from this analysis and perhaps not surprisingly, both relationships were specific to their respective modalities. That is, a better performance on the Bells Right task (a visual task) was associated with achieving a higher visual task on APT and a better performance on both trials of the PASAT (an auditory task), was associated with achieving a higher auditory task on APT. Although these findings are of interest, the presence of only four significant relationships is not sufficient data to argue that neuropsychological performance, at least in this study, influenced performance on APT. It does however provide consideration for modality-specific training where the participant is initially assessed for impairment in either or both auditory and visual modality and rehabilitation is provided accordingly.

#### **Attention abilities of stroke survivors in the early stages of recovery.**

The current study also provided an opportunity to investigate the attention abilities of stroke survivors in the early stages of recovery. As part of the inclusion criteria for the study all participants had been found to have an attention deficit as determined by their score of one standard deviation or more below the normative mean on any scale of the IVA-CPT, the Trail Making Test Part A or B,

either trial of the PASAT or if they missed more than 3 bells on the left or right of the Bells Cancellation Test. A series of correlations showed that some baseline attention scores were found to have significant positive relationships with “Time since Stroke” and scores on the Barthel Index and the Mini Mental State Exam. The association between “Time since Stroke” and an improved performance on baseline attention measures has already been discussed in this chapter within the context of spontaneous recovery. A significant correlation between the Barthel Index and six attention measures was found which is somewhat surprising, given that an often cited weakness of the Barthel Index is its failure to measure activities that require cognitive abilities. According to Herndon (2006), patients can have significant cognitive impairment and still obtain a perfect score on the Barthel Index. However, in this study the Barthel Index was one of the factors of randomisation and therefore properties of this measure would have been balanced across the APT and SC groups.

The significant correlations that were found between the MMSE and six baseline attention measures was not surprising given that it is a measure of cognitive functioning and attention is one of the five areas of cognition that is specifically tested by this instrument (Folstein et al., 1975). All participants had to have obtained  $\geq 20$  on the MMSE for inclusion into the study.

Also of interest was the interplay of baseline attention measures with neuropsychological measures of memory, language and executive functions. Of all the neuropsychological measures, performance on the ROCF was associated with more measures of attention than any other measure. The significant correlations between the delay and recognition trials of the ROCF and attention measures is consistent with the argument that attention is a precursor to other

cognitive functions such as memory (Cowan, 1995) and also corresponds with the statement that a good performance on the short delay trial of the ROCF is reliant on intact working memory (Lezak, 1995). Furthermore, most of the attention measures that correlated significantly with the ROCF were in the same modality, i.e. visual. However, an interesting finding was that the BNT, a measure of the verbal modality, correlated significantly with six of ten attention measures that were in the visual modality.

The variability of the performances of stroke patients as measured by neuropsychological tasks is largely dependent on the many factors that affect the presentation and experience of the disease and the stage at which the person is in the recovery process. Factors that influence recovery rate are wide and varied and can include; type of stroke, severity of stroke, site of the lesion, previous stroke, neurological deficits, loss of consciousness, type and amount of therapy provided, timing of therapy, the presence of neglect (especially in cancellation tasks), awareness of the neglect (anosognosia), cognitive status, as well as demographic characteristics such as age, gender and education (Barker-Collo & Feigin, 2006; Brandt, 2007; Cumming, Plummer-D'Amato, Linden, & Bernhardt, 2009; Gialanella, Monguzzi, Santoro, & Rocchi, 2005; Kumar, 2003; Menon-Nair, Korner-Bitensky, & Ogourtsova, 2007; Stone, Patel, Greenwood, & Halligan, 1992), although this is by no means an exhaustive list of variables that affect the path of recovery.

The preceding paragraphs provide some insight into the attention and wider neuropsychological abilities specific to the study sample. It would be useful if future research built on this data so as to develop a knowledge base of the



attention profile of stroke patients sub-groups. This information would then be useful data for comparing across studies.

### **Limitations**

There were a number of limitations identified in this study, the main one being the failure to ensure a balance for the primary outcome at baseline across the two groups. This oversight makes it difficult to draw firm conclusions regarding the efficacy of APT. It is uncertain whether or not the APT group would have demonstrated as much improvement if their baseline measure was equivalent to the SC group. Future studies should ensure that baseline measures for the primary outcome are equivalent across the groups.

Another possible confounding variable in this study was the use of one standard deviation on the primary outcome measure as the criteria to determine if the intervention was effective. The authors of the IVA-CPT reported a test re-test correlation for the composite quotient scores as ranging from .37 to .75 (Sandford & Turner, 2009). These relatively weak coefficients make it difficult to reconcile that an observed effect of just one standard deviation should be interpreted as not likely to be due to random fluctuations. Future studies may consider increasing the criteria for an observed effect to more than one standard deviation.

Another limitation of this study was the relatively narrow inclusion criteria of the sample. Only patients who had experienced first-ever strokes were included into the study thereby limiting the findings to that particular group of stroke survivors. Furthermore, restricting recruitment to patients who had suffered their stroke within the previous 30 days means that the findings are not able to be generalised to non-acute stroke survivors. Of course, in contrast, the advantage of the rather strict criteria is that it provided some control for the wide

variability in stroke patients meaning that the findings can be regarded as representative of this particular group of stroke patients.

This study examined the movement across categories and as such provided useful information in terms of a more clinical interpretation of the findings, however, this approach does have some limitations which will now be discussed. Using this approach, there is the possibility that a participant's true change may not be evident. For example, a participant may make minimal improvement from baseline to post however if at the baseline stage that participant is sitting at the high end of a category the minimal improvement may be enough to re-classify the participant into the low end of the next category. In another scenario, a participant may make a significant improvement from baseline to post-treatment yet not necessarily be re-classified into a higher category. This situation is more likely to occur if at the baseline measure the participant is sitting at the low end of the category. This condition was most clearly depicted in this study where the 'average range' category, based on the normal probability curve, included those scores that fell between one standard deviation below the mean and one standard deviation above the mean, thereby covering a range of two standard deviations. This created a situation where a participant's performance from baseline to post-treatment may have been statistically significant however no change of category would necessarily have occurred.

Another method of measuring the effectiveness of an intervention is to calculate the effect size which, as the name implies, is a statistic that conveys the strength of a treatment effect. Effect size is a useful adjunct to inferential statistics by providing substantive evidence that further informs decision making around meaningful treatment plans for clients. The z-scores of a standard normal

distribution as utilised in this study are an example of an effect size (Faraone, 2008). Thus comparing z-scores from pre to post-treatment in this study would have also provided a clinical interpretation of the effectiveness of APT. Effect sizes can be classified as portions of a z-score with “small effect size” being (0.2 to 0.5), “medium effect size” (0.5 to 0.8) or “large effect size” (0.8 and higher; Coe, 2002).

The Reliability Change Index (RCI) provides another method for calculating clinical significance. The RCI is the difference between a participant’s pre-test and post-test scores divided by the standard error of the difference (Jacobson & Truax, 1991). Cut off scores are established to determine any clinical change that may occur.

Thus, although this study examined the changes in categories to determine a clinically significant change in performance, it can be seen that this method of analysis is not without its problems. Future studies may consider utilising an alternative method thereby avoiding the problems associated with the category-change method.

Another limitation of the study may have been that the lack of evaluation for depression or anxiety. Given that both of these states have the potential to impact on cognitive performance, future studies should control for this possibility. The Beck Depression Inventory-II and the Hamilton Rating Scale for Depression are commonly used for screening depression in stroke populations however they were not developed specifically with a stroke population in mind and as such overlook some of the complex neurological problems involved in this disease. In response to this problem, Gainotti et al. (1997) developed the Post-Stroke Depression Rating Scale an instrument that is specifically designed to investigate affective

disorders of stroke patients who suffer various degrees of severity of depression. As such this would be an appropriate tool to use in future studies when screening for depression.

The APT did not impact on a health related quality of life measure at this early stage however it may have been more appropriate to have conducted a follow-up assessment of this domain at a slightly later stage when participants were back in their usual environment and some of the questions that make up the SF-36 would have been more relevant. Alternatively, the CFQ, which was administered at baseline and again at 6 months for the START study, may have been more useful at the 5 week post-stroke point compared to the SF-36, given that the former is a measure more reflective of underlying neuropsychological impairment.

### **Implications of this study**

There is now general consensus within the practise of neurological rehabilitation that cognitive rehabilitation is necessary to meet the needs of people who suffer brain injury. Historically rehabilitation models were developed with the primary purpose of addressing physical impairment and there still remains a lack of empirically validated interventions routinely available to clinicians, for the numerous cognitive impairments that affect this population. While the understanding of non-spatial attention is grounded in theoretical models that have developed and evolved over many decades, there are few theoretically-based models of this phenomenon with a clinical application. Sohlberg and Mateer's contribution to this area of cognitive rehabilitation, with the development of APT has been an important and significant step in addressing this gap and the current

research has further sought to improve this situation by providing a well-designed study for the evaluation of APT.

However, in many settings the rehabilitation programme continues to remain wholly focussed on the physical consequences of stroke despite considerable support for a multi-disciplinarian approach such as the stance taken by the American Stroke Association (Schwamm et al., 2005). The inclusion of cognitive rehabilitation into rehabilitative programmes will place demands on already stretched resources so it is vital that studies such as the current one continue to provide empirical evidence for the rationale and justification of cognitive rehabilitation if it is to become a respected and integral component of post-stroke rehabilitation.

While the overall findings of this study add to evidence-based practises, the addition of qualitative findings is also encouraging to clinicians as it provides further insight into the actual improvement of attention changes that occurred for participants. Not only does this information increase the clinician's confidence in the intervention but it also provides a framework for measuring change that can be utilised in a rehabilitative setting.

Furthermore, this study provides support for the assertion that rehabilitation for cognitive deficits should be initiated in the early stages of stroke recovery. Hochstenbach et al. (1998) argue that advice and treatment for cognitive deficits needs to be initiated earlier than three months post-stroke, however, early neuropsychological assessment of stroke patients is not routinely carried out in most hospitals (Hoffman, Schmitt, & Bromley, 2009).

## Conclusions

APT appears to be a viable option for the remediation of attention deficit for those people who are in the early stages of recovery from stroke. Significant improvement was obtained on the primary outcome measure although confounding factors rendered this particular result inconclusive. However, improvements were also obtained on two other measures of attention after administration of the APT. The impact of APT on quality of life measures at such an early stage in the recovery process was not discernible. Factors that appeared to influence the performance of participants when undertaking APT in this study are fatigue and motivation levels so formal assessment of these issues are recommended in future research in order to determine optimal application parameters for this cognitive intervention.

