Long-term functional and neuropsychological outcomes of subarachnoid haemorrhage (SAH) survivors

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A thesis submitted in partial fulfilment of the requirements for the degree of Doctor of Clinical Psychology, Department of Psychology, The University of Auckland, 2010.

Abstract

Stroke is the second commonest cause of death worldwide and the most frequent cause of disability in adults. Although Subarachnoid Haemorrhage (SAH) accounts for a small percentage of stroke, it impacts people at a younger age and with debilitating consequences, in the areas of mood, functioning and cognitive deficits which may persist for years after the SAH. However, very little research has been done to examine the long-term (beyond 1-3 years) outcomes of SAH. Furthermore, previous studies have not been population based, nor have they examined the impact of long-term cognitive outcomes using a battery of neuropsychological tests.

The current population based study examined long-term neuropsychological and functional outcomes of SAH survivors (n=27). A control group (n=26) matched on age, gender and ethnicity was used to compare the mood, functional (i.e., disability, handicap, quality of life) and neuropsychological outcomes (i.e., verbal memory, visual memory, executive functioning, language, processing speed and visuoperceptual abilities) of SAH survivors.

As compared to the controls, the SAH group was more depressed and was significantly impaired in the areas of disability, handicap, quality of life; particularly in the areas of mental health with females reporting poor mental health than males within the SAH group. Furthermore, the SAH group had significant deficits across cognitive domains (i.e., language, memory, visuoperceptual abilities, executive functioning/attention and information processing) when compared to controls. Although impairments were noted at 5-years post-SAH, over time (i.e., from acute stage) significant improvement had occurred in the areas of functioning (i.e., disability, health related quality of life and well-being) of SAH survivors. Depressed mood and baseline functioning were related to worse functional outcomes at 5-years post-SAH. Whilst poor

cognitive functioning impacted the long-term functional outcomes of SAH survivors only, visual memory and language were found to independently relate to worse functional outcomes of SAH-survivors.

The finding that long-term neuropsychological impairment in SAH-survivors is independently associated with the functional outcomes at 5-years post-SAH provides a new direction for the rehabilitation efforts which have traditionally focussed on physical functioning and activities of daily living. Thus, these findings are of relevance to clinicians to help them understand the expected cognitive deficits and their potential to impact on wider functional outcomes of SAH survivors, thereby allowing the clinicians to plan appropriate interventions for rehabilitations.

-Dedicated to my family-

Acknowledgements

The following people provided valuable input and contributed in the completion of this thesis. I gratefully acknowledge their help:

I am deeply indebted to all the participants and their families who devoted their time and energy to participate in the research.

Firstly I would like to thank my primary supervisor Dr Suzanne Barker-Collo for her invaluable guidance, support and motivation. Suzanne-I really appreciate that you were always available with your expert advice and efficient feedback. I wish to thank you for the energy and commitment you gave through the course of this thesis. I would also like to thank Dr Valery Feigin, my secondary supervisor for his valuable input in this research.

I would like to show my gratitude to the staff at Clinical Trials Research Unit and particularly, Elizabeth Glenn for her help in recruiting participants for this study.

I am grateful to the Health Research Council of New Zealand and the University of Auckland Doctoral Scholarship for providing me with funding towards tuition, research and conference travel costs.

I can not thank enough my friends in the doctoral course who have been a huge support for me during this thesis and have shared this journey with me. In particular, Madhu, Helen and Tina-thank you for all the emotional support you provided and for helping me get through the difficult times.

I would like to acknowledge my family and friends in New Zealand and overseas who reminded me that life exists beyond the thesis. Lastly, and most importantly, I wish to express my sincerest appreciation to my, husband Bobby for his encouragement, and for providing his invaluable support in numerous ways.

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List of Abbreviations

Abbreviation	Full Expression
ACROSS	Australasian Cooperative Research on Subarachnoid Haemorrhage Study
ADLs	Activities of Daily Living
ANOVA	Analysis of Variance
ARCOS	Auckland Regional Community Stroke Study 2002-2003
AVM	Arteriovenous Malformation
BD	Block Design
BDI	Beck Depression Inventory
BI	Barthel Index
BMI	Body Mass Index
BNT	Boston Naming Test
BP	Body Pain (Outcome domain of the Short Form-36)
CAMCOG	Cambridge Examination for Mental Disorders of the Elderly
CF	Consent Form
CI	Confidence Interval
COWA	Controlled Oral Word Association
CT	Computed Tomography
CVLT-II	California Verbal Leaning Test-Second Edition
FAI	Frenchay Activity Index
GCS	Glasgow Coma Score
GDS-SF	Geriatric Depression Scale-Short Form
GH	General Health Perceptions (Outcome domain of the Short Form-36)
GHQ-28	General Health Questionnaire-28
GOS	Glasgow Outcome Scale
HADS	Hospital Anxiety and Depression scale
HAMT	Hodkinson Abbreviated Mental Test
HRQoL	Health Related Quality of Life
ICD	International Classification of Diseases
ICF	International Classification of Functioning, Disability and Health
ICH	Intracerebral Haemorrhage
ICIDH	International Classification of Impairments, Disabilities and Handicaps
IS	Ischemic stroke
IVA-CPT	Integrated Visual and Auditory Continuous Performance Test

LHS	London Handicap Scale
LM	Logical Memory
MCS	Mental Component Summary score (Outcome domain of the Short Form-36)
MH	Mental Health (Outcome domain of the Short Form-36)
MMSE	Mini-Mental State Examination
MR	Matrix Reasoning
MRS	Modified Rankin Scale
NIHSS	National Institute of Health Stroke Scale
PCS	Physical Component Summary score (Outcome domain of the Short Form-36)
PF	Physical Functioning (Outcome domain of the Short Form-36)
PIS	Participant Information Sheet
PSA	Post Stroke Anxiety
PSAHD	Post Subarachnoid Haemorrhage Depression
PSD	Post Stroke Depression
RE	Role Emotional (Outcome domain of the Short Form-36 [limitations due to emotional health])
RP	Role Physical (Outcome domain of the Short Form-36 [limitations due to physical health])
ROCF	Rey-Osterrieth Complex Figure
RR	Relative Risk
SAH	Subarachnoid haemorrhage
SF	Social Functioning (Outcome domain of the Short Form-36)
SF-36	Short Form-36
TMT	Trail Making Test
VPA	Verbal Paired Associates
VST	Victoria Stroop Test
VT	Vitality (Outcome domain of the Short Form-36)
WAIS	Wechsler Adult Intelligence Scale
WHO	World Health Organisation
WMS	Wechsler Memory Scale

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CHAPTER-I

INTRODUCTION

SYNOPSIS

This study examines long-term (5-year) functional and neuropsychological outcomes of subarachnoid haemorrhage (SAH) survivors in Auckland, New Zealand. As such the following literature review provides an overview of the definitions, prevalence and risk factors for stroke, with a focus on SAH. This is followed by a section on stroke outcomes again focussing on outcomes of SAH. This includes examination of the WHO models of outcomes and is organised to cover various aspects of WHO (2001) model which was used to guide the study design. As this is a New Zealand based study, data from New Zealand are presented where available.

INTRODUCTION TO STROKE AND ITS EPIDEMIOLOGY

Stroke: Definitions and subtypes

Stroke is an acute vascular injury to the brain and is defined as a sudden (within seconds) or at least rapid (within hours) episode of focal neurological dysfunction caused by a blood clot blockage, by narrowing of the blood vessels (clogging), by both a blockage and narrowing, or rupture of a blood vessel into the brain (Feign, 2004). Disruption of circulation results in inadequate blood supply which is evident in clinical signs and symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than of vascular origin (World Health Organisation [WHO], 1989). This definition includes SAH, however excludes cerebral tumour, subdural hematoma, post-seizure palsy, brain trauma, syncopes of cardiac or other origin where no focal cerebral symptoms are present and transient ischemic attacks (TIAs; WHO, 1989). Stroke is a hetrogeneous disorder that consists of two major pathological types; ischaemic stroke

(IS); and haemorrhagic stroke of which there are two types, intracerebral haemorrhage (ICH) and SAH. An undetermined stroke is a stroke "in which a patient had not undergone Computed Tomography (CT) scanning within 28 days of the onset of symptoms and an autopsy had not been performed" (Thrift et al., 2001, p.1733).

Acute IS or cerebral infarction refers to strokes caused by thrombosis, where a solid mass or clot is formed in vessels, composed of the constituents of the blood; or embolism, where an emboli (debris) migrates from another part of body via the circulatory system and causes blockage of a blood vessel in another part of body (Feigin, 2004). By contrast haemorrhagic strokes involve bleeding into the brain tissue (i.e., intracerebral haemorrhage) or into the narrow space between the brain surface and the layer of tissue that covers the brain (i.e., SAH). Intracerebral haemorrhage is defined as "a stroke in which a CT scan demonstrated an area of hyperdensity within the brain parenchyma with or without extension into the ventricles or subarachnoid space" (Thrift et al., 2001, p.1733). SAH is a haemorrhagic stroke caused by bleeding into the subarachnoid space which surrounds the brain and is beneath the arachnoid membrane and pia mater; and can be defined as "as an abrupt onset of severe headache and/or loss of consciousness, with or without focal neurological signs, with CT, neuropsy, or lumbar puncture evidence of focal or generalized blood in the subarachnoid space" (The ACROSS Group, 2000, p.1844). IS and haemorrhagic stroke are shown in Figure 1. As the focus of this research is SAH, the following section will review the causes of SAH; which include ruptured aneurysm (85%); non-aneurysmal perimesencephalic haemorrhage (10%); and a variety of rare conditions such as cerebral arteriovenous malformation (AVM); arterial dissection, vascular lesions around spinal cord, cocaine abuse, and trauma (5%; van Gijn & Rinkel 2001).