This study in context with previous research evaluating APT contributes to a steadily growing bank of data for the efficacy of APT since it was first developed by Sohlberg and Mateer in 1997. This has been the first randomised controlled trial of this intervention in a relatively large stroke sample and provides encouraging data for its usefulness in neurologically impaired patients other than those for whom the programme was first developed. The strength of this study was in its design, in that it was a single blind randomised control trial. Inclusion criteria ensured homogeneity of the sample group. The selection of outcome measures resembling the training tasks did provide for an accurate gauge of any changes that may have occurred for that specific skill although without other measures in place generalising the findings to functional improvement was not achievable.

## **Future Research**

Hopefully, the results of this study provide incentive for further randomised controlled trials for the efficacy of APT in acute stroke patients. The reasonably strict inclusion criteria of the current study was intended in order to increase confidence that any observed effects of the intervention, apply to this particular group of stroke patients. However, more evaluation of APT with other sub groups of stroke patients is also necessary. Patients with particular lesion sites, long term stroke patients or survivors of recurrent stroke, are all examples of the different stroke sub groups where the efficacy of APT needs further investigation in order to determine its suitability for those particular groups. It is recommended that psychological and physical problems often associated with stroke patients should be considered and addressed in order to optimise the full benefits of this intervention.

Future studies evaluating APT should also include follow-up measures particularly to assess any real-life benefits to the individual. This could be achieved by including measures of HRQoL, ADL's, functional measures, self-reporting questionnaires, and caregiver questionnaires.

This study briefly looked at other investigations of APT whose samples included patients with schizophrenia ADHD, TBI and encephalitis. However, there are many other neurological disorders such as Multiple Sclerosis, Korsakoff's syndrome, Parkinson's disease, Huntington's disease and dementia, plus other neurological conditions that may also result in attention deficit. Research into the efficacy of APT as a viable rehabilitation intervention for those conditions is needed.

There has also been little research conducted with children primarily because the APT programme was designed for adults. However, Kerns Eso, and Thompson (1999) and Semrud-Clikeman (1999) have conducted studies with children using interventions based on APT with both studies producing results of improved attention. However, given the maturing nature of a child's brain, APT needs to be evaluated within narrow age bands in order to evaluate its efficacy with each age band.



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## **Appendix A**

### **Stroke Risk Factors**

#### **Non-Modifiable Risk Factors**

This section will provide some background information on those risk factors that are considered to be significant precursors of stroke.

##### **Age.**

Advancing age is strongly associated with an increase and prevalence of stroke and is the principal non-modifiable risk factor for this disease (Falcone & Chong, 2007; Petrea et al., 2009; Sacco et al., 1997). For each decade after the age of 55, for both men and women, the risk of stroke more than doubles (Cubriilo-Turek, 2004). Reasons for this increase include the increased exposure to environmental risk factors and higher prevalence of risk factors associated with age including atrial fibrillation, hypertension, diabetes, and coronary heart disease (Wattigney, Mensah, & Croft, 2003).

##### **Gender.**

To date, research has produced contradictory findings for gender differences in the epidemiology, outcomes and treatment of stroke. A recent worldwide systematic review of the literature on gender differences in stroke epidemiology concluded that although stroke is more common among men, women are more severely ill. A number of studies have found that not only do men have higher age-specific rates than women but compared to women men are also more likely to have their first-ever stroke at a younger age (Appleros, Nydevik, & Viitanen, 2003; Petrea, et al., 2009; Roquer, Campello, & Gomis, 2003). Conversely, other researchers conclude that due to a significant increase in the incidence of stroke for women over the age of 84, women overall, experience more strokes (Niewada,



Kobayashi, Sandercock, Kaminski, & Czlonkowska, 2005; Reeves et al., 2008).

This large increase in incidence in elderly women raises the possibility of biological explanations such as the neuro-protective effect of estrogen. This theory presents a challenging subject area for further research (Suzuki, Brown, & Wise, 2009).

There is a growing body of research implicating gender as a confounding factor for stroke sub-type, but again the findings are diverse and inconsistent. For example, Appleros et al. (2003), Ayala et al. (2002), and Reeves et al. (2008), provide evidence for higher incidence of subarachnoid haemorrhages in women than men, although in another study, Bogousslavsky, Van Melle, and Regli (1988), found a greater association with men than women for haemorrhagic stroke in patients younger than 50 years old. In another study of residents of a European city, the authors found the incidence rate for large-artery atherosclerosis, to be twice as high for men than for women (Blacker & Brown 2002; Grau et al., 2001; Kolominsky-Rabas, Weber, Gefeller, Neundoerfer, & Heuschmann, 2001) although other studies have found age-related large-artery atherosclerosis to be greater in post-menopausal women (Ahimastos, Formosa, Dart, & Kingwell, 2003; Goto, Baba, Ito, Maekawa, & Koshiji, 2007; Kingwell et al., 2001). Possible explanations for these gender differences lie in genetic and hormonal modulations, although a clearer understanding of these differences needs further investigation.

A number of studies have consistently identified women as experiencing poorer outcomes and greater dependency post stroke (Falcone & Chong, 2007; Petrea, 2009; Reeves, et al., 2008; Roquer et al., 2003). The causes for these disparities are explained by a number of factors. Overall women are older when

they have their stroke, they tend to have poorer pre-stroke functioning, more comorbidity, such as depression, are less likely to have social support and more likely to be widowed or divorced (Reeves et al., 2008). Gender has also been found to be a factor in other areas of stroke including, presentation (Falcone & Chong, 2007), diagnosis (Smith, Lisabeth, Brown, & Morgenstern, 2005) and the type of treatment used in acute stroke therapy (Smith, Murphy, Santos, Philips, & Wilde, 2009). Indeed, while it appears gender is likely a marker for multiple medical, genetic, and socio-economic factors concerning stroke, some researchers caution that these differences are over-estimated and there is general consensus that further studies are required to elucidate the situation (Falcone & Chong, 2007; Smith et al., 2009; Zhang et al., 2007).

### **Ethnicity.**

Ethnicity is a complex and heterogenous concept incorporating a wide range of characteristics such as biology, history, culture, language, and religion. Fustinoni and Biller (2000) in their editorial on ethnicity as a variable in stroke research, caution against assumptions and possible bias when classifying ethnicity. For example, ethnicity is largely influenced by cultural attitudes; “what is black to someone from the United States may be white to a Brazilian or a Caribbean islander”. A number of studies have used self-classification in their methodology (Bonita, Broad, & Beaglehole, 1997; Carter et al., 2006; Sacco et al., 1998), however, the authors also point out flaws in this process such as misinterpretation, confusion and self-reclassification. They give the example of some respondents thinking that “South and Central American” referred to natives of the south and central United States. They also discuss how the influence of socio-economic and associated risk factors might be hidden within ethnic groups,

with the example of affluent “blacks” from the US East Coast not necessarily developing the same disease patterns as poor inhabitants from a comparable group in the southern states. They suggest genetic research to determine common ancestry may provide some clarity to the heterogeneity of ethnicity, which in itself is a confounding variable. Fustinoni & Biller (2001) express concern that flawed ethnic research might lead to an assumption that ethnic minorities are a social health burden compared to “white” populations, thus adding fuel to racial prejudices.

Harwood, McNaughton, McPherson, and Weatherall (2000), state that despite the heterogeneity of ethnicity, it has nevertheless consistently been shown to be a significant variable in increasing risk of stroke and has served particularly well for providing a framework of understanding of difference in stroke outcomes in the Pacific Rim region. They propose that research focusing on the inequities of access and quality of stroke care for ethnic groups will be more rewarding than research into genetics. Furthermore, they suggest that all viewpoints of ethnic disparities in stroke incidence are worthy of further discussion and investigation however in accordance with good research practice and to ensure the validity of data particularly when comparing studies, researchers should clearly describe the logic behind their "ethnic" groupings (Senior & Bhopal, 1994).

Evidence for ethnic disparities in stroke incidence, severity, and mortality has continued to mount in recent years. Substantial evidence highlighting an increased risk of stroke and increased mortality for ethnic minority groups has emerged from a number of countries including the United States, the United Kingdom and New Zealand. A number of studies have provided evidence that ethnic minority groups tend to have their strokes at a younger age, (Bonita et al.,

1997; Bravata et al., 2005; Carter et al., 2006; Fink, 2006; Markus, et al., 2007; Sacco et al., 1998; Wolf et al., 2002; Wolf & Kannel, 2007) even after socioeconomic status had been factored out (Bravata et al., 2005; Howard et al., 1995; Markus et al., 2007; Stewart et al., 1999). However, other authors have found that socioeconomic status is the main contributing factor in the higher incidence and mortality rate of stroke in ethnic minorities (Bravata et al., 2005; Gillum & Mussolino, 2003; Maheswaran, Elliott, & Strachan, 1997). Howard et al. (1995) argued however that although socioeconomic status is important in stroke mortality risk, it is but one component of a complex picture that may include a range of compounding factors. Other researchers also attribute a range of risk burden factors such as, possible genetic susceptibility, hypertension, hyperlipidaemia, diabetes, obesity, tobacco smoking and physical inactivity, as determinants for the higher stroke incidence consistently found in ethnic minorities (Carter et al., 2006; Feigin et al., 2006; Fink, 2006; Stewart et al., 1999; White et al., 2005). Fink (2006), states that reduced stroke incidence observed among European populations in New Zealand in the last 20 years can be credited to improved primary and secondary prevention of cardiovascular disease, however as he points out these are improvements that have not been achieved among Maori and Pacific people in New Zealand. A similar observation is made by Heuschmann, Grieve, Toschke, Rudd, and Wolfe (2008) in their study investigating a 10-year ethnic trend in stroke incidence with the South London Stroke Register, where they attribute the decrease in whites' incidence of stroke compared to blacks, to changes in prior-to-stroke risk factors. Compared to whites, blacks had made fewer gains in reducing rates of hypertension, diabetes, smoking and atrial fibrillation.

Epidemiological studies of migrating populations also consider the management of risk factors as major determinants in stroke incidence for ethnic groups. For example, Khaw (1996) points out that rates of strokes for Japanese populations in the United States are closer to American white populations than Japanese populations in Japan and suggests better medical care, changing severity of disease and reduced case fatality are responsible for the lower rates in the United States.

### **Modifiable Risk Factors**

#### **Hypertension.**

Hypertension, also known as high blood pressure, is believed to be responsible for about 60-70 % of all strokes world-wide (Cubrillo-Turek, 2004; WHO, 2009). Over time, damage to the arteries caused by high blood pressure impairs blood flow by the rupturing of the blood vessel or by causing plaque to build up thus creating a clot which can then cause a stroke. Hypertension is considered to be the most important modifiable factor in reducing the incidence of stroke. (Aszalos, Barsi, Vitrai, & Nagy, 2002; Herekar & Hilal, 2008; Khan, Rehman, Shah, & Jielani, 2006; SHEP Cooperative Research Group, 1991; Straus, Majumdar, & McAlister, 2002). In their statement for the Primary prevention of Stroke, professionals from the Stroke Council of the American Heart Association identified elevated systolic blood pressure with or without an accompanying elevation in diastolic blood pressure, as increasing the risk of stroke (Goldstein et al., 2001). Several other authors have implicated elevated systolic blood pressure in increased risk of stroke (He & Whelton, 1999; Kannel et al., 1981; Kurl et al., 2001). The treatment of hypertension has been consistently demonstrated for lowering blood pressure yet despite this knowledge,

a significant proportion of people remain undiagnosed or inadequately treated for hypertension (Goldstein et al., 2001; Hypertension Detection and Follow-up Program (HDFP), 1982).