SAH types

The most common cause of SAH, aneurysms, develop at the site of a defect in the wall of the intracranial blood vessels. The weakened wall balloons out to form a blood filled sac, known as a saccular aneurysm, usually at the base of the brain, either on the circle of Willis or at a nearby branching point. As the bulge grows, its' walls becomes thinner, weaker, and unstable and may rupture causing haemorrhage into and around the brain structures (van Gijn, Kerr & Rinkel, 2007). As noted above, ruptured aneurysm is the most common cause of SAH, and the most common sites of a rupture include the anterior communicating artery (29%), followed by internal carotid artery-posterior communicating origin (23%), middle cerebral artery (23%), multiple lesions (15%), vertebrobasilar circulation (5%), internal carotid artery bifurcation (3%) and distal position of anterior cerebral artery (2%) (Al-Shahi, White, Davenport & Lindsay, 2006; Richardson, 1969). In patients with multiple aneurysms it is important to determine the location of aneurysm rupture with the help of CT scan (Al-Shahi et al., 2006). It is noteworthy that approximately 2% of people have unruptured aneurysms (Rinkel, Djibuti, Algra & van Gijn, 1998), with a 1.2% (follow-up <5 years) risk of rupture, and this risk increases with age and female gender (Wermer, van der Schaaf, Algra & Rinkel, 2007).

Non-aneurysmal perimesencephalic haemorrhage, the second most common cause of SAH, occurs when the leaked blood is confined to the cisterns around the mid brain, and the centre of bleeding is immediately anterior to the midbrain (Schwartz & Solomon, 1996). While it is difficult to clinically distinguish between this type of haemorrhage and one caused by ruptured aneurysm as the presenting symptoms are a similar in both types (van Gijn & Rinkel, 2001), in non-aneurysmal haemorrhage the onset of headache is generally more gradual (minutes rather than seconds) (Linn, Rinkel,

Algra & van Gijn, 1998). Furthermore, on admission patients with non-aneurysmal haemorrhage are usually alert and only a few are disoriented (Schwartz & Solomon, 1996).

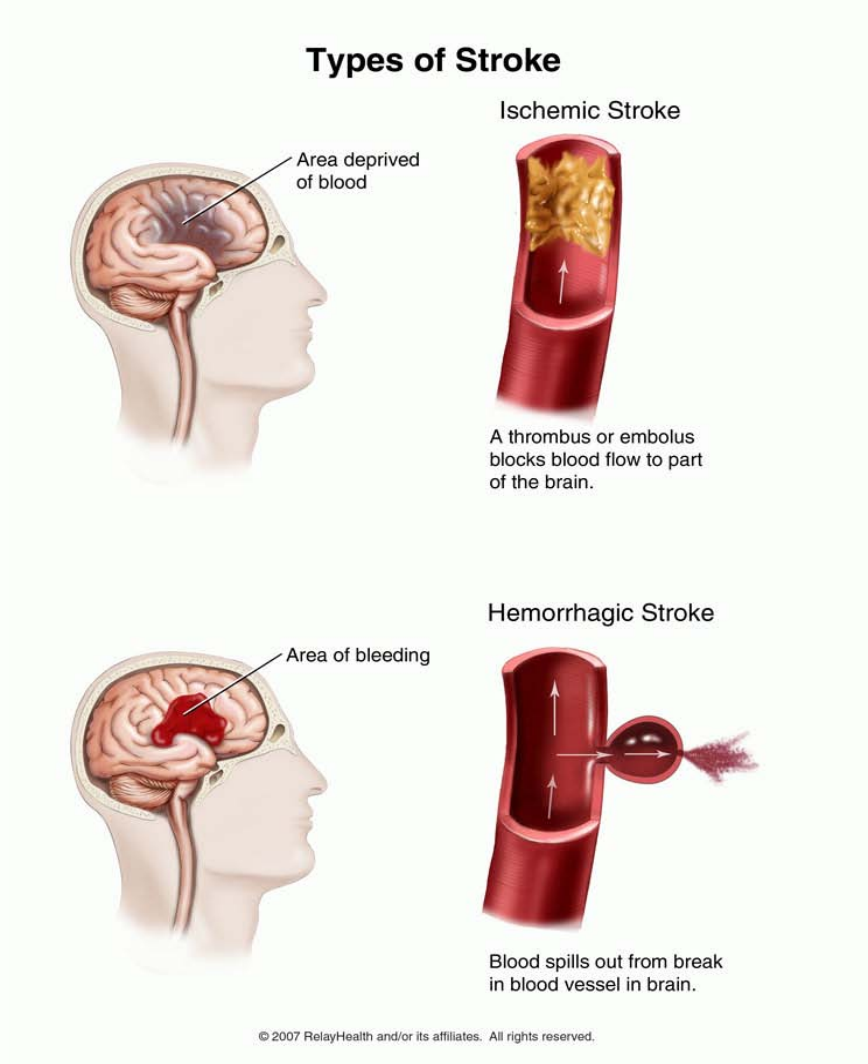

Figure 1

Types of stroke

Arteriovenous malformation (AVM) is a congenital disorder characterized by a complex tangled web of arteries and veins. AVMs rarely rupture (<3%), with the blood

leakage remaining in the subarachnoid space without intracerebral haematoma (Hernesniemi et al., 2008). While aneurysm rupture is the most common cause of SAH, it accounts for only 4% of all strokes. A community based Italian study found that SAH was more prevalent in stroke patients <45 years of age (42.7%), than older patients (15.7%); and that most SAH cases (52.6%) were due to aneurysms and arteriovenous malformations (Marini et al., 2001). As alluded to above, people suffering from stroke often experience non-specific symptoms such as headache, dizziness, nausea, vomiting, and unsteady gait and may show subtle or absent neurological signs (i.e., dysarthia, ataxia; Edlow, Newman-Toker & Savitz, 2008). In contrast, the most characteristic feature of SAH is sudden and severe headache whose onset is within a few seconds. This distinct headache is often accompanied by photophobia, vomiting, low grade fever, loss of consciousness; and face, neck and back pain (Kopitnik & Samson, 1993; Suarez, Tarr & Selman, 2006). SAH may be misdiagnosed when these typical signs and symptoms are absent (Edlow & Caplan, 2000), though head CT scan performed within 12-24 hours of symptom onset helps in clarifying the diagnosis. In some patients with sudden headache and normal CT scan, it is necessary to perform lumbar puncture to confirm the diagnosis of SAH (Edlow & Caplan, 2000; Suarez et al., 2006).

Having reviewed definitions of stroke and its subtypes as well as the most common mechanisms of SAH, the following sections examine the incidence and risk factors for stroke, with an emphasis on SAH.

Epidemiology of stroke

In a population based study conducted in Melbourne (Thrift et al., 2001) the incidence of first ever stroke was reported as 72.5% ischemic, 14.5% intracerebral haemorrhage, 4.3% SAH, and 8.7% undetermined stroke. A recent review of population based studies of stroke incidence reported that from 2000 to 2008, the highest crude

stroke type-specific incidence rates were reported in Italy (IS 174 per 100,000 and primary ICH 23 per 100,000) and Auckland (SAH 10 per 1000,000; Feigin, Lawes, Bennet, Barker-Collo & Parag, 2009). Over the last four decades, stroke incidence in high-income countries (using World Bank country classification) decreased by 42%; whereas in low to middle income countries incidence increased by 100% (Feigin et al., 2009). Based on follow-up of 18 European populations in the Monica Risk, Genetics, Archiving and Monograph project, Asplund et al. (2009) reported that stroke risk increased with increasing age and each year of age increased the risk of stroke (fatal or non fatal) by 9% (95% CI, 9% to 10%) (Nilsson, Lindgren, Ståhl, Brandt, & Säveland, 2000). Feigin et al. (2009) reported that in high income countries a significant decrease in incidence rates was observed in people of all ages, while in low to middle income countries, there was a significant two times increase incidence in the younger (<75 years) people and almost four times increase in the older (≥75 years) people.

Recent data from the United Kingdom and China suggest that reduced stroke incidence can be attributed to preventive treatments and reduction of risk factors (e.g. hypertension, artiral fibrillation, smoking) at the population level (Heushman, Grieve, Toschke, Rudd & Wolfe, 2008; Wang et al., 2007). Increased stroke incidence in low to middle income countries is attributed to health and demographic transitions in these countries including exposure to cardiovascular risk factors (e.g., smoking, raised blood pressure, high salt and fat diet; Connor, Walker, Modi & Warlow, 2007; Reddy, 2004). In New Zealand stroke incident rates were stable from 1981-1982 to 1991-1992 and then declined to 2002-2003, thus generating a slight overall decline of 11% (95% CI, 1 to 19%) in standardized incidence (Anderson et al., 2005). However, the Perth Community Stroke Study (Islam et al., 2008), reported that stroke incidents rates declined by 43% in 2000-2001 as compared to 25% in 1989-1990. The lesser decline rates in Auckland

could be attributed to the greater proportion of minority ethnic people in the population in Auckland (i.e., Maori, Pacific Island) who are at greater risk of stroke (Carter et al., 2006; Islam et al., 2008).

In addition to overall incidence of stroke, epidemiological studies commonly report case-fatality. Stroke is the second commonest cause of mortality in the world (Murray & Lopez, 1997; Sarti, Rastenyte, Cepaitis, & Tuomilehto, 2000). Case fatality (28-day) has been reported as 12% for IS, 45% for intracerebral haemorrhage, 50% for SAH and 38% for stroke of undermined type (Thrift et al., 2001). In the absence of appropriate interventions, the number of deaths due to stroke worldwide is expected to rise to 6.5 million in 2015 and to 7.8 million in 2030 (Strong, Mathers & Bonita, 2007). Epidemiological studies show that early stroke case-fatality rates (21 day to 1 month) have decreased in both high-income and low to middle income countries, but overall early stroke case-fatality in low to middle income countries in the past decade is 25% higher than in high-income countries (Feigin et al., 2009). From 2000 to 2008 early case-fatality ranged from 17% to 30% (13-23% for IS, 25-35% for primary ICH, and 25-35% for SAH) in high income countries and from 18-35% in low to middle income countries (13-19% for ischemic stroke, 30-48% for primary intracerebral haemorrhage, and 40-48% for SAH; Feigin et al., 2009). Having discussed the incidence and case fatality of stroke more generally, in the ensuing section the incidence and prevalence of SAH will be reviewed in more detail along with a review of stroke risk factors again with a focus on SAH.

Epidemiology of SAH

SAH constitutes approximately 5% of all strokes (Longstreth, Koepsell, Yerby & van Belle, 1985) and affects 21,000-33,000 people every year in the United States (Rosamond et al., 2007). The incidence of SAH has been reported to be around 6 cases

per 100,000 patients (van Gijn & Rinkel, 2001).

Two previous population based stroke incidence studies in Auckland, New Zealand (1981-1982 and 1991-1992) showed that the incidence of SAH declined from 1981-1983 (14.6 per 100,000) to 1991-93 (11.3 per 100,000) (Truelsen, Bonita, Duncan, Anderson & Mee, 1998). The same trend can be seen in a recent population based study (Australasian Cooperative Research on Subarachnoid Haemorrhage Study; ACROSS) in New Zealand and Australia, which found that the crude annual incidence of SAH (1995-1998) has reduced for the total population (8.1 per 100,000; The ACROSS Group, 2000). However, while studies show a decline in incidence of SAH, this may be an artifact of assessment procedure with the actual incidence of SAH remaining unchanged. That is, there may be a diagnostic bias with a large proportion of patients currently investigated with CT scan, providing more accurate distinction between SAH and intracerebral haemorrhage than was previously possible (Linn et al., 1996; van Gijn et al., 2007). The standardized incidence of SAH varies across regions with incidence in Finland almost thrice that of other parts of the world (Linn et al., 1996). Auckland New Zealand has the highest incidence of SAH (10.3 per 100,000) as compared to other cities in Australia (i.e., Adelaide, Hobart and Perth; The ACROSS group, 2000). Early case-fatality (21 days to 1 month) of SAH in low to middle income countries has been reported to be 31.7% higher than in high income countries (43.9% vs 30.0%; Feigin et al., 2009) which could be due to poorer management in low to middle income countries (Ingall, Asplund, Mähönen & Bonita, 2000). A review of SAH studies showed that from 1995 to 2007 case-fatality rates have decreased by 0.8% per year (CI 0.2 to 1.3); a 23% decrease in case-fatality over the 30 years studied (Nieuwkamp et al., 2009).

RISK FACTORS FOR STROKE AND SAH

Numerous conditions and life-style elements have been identified as risk factors for stroke, which can be classified into modifiable and non-modifiable factors (Feigin et al., 2005; Lindner, Bor & Rinkel, 2010; Romero, 2007; Sacco et al., 1997). While review of all risk factors is beyond the scope of this study, the following section reviews the main stroke risk factors. Information on stroke in general is presented and, where available, specific risk for SAH is reported.

Non-modifiable risk factors

Non-modifiable risk factors (i.e., ethnicity, age, sex, family history) help identify individuals at greater risk for stroke. In regards to *ethnicity*, stroke incidence and mortality rates vary considerably (Wolfe et al., 2002), with studies in the United States showing significantly higher mortality rates in blacks as compared to whites (Mensah, Mokdad, Ford, Greenlund & Croft, 2005). In New Zealand, significant ethnic disparities were observed in stroke incidence and incidence trends, with stroke incidence rates declining in Europeans from 1981-1982 to 2002-2003; yet remained high or increasing in other ethnic groups (e.g., Maori and Pacific people 66% increase [95% CI; 11% to 225%]; Carter et al., 2006). The Auckland Regional Community Stroke Study, 2002 to 2003 reported similar rates of SAH across ethnic groups (i.e., European, Maori, Pacific Island, 'Asian and others'; Feigin et al., 2006). Carter et al. (2006) noted consistent declines in 28-day fatality rates across all ethnic groups (i.e., New Zealand European, Maori, Pacific Island and 'Asian and others'), although this trend was not significant for Maori, and the largest decline occurred for Pacific Island (55% decline) and 'Asian and other' (70% decline) groups. In regards to SAH, a recent population based American study reported that SAH disproportionately affects Mexican Americans and non-Hispanic whites, with the 7-year cumulative incidence of SAH being higher in

Mexican Americans (60% of cases versus 48% of the population) as compared to non-Hispanic whites (40% of cases versus 53% of population; Eden et al., 2008). The South London Stroke Register study also studied ethnic disparities reporting that the incidence of SAH was higher in black as compared to white population (0.18 [95% CI 0.04-0.31] and 0.08 [95% CI 0.05 to 0.10], respectively (Wolfe et al., 2002). From 1995 to 2004, a substantial decrease in the SAH incidence (incident rate ratio, 0.12, 95% CI 0.02 to 0.59) was observed in black patients as compared to whites (Heushmann et al., 2008).

Age is also associated with stroke (Harmsen, Lappas, Rosengren & Wilhelmsen 2006; Romero, 2007). In New Zealand, from 1981-82 to 2002-03, the average age of stroke patients increased in New Zealand European, Pacific Island people and Maori, but not for 'Asian and others' (Cartel et al., 2006). In Maori and Pacific Island people, stroke occurs at a much younger age (means=60.7 years and 64.5 years, respectively) as compared with New Zealand Europeans (mean=75.6 years; Carter et al., 2006). This relationship between SAH and age is noted in several population based studies (The ACROSS Group, 2000; Nilsson et al., 2000). Although there is increased incidence of SAH with age ranging from 0.8 per 100,000 (15-24 years) to 26.5 per 100,000 ($\geq$ 85 years), over half of SAH survivors are <55 years of age (The ACROSS Group, 2000; Marini et al., 2001; Nieuwkamp et al., 2009; Nilsson et al., 2000). A population based New Zealand study reported a continuous rising trend for incidence with increasing age in females, however for males the trend was bimodal, being highest in younger males (aged 34-54 years) and older age ($\geq$85 years) (The ACROSS Group, 2000).

G*ender* is also associated with stroke incidence, with lifetime risk of stroke in women (1 in 5) greater than that for men (1 in 6) (Carter et al., 2006)**.** In New Zealand, women on average have their first stroke later than men with mean age of onset of stroke being 75.8 years and 73 years, respectively (Anderson et al., 2005; Dyall et al.,

2006). For males there was a significant 16% (95% CI, 2 to 27%) decline in stroke incidence and 14% (95% CI, 2 to 24%) decline in attack rates from 1981 to 2003 while for women, standardised stroke incidence remained stable (Anderson et al., 2005). However, a significant decline of 14% (95% CI, 2%-29%) for first ever stroke rates was found between 1991-92 and 2002-03 in women.

Prominent gender differences have also been reported for SAH, with the incidence of SAH being 1.6 times higher in women than men (Linn, Rinkel, Algra & van Gijn, 1996). A review of population based studies indicated that those aged 25-45 years, incidence of SAH was significantly higher in men than women, however those aged 55-85 years, incidence of SAH was significantly higher in women (de Rooij, Linn, Plas, Algra & Rinkel, 2007). The ACROSS study similarly indicates higher incidence rates for females as compared to males in Australia and New Zealand; with a similar trend noticed specifically in Auckland, New Zealand (The ACROSS Group, 2000).

Family history of stroke is also a risk factor (Woo et al., 2009). In a prospective study, history of parental stroke was associated with an increased incidence of stroke in their children (hazard ratio, 2.79; 95% CI, 1.68 to 4.66; $p<0.001$), with parental stroke before 65 years of age associated with a 3-fold increase in risk (Seshadri et al., 2010). This risk may be due to a genetic predisposition, or presence of other risk factors (e.g., diabetes, hypertension, obesity) (de Freitas, Bezerra, Maulaz & Bogousslavsky, 2005; Yamada et al., 2006). Woo et al (2009) noted that a family history of brain aneurysm/SAH has been associated with increased risk of experiencing SAH ($p=0.03$). A recent study reported odds ratio of SAH individuals with one affected first-degree relative was 2.15 (95% CI 1.77-2.59) and for the two affected first degree relatives, 51.0 (95% CI 8.56-1117; Bor et al., 2008).

Modifiable risk factors

Modifiable risk factors (i.e., hypertension, diabetes mellitus, cigarette smoking, obesity, alcohol consumption, heart disease, carotid stenosis) have a potential to change and thus can reduce stroke risk. For example, *hypertension* is one of the most common modifiable risk factors for stroke (Seshadri et al., 2006; Wolf, D'Agastino, Belanger & Kannel, 1991; Woo et al., 2004) and can be controlled by medication and life style changes (e.g., diet, weight loss, reduced alcohol consumption; Ong, Cheung, Man, Lau & Lam, 2007). Both systolic and diastolic blood pressures have a continuous and graded influence on stroke (Kannel, Vasan & Levy, 2003; Sturgeon et al., 2007). A 10mm Hg reduction in systolic or a 5 mm Hg reduction in diastolic blood pressure results in 40% lower risk of stroke death (Lewington, Clarke, Qizilbash, Peto & Collins, 2002). People with normal BP have half the lifetime risk of stroke compared to those with high blood pressure (Seshadri et al., 2006). Overall, 32% (95% CI, 14% to 45%) of all strokes are attributable to uncontrolled high blood pressure (Klungel et al., 2000). Uncontrolled blood pressure among treated hypertensive people is a noted cause of hemorrhagic strokes 57% (95% CI, 26% to 75%). A review of studies reported that hypertension increases the risk of SAH by approximately 2.5 times and was 30% more hazardous in women than men (Feigin et al., 2005).