### **Atherosclerosis.**

Atherosclerosis refers to the thickening of the artery walls as a result of the build up of plaque which is made up of fatty materials such as cholesterol, fat, calcium and other substances found in the blood. When the arteries supplying the brain are partially or totally narrowed, blood flow is obstructed resulting in a stroke. Alternatively, pieces of the plaque itself or clot that forms on the plaque can break off and block off smaller arteries downstream (Di Tullio, Homma, & Sacco, 2008; O'Leary et al., 1999). When such narrowing of the arteries occurs in the head, the condition is called intracranial atherosclerosis and is the dominant cause of stroke in over 70% of the world's population (Kim, Caplan, & Wong, 2008).

Atherosclerosis and Stroke share many of the same risk factors including those that are modifiable such as; high blood pressure, diabetes, hyperlipidaemia, tobacco use, heavy alcohol consumption, obesity, physical inactivity and an unhealthy diet, as well as non-modifiable factors including advancing age, gender and a family history of early atherosclerosis (Blacker & Brown, 2002; Hobson, Wilson, & Veith, 2004)

### **Atrial Fibrillation.**

Of all the cardiac diseases atrial fibrillation is considered to be the most powerful and treatable precursor of stroke. Atrial Fibrillation describes the rapid irregular beating of the upper chamber of the heart which can result in slow blood flow and the subsequent formation of blood clots. Anticoagulant and antiplatelet

medications are often used to thin the blood and reduce the likelihood of clotting which leads to stroke. Atrial fibrillation is known to rise markedly with increasing age (Frost, Andersen, Godtfresden, & Mortensen 2007; Spence, 2006; van Walraven et al., 2009; Wolf, Abbott, & Kannel, 1987) and has been well identified as an independent risk factor for stroke (Kannel et al., 1981; Rastas et al., 2007; Tsang et al., 2003; Wolf et al., 1987). A stroke may result when a blood clot (embolus) breaks off, travels through the blood stream and lodges in the artery leading to the brain. Atrial fibrillation has been estimated to increase the risk of stroke about five-fold, particularly in the elderly in whom the prevalence of atrial fibrillation is high (Lancaster, Mant, & Singer, 1997; Wolf, Abbott, & Kannel, 1991). Data from the Framingham study also showed that ischaemic stroke that occurred with atrial fibrillation is almost twice as likely to be fatal than stroke without atrial fibrillation (Lin et al., 1996).

### **Diabetes.**

Diabetes mellitus is a disorder of metabolism which results in the body accumulating glucose in the blood. The excess glucose can then attach to proteins in the blood vessels creating a build up of plaque which causes them to become thicker and less elastic making it hard for blood to flow through. Diabetes has been well established as a risk factor for stroke (Burchfield et al., 1994; Davis, Millns, Stratton, Holman, & Turner, 1999; Goldstein et al., 2001; Lukovits, Mazzone, & Gorelick, 1999; Manson et al., 1991), with several studies indicating a two-fold increase of stroke risk with higher associated mortality and morbidity (Abbott, Donahue, McMahan, Reed, & Yano, 1987; Jeerakathil, Johnson, Simpson, & Majumdar, 2007; Kissela et al., 2005). Cubrilo-Turek (2004) suggests that the population attributable risk for diabetes causing stroke is 15-20%.

Furthermore, diabetic stroke patients have a worse prognosis than non-diabetic stroke patients, with a two-fold increase in the likelihood of recurrent stroke.

While advanced age is the single most non-modifiable risk factor for stroke in the general population, in patients with diabetes younger than 55 years, the risk of stroke increases ten-fold (You et al., 1997). In their study of ischemic stroke patients Kissela et al. (2005), found those patients with diabetes to be not only younger than non-diabetic patients but also more likely to be African American, to have hypertension, myocardial infarction, and high cholesterol. Indeed, people with diabetes are often more susceptible to other stroke risk factors such as hypertension, abnormal cholesterol levels, atherosclerosis and ischaemic heart disease (Cubriilo-Turek, 2004; Lehto, Ronnema, Pyorala, & Laakso, 1996; Lithner et al., 1998). Therefore, as well as management of blood glucose, diabetes care needs to include management of blood pressure and cholesterol in order to reduce the risk of stroke.

### **Migraine.**

A number of studies have shown an increase in the risk of stroke among people who have a history of migraine (Chang, Donaghy, & Poulter, 1999; Connor, 1992; Dorfman, Marshall, & Enzmann, 1979; Tzourio, et al., 1995). This association has been linked predominantly to migraine with aura (Bousser & Welch, 2005; Diener & Kurth, 2005; Rothrock et al., 1993) although not always (Carolei, Marini, & De Matteis, 1996). Some suggest that the relationship between migraine and stroke is bidirectional, i.e. cerebral infarction can also cause migraine (Bousser & Welch, 2005; Thomas, 2005). The mechanisms between these two conditions is unclear as is the pathophysiology of migraine, however one theory suggests that because migraine is essentially the result of



haemodynamic changes within the brain, it is believed that stroke arises from persistent decreased blood flow (Estol, 2001).

Another theory implicates Patent Foramen Ovale (PFO), (an incomplete closure of the wall between the two upper chambers of the heart, also known as “hole in the heart”), which is present in approximately 50% of individuals who experience migraine with aura. The prevalence of PFO among stroke patients with migraine associated with aura is also high (Mohammed, Ormerod, & Downes, 2006). If the PFO is surgically closed the occurrence of migraine has been shown to either cease or significantly reduce in intensity and frequency (Jesurum et al., 2008; Sztajzel, Genoud, Roth, Mermillod, & le Floch-Rohr, 2002; Tobis & Azarbal, 2005). The closure of the PFO prevents a clot that may have formed in the vein from passing across the heart chambers into the arterial system and travelling up to the brain where it may block a vessel and cause a stroke (Rakhit, 2003).

#### **Sickle cell anaemia.**

Sickle cell disease is a heritable condition characterised by chronic anaemia and episodes of pain. It is a blood disorder in which red blood cells mutate assuming an abnormal rigid sickle shape. Although sickle cell disease is more common in people of African and Mediterranean descent, it also presents in people from South and Central America, the Caribbean and the Middle East (Lee et al., 2006; WHO, 1996). Passage of cells through the blood vessels can become difficult and the sickle cell may become jammed causing a blockage that impedes the passage of oxygen and nutrients subsequently leading to stroke. The internal carotid artery and the middle cerebral arteries are frequently affected and commonly produce severe neurological deficits with even more disastrous results

in those who suffer recurrent strokes. Recent reports put the prevalence of stroke in sickle cell disease as ranging from 4% to 8% (Balkaran et al., 1992; Johnson, Unwin, & Graybeal, 2001; Ohene-Frempong, 1991). Approximately 10% of children with sickle cell disease suffer a stroke making it the most common form of stroke in children. 24% of patients with sickle cell anaemia will experience a stroke by the time they are 45 years old (Switzer, Hess, Nichols, & Adams, 2001; Wethers, 2000).

Ischaemic stroke is more common in children and older patients, whereas haemorrhagic stroke has been found to occur more often in late adolescence and early adulthood (Adams, 2001; Ohene-Frempong et al., 1998; Sarniak & Lusher, 1982, cited in Johnson et al., 2001). Death from cerebral infarction is rare however haemorrhagic stroke are fatal in approximately 25% of those patients with sickle cell anaemia (Switzer et al., 2001). Since the 1970's researchers have been aware that patients who have suffered a stroke are at high risk of recurrence and that chronic maintenance transfusion is highly effective in preventing that recurrence (Josephson, Su, Hillyer, & Hillyer, 2007; Platt, 2006; Riddington & Wang, 2002; Russell et al., 1984; Stockman, Nigro, Mishkin, & Oski, 1972). Although there is no cure for this disease, it is nevertheless a treatable condition. A healthy life style together with available treatments including pain management, blood transfusions, and pharmaceutical interventions can help people with sickle cell disease live with reasonably good health much of the time.

### **Behaviour/Lifestyle Changes**

#### **Tobacco.**

Tobacco use is well established as a significant independent risk factor for the occurrence of stroke (Center for Disease Control and Prevention. 2004;

MacKay & Mensah, 2004; Manolio, Kronmal, Burke, O'Leary, & Price, 1996; Wolf et al., 1988). The chemicals in tobacco smoke increase the build up of plaque in artery walls and promote the development of blood clots that can cause strokes. According to Cubrilo-Turek (2004), smoking doubles the risk of stroke there is a clear relationship between the number of cigarettes smoked and the risk of stroke. Kurth et al. (2003), provided evidence that smoking 15 cigarettes per day increases the risk of stroke by up to four times. A number of researchers have also found a positive correlation between the length of time smoking and increased stroke risk (Adams, 2006; Bhat et al., 2008; Tamargo & Conway, 2006). As well as increasing the risk of ischaemic stroke, tobacco use has also been demonstrated to increase the risk of subarachnoid haemorrhage (Isaksen, Egge, Waterloo, Rommer, & Ingebrigsten, 2002; Qureshi et al., 2001) and haemorrhagic stroke in both men and women (Kurth et al., 2003). The authors of the American Heart Association and the American Stroke Association published guidelines on the Primary Prevention of stroke in their systematic review of the literature, associated cigarette smoking with a 2 to 4-fold increased risk of haemorrhagic stroke (Broderick et al., 2003). They suggest that smoking contributes to 12% to 14% of all stroke deaths. Second-hand smoking has also been firmly established as contributing to the incidence of stroke (Bonita, Duncan, Truelsen, Jackson, & Beaglehole, 1999; Glymour, DeFries, Kawachi, & Avendano. 2008; You, Thrift, McNeil, Davis, & Donnan, 1999).

### **Alcohol.**

Heavy drinking is associated with hypertension and atrial fibrillation and as such alcohol has been correlated with increased risk of stroke (Furie & Kelly, 2004; Marmot & Poulter, 1992; Mukamala et al, 2005). However, research

investigating a direct relationship between alcohol and stroke has produced mixed results. In 1989, Gorelick failed to find any association between alcohol consumption and cerebral infarction in middle-aged and elderly patients.

Conversely, other authors have found a positive relationship between heavy alcohol consumption and increased risk for stroke (Caicoya, Rodriguez, Corrales, Cuello, & Lasheras, 1999; Gill et al., 1991; Mohr, Choi, Grotta, Weir, & Wolf, 2004; Mukamala et al., 2005; You et al., 1997). A 2003 meta-analysis reviewing 35 studies indicated that >60 g of alcohol per day increased the risk of stroke although light or moderate consumption, i.e. <24 g per day decreased the risk of stroke when compared with abstainers (Reynolds et al., 2003). There has been further evidence for an association between moderate consumption and a reduced risk of stroke (Berger et al., 1999; Carmago, 1989; Elkind et al., 2006; Sacco et al., 1999; Stampfer, Colditz, Willet, Spaizer, & Hennekens, 1988). Gill, Zezulka, Shipley, Gill, and Beevers (1986), found those drinkers whose consumption was 10 to 90 g of alcohol weekly had a 0.5 lower relative risk of stroke than non-drinkers. In an epidemiological review of the literature Camargo (1989) reported the same relationship in predominantly white populations however minimal evidence of any such association was found within a Japanese population. There is also evidence for an association between heavy drinking and an increased risk of haemorrhagic stroke (Ebrahim et al., 2006; Iso et al., 2004) although Klatsky, Armstrong, Friefman, and Sidney (2002) only found a weak correlation.

However, unlike the protective effect of moderate drinking for the risk of ischaemic stroke, (Daniel & Berezki, 2004), this did not appear to be the case for the occurrence of haemorrhagic stroke. The association between heavy drinking and subarachnoid haemorrhage has also been firmly established (Hillbom &

Kaste, 1981; Juvela, Hillbom, Numminen, & Koskinen, 1993; Longstreth, Nelson, Koespell & van Belle, 1992). There is a growing pool of evidence to suggest that the type of alcohol consumed may alter stroke risk factors. For example, wine may convey greater benefit than other type of alcohol (Malarcher et al., 2001; Truelsen, Gronbaek, Schnohr, & Boysen, 1998). Mukamala et al. (2005) showed that men who drank red wine had a 23 percent lower risk of a stroke compared with those who drank other types of alcohol although further research was recommended in order to validate their findings.

### **Physical Inactivity.**

A clear association between physical inactivity and an increase in the risk of stroke has been established (Abbot, Rodriquez, Burchfield, & Curb, 1994; Gillum, Mussolino, & Ingram, 1996; Goldstein & Amarenco, 2005; Hu, Tuomilehto, Silventoinen, Barengo, & Jousilahti, 2004). Several studies have also demonstrated an inverse relationship between increased physical activity and stroke risk factors such as hypertension, high cholesterol, obesity, diabetes and the development of atherosclerosis (Gorelick & Alter 2002; Heart and Stroke Foundation of Ontario website; Warburton, Nicol, & Bredin, 2006). There is some evidence of a dose-response gradient of reduced stroke with increasing activity (Dishman, Washburn, & Heath, 2004), although Kiely, Wolf, Cupples, Beiser, and Kannel (1994), only found a protective effect of medium and high levels of physical activity for males and not for the females in their study. A meta-analysis of the literature covering 1966 to 2002 concluded that there was a lower risk of both ischaemic and haemorrhagic stroke in moderate and highly active individuals (Lee, Folsom, & Blair, 2003), a finding that was replicated in a more recent meta-analysis of 31 observational studies (Wendel-Vos et al., 2004).

**Obesity.**

The relationship between obesity and stroke remains controversial primarily because of methodological inconsistencies across studies. Those studies using Body Mass Index (BMI) as an indicator have wrongly included short well-muscled individuals as overweight while other studies have used waist-to-hip ratio as the indicator. Nevertheless, obesity and in particular abdominal adiposity and waist circumference and the occurrence of ischaemic stroke have a positive relationship (Godfrey & Sacco, 2009; Grau et al., 2001; Hu, 2008; Mohr et al., 2004; Suk et al., 2003). The occurrence of stroke as a result of obesity is thought to occur through the mechanics of hypertension and diabetes (Shinton, Sagar, & Beevers, 1995). The implications of obesity for other stroke types also remain unclear although Kurth et al. (2002) found a positive correlation between BMI and occurrence of ischaemic and haemorrhagic stroke independent of confounding risk factors such as hypertension, diabetes and high cholesterol. The World Health Organisation warns against a potential surge in stroke incidence as the obesity epidemic spreads across developed countries.

**Diet.**

Due to the association of diet with obesity and its complex interactions with other lifestyle factors, e.g. people with nutritional diets tends to smoke less, it is difficult to exactly determine the extent to which diet alters the risk of having a stroke. A number of studies have demonstrated that healthy nutritional habits play a significant role in mitigating other stroke risk factors including hypertension, high cholesterol, diabetes and cardiac disease (Amarenco, Labreuche, & Touboul, 2008; Spence, 2006). A Western style diet that is typically high in fat, sugar and salt has been found to increase the risk of stroke

(Metzger, Kotulak, & Brick, 2006) by raising triglycerides and increasing blood pressure. Conversely, several studies examining diets that are low in saturated fats and sodium and high in fruit and vegetables have been shown to significantly reduce the risk of stroke (Dauchet & Dallongeville, 2008; Gillman et al., 1995; Sauvaget, Nagano, Allen, & Kodama, 2003; Van Duyn & Pivonka, 2000). Fung et al. (2009) found that the Mediterranean diet comprising of fresh fruit, an abundance of plant foods, dairy foods (cheese and yoghurt), olive oil, moderate amounts of fish and poultry, low amounts of red meat, zero to four eggs consumed weekly and moderate amounts of wine, was associated with a lower risk of stroke in women.

Research into diet and stroke subtype is scarce although there is some evidence that consumption of baked or boiled fish is associated with a decreased risk of ischaemic stroke (He et al., 2002; He et al., 2004). Mozaffarian et al. (2005) had a similar outcome in their study investigating fish consumption in an elderly population although in the same group they found that fish that had been fried increased the risk of haemorrhagic stroke.

## Appendix B

### Outcomes of Stroke

#### Survival

Survival from stroke is influenced by various factors including stroke subtype, the extent of the neurological damage, level of consciousness; the presence of cardiac disease, hypertension and diabetes; previous stroke history, impact of disability, and level of supportive and rehabilitative care, to name a few (Sacco, 2005; Williams & Jiang, 2000).

Survival rates for patients vary considerably between studies however the greatest risk of death appears to occur in the first 30-days post-onset and is related to type of stroke (Rundek & Sacco, 2004; Warlow, Dennis, van Gijn, Hankey, & Sandercock, 2001). Fatality rates are greater for patients with intracerebral haemorrhage than patients with cerebral infarction (Boden-Albala & Sacco, 2004; Vernino et al., 2003; Wang & Talkad, 2009). In the Rochester study covering the course of stroke from 1955 through to 1969, the overall 30-day survival rate was 72% (Matsumoto, Whisnant, Kurland, & Okazaki, 1973). The same survival rate was found in an analysis of the Framingham study covering the years 1971 through 1981 (Kelly-Hayes et al, 1988) and in the Oxfordshire Community Stroke Project, the overall stroke survival rate for the years 1981 through 1986, was 81% (Bamford et al., 1990). In all three studies, survival rates for patients who sustained an ischaemic stroke were considerably higher than those who had suffered haemorrhagic stroke. In a recent systematic review of worldwide stroke incidence and case fatality, 21-day to 1-month survival rates for all strokes in high income countries for the period 2000 through 2008, was between 17 and 30% and 18 to 35% in low and middle income countries. Again, there was a significantly



higher fatality rate for haemorrhagic stroke than for ischaemic stroke (Feigin et al., 2009).

Most recent studies have failed to establish a gender difference in both short-term and long-term survival rates of stroke (Appleros et al., 2003; Carlo et al., 2003; Glader et al., 2003; Terent, 2003). However, some studies have demonstrated better survival rates for either men (Arboix et al., 2001) or women (Andersen, Andersen, Kammersgaard, & Olsen, 2005; Niewada, et al., 2005; Olsen, Dehlendorff, & Andersen, 2007).

The “clot busting” agent tissue plasminogen activator (tPA) has been shown to be an effective treatment resulting in increased survival for ischaemic stroke patients (Lansberg Bluhmki, & Thijs, 2009; NINDs, 1995; Marler, 2005; Wardlaw, Lindley, & Lewis, 2002). However, there is a narrow 3 to 4.5 hour time-to-treat window with greater benefits achieved the sooner the treatment takes place (Hacke et al., 2004; Temporal, 2005). In a randomised double-blind trial of 291 ischaemic stroke patients who were given either tPA or a placebo, the former group performed better at 3 months across four measures including the Barthel Index, the Modified Rankin scale, The Glasgow Outcome Scale and the NINDS (National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group, 1995). These findings were replicated in a similar more recent study providing further encouragement for the use of tPA for the treatment of acute ischaemic stroke (Schmulling, Grond, Rudolf, & Heiss, 2000).

In a New Zealand study, improved technology and an increase in hospital admissions were two factors credited for a rise in survival rates and better outcomes for stroke patients in recent years, a trend that was observed in the USA in the 1980s and in other developed high-income countries

(Carter, Anderson, Hackett, Barber, & Bonita, 2007; Feigin et al., 2009; Lakshminarayan, Anderson, Jacobs, Barber, & Luepker, 2009; McCarron, Smith, & McCarron, 2006; Shahar et al., 1995).

### **Recurrence**

Stroke recurrence rate is an important outcome measure, as patients who suffer recurrent stroke have poorer outcomes than those with first-ever strokes (Ng, Jung, Chiong, & Lim, 2007). A 10-year prospective study of patients with suspected acute stroke or TIA, found the risk for recurrent stroke to be 6 times greater compared to the general population (Hardie, Hankey, Jamrozik, Broadhurst, & Anderson, 2004). Other authors have suggested a stroke recurrent rate of 25% to 35% (Diller, 1999; Williams et al., 1999) although most studies indicate variance at different stages post-stroke. For example, large community-based studies have found recurrence rates of stroke to be 1.7% to 4% in the first 30 days, from 6% to 13% in the first year and from 5% to 8% per year for the next 2 to 5 years, with a cumulative risk of 19 to 42% over the first 5 years (Petty et al., 1998; Sacco, Shi, Zamanillo, & Kargman, 1994). Another study of patients on the South London Stroke Register found the cumulative risk of stroke recurrence at 1 year, 5 years and 10 years was 7.1%, 16.2% and 24.5%, respectively (Mohan, Richton, Grieve, Wolfe, & Heuschmann, 2009). Furthermore, mortality after recurrent stroke was found to be almost doubled compared with patients with a first-ever stroke (Jerrgensen, Nakayama, Reith, Raaschou, & Olsen, 1997). Although research into the risk for the recurrence of stroke has produced contrary findings, largely because of methodological differences, it is nevertheless evident that stroke recurrence

is frequent and responsible for a major portion of overall stroke morbidity and mortality (Sacco, 1994).