Diabetes mellitus is a further independent risk factor for stroke (e.g., Harmsen et al., 2006; Feldmann et al., 2005). Epidemiological studies show that diabetic patients have a 1.8 to 6 times increased risk of stroke (Goldstein et al., 2001; Giorda et al., 2007). However, diabetes does not appear to increase risk of aneurismal SAH (Adams, Putman, Kassell & Torner, 1984; Qureshi et al., 2001), and some studies even suggest that diabetes is associated with significant reduction of SAH risk (Feigin, 2005; Inagawa, 2005).

Cigarette Smoking is a recognised independent risk factor for all stroke types (Sacco et al., 1997; Feldmann et al., 2005), increasing the risk of stroke to four times that of people who never smoked (Bonita, Duncan, Truelsen, Jackson & Beaglehole, 1999). In current smokers increased number of cigarettes smoked increases the risk of haemorrhagic stroke (Kurth et al., 2003). Ueshima et al. (2004) found that for men and women who smoked 21 cigarettes/day or more, the relative risk (RR) for all strokes were 2.17 (95% CI, 1.09 to 4.30) and 3.91 (95% CI, 1.18 to 12.90), respectively; while their RRs of fatal stroke ($\geq$21 cigarettes/day) were 2.17 (95% CI, 1.09 to 4.30) and 3.91 (CI, 1.18 to 12.90), respectively. In New Zealand, the risk of SAH is 3 times higher in current smokers, though this risk reduces within a few years after smoking stops (Anderson, Feigin, Bennett, Lin, Hankey & Jamrozik, 2004). Current smoking with a family history of aneurysmal SAH compounds SAH risk to six times as compared to those with neither risk factor (Woo et al., 2009).

Obesity is known to increase the risk of stroke (Kruth et al., 2005). In a cohort study, of middle aged men, elevated Body Mass Index (BMI $\geq$ 30 kg/m^2) and low levels of physical activity predicted stroke at 28-year follow-up (Harmsen et al., 2006). The RR of IS is 1.06 (95% CI, 1.04-1.07) per 1 kg/m2 increase in BMI (Carron, Smith & McCarron, 2006). The relationship between BMI and SAH is unclear (Feigin et al., 2005). One longitudinal study reported that lean BMI decreases risk of SAH in men by 70%, whereas case control studies report that lean BMI increases this risk (Feigin et al., 2005). Sandvei, Romundstad, Müller, Vatten & Vik (2009) report that overweight people (BMI, 25 to 29.9 kg/m^2) were at lower risk of SAH as compared to the normal weight (BMI, 18.5 to 24.9 kg/m^2).

Alcohol consumption is also associated with increased risk of stroke (Ariesen, Claus, Rinkel & Algra, 2003). Excessive alcohol consumption (>150g per week)

increases risk of SAH by approximately two times (Feigin et al., 2005; Feldmann et al., 2005; Ruigrok, Buskens & Rinkel, 2001). Drinking 100 to 299 g/wk accounts for 11% of the cases of SAH, whereas drinking ≥300 g/wk accounts for 21% of the SAH cases (Ruigrok et al., 2001).

Carotid Stenosis, a narrowing of the inner surface of the carotid artery (Feigin, 2004) is associated with increased risk of stroke (Sacco, 2001; Schwartz et al., 1995). Patients with severe (>75%) and progressing carotid stenosis have more frequent ischemic events (Chambers, & Norris, 1986). Carotid wall thickness is associated with the risk of stroke and hazard ratio for each 0.1 mm of intima-media thickness results for 1.47 (95% CI 1.16-1.87) stroke events (Silvestrini et al., 2010). An association has also been reported between carotid stenosis and SAH (Kleinig, Kimber & Thompson, 2009).

Summary

While only accounting for a small percentage of all strokes, SAH tend to impact at a younger age. Particular risk factors for SAH include ethnicity, age, gender, family history, hypertension, diabetes mellitus, cigarette smoking, obesity, alcohol consumption, heart disease, and carotid stenosis. Having reviewed the definitions and epidemiology of stroke, with a focus on SAH, the following section shifts focus to outcomes. As the literature specific to SAH is sparse, the more general literature on outcomes of stroke is reviewed with any available literature on SAH presented within this wider context. This is first placed within the context of models of health outcomes.

HEALTH OUTCOMES

In health outcomes research, outcomes are measured from different perspectives. This section reviews different ways in which health outcomes have been conceptualised, focussing on the development of the International Classification of Impairments, Disabilities and Handicaps (ICIDH; WHO, 1980) and its' replacement the International

Classification of Functioning, Disability and Health (ICF; WHO, 2001); and their criticisms. This is followed by a review of literature examining the long-term neuropsychological and functional outcomes following stroke and SAH. This includes a review of literature relating to body structure and impairments; including neurological impairments, cognitive, and mood and emotional outcomes after stroke and SAH. The final section includes a review of literature examining the long-term functional outcome, including activity and participation, in stroke and SAH survivors.

Models of health outcomes

A number of models of human functioning and disability have been applied in the context of health outcomes (Albrecht, Sleeman & Bury, 2001; Jette, 2006). According to the bio-medical model, an illness/disease is caused by an abnormality within the structure of a specific body organ. This organic process creates certain signs and symptoms which require intervention by professionals. The patient has no responsibility for the presence of the disease and is a passive recipient of treatment, although cooperation is expected (Wade & Helligan, 2004). The main flaw of this model is its over-emphasis of the medical condition, incorrectly assuming a causal link between impairment and disability, and not considering the impact of personal factors or environment on ability to function (Engel, 1977).

According to the social model, disability is not a result of a medical illness/disease but due to restrictions imposed by society through its organisation for healthy and able individuals (e.g., lack of wheelchair access, discrimination, attitudes towards disabled). Thus, "the underlying problem is created by an unaccommodating or inflexible environment brought about by the attitudes or features of the social and physical environment itself, which calls for a political responses or solution" (Jette, 2006, pp.727). The main criticism of this model is that even though society plays an

important role in peoples' lives, disability is not an outcome of social barriers alone. Thus, non-socially imposed restrictions on activity can be better understood within the context of impairments (Shakespeare & Watson, 2001).

A third model, the bio-psycho-social model of disability, attempts to combine medical and social models of disability; viewing disability as an interaction of biological, personal and social factors (Engel, 1977). In this model, 'biological' refers to physical or mental health conditions, while 'psycho' refers to the personal/psychological factors that influence functioning, and 'social' refers to the impact of social context on illness behaviour. This is an individual-centred model which considers that function is a complex interaction of the above mentioned factors. Although this model represents the dominant perspectives behind contemporary models of disability used, it does not fully clarify or differentiate the terms 'psycho' and 'social' (Waddell, Burton & Aylward, 2008).

WHO models of health outcomes

The WHO recognised that the existing medical model and the International Classification of Diseases (ICD) was useful for classifying medical diagnosis, but inadequate to address disability as it did not address the consequences of the disease (Fougeyrollas, 1995). Thus, in the absence of a consensus on the understanding of health outcomes, the WHO developed the International Classification of Impairments, Disabilities, and Handicaps (ICIDH) 'for trial purposes', to be used in conjunction with the ICD (WHO, 1980). The main aim of the ICIDH was to clarify confusions in terminology and present a model of cause and effect relationships between conceptual levels. According to this model, injury/disease leads to functional and organic impairment, which in turn results in disability in an individual's behaviour and activities, which generates handicap(s) and disadvantages, with respect to roles (WHO, 1980). To

define these further, "In the context of health experience, an *impairment* is any loss or abnormality of psychological, physiological, or anatomical structure or function" (WHO, 1980, p.27) and is associated with 'signs and symptoms'. For example, common neurological impairments such as weakness in, or loss of a limb; pain, double vision, ataxia and reduction in movement at a joint. In contrast, "In the context of health experience, a *disability* is any restriction or lack (resulting from an impairment) of ability to perform an activity in the manner or within the range considered normal for a human being" (WHO, 1980, p. 28). For example, disturbances in behaviour, in personal care (e.g., inability to dress), or other activities of daily living and in locomotion (e.g., inability to walk). Finally, "a *handicap* is a disadvantage for a given individual, resulting from an impairment or a disability, that limits or prevents the fulfilment of a role that is normal (depending on age, sex, and social and cultural factors) for that individual" (WHO, 1980, p. 29). Thus, handicap is a result of impairments and disabilities and is seen within the social context where an individual is placed at a disadvantage in relation to their peers; such as job loss or marital breakdown arising as a consequence of the disease. To illustrate these concepts, an individual who suffered a SAH may experience impairments in right upper extremity mobility and fine motor coordination. These impairments may result in disabilities such as reduced writing ability and requiring assistance to dress. The resulting handicap may include inability to return to work.

Although ICIDH is an important framework to classify function and disability associated with health conditions and has been widely used (Badley, 1993), it failed to receive endorsement from the World Health Assembly (Stucki, 2005). The major criticism was in the linearity of the model as it progressed from biomedical to psychosocial constructs (i.e., from impairments to disabilities to handicaps) and did not

clarify the causal and temporal relationship between the three dimensions (Gray & Hendershot, 2000; Johnston & Pollard, 2001). Furthermore, the three constructs lacked clarity and overlapped (Badley, 1993). For example, it is not clear when a particular function (e.g., skeletal impairment) becomes an activity (e.g., locomotor disability). Another criticism was that the ICIDH did not pay sufficient attention to the role of the environment on an individual's outcome (Fougeyrollas, 1995). For example, an individual with a walking disability can mobilize using a wheelchair. However, a rural setting without wheelchair access or obstacles to wheelchair use will impact his/her ability to perform 'social roles' such as going to work.