Studies comparing the recurrence of specific stroke subtypes found that patients with atherothrombotic infarcts and cardioembolic stroke have a higher risk of recurrence compared to patients who have suffered a lacunar infarct (Hata et al., 2005; Modrego, Mainar, & Turull, 2004; Petty et al., 2000; Sacco et al., 1989). For example, Modrego et al. (2004) found the cumulative recurrence rates for atherothrombotic stroke at 1 month, 1 year and 5 years to be 2%, 11% and 28% respectively; 3.5%, 12.5%, and 25% for cardio-embolic stroke and 1.2%, 9%, and 22% for lacunar stroke. In their longitudinal study of ischaemic stroke patients, Petty et al. (2000), estimated rates of recurrent stroke at 30 days was 18.5% for athero-thrombotic stroke, 5.3% for cardio-embolic strokes and 1.4% for lacunar strokes. Two recent studies found that patients with posterior circulation TIA or stroke, although less common than stroke involving the anterior circulation, have a higher recurrent stroke risk (Azarpazhooh et al., 2008; Gulli, Khan, & Markus, 2009).

Mohan et al. (2009) found that prior-to-stroke risk factors were also significant determinants of recurrent stroke. Other risk factors associated with greater recurrence of stroke include; previous TIA, atrial fibrillation, ischemic heart disease, hypertension, and diabetes (Alter et al., 1987; Yokota, Minematsu, Hasegawa, & Yamaguchi, 2004). Tatemichi et al. (1993) also found the presence of cognitive impairment to be associated with increased risk of recurrent stroke.

It is critical that appropriate management of risk factors for recurrent stroke are addressed soon after the event. A number of treatments have been recommended to reduce the recurrent of secondary stroke including, placing all stroke survivors on aspirin or an alternate antiplatelet drug (Norris, 2005) lowering blood pressure, treating hypertension, or placing the stroke patient on statin therapy (Goldstein et al., 2009; PROGRESS Collaborative Group, 2001; Sacco et al., 2006),

Stroke outcomes are largely determined by site and severity of the lesion although many other variables such as age, co-morbidities, general well-being and previous stroke history can also influence the effects of the stroke. The following sections will provide a brief outline of the physical, perceptual, psychological or behavioural effects of stroke?

### **Visual Field Deficits of Stroke**

#### **Homonymous Hemianopia.**

Homonymous Hemianopia refers to partial blindness which results in a loss of vision in the same visual field of both eyes and can arise out of many types of injuries to the brain including tumour, trauma, infection, epilepsy, arteriovenous malformation, or stroke, to name a few. However, it is most commonly a neuro-ophthalmological manifestation of stroke (Gray et al., 1989; Kelley & Kovacs, 1986; Peli, 2000; Rowe et al., 2009). This condition which usually affects the peripheral vision, results in problems with reading and visual scanning because people fail to notice stimuli in the affected field. For example, a lesion of the right occipital lobe will affect the left visual fields of each eye. It may also cause some individuals to run into objects, trip or fall, knock things over, or be surprised by objects that

seem to appear out of nowhere (Anderson, 2002). The ability to drive a vehicle safely is compromised and indeed in some countries, individuals with homonymous hemianopia are not legally able to do so.

In Australia, a study of the implications of post-stroke homonymous visual field (PSHVF) loss for driving implications, found a prevalence rate of 16% for homonymous visual field defects amongst sixty one stroke patient admissions (Townsend et al., 2007). A more recent study in the UK investigating the prevalence of visual impairment following stroke found 29.4% of their 323 study sample suffered homonymous hemianopia (Rowe et al., 2009). Rossi, Kheyfets and Reding (1990), suggest as many as a third of all stroke patients may suffer homonymous hemianopia. This disorder has been found to have an adverse effect on recovery with considerable social and vocational consequences (Kalra, Smith, & Crome, 1993).

Visual neglect is sometimes confused with homonymous hemianopia as they are both visual deficits. However, the latter is a physical loss of visual field to one side, while the former is a problem of inattention. The patient may or may not suffer from homonymous hemianopia but due to the visual neglect, cannot learn to compensate because they cannot mentally attend to that side. An in-depth discussion of visual neglect is provided later in this chapter in the section on attention deficits.

### **Diplopia.**

Diplopia is the medical term for what is commonly known as double vision (i.e., the individual sees two images of a single object at the same time). The condition is not a problem with the mechanics of the eyes but rather results from damage to those parts of the brain that control and co-

ordinate eye movements. Aetiology of diplopia is wide and varied however, sudden onset is typical when a person experiences a stroke that affects the vertebrobasilar system which usually affects the brainstem, cerebellum, and occipital lobe (Bogousslasky & Caplan, 2001). Diplopia may be intermittent or constant, horizontal, vertical or tilted and may also be distance dependent (Caplan, 2005; Stein, 2004; Warlow et al., 2001; Wiebers, Feigin, & Brown, 2006). A number of authors have shown a high incidence of visual disturbance, including diplopia, following stroke (Clisby & Cox, 1999). This disorder can present considerable problems for functional recovery. For example, Rathore, Hinn, Cooper, Tyroler, and Rosamund, (2002), in their study of 474 confirmed stroke hospitalised patients found a 5.5% occurrence of diplopia. Diplopia can be a major handicap to good functional outcomes (Barker-Collo & Feigin, 2006).

### **Motor Deficits**

#### **Hemiplegia/Hemiparesis.**

Hemiplegia refers to the total paralysis of the arm leg and trunk while Hemiparesis is weakness of one side of the body. Both disorders affect the contralateral side of the body to the brain lesion because the corticospinal tract which runs down from the cortical neurons of the frontal lobe to the motor neurons of the spinal cord crosses to the opposite side at the lowest point of the medulla known as *the pyramids*. Hemiplegia is common following stroke, occurring in as many as 75% to 88% of stroke survivors at 30 days with a high incidence of associated neurological deficits in the acute stage (Diller, 1999). Hemiparesis is also common with some authors estimating occurrence between

80-90% of all patients with stroke (Bogousslavsky et al., 1988; Herman et al., 1982; Libman, Sacco, Shi, Tatemichi, & Mohr, 1992)

### **Ataxia.**

Ataxia is the term used to refer to a neurological deficit characterised by a gross lack of muscle co-ordination manifested by disjointed or jerky movements resulting in problems with walking or picking up objects. Ataxia occurs as a consequence of damage to that part of the nervous system that coordinates movement, such as the cerebellum (Timmann & Diener cited in Bogousslavsky & Caplan 2001 p. 53). Speech, eye movement and ability to swallow can also be affected by this disorder (Bendheim & Berg, 1981; Deluca et al., 2011).

### **Dysarthria.**

Dysarthria is an acquired motor speech disorder characterised by dysfunction in the initiation, control and co-ordination of those articulatory structures involved in speech output. It is a clinical manifestation of cerebral ischaemia and has been observed in 8 – 30% of all patients in a large number of stroke studies (Urban et al., 2006). Dysarthria has been identified with poorer outcomes following stroke (Tilling et al., 2001).

### **Dysphagia.**

The initiation of swallowing is a voluntary action that involves the integrity of the motor and sensory areas of both cortices while the reflexive component of swallowing is mediated by swallowing centres in the brain stem (Martin & Sessle, 1993; Miller, 1982; Singh & Hamdy, 2006). Disruption or difficulty of the swallowing process is termed dysphagia. The most common symptom of dysphagia post-stroke is difficulty trying to

swallow, although other symptoms such as choking or breathing saliva into the lungs while swallowing, coughing while or after swallowing, regurgitating liquid through the nose, excessive throat clearing, breathing in food while swallowing, weak voice and weight loss, may also be present (Edmans et al., 2010). Disturbance of swallowing symptoms is related to the neurological lesion site (Steinhagen, Grossman, Benecke, & Walter, 2009).

The incidence of dysphagia following stroke is high (Bougousslavsky & Caplan, 2001; Gordon, Hewer, & Wade, 1987; Langdon, Lee, & Binns, 2007; Mann, Hankey, & Cameron, 2000; O'Neill, 2000; Martino et al., 2005). In their systematic review of the literature Martino et al. (2005) found that the frequency of dysphagia following stroke, ranged from 37% to as high as 78% with higher rates detected when more accurate instrumental screening tools, as opposed to clinical assessment, were used. Different inclusion criteria across studies also impacted on the rates of dysphagia found across studies. Recovery time from dysphagia depends on numerous factors including severity and site of the lesion, comorbidities, assessment protocols and treatment regimes. Although symptoms will often resolve in the acute stage of recovery, dysphagia has also been found to be a predictor of delayed recovery, poorer outcomes and serious complications such as aspiration pneumonia. (Foley, Teasell, Salter, Kruger, & Martino, 2008; Langdon et al., 2007; Mann, et al., 2000; Martino et al., 2005; Obara, Tomite, & Doi, 2008; O'Neill, 2000; Sharma, Fletcher, Vassallo, & Ross, 2001; Singh & Hamdy, 2006; Smithard et al., 1996).



### **Aspiration pneumonia.**

Aspiration pneumonia is a lung infection caused by the inhalation of foreign material into the lungs. These tiny particles are normally prevented from entering the airways by a complex physiological mechanism which is compromised in the patient with dysphagia thus allowing oral contents to drop into the trachea and then into the lungs. The contaminated contents enter the more sterile environment of the lungs causing infection and inflammation (Kaste & Roine, 2004; Marik, 2001; Mohr et al., 2004; Speiker, 2001). Stroke survivors who are diagnosed with aspiration pneumonia have an increased risk of dying when compared to patients who do not have pneumonia (Hreib, 2008; Meng, Wang, & Lien, 2000; Reynolds et al., 1998), however studies have shown a number of protective measures such as early assessment, improved oral and dental care, positioning of patients, treatments of reflux, unidirectional speaking valves and nasogastric feeding as been effective in reducing the risk of aspiration pneumonia (Drakulovic et al., 1999; Jones, 1993; Mamum & Lim, 2005; Marik & Kaplan, 2003; Terpenning et al., 2001; Ramsey, Smithard, & Kalra, 2003; Rhinehart & Friedman, 2005; Sarin, Balasubramaniam, Corcoran, Laudenbach, & Stoopler, 2008).

### **Verbal Deficits**

#### **Aphasia.**

The term aphasia literally means “complete loss of language” however in most cases of neuropsychological impairment some linguistic abilities are retained and therefore “dysphasia” meaning a “partial lack of language”, is a more accurate term for use (Sims, 2003). Disturbance in comprehension or

production of spoken written or signed language are the hallmarks of this set of disorders. Locating specific areas of the brain in relation to aphasic symptoms has been problematic as the production and comprehension of language depends on a complex and dynamic interaction between vast regions of the brain. Nevertheless, damage to particular cortical regions, has been found to produce a cluster of aphasic symptoms. Broca's Area, sited in the left frontal lobe and Wernicke's Area sited in the left temporal lobe have long been known as the major areas of the brain responsible for language. The group of fibres running deeply into the white matter of the temporal, parietal, and frontal regions that connect these two areas is known as the arcuate fasciculus (Bruni & Montemurro, 2009; Eysenck & Keane, 2000).

More recently, a third area named Geschwind's territory, which connects Broca's and Wernicke's areas via a region of the parietal cortex has been identified as playing an important role in language acquisition in children. These areas are found in the left hemisphere of 99% of right-handed people and 60-70% of left-handed people, which is dominant for language (Ganong, 2005; Roth & Heilman, 2000).

The four general types of aphasia are; Broca's Aphasia, Wernicke's Aphasia, Anomic Aphasia, and Global Aphasia. Stroke is the most common cause of aphasia (Kirshner, 2004; Ruiz, 2000; Wade, Hewer, David, & Enderby, 1986), although some other events that can cause lesions leading to aphasia are traumatic brain injury, degenerative neurological disease, and certain chronic neurological disorders such as migraine, epilepsy, or brain tumour.

Although the prevalence and course of post-stroke aphasia is dependent on the diagnostic criteria and the time of first evaluation, research estimates the incidence of aphasia post-stroke as varying from 17% to 40% (Hier, Yoon, Mohr, Price, & Wolf, 1994; Nicholas, 2005; Pedersen, Jorgensen, Nakayama, Raaschou, & Olsen, 1996). Typically, patients with Broca's aphasia fully understand spoken and written language however, are unable to speak fluently themselves even though they know what they want to say. There is a delay in the formation of sentences which are often delivered as broken phrases or with words often been communicated one at a time. There is diminished vocabulary, and speech lacks correct grammatical conventions and there is particular difficulty using adverbs and prepositions. Stress patterns and intonation are absent so the individual speaks in a flat and mono-pitched tone (Dronkers & Larsen, 2001).

Primarily, people who suffer from Wernicke's aphasia have difficulty understanding the speech of others including simple and complex statements. They cannot follow directions or answer questions. For example, if you are having coffee with a patient and you ask for the sugar, the patient may not respond or may pass you the cream. However, patients with Wernicke's aphasia can also have problems expressing themselves and their speech may be characterised by the use of jargon, that is, the words they use have the wrong or no meaning at all (Tanner, 2007). These Wernicke's patients suffer greater disability than sufferers of Broca's aphasia only.

Global aphasia, caused by lesions to both Broca's and Wernicke's areas results in the patients experiencing significant difficulty in all

modalities of language both spoken and written. This condition is most prevalent after strokes that have involved the internal carotid artery or the middle cerebral arteries (Ozeren, Koc, Demirkiran, Sonmezler, & Kibar, 2006).

Word finding difficulty is a problem present in a number of aphasias however when this deficit occurs in isolation, the term “Anomic aphasia” is applied (Schwartzman, 2006). Preservation of speech characteristics and auditory comprehension remain intact. Many patients present with specific-category naming deficits, for example difficulty with verbs and not nouns or the reverse. Other patients may have difficulty naming animate objects but not inanimate objects (Benson & Ardilla 1996; Nicholas, 2005).

The type of aphasia presenting in the acute stage of stroke was investigated by Croquelois Godefroy, and Bogousslavsky (2007). They concluded that Global aphasia and unclassified aphasias (mainly anomic plus aphasia of mild severity) accounted for half of the aphasic syndromes while Wernicke’s and Broca’s aphasia accounted for 40%.

In two recent studies, gender was not found to be an independent determinant in the presentation of aphasia post-stroke (Engelter et al., 2006; Law et al., 2009; Pedersen, Jorgensen, Nakayama, Raaschou, & Olsen, 1995), however Kyrozis et al. (2009) found female gender to be an independent prospective predictor of post-stroke aphasia. In two other studies evidence for an anterior-posterior difference of extension of lesion was found between women and men (Hier et al., 1994; Lang & Moser, 2003). The role of gender as an independent factor of aphasia post-stroke remains unclear and debatable.

## **Perceptual Deficits**

### **Apraxia.**

Apraxia is a neurological disorder usually associated with lesions to the left cerebral hemisphere, affecting the ability to plan the steps involved in a complex task and carry out those steps in the correct sequence despite having the desire and physical ability to do so (Roth & Heilman, 1997). For example, when a person is asked to pretend to blow out a match”, they fail to do so, but when a lit match is placed before them they blow it out successfully because the presence of the lit match “made it possible to retrieve the movement memories” (Roth & Heilman, 1997, p. 1). Apraxia presents on a continuum of severity and the milder forms of this disorder are referred to as dyspraxia.

Neuropathology of apraxia as a consequence of stroke suggests that lesions that occur in the left hemisphere are the most common cause of this disorder (Duffy, 2005). It has been found to be a persistent disorder with adverse effects on recovery of activities of daily living (Donkervoort, Dekker, & Deelman 2006). Frequency of apraxia in post-stroke patients differs considerably between studies. One reason for this disparity is because subtle apraxic impairments have not been detected resulting in an underestimation of the presence of this condition. For example, in one study (Haaland & Flaherty, 1984), only 13% of the sample group were found to have apraxia however, in another study (Schnider, Hanlon, Alexander, & Benson, 1997) that used more sensitive measures, 45% of the sample group fell into the apraxia range.

## **Behavioural Outcomes**

### **Fatigue.**

After a stroke, many people feel unusually tired because of the extra energy they use to cope with physical and emotional changes. However post-stroke fatigue is more than just common tiredness, as expressed by Barker-Collo, Feigin, & Dudley (2007, p.1), who consider post-stroke fatigue (PSF), to be “a complex interaction of biological, psychosocial, and behavioural phenomena”. Staub and Bogousslavsky (2001), propose a concept that links PSF to attentional deficits and involves damage to structures involved in the subcortical attentional network. Other common causes of fatigue include; pain, disease, anaemia, inactivity or other health problems (Glaus, Crow, & Hammond, 1996). Whatever the aetiology, fatigue is reported as persisting for months and even years after the stroke.

Prevalence of fatigue in stroke patients is known to be high although the lack of a common definition, different sample populations and the use of a wide variety of assessment tools, has resulted in considerable variance across studies. However, increased fatigue has been associated with insititutionalisation and case-fatality and decreased functional independence (Glader, Stegmayr, & Asplund, 2002). Furthermore, PSF has been shown to have a detrimental effect on rehabilitation, quality of life and returning to work and correlates significantly with functional disability and neuropsychological deficits (Glader et al., 2002; Ingles, Eskes & Phillips, 1999; Michael, 2002). Given that the causes of PSF are multi-factorial it has been suggested that strategies to alleviate this problem need to be individualised (Choi-Kwon, Han, Kwon, & Kim, 2005; Staub & Bogousslavsky, 2001).

**Incontinence.**

The incidence of urinary incontinence (UI), among survivors of stroke is high and has found to be associated with the size of the infarct or cerebral haemorrhage and severity of the stroke (Brittain et al., 2006; Brittain, Peet, & Castleden, 1998; Brocklehurst, Andrews, Richards, & Laycock, 1985; Gelber, Good, Laven, & Verhulst, 1993). Prevalence studies of UI in the acute stages post-stroke have produced rates ranging from 32% (Kamouchi et al., 1995; Patel, Coshall, Rudd, & Wolfe, 2001), to as much as 83% (Luft & Vriheas-Nichols, 1998). However, the transient nature of incontinence was demonstrated in Nakayama, Jorgensen, Pedersen, Raaschou, and Olsen (1997) who found that the majority of patients gained bladder control within 6 months post-stroke. This transiency was again demonstrated in a 2-year study where Patel et al. (2001), found the incidence of UI in their population at the acute stage, 3 months, 1 year and 2 year post-stroke to be, 40%, 19%, 15% and 10%, respectively. In a review of the prevalence of incontinence studies ranging from 1985 to 2002, Barrett (2002), the improvement of incontinence over time is said to have been clearly demonstrated.

Stroke patients who suffer from UI have a higher risk of mortality, morbidity and a high rate of discharge to nursing homes (Brittain et al., 1998; Kolominsky-Rabas, Hilz, Neundoerfer, & Heuschmann, 2003; Patel et al. 2001; Thom & Van Den Eeden, 1997). Its presence is used as a prognostic factor for stroke patients (Hamann, Rogers, & Addington-Hall, 2004; Kobayashi, Hara, & Morita, 2005; Mant, Wade & Winner, 2004; Wade & Hewer, 1985) although as Shuji, Mizuho, and Akiko (2005) point out the changing condition of the incontinence needs to be considered when predicting outcomes. Effective

treatment of UI has the potential to improve participation in rehabilitation and ultimately the outcomes of rehabilitation. Early assumption of the standing position and early ambulation promotes continence (Taub, Wolfe, Richardson, & Burney, 1994). Other treatments commonly utilised for this condition include pelvic floor muscle training, timed voiding, pharmacotherapy and hormonal interventions and specialised professional input (Thomas et al., 2007).

Studies published in 1987 and 1997 found that between 31% and 40% of stroke patients experienced fecal incontinence on admission to hospital (Brittain et al., 1998; Nakayama et al., 1997; Wade & Hewer, 1987). Functional limitations and age have been found to influence the presence of fecal incontinence (Brittain et al., 2006; Kovindha, Wattanapan, Dejpratham, Permsirivanich, & Kuptniratsaikul, 2009) although in one study age was not found to be a significant factor (Edwards & Jones, 2001). Fecal incontinence is also negatively associated with mortality and institutionalisation as well as having a detrimental influence on functional outcomes (Baztan, Domenech, & Gonzalez, 2003).

### **Double Incontinence.**

Double Incontinence is also commonly present after stroke. In one study double incontinence was more prevalent than either urinary or fecal incontinence alone (Kovindha et al., 2009), although in a postal survey of stroke survivors, 4.3% reported double incontinence as opposed to 5% with major fecal incontinence alone (Brittain et al., 2006). However, the same study found double incontinence to be four times greater in stroke survivors than in the general population.



**Pain.**

Pain is a frequent and often long-term consequence of stroke occurring in 19 to 74% of stroke patients (Henon, 2006; Kim, 2009; Milani, 2009), often having a profound effect on patient's well-being. A recent study reported one third of their patients suffered from moderate to severe pain 4 months after onset and that the intensity of the pain had increased over time (Henon, 2006). The persistency of this condition has been shown in other studies (Widar, Samuelsson, Karlsson-Tivenius, and Ahlstrom (2002) where intramuscular electrical stimulation treatment was shown to be less effective when administered later rather than earlier post-stroke (Chae et al., 2007). In another recent study, although the number of patients experiencing pain at 4 and 16 month intervals dropped from 32% to 21%, the pain intensity was reported as being more severe at the later interval (Jonsson, Lindgren, Hallstrom, Norrving, & Lindgren, 2005). Other types of pain can be traced to nerve damage, bed sores, or a joint that doesn't move.