To address criticisms of ICIDH, the WHO developed a revised classification, the International Classification of Functioning, Disability and Health (ICF). The ICF provides a framework and standard language for classifying and describing health and health related states, both at individual and population levels (Jette, 2006; Stucki, 2005) and was endorsed by the 54th World Health Assembly (WHO, 2001).

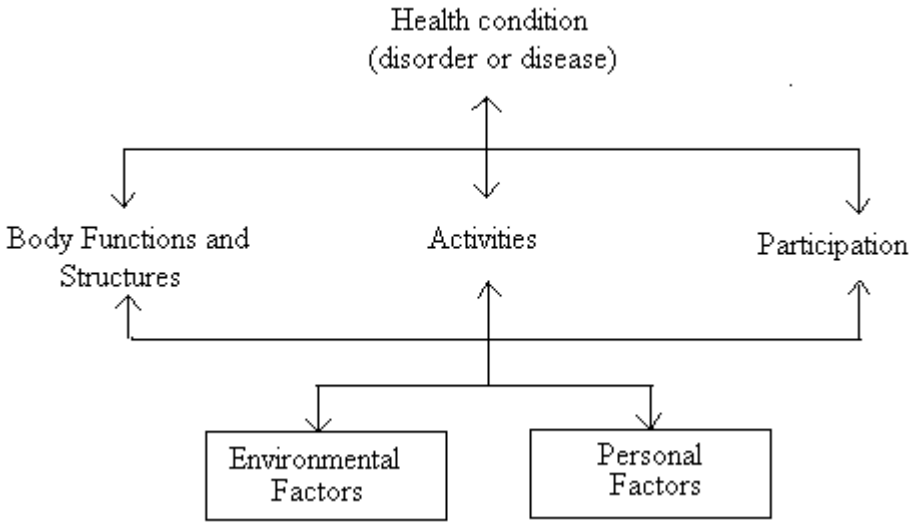

Figure 2

The current framework of Functioning, Disability and Health (ICF)

The ICF model (Figure 2) is based on the bio-psycho-social model encompassing three key components of functioning and disability- *body functions and structures, activity* and *participation*. These components may interact with the individual's health condition and contextual factors (i.e., personal and environmental). In this model 'functioning' denotes positive aspects and 'disability' denotes negative aspects of outcomes (impairments, limitations in activities, and restrictions in participation).

'*Body functions*' are physiological and psychological functions of the body systems (e.g., eye hand coordination, working memory), while anatomical parts of the body are referred to as '*body structures*' (e.g., lobes of the brain, limbs). Abnormality in body functions or structures is referred to as an impairment. In contrast, '*activity'* is the execution of a task by the individual, including their capacity to do it and actual performance of the task. Difficulties executing activities is referred as the 'activity limitation' (e.g., difficulty in walking due to mobility limitations). Finally, *'participation'* refers to involvement in life situations. Problems experienced by an individual with such involvements are called 'participation restriction' (e.g., due to walking difficulty being restricted in social life).

Environmental factors include physical, social and attitudinal environment such as products and technology, supports, relationships, attitudes, available services, social systems and policies which can facilitate or hinder a person's level of function and disability. *Personal factors* include the individual's background and do not include their health condition (e.g., age, sex, ethnicity, health conditions, coping style).

Thus, the focus of the ICF is on activities of an individual and their participation and contribution to the social environment. The ICF framework is comprehensive, with universally accepted terminology for functioning and disability (Stucki, 2005) and has been implemented in many countries in various sectors (Cieza et al., 2006; Jette, 2006;

Stucki, Cieza & Melvin, 2007; WHO, 2001). One of the main criticisms of the ICF is that it cannot be consistently applied to clinical practice as it contains over 1400 categories (Üstün, Chatterji & Kostanjsek, 2004). To improve clinical applicability, ICF Core Sets were developed "to select sets of categories out of the whole classification, which can serve as minimal standards of assessment, communication and reporting of functioning and health for clinical studies, clinical encounters and multi-professional comprehensive assessment and management purposes" (Grill, Ewert, Chatterji, Kostanjsek & Stucki 2005, p 362). A comprehensive ICF Core Set for stroke has been developed, which includes 41 body functions, 5 body structures, 51 activities and participation, and 33 environment factors (Geyh et al., 2004). However, this core set needs to be validated. Thus far, inter-rater reliability of the ICF core sets for stroke has been found to be moderate (Starrost et al., 2008). However, a recent study suggests the ICF stroke core set be reduced to 28 body functions, and 41 activities and participation components to enhance feasibility for assessment (Algurén, Lundgren-Nilsson & Sunnerhagen, 2010). A recent study supported the construct validity of the 'functioning' part of the ICF stroke core set (Ewert, Allen, Wislon, Üstün & Stucki, 2010).

When planning for the current study began (2005), the core sets for stroke had not been fully developed. Thus, in the current study, standardised outcomes measures were selected to serve as indicators of ICF categories of body functioning (measures of impairment: neuropsychological tests and mood), activity (measures of disability), and participation (measures of handicap and health related quality of life [HRQoL]). In the ensuing section although outcomes of stroke are reviewed within the ICF framework, to avoid confusion and for consistency, the following labels are used: impairment (body functioning), disability (activity) and handicap (participation).

STROKE AND SAH OUTCOMES

Stroke results in enormous burden to society and changes live for individuals and families in many ways. It has a massive impact on the individual, family, health care system and society. Although it is important to examine short-term (≤1 year) outcomes following stroke; it is also important to study long-term (>1 year) outcomes in order to plan and provide appropriate rehabilitation and care. In addition, cessation of active rehabilitation in most 5 to 6 years stroke survivors (compared to 1-3 year survivors) may adversely impact outcomes, and there is evidence that factors affecting long-term outcomes are different from those affecting short-term outcomes post-stroke (Woo, Kay, Yuen & Nicholls, 1992).

As SAH outcomes tend to change overtime (Haug et al., 2007; Koivisto et al., 2000), studying SAH is important from the stand-point of better understanding its natural course and its impact. Studying long-term outcomes of SAH in a population based context is important, yet there are few population based studies of SAH survivors beyond 12 months (Hackett & Anderson, 2000) and even fewer examining wider outcomes such as neuropsychological outcomes, quality of life (Mocco et al., 2006), disability (Hop, Rinkel, Algra & van Gijn, 1997), and mood (Powell, Kitchen, Heslin & Greenwood, 2004). The following section reviews the major areas of long-term outcomes of stroke with a focus on SAH. Greater attention is given to cognitive and functional outcomes and studies which are population based as these are the focus of the current study.

Body Functioning and Structure Impairments

Neurological outcomes

Several scales are used to measure the neurological impact of stroke such as the National Institute of Health Stroke Scale (NIHSS; Brott et al., 1989), the Canadian

Neurological Scale (Cote, Hachinski, Shruvell, Norris & Wolfson, 1986) and European Stroke Scale (Hantson et al., 1994). These scales are often used in conjunction with functional outcome assessments to examine stroke impact. Common neurological outcomes of stroke include recurrence, visual field deficits, motor deficits, verbal and perceptual deficits and behaviour change (Carlo et al., 1999; Lawrence et al., 2001; Taub, Wolfe, Richardson & Burney, 1994). Within this section, prevalence of neurological impairments post-stroke and SAH are outlined followed by a review of their relationship to functional outcomes.

The prevalence of various acute stroke impairments were studied by Lawrence et al. (2001) in a multi-ethnic population based study (n=1299); who reported that almost all the patients (98.2%) had some impairment. Common impairments were limb weakness (upper limb 77.4%, lower limb 72%), urinary incontinence (48.2%), impaired consciousness (44.7%), and dysphagia (44.7%). Impairments such as paralysis, urinary incontinence, dysphagia and gaze perasis at acute stage were associated with severe disability and death at 3-months (Carlo et al., 1999; Lawrence et al., 2001). Similar findings are reported by other authors at 1-year post-stroke (Taub et al., 1994) and 3-months post-stroke (Carlo et al., 1999). A multi-centre European study (Carlo et al., 1999) examined clinical presentation and outcomes post-stroke reporting that clinical state was more severely compromised in older (>80 years) patients and they presented more frequently with urinary incontinence (41.7%), paralysis (39.3%), dysarthria (33.7%), aphasia (32.6%), and swallowing problems (26.9%).

In a population based study, determinants of long-term HRQoL were stroke severity markers (dysphasia, loss of consciousness, neglect, and dense hemopresis [loss of power against gravity in at least 1 limb]) as measured by NIHSS (Sturm et al., 2004b). More recent studies have reported that incomplete motor recovery

(Franceschini, La Porta, Agosti & Massucci, 2009) and incontinence are important factors in reduced HRQoL 4-years post-stroke (Haacke et al., 2006).

Common impairments in SAH patients include urinary incontinence (71%), dysphagia (68%), limb weakness (66%), and gaze paresis (46%; Lawrence et al., 2001). Powell, Kitchen, Heslin and Greenwood (2002) reported that 61% of SAH participants (n=52) had at least one impairment (23.5% impaired visual acuity, 8% visual field deficit, 6% diplopia, 15.7% tinnitus, 27.5% dysphasia, and 12% vertigo). None of the participants suffered from ataxia, or locomotor deficits. Dysphasia 3-months post-SAH predicts adverse psychosocial outcomes at 9-months follow-up (Powell et al., 2002).