Central post-stroke pain (CPSP) also referred to as thalamic pain or neurogenic pain, is a syndrome characterised by pain in the part of the body corresponding to the brain territory where the lesion has occurred. CPSP occurs most frequently following strokes on the right side of the brain, affecting the left side of the body. Approximating the prevalence of CPSP is difficult partly due to the difficulty in distinguishing it from other pain types that occur post-stroke (Klit, Finnerup & Jensen, 2009) although between 8% and 14% prevalence rates have been quoted by some authors (Bowsher, 2001; Kumar, Kalita, Kumar, & Misra, 2009).

Shoulder pain is also a common consequence of stroke especially in patients with severe sensorimotor deficit (Ratnasabapathy et al., 2003). Two prospective

studies demonstrated an incremental increase of incidence of shoulder pain in their study sample over the first 6 months post-stroke (Langhorne et al., 2000; Ratnasabapathy et al., 2003). A more recent study revealed 52% of the sample of 122 stroke survivors suffered shoulder pain over 12 months. The authors suggest that such a high percentage of individuals were identified because, unlike previous studies, their study did not exclude patients with language or cognitive impairment (Sackley et al., 2008).

### **Emotional Lability.**

Emotional lability is the term used to describe the very common effect where stroke survivors lose control over their emotions (Horrocks, Hackett, Anderson, & House, 2004). About 20% of stroke patients are believed to suffer from this syndrome, which has a number of characteristic features including feeling angry or irritable with little provocation or sudden and unexpected episodes of laughing or crying that do not correspond to the underlying emotional feeling (House, 1987; Robinson, 1997). Emotional lability may co-exist with depression and according to Starkstein and Robinson (2000), patients respond well to tricyclic antidepressants. In their systematic review of the literature, Horrocks et al. (2004), found evidence to suggest pharmacological intervention reduces crying emotionalism post-stroke. The authors however are guarded of the findings citing vast methodological differences as having a negative effect on the validity of their findings.

### **Anxiety.**

Anxiety post-stroke is common and has a negative impact on rehabilitation, daily functioning and quality of life in general (Fruhwald, Loffler, Eher, Saletu, & Baumhackl, 2001; Kumar, Lavretsky, & Haroon, 2005; Sagen et al., 2009; Tanner,

2007). In 2007, Barker-Collo, found 21.1% of her sample of stroke patients from an inpatient rehabilitation unit suffered from anxiety, a result which is consistent with other studies examining the frequency of post-stroke anxiety PSA (Castillo, Starkstein, Fedoroff, Price, & Robinson, 1993; De Wit et al., 2008; Leppavuori, Pohjasvaara, Vataja, Kasate, & Erkinjuntti, 2003; Robinson, 1998).

In their longitudinal study, Morrison, Pollard, Johnston, and MacWalter (2005), found PSA to be present at 10-20 days, 1 month, 6 months and 3 years after the stroke to be more common amongst females. This relationship is not uncommon as suggested by a number of other studies that have also found a higher correlation between PSA and being female than between PSA and being male (Kadojic et al., 2005; Kuroda, Kanda, & Sakai, 2006; Wilz & Kalytta, 2008). Current data about the relationship of anxiety with location site are not conclusive although some studies have implicated the right hemisphere (Astrom, 1996; Astrom, Adolfsson, & Asplund, 1993; Barker-Collo, 2007) while other studies indicate left hemisphere involvement when the anxiety is comorbid with depression (Castillo et al., 1993; Robinson 2006).

### **Depression.**

A significant portion of stroke survivors experience psychological sequelae which can include anxiety, depression, grief reaction, irritability, sadness or unhappiness (Barker-Collo, 2007; Burvill et al., 1995; Ebrahim, Barer, & Nouri, 1987; Newberg, Davydow, & Lee, 2006; Saxena, 2006). Studies investigating post-stroke depression (PSD) have produced a wide range of prevalence rates. For example, Robinson (2003) in a summary of studies examining the prevalence of stroke, found between 14% and 19% of individuals experienced PSD as opposed to 10% in the general population. An earlier review of studies identified

as many as 65% of stroke survivors suffer from PSD (Primeau, 1988). However, prevalence rates between 20% and 50% have been found in most studies on this issue (Barker-Collo, 2007).

Some authors report a correlation between people suffering PSD and increased fatality (Bogousslavsky, 2003; Burvill et al., 1995; Feigin, 2004; Morris, Robinson, Andrezejewski, Samuels, & Price, 1993; Morris, Robinson & Samuels, 1993; Paul, Srikanth & Thrift, 2007; Williams, Ghose, & Swindle, 2004). The relationship between site of lesion and the incidence of PSD gravitates towards a link with left hemisphere lesion (Barker-Collo, 2007; Morris, Robinson, & Raphael, 1992; Starkstein, Robinson, & Price, 1987; Sulaiman, Zainal, Tan, & Tan, 2002). A meta-analysis of 52 studies found a weak relationship between right hemisphere lesion and PSD (Yu, Liu, Chen, Wang, & Yu, 2004).

Conversely, Vataja et al. (2005), found that a brain infarct affecting structures of the frontal-subcortical circuits, especially on the left side, predispose stroke patients to depression. Indeed, there is compelling evidence associating PSD with left-hemispheric damage (Astrom et al., 1993; Barker-Collo, 2007; Herrmann, Bartels, Schumacher, & Wallesch, 1995; Hosking, Marsh, & Friedman, 2000; Jorge, Robinson, Starkstein, & Arndt, 1993).

A temporal association between lesion location and PSD has also been found by Shimoda and Robinson (1999) and Bhogal, Teasell, Foley, and Speechley (2004). These findings suggest that individuals may experience PSD symptoms immediately after the stroke as a sequelae of neurological alteration or at a later stage, as a psychological development as their awareness of the impact of the stroke increases. The substantial discrepancy in the findings regarding the association between lesion location and PSD can be largely attributable to

methodological procedures. Patient selection (in-patient or out-patient), time since onset and the assessment of depression tools to measure post-stroke depression have all contributed to the heterogeneity of the findings (Bhogal et al., 2004; Toso et al. 2004).

The effects of PSD are widespread and are considered by some to be the strongest predictor of quality of life in stroke survivors (Kim, Warren, Madill, & Hadley, 1999; King, 1996). Moreover, PSD has been associated with increased disability (Kotila, Numminen, Waltimo, & Kaste, 1999; Pohjasvaara, Vataja, Leppavuori, Kaste, & Erkinjuntti, 2001), increased risk of falls (Jorgensen, Engstad, & Jacobsen, 2002) and with worse rehabilitation outcomes (Gillen, Tennen, McKee, Gernert-Dott, & Affleck, 2001; Paolucci et al., 1999; Sinyor et al., 1986).

There is a large body of compelling evidence that PSD is associated with cognitive impairment (Kauhanen et al., 1999; Murata, Kimura, & Robinson, 2000; Narushima, Chan, Kosier, & Robinson, 2003; Robinson, Bolla-Wilson, Kaplan, & Lipsey, 1986; Saxena, 2006; Talelli et al., 2004; Yoo, Jeong, & Kim, 2009) and linked to left hemispheric lesion (Barker-Collo, 2007; Robinson et al., 1986; Robinson, Kubos, Starr, Rao, & Price, 1984). Studies investigating the relationship between PSD and specific cognitive functions have implicated memory, non-verbal problem solving, attention and psychomotor speed (Barker-Collo, 2007; Hoskings et al., 2000; Kauhanen et al., 1999), visual perception and construction, and language (Nys, 2005).

## Appendix C Participant Information Sheet



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**Project Title:** Does Attention Process Training improve functional outcomes of stroke?

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### **An invitation...**

You are invited to take part in a new research study called:

#### **“Stroke Attention Rehabilitation Trial (START)”**

We would welcome your help in this important project, however, your participation is entirely voluntary (your choice).

What is the purpose of the study?

The purpose of this study is to evaluate a new therapy called ‘Attention Process Training’ or APT as a form of rehabilitation for stroke survivors. If you have experienced a stroke within the past 4 weeks, you may be able to join this study.

### **Why do we need this study?**

The most common cognitive problem after stroke is reduced attention. A reduction in the ability to pay attention may have a negative impact on a person’s ability to function and on their quality of life. A new form of rehabilitation to address attention difficulties after stroke has been developed. It is called ‘Attention Process training’ or APT. We do not know if APT does improve attention and function of people who have experienced a stroke.. and so we will be testing APT in this study. This study will examine the effectiveness of Attention Process training (APT) by comparing persons who have recently suffered a stroke who receive APT with persons who have recently suffered a stroke and receive usual care.

**What is Attention Process Training?**

APT is a comprehensive programme specifically designed to treat impairments in paying attention. It is a series of tasks delivered by a trained neuropsychologist using a paper and pencil or an audio tape. Each paper/tape contains exercises and activities to be completed by the stroke survivor, to retrain the stroke survivor on how to pay attention. The exercises are tailored to meet the ability of the stroke survivor. If you are randomised to the group that receives the Attention Process Training, the session will take one and a half hours, Monday to Friday for 4 weeks. This is flexible based on your health, and you will be allowed the opportunity to rest if you wish too. The training will begin in hospital, and if you are discharged prior to the training being completed, it will continue at your place of residence.

**Who can participate in this study?**

People who have experienced a stroke within the past 4 weeks and have been assessed as having a decrease in their ability to pay attention. We will be seeking 169 stroke survivors for this study.

**What is involved?**

If you decide that you would like to take part, you will undergo an initial interview at hospital with a researcher who is a neuropsychological trainee or neuropsychologist, to assess if you have a decrease in your ability to pay attention and if you are well enough to complete the APT programme. If they are willing, we will also be asking questions from the caregiver (family member or friend) who provides you with the most support.

You will be required to complete a questionnaire and a series of tests with a researcher to assess your cognitive functioning (ability to pay attention), current state of health and your medical history. The initial interview will take about 30 minutes. We will also seek permission to access your medical and hospital records to collect information on your medical history, the medications you are taking, and the type of therapies you have received. If you are eligible for the study, you will have three assessments: on the day of enrolment into the study, and then after 5 weeks, and at 6 months. The assessment at 5 weeks is similar to the initial interview, and will take about 30 minutes.

For the assessment on the day of enrolment and at 6 months, you will be required to complete a more extensive set of tests with a researcher to assess your cognitive functioning (ability to pay attention, solve problems and remember things), and questionnaires about your current state of health, your medical history, your ability to do everyday things and your quality of life. The assessment will take about 150 minutes, and you will have an opportunity to rest if you require it.

**What is meant by the term ‘randomisation’**

If you agree to be part of this study you will be randomised either to receive the Attention Process Training, or usual care rehabilitation. Randomisation is like a flip of a coin, you have an equal chance that you will be in either the Attention Process Training Group or receiving standard care. Currently, we do not know which health service is the most effective for stroke rehabilitation; which is why

we are conducting the study. Randomisation allows us to compare the two services with other.

**What is APT?**

Attention Process Training is a rehabilitation package used to improve difficulties with attention. It includes a number of activities which get more difficult over time. The program activities are not functional and resemble laboratory tasks, for example listening to a list of words and pressing a button every time you hear the word “and”. Participants in the APT group will receive daily individual APT treatment on weekdays for a period of 4 weeks.

**What are the expected benefits?**

You may not directly benefit from the study, as we do not know if APT works. However, you will help the people who fund, provide and deliver health services for stroke rehabilitation. This study will be of benefit to the wider population. There is no guarantee that you will benefit directly from being involved in this study.

**What are the potential risks and discomforts?**

Taking part in this study will take some of your time and require you to answer a series of questions. There are no known risks caused by this study. As we are only asking questions and getting you to perform mental tasks, there are no risks with this study. You may find some of the tasks tiring, but can take a break at any time. You will not be asked to perform any tasks that make you feel uncomfortable or that you do not feel that you can complete. You will continue to receive care from your doctor and other health services. Your usual medical care will not be affected in any way by participating in this study, or by declining to participate or withdrawing from the study at any stage. Your participation in this study will be stopped should any harmful effects appear or if the doctor feels it is not in your best interest to continue. Similarly your doctor may at any time provide you with any other treatment he/she considers necessary.

**Confidentiality**

All data generated from this study will be treated with utmost confidentiality without reference to your name. It is very important that the data collected are accurate and therefore, it will need to be checked against your medical records. You are therefore asked to give permission for the researchers to look at your medical records to help them carry out these checks and to access information on what health services you receive during the trial from the District health Board and their agencies. Naturally, the information will be kept strictly confidential and will be used only for statistical purposes of this study. Your identity will be kept confidential. In the study documents you will only be identified by your initials, date of birth, and a study number. The data will be kept for the duration of the study at the Clinical trials Research Unit, The University of Auckland and destroyed after 16 years according to national research guidelines. Any information provided to interviewers will not be acted upon unless there are concerns about the participant’s safety or the safety of others.



**Withdrawal from the study**

Your participation in the study is entirely voluntary. You may withdraw at any time, and you do not have to give a reason for doing so, although it would be helpful if you did and to participate in the final assessment visit if at all possible. Your doctor may also suggest that you withdraw if he/she has any concerns about your participation. You may also be withdrawn if you are not able to comply with the study procedures or for other administrative reasons. If you do withdraw, this will in no way affect any future treatment you may require.

**Costs**

There will be no charge made to you for any attendance or tests conducted during this study. Your doctor will not be paid or incur and study related costs for your participation.

**Compensation for Physical injury**

In the unlikely event of a physical injury as a result of your participation in this study, you may be covered by ACC under the Injury Prevention, Rehabilitation and Compensation Act. ACC cover is not automatic and your case will need to be assessed by ACC according to the provisions of the 2002 Injury Prevention Rehabilitation and Compensation Act. If your claim is accepted by ACC, you still might not get any compensation. This depends on a number of factors such as whether you are an earner or non-earner. ACC usually provides only partial reimbursement of costs and expenses and there may be no lump sum compensation payable. There is no cover for mental injury unless it is a result of physical injury. If you have ACC cover, generally this will affect your right to sue the investigators.

If you have any questions about ACC, contact your nearest ACC office or the investigator.

**Further Information**

You are encouraged to ask questions at any time during the study. If you have any questions at any time during the study please do not hesitate to ask your general practitioner or the research staff associated with the study.

If you have any queries or concerns regarding your rights as a participant in this study you may wish to contact a Health and Disability Advocate on 0800 423 638 Northland to Franklin – free fax 0800 2787 7678.

**You will receive a copy of this information sheet. If you want more information, please contact either the Principal Investigator Dr Suzanne Barker-Collo at the Department of Psychology, University of Auckland on 09 373-3=7599 ext 88517 or Ms Margaret Dudley on 021 164 9453.**

This study has received ethical approval from the Northern X Regional Ethics Committee.

## Appendix D

### Consent Form



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Project Title: Does Attention process Training improve functional outcomes of stroke?

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### CONSENT FORM

Registration Number

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English	I wish to have an interpreter	Yes	No
Maori	E hiahia ana ahau ki tetahi tangata hei korero Maori ki ahau	Ae	Kao
Samoan	Oute mana'o e iai se fa'amatala upu	Loe	Leai
Tongan	'Oku fiema'u ha fakatonulea	Lo	Ikai
Cook Island	Ka inangaro au i tetai tangat uri reo	Ae	Kare
Niuean	Fia manako au ke fakaaoga e tagata fakahokohoko vagahau	E	Nakai



I, \_\_\_\_\_, acknowledge that I have had explained to me by the Neuropsychologist Trainee, the nature and procedures involved with this research study.

I have read and I understand the information sheet dated 24/08/2006 for volunteers taking part in the study designed to evaluate the effects of Attention process Training on stroke survivors.

I have had the opportunity to discuss this study. I am satisfied with the answers that I have been given.

I've had the opportunity to us whanau support or a friend to help ask questions and understand the study.

I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time and this will in no way affect my future and continuing health care.

I understand that my participation in this study is confidential and that no material which could identify me will be used in any report on this study.

I am aware that the exception to confidentiality will be applied in situations where the interviewer has significant concerns about the safety of myself or others.

I understand the compensation provisions for this study.

I have had time to consider whether to take part.

I know who to contact if I have any problems from taking part in this study.

I know who to contact if I have any questions about the study.

I give consent for the researchers to access my medical records and information on the health services I receive during the study from the health service funder or provider YES/NO

I would like to receive a copy of the results of the study YES/NO

I consent to take part as a subject in this research YES/NO

Signature\_\_\_\_\_

Date\_\_\_\_\_/\_\_\_\_\_/\_\_\_\_\_

Project explained by\_\_\_\_\_.

**Appendix E**  
**Tables showing correlations between baseline and post-intervention measures with the highest tasks reached on APT.**

Table E-1

*Correlations between baseline demographics and highest auditory and visual task reached*

Baseline Demographic	Highest Auditory Task	Highest Visual Task
Age	0.26	-0.04
Gender	-0.25	0.01
Education	-0.09	0.07
Time since stroke	-0.12	-0.12
MMSE	-0.20	-0.22
Barthel Index	-0.18	-0.27

Table E-2

*Correlations between baseline attention measures and highest auditory and visual task reached*

Baseline Attention Measure	Highest Auditory Task	Highest Visual Task
FSAQ	-0.06	-0.05
AAQ	-0.12	-0.08
VAQ	-0.10	-0.13
FSRQ	0.20	-0.02
VPQ	0.28	-0.04
APQ	0.27	0.09
TMT		
A	0.20	-0.15
B	0.26	-0.18
PASAT		
2.4	<b>0.48*</b>	0.29
2.0	<b>0.57*</b>	0.32
Bells		
Left	0.04	-0.13
Centre	0.01	0.10
Right	0.04	<b>-0.43*</b>

**\*Correlation is significant at the 0.05 level**

Table E-3  
*Correlations between baseline neuropsychological measures and highest  
 and visual task reached*

Baseline Measure	Highest Auditory Task	Highest Visual Task
Stroop		
Dot	0.27	-0.01
Word	0.17	-0.06
Colour	0.36	0.16
ROCF		
Copy	0.14	-0.12
SD	0.35	0.09
LD	<b>0.43*</b>	0.10
Recognition	0.14	0.09
VPA		
Learning	0.14	0.09
Delayed	0.11	0.11
BNT	0.34	0.25
LM1	-0.03	0.10
LM11	0.06	0.06
COWA	0.14	0.17
CVLT		
SD Free	0.03	-0.10
LD Free	0.05	-0.09
Recognition	-0.09	0.07

**\*Correlation is significant at the 0.05 level**

Table E-4  
*Correlations between post-intervention attention measures and highest  
 auditory and visual task reached.*

Post-intervention Attention Measure	Highest Auditory Task	Highest Visual Task
FSAQ	0.07	0.14
AAQ	-0.10	-0.01
VAQ	0.13	0.11
FSRQ	0.11	0.13
VPQ	0.05	-0.00
APQ	0.00	0.09
TMT		
A	0.04	-0.05
B	0.13	-0.06
PASAT		
2.4	0.21	0.17
2.0	0.10	0.14
Bells		
Left	0.28	0.15
Centre	-0.02	-0.19
Right	-0.01	-0.26

**Appendix F**  
**Tables showing correlations between baseline and post-intervention  
measures with total hours of APT completed**

Table F-1

*Correlations between demographic measures and total  
hours of APT completed*

Baseline Demographic	Total Hours
Age	0.27
Gender	-0.26
Education	-0.07
Time since stroke	0.02
MMSE	<b>-0.40*</b>
Barthel Index	-0.29

**\*Correlation is significant at the 0.05 level**

Table F-2

*Correlations between baseline attention measures and  
total hours of APT completed*

Baseline Attention Measure	Total Hours
IVA-CPT	
FSAQ	-0.11
AAQ	0.01
VAQ	-0.22
FSRQ	0.19
VPQ	0.23
APQ	<b>0.41*</b>
TMT	
A	-0.16
B	0.13
PASAT	
2.4	0.05
2.0	0.07
Bells	
Left	-0.05
Centre	0.07
Right	-0.05

**\*Correlation is significant at the 0.05 level**

Table F-3  
*Correlations between baseline neuropsychological measures and total hours of APT completed*

Baseline Measure	Total Hours
Stroop	
Dot	0.00
Word	-0.03
Colour	-0.06
ROCF	
Copy	-0.03
SD	0.05
LD	0.06
Recognition	-0.12
VPA	
Learning	-0.09
Delayed	-0.16
BNT	0.30
LM1	-0.08
LM11	0.02
COWA	-0.05
CVLT	
SD Free	-0.08
LD Free	0.01
Recognition	-0.28

Table F-4  
*Correlations between post-intervention attention measures and total hours of APT completed*

Post-intervention Attention Measure	Total Hours
IVA-CPT	
FSAQ	-0.07
AAQ	-0.21
VAQ	-0.01
FSRQ	-0.08
VPQ	0.12
APQ	0.20
TMT	
A	-0.10
B	-0.26
PASAT	
2.4	-0.38
2.0	-0.40
Bells	
Left	0.14
Centre	-0.07
Right	-0.07
SF-36	
MCS	-0.03