Besides disability and handicap, neurological deficits are associated with depression post-stroke and SAH (Kauhanen et al., 1999; Powell et al., 2002; Townend et al., 2007). Åström, Adolfsson and Asplund (1993) studied depression in a 3-year longitudinal study and reported that dysphasic patients (24%) were significantly more likely to be depressed than non-dysphasic patients. Similar findings are reported for aphasic patients (Damecour & Caplan, 1991; Kauhanen et al., 2000), and in relation to fatigue and impaired motor function of arm and leg (van De Port, Kwakkel, Bruin & Lindeman, 2007). Besides incontinence, behavioural outcomes such as fatigue are a common complaint for post-stroke survivors and negatively impacts their daily functioning and HRQoL (de Groot, Phillips & Eskes, 2003).

Cognitive outcomes

Neuropsychological impairments are an important sequel of stroke, occurring in 30% to 65% of the stroke survivors (Ballard, Rowan, Stephens, Kalaria & Kenney, 2003; Srikanth et al., 2003). Neuropsychological functioning may be defined as the way in which brain functioning is reflected in behaviour (Lezak, Howieson & Loring, 2004) including the ability to plan, memory, information processing speed (IPS), and language.

Previous researches show that although 70-80% of the SAH patients survive through surgery and show a positive neurological outcome (Uski et al., 2000), approximately 60% have considerable cognitive impairments (Hütter, Gilsbach & Kreitschmann-Andermahr, 1995; Saciri & Kos, 2002).

Stroke: Even though stroke survivors experience ongoing problems, recovery of cognitive functions is a continuous process with improvements across time (Hochstenbach, Otter & Mulder, 2003). Yet most studies of cognition to date have focused on relatively short periods (≤1 year) post-stroke (Sachdev, Brodaty, Valenzuela, Lorentz & Koschera, 2004; Srikanth et al., 2003; Haug et al., 2010). Hence there is little information regarding long-term cognitive functioning post-stroke.

Although a few population-based studies have examined long-term cognitive functioning post-stroke, these studies often used brief measures such as Mini-Mental State Examination (MMSE). For example a population-based study in South London (Patel, Coshall, Rudd & Wolfe, 2003) reported that cognitive impairments were present in 32% of the patients at 3-years post-stroke. A recent prospective, community based study assessed patients 5-years post-stroke and reported that 58.9% were cognitively impaired and 7.9% developed severe dementia (Chausson, Olindo, Cabre, Saint-Vil & Smadja, 2010). As these studies did not administer a comprehensive neuropsychological battery to assess cognitive outcomes, they are likely to under-identify impairments as measures such as the MMSE are cognitive screening measures only designed to identify those with significant impairments. MMSE has been found to be insensitive to detect post-stroke cognitive impairment as compared to comprehensive neuropsychological battery (Nys et al., 2005). In a comprehensive literature review of unruptured intracranial aneurysms (the most common cause of subarachnoid haemorrhage), Togwood, Ogden and Mee (2004) strongly suggest inclusion of a comprehensive battery

of neuropsychological tests for inpatient assessments. Some studies have used a neuropsychological battery to examine long term (>1 year) cognitive outcomes following stroke (Rasquin, Lodder & Verhey, 2005; Srikanth, Quinn, Donnan, Saling & Thrift, 2006), however the follow-up period in these studies was only up to 2-years post stroke. Furthermore, these studies excluded participants with severe neurological outcomes, aphasia, dysphasia, hearing and vision problems and major psychiatric disorders.

As the current study focuses on outcomes at 5-years post-stroke, in the following section, long-term (>15 months) cognitive outcomes assessed by neuropsychological batteries are examined in more detail (Table 1). As seen in Table 1, post-stroke cognitive impairments persist for over 1-year and may range from global cognitive decline to domain specific impairments such as memory, language orientation, attention, mental speed, visuospatial, and executive functioning (Ballard et al., 2003; Engstad, Almkvist, Viitanen & Arnesen, 2003; Rasquin, Verhey, Lousberg & Lodder, 2005; Sachdev et al., 2009; Srikanth et al., 2006). Perhaps some of this is due to variability in location of lesions post-stroke which would expect to impact whatever areas are affected. However, the exact nature of the cognitive impairments among stroke survivors is not known, as studies have not attempted to assess the full breadth of cognitive deficits of stroke survivors. As noted previously, some studies have assessed global cognitive profile by using screening measures such as the MMSE and/or Cambridge Examination for Mental Disorders of the Elderly (CAMCOG) in conjunction with a few more specific neuropsychological tests (Ballard et al., 2003; Dik et al., 2000). Whereas, others have assessed cognitive domains using an extensive neuropsychological battery of tests (e.g., Hochstenbach et al., 2003).

Table 1

Neuropsychological outcomes using battery of tests >15 months post-stroke

Author/Study	Sample	Time post-stroke	Measures	Outcomes
Change over time				
Ballard et al. (2003) *Longitudinal*	N=115 stroke >75y $\bar{x}$=80.4	3-15 mnths	MMSE, CAMCOG, CDR (Simple Reaction Time, CRT, Digit Vigilance, Memory Scanning and Spatial Memory), BNT, COWAT	Over time: > 30 % ↓ cognition -9% developed dementia (sig ↓ global cognition, memory, attention) -50% ↑ global cognition
Sachdev et al. (2009) *Longitudinal*	N=198 IS & TIA =106 controls	3-6 mnths & 36 mnths	NART-R, WMS-R-LM I & II, VR I & II; WAIS-R-(DS, Arithmetic, BD, Similarities, PC, BNT-15 item), TMT- A/B, SDMT, CFST, COWAT, WAB, finger gnosis and stereognosis	3-6 mnths ps= 18.2% have VaD, 40.9% vaMCI and 40.9% NCI -VaD with ↓ working memory, processing speed, executive functioning 36 mnths ps = ↓ language, memory (verbal and visual), executive functions -Incident dementia= 24.3% of vaMCI; 8.5% NCI -Controls=0% dementia
Alfieri et al. (2004) *Longitudinal*	N=191 IS & ICH $\bar{x}$=71.3 yrs	12, 24, 36 & 48 mnths	AVLT, RCF, Corsi Block-Tapping Test, SCWT, TMT, WCST, Coloured Progressive Matrices, COWAT	-incidence post-stroke dementia= 6.3% (12 mnths), 21.5% (48 mnths)
Hochstenbach et al. (2003) *Cohort home based*	N= 57 IS =8 Haemorrhages =33 Controls $\bar{x}$=56.4 yrs	$\bar{x}$=2.3 & 27.7 mnths	AVLT (Dutch version), RBMT, TMT, WAIS-II (DS, DSy, Letter Cancellation, Similarities, BD), BIT (copy, photograph scanning), Bobertag structural clock test, CDT, Money's road map test, DAS (naming, verbal fluency, word and sentence comprehension), Aspects of handling money (recognizing, counting, and arithmetic)	-Significant ↑ in all cognitive domains -Biggest ↑ for attention & language -Least for memory

Author/Study	Sample	Time post-stroke	Measures	Outcomes
Serrano et al. (2007) *Longitudinal*	N=327 Stroke	3, 12 & 24 mnths	MMSE, SPMSQ, hearing and simple/random visual reaction time, Bells Test, COWAT, WAIS-R-(PR, WR, LM, BD, Similarities), BDAT, TT	-3-mnths: 26.9% Cognitive Impairment No Dementia -24-mnths: 36.6% Cognitive Impairment No Dementia
van Zandvoort et al. (2005) *Longitudinal*	N=57 IS only $\bar{x}$= 56 yrs	$\bar{x}$=11.2 days & 20.9 mnths	WAIS (vocabulary), RAPM, BNT, COWAT, CBTT, RAVLT, Doors Test, RCFT, JOLO, TFP, TMT	Long-term: maximum ↓: visuo-perception and attention/psychomotor -At 20.9 mnths: ↑intellectual ability, language, memory, attention, perception, visuospatial construction -Maximum↑= language & memory
Controlled Comparisons				
Desmond et al. (2002) *Longitudinal*	N=575 Stroke=334 Control=241	$\bar{x}$=21.1 mnths	MMSE (orientation), SRT, BVRT (multiple-choice recognition), BNT, BDAE, RDT, BVRT, WAIS-R Similarities, MDRC (identities, oddities), Cancellation Tasks	-dementia incidence (stroke)=8.49 cases/100 persons-year; Controls-1.37 cases/100 persons-year -RR of dementia ps=3.83 (95% CI, 2.14-6.84)
Srikanth et al. (2006) *Population-based*	N=198 Stroke=99; Controls=99 $\bar{x}$ Age= 69.9 yrs	$\bar{x}$=2.14 yrs	WAIS-R (Ward SF)-Information, DS, Similarities, Arithmetic, PC, BD, DSy, K-SNAP, Gestalt Closure (number recall, 4-letter words), MMSE, RAVLT, RBMT, RCFT, CDT, COWAT, IQCODE	-2 yrs-Recurrent stroke ↑ risk of dementia. -37% with ↓ cognition developed dementia -Stroke=↓ spatial ability, memory, attention/speed, executive functioning, orientation/knowledge, compared to controls - 10.6% cognitively normal post-stroke
Rasquin et al. (2005) *Prospective*	N=156 strokes $\geq$ 40 yrs	24-mnths	CAMCOG, AVLT, CST, Stroop (Colour, Word), GIT (Calculation and Mental Rotation)	-↓: memory, mental speed, executive functioning, orientation, attention, language, praxis, visuospacial abilities and calculation -Most impaired= speed (44.2%); executive functioning (25.2%) - Least impairment= orientation (7.2%).

Author/Study	Sample	Time post-stroke	Measures	Outcomes
Engstad et al. (2003) *Cross-sectional*	N=223 199=IS and ICH 24=Controls	$\bar{x}$=8.9 years	MMSE, ROCF, CAT supermarket items, FAS, DS, TMT-A/B, TAPDOM, TAPDON, ANS	-Stroke=cognitively impaired, ↓motor speed, visuospacial, episodic memory, and verbal fluency
Dik et al. (2000) *Longitudinal*	N=1246 Stroke=75 Control=1171 Age 55-85 yrs	3-yrs	MMSE, RAVLT, Coding Task	-Sig↓ global cognition & processing speed
del Ser et al. (2005) *Longitudinal*	N=193 Stroke $\bar{x}$=66.8 yrs	24-mnths	SPMQ, SS-IQCODE, MMSE, Visual and Hearing reaction time, Bell Test, COWAT, WAIS-R (Picture Recognition, Word Learning, LM, BD, Similarities), BDAE, TT, Lowton-Brody Scale	-Cognitive status stable=78.2% -Cognition ↓=14% -Cognition ↑ =7.8% (↑ language)

ANS=Aachener Naming Subtest; AVLT=Auditory Verbal Learning Test; BD=Block Design; BDAE=Boston Diagnostic Aphasia Examination; BIT=Behavioural Inattention Task; BNT=Boston Naming Test; BVRT=Benton Visual Retention Test; CAMCOG=Cambridge Examination for Mental Disorders of the Elderly; CBTT=Corsi Block-Tapping Task; CDR=Cognitive Drug Research; CDT=Clock Drawing Test; CIND=Cognitive Impairment No Dementia; CFST=Colour Form Sorting Test; CST=Concept Shifting Test; COWAT=Controlled Oral Word Association Test; CST=Concept shifting Test; DAS=Dutch Aphasia Society; DS= Digit Span; DSy=Digit Symbol; GIT=Groninger Intelligence Test; ICH=Intracerebral Haemorrhage; IS=Ischemic Stroke; JOLO=Judgement of Line Orientation; K-SNAP=Kaufman Short Neuropsychological Assessment; LM=Logical Memory; NCI=No cognitive Impairment; MDRT=Mattis Dementia Rating Scale; MMSE=Mini Mental State Examination; PC=Picture Completion; PR=Picture Recognition; PS=Post-stroke; RAPM=Raven Advanced Progressive Matrices; RAVLT=Rey Auditory Verbal Learning Test; RBMT=Rivermead Behavioural Memory Test; ROCFT=Rey-Osterreith Complex Figure Test; RDT= Rosen Drawing Test; SCWT=Stroop Colour Word Test; SDMT=Symbol Digit Modalities Test; SF=Short Form; SPMSQ=Short Portable Mental Status Questionnaire; SRT=Selective Reminding Test; SS-IQCODE= Short Spanish version-Informant Questionnaire on Cognitive Decline in the Elderly; TAPNOM=Finger-Tapping Performance of Non-Dominant Hand; TAPDOM=Finger-Tapping Performance of Dominant Hand; TFP=Test of Facial Perception; TIA-Transient Ischemic Attack; TMT=Trail Making Test; TT=Token Test; VaD=Vascular Dementia; vaMCI=Vascular Mild cognitive impairment; VR=Visual Reproduction; WAB=Western Aphasia Battery; WAIS-R=Wechsler Adult Intelligence Scale-Revised; WCST=Wisconsin Card Sorting Test; WL=Word Learning; WMS-R=Wechsler Memory Scale-Revised; $\bar{x}$=Mean.

As seen in Table 1, studies have documented that cognitive impairment persists for many years and may progress to dementia over time (Alfieri et al., 2004; Ballard et al., 2003; Desmond, Moroney, Sano & Stern, 2002; Sachdev et al., 2009; Srikanth et al., 2006). Many longitudinal studies have focused on post-stroke dementia; however the specific cognitive domains most severely impacted by stroke have not been adequately investigated (Desmond et al., 2002). Although extensive neuropsychological batteries have been used by some studies, they have not provided a detailed neuropsychological profile of the participants (Serrano Domingo, Rodrìguez-Garcia, Castro & del Ser, 2007). Furthermore, such studies also suffer from methodological limitations such as random sample selection, small sample size and exclusion of very old patients or those with serious clinical condition (Alfieri et al., 2004; Sachdev et al., 2009) or aphasia; and considerable loss to follow-up (Srikanth et al., 2006). Also, testing time was not consistent in long-term studies, varying from 0-43 years (Engstad et al., 2003); thus not providing a clear picture of the course of recovery or of recovery at a given time.

While the research examined here has shown that post-stroke cognitive impairment progresses over time, studies have also reported that patients with cognitive deficits in the acute phase (1-6 months) after stroke showed global improvement at follow-up assessments 15-24 months later (Ballard et al., 2003; Rasquin et al., 2005; del Ser et al., 2005); indicating that long-term improvement in generalized cognitive function does take place. Studies report that those who show cognitive improvement over time are typically younger, more educated, with less medial atrophy on CT scan, while those who decline tend to be significantly older, illiterate, had more frequent drug intake and are more cognitively impaired before stroke (Sachdev et al., 2004; del Ser et al., 2005).

Table 2

Neuropsychological outcomes using battery of tests > 1 year post-SAH

Author/study	Sample	Time post-SAH	Measures	Outcome
Haug et al. (2009) *Prospective study*	N=46 Aneurysmal SAH (ACoA, MCA only) $\bar{x}$ Age =53 yrs	1-yr	CVLT-II, CVMT, Grooved Pegboard, WAIS-III (DS, DSy), WASI-R (Vocabulary, Similarities, BD, Matrices), D-KEFS (verbal fluency, design fluency, colour word interference, TMT, sorting test)	-mild to moderate↓: Memory, motor functioning, attention, psycho-motor, intellectual, executive functioning -Better functioning in MCA sample
Egge et al. (2005) *Prospective study*	N=42 SAH $\bar{x}$= 28-69 yrs	1-yr	HRTB (SRT, TMT-A/B), WMS-R (verbal and visual paired associates), COWAT, WCST (computerised), Grooved Pegboard	-normal cognition: 7% -93% ↓: memory, information processing, cognitive flexibility, problem solving
Ogden et al. (1990) *Prospective study*	N= 16 SAH $\bar{x}$ =50 yrs	5-yrs	NART, WAIS-R (Vocabulary, comprehension, BD, DS, DSy), WMS (LM, VR, PA), RCFT, WCST (modified), TT, visuospatial neglect tests, MTDDA, EHT	-Cognition ↓ (mild-severe): memory, visuospatial, executive functioning -37.5% ↓ in < 2 domains -25% ↓ in 3-4 domains -37.5% ↓ in > 5 domains
Hütter and Gilsbach (1993) *Retrospective study*	N= 31 Aneurysmal SAH $\bar{x}$=42.9 yrs	$\bar{x}$=3-yrs	WIF, d2 (concentration), LPS-LD, LPS-RV, IST, BT, FWIT, TT, WD, HWIE	-Cognitive ↓ =7%-62% -54% ↓ in >3 domains -25% ↓ in 5-7 domains -Maximum ↓: attention followed by memory -No ↓ : general intelligence
Hütter et al. (1995) *Retrospective study*	N=58 SAH $\bar{x}$= 46 yrs	3-yrs	WIF, d2 (concentration), LPS-LD, LPS-RV, IST, BT, FWIT, TT, WD, HWIE	- 42% cognitively ↓ -Maximum ↓: visual short term memory (46%), attention (65%), verbal long-term memory (28%), concentration (13%), & language (11%)

Author/Study	Sample	Time post-SAH	Measures	Outcomes
Alfieri et al. (2008) *Prospective study*	N=30 SAH $\bar{x}$=41.7 years Non-Aneurysmal SAH	5-yrs	ZVNC, D2TA, WAIS-III (DS), WASI (BD, MR), RAVLT, BVRT, RRT, TLT, ROCF, WCST (modified)	-Sig ↓ in all cognitive domains - ↓ in attention and memory
Cheng et al. (2006) *Prospective study*	N=37 Aneurysmal SAH $\bar{x}$ =46.2	1 day-5-yrs	WAIS-R (Arithmetic, DS, DSy), WMS-R (PC, VR), COWAT, TMT, MMSE	- 29.7% no cognition ↓ -70.3% ↓: attention, memory, executive functions -37.8% -1 deficit, 32.4% > 2 deficits
Wong et al (2009) *Prospective study*	N=40 SAH	9-27-mnths	FAB, RBMT, ADAS-Cog	-global cognition ↓ in 35% - ↓ executive functioning 27.5% - ↓ memory in 43.6%
Madureira et al. (2000) *Prospective study*	N=22 Perimesencephalic SAH $\bar{x}$=54 years	39-mnths	MMSE, LC, WFT, Alternating Sequences, Proverb Interpretation, RPM, WMS, CD, WAIS (BD, Information), Simple Arithmetic Operations, temporal, spatial, personal orientation test	-72% ↓ in at least 1 domain -Most ↓ visual memory (39%), abstract reasoning (33%)
Haug et al. (2010) *Prospective study*	N=26 Aneurysmal SAH $\bar{x}$ =49 yrs	1-yr	GPT, WAIS-III (DSy, DS), D-KEFS (CWIT, TMT, COWAT, DFT, CWIT), CVLT, CVLT-II, ROCFT, WASI-R (vocabulary, Similarities, BD, Matrices)	-Mild ↓ (53%)= psychomotor, executive, attention & intellectual function -Moderate-severe ↓ (47%) =motor, verbal and visual memory
Vilkki et al (2004) *Prospective study*	N=138 Aneurysmal SAH $\bar{x}$=49.7 yrs	1-yr	WAIS-R (DS, Similarities, BD), CFIT, Face Searching, Face Recognition, MBVRT, Word list learning	-↓ cognition: attention, general intelligence, & memory
Hütter & Gilsbach (1992) *Prospective study*	N=48	2.8-yrs	WIP, d2, LPS-LD, LPS-RV, IST-memory, Benton-test, Stroop Test, TT, WD	-↓ cognition: concentration, logical thinking, spatial abilities, language, memory, attention

Author/Study	Sample	Time post-SAH	Measures	Outcomes
Change over time				
Ogden et al. (1993) *Prospective study*	N=89 SAH $\bar{x}$=16-69 yrs	12 wks & 12-mnths	WAIS-R (Vocabulary, Similarities, Comprehension, DS, PC, BD, PA, DSy), RMT (Words, Faces), WMS (Associate Learning, LM), ROCFT, OSR (Form I), TT, NART, GNT, MTDDA, VNT, TMT, WCST (modified)	-12 mths=↑ across all domains -↓ attention, visuospatial construction, memory, psycho-motor speed, mental flexibility -Maximum ↓=verbal cognition & memory
Koivisto et al. (2000) *Longitudinal*	N= 109 Aneurysmal SAH	3 mnths & 12-mnths	WAIS-R (Similarities, Vocabulary, DS, PC, BD), BNT, COWAT, WMS (LM, VR), ROCFT, Stroop Test, TMT	-12 mnths: ↓ performance in all domains
Haug et al. (2007) *Prospective study*	N=32 Aneurysmal SAH $\bar{x}$=54 yrs	3,6 & 12-mnths	GPT, WAIS-III (DS), CVLT-II, ROCFT, D-KEFS (DS), WASI-R (Similarities, BD), BNT-short version	-Cognition still ↓ at 12 mths - At 3 mnths - 6 mths= ↑ Motor functioning -At 12 mnths =↓ Motor functioning ↑ verbal memory, - ↓ verbal intelligence, aphasia, psychomotor & executive functioning, visuospatial -No↑ in attention
Samra et al. (2007) *Longitudinal*	N=185 SAH =45 Controls	3, 9 & 15-mnths	BVRT-R, COWAT, GPT, ROCFT, TMT, NART-R	SAH group: Cognitive occurred over time -cognition ↓:3-months=35.7%; 9-months= 25.8%; 15-months= 23.3%
Controlled comparisons				
Powell et al. (2004) *Cross sectional*	N=49 SAH =49 Controls $\bar{x}$=46 yrs	18-mnths	DS, COWAT, & test of prose recall	-18-mths: ↓ attention, verbal fluency & memory
Ravnik et al. (2006) *Cross sectional*	N=10 SAH =10 Controls $\bar{x}$= 55 yrs	41-mnths	WCST, TLT, WMAS (verbal span, list acquisition, list recall, visual span, faces, visual reproduction), Stroop Test, D2TA, CTT	-Mild degree of long-term cognitive ↓ -Maximum ↓:memory & executive functioning

Author/Study	Sample	Time post-SAH	Measures	Outcomes
Germanò et al. (1998) *Cross sectional*	N=20 SAH =20 Controls $\bar{x}$= 45.4 years	1-yr	WMS-R (LM, VPA, VDR, DS), TPT, WAIS-R (DS), TT, COWAT, BVRT-revised	-No sig cognitive differences in SAH and controls
Hadjivassiliou et al. (2001) *Prospective*	N= 80 SAH =31 Controls	1-yr	NART, WAIS-R (Vocabulary, DS, Arithmetic, PA, BD), story recall, face recognition, drawing complex figure-check if ROCFT, CANTB (CBS, ID/ED test, TLT), BNT, test of form discrimination, TMT	-Sig ↓: IQ, memory, executive functions, language, visual perception, attention, as compared to controls
Mavaddat et al. (1999) *Cross sectional*	N= 47 SAH =20 Control $\bar{x}$=51.5 yrs Aneurysmal	6-24 mnths	MMSE, CANTB	-83% SAH cognition ↓: verbal fluency, pattern recognition and spatial working memory

Note: ACoA=Anterior Communicating Artery; ADAS-cog=Cognitive subscale of Alzhiemer Disease Assessment Scale; BD=Block Design; BT=Benton Test; BNT=Boston Naming Test; BVRT-R=Boston Visual Retention Test-Revised; CANTAB=Cambridge Automated Neuropsychological Battery; CD=Clock Drawing; COWAT=Controlled Oral Word Association Test; CTT=Color Trail Test; CVLT-II=California Verbal Learning Test; CWIT=Color Word Interference Test; D2TA=D2 Test of Attention; DS=Digit Span; DSy=Digit Symbol; D-KEFS=Delis-Kaplan Executive Functional System; EHT=Edinburgh Handedness Test; FAB=Frontal Assessment Battery; FTT=Finger-Tapping Test; GPT=Grooved Pegboard Test; HWIE=Hamburg Wechsler Intelligence Examination; ID/ED shift test=Intradimensional/Extradimensional Shift Test; IST=Intelligenz Struktur Test; IQ=Intelligent Quotient; LC=Letter Cancellation; LM=Logical memory; LPS-LD=Leistungsprüfsystem-Lugisches Denken; LPS-RV=Leistungsprüfsystem Röumliche Vorstellungsvermögen; MBVRT=Modified Benton Visual Retention Test; MCA= Middle Cerebral Artery; MMSE=Mini Mental State Examination; MWCST=Modified Wisconsin Card-Sorting Test; PA=Picture Arrangements; PC=Picture Completion; RBMT=Rivermead Behavioural Memory Test; ROCFT=Rey-Osterreith Complex Figure Test; RPM=Ravens Progressive Matrices; RRT=Rey Recovery Test; NART=National Adult Reading Test; PA=Paired associates; RMT=Recognition Memory Test; TLT= Tower of London Test; TMT-Trail Making Test; TT=Token Test; VNT= Visual Neglect Test; VPA=Visual Pared Associates; VR=Visual Reproduction; WAIS-R=Wechsler Adult Intelligence Scale-Revised; WASI-R= Wechsler Abbreviated Scale of Intelligence-Revised; WD= Weiner Determinationsgerät; WIF=Wechsler Intelligenz Prüfung; WMS-R= Wechsler Memory Scale-Revised; WCST= Wisconsin Card-Sorting Test

SAH: Cognitive impairment has been reported in up to 83% of SAH survivors (Mavaddat, Sahakian, Hutchinson & Kirkpatrick, 1999). Despite good neurological outcome, persistent cognitive consequences may manifest years after SAH (Mavaddat et al., 1999), impacting psychosocial functioning (Ravnik et al., 2006). Even though SAH survivors experience ongoing problems, recovery of cognitive function is a continuous process with different time courses for different areas of impairment (Haug et al. 2007; Koivisto et al. 2000; Ogden, Mee & Henning, 1993), which involves developing appropriate coping skills and having a positive attitude towards recovery (Ogden, Utley & Mee, 1997). For example, Frazer, Ahuja, Watkins and Cipolotti (2007) examined the cognitive outcome of SAH survivors at post acute and 6-months follow-up, reporting significant improvements in intellectual functioning, memory, executive functions, and IPS. Cognitive performance of SAH patients improves across neuropsychological assessments done at 3 months and 12 months post-SAH (Koivisto et al., 2000). Studies have reported that cognitive recovery continues beyond 3-months; with a plateau starting at 9-months and lasting until 15-months (Samra et al., 2007). However, cognitive deficits are still present up to 5-years post-SAH (Cheng, Shi & Zhou, 2006). However, as compared to the acute phase of SAH, very little data is available on its long-term consequences as most studies are limited to short-term ($\leq$6-months) follow-up (Mayer et al., 2002; Uski et al., 2000) and selection bias with participants being referred to hospital and/or rehabilitation settings; and have small sample sizes (e.g., N=16; Ogden, Levin & Mee, 1990).

Since SAH outcomes are thought to change over time, studying SAH longitudinally is important for better understanding its natural course, the changing needs of survivors, and its impact on family and society. For this, a detailed neuropsychological assessment is extremely valuable. The following section reviews

long term (>1-year) neuropsychological outcomes of SAH survivors focusing on studies that used a neuropsychological battery rather than screening measures such as MMSE (Table 2). Most of the existing long-term studies on SAH exclude the very old (e.g., Huag et al., 2009; Hütter et al., 1995) and those with severe grades of SAH (e.g., Ravnik et al., 2006). There is evidence that cognitive outcomes in SAH survivors may vary due to injury characteristics such as impact of bleed, GCS, recurrent SAH and demographic variables such as age, race, ethnicity, and education (Egge et al. 2004; Haug et al., 2007; Ogden et al., 1993; Scharbrodt, Stein, Schreiber, Böker & Oertel, 2009). It is becoming increasingly clear that SAH survivors who make a good clinical recovery or do well on global outcome measures (e.g., Glasgow Outcome Scale Score=4-5) still exhibit cognitive deficits (Hadjivassiliou et al., 2001; Ogden et al., 1990; Powell et al., 2004; Ravnik et al., 2006). Mild to moderate cognitive impairment is associated with younger and more educated patients who are most likely to be functioning at higher level before the SAH (Haug et al., 2010; Powell, et al., 2004). Yet the presence and patterns of cognitive impairments in patients post-SAH varies across studies. Some investigators have found that individuals experience little or no persisting significant neuropsychological deficits accompanying uncomplicated SAH (Germanò et al., 1998; Haug et al., 2009); while others report significant impairment across cognitive domains (Alfieri et al., 2008; Hadjivassiliou et al., 2001; Haug et al., 2010; Ogden et al., 1990). The reasons for these inconsistencies are likely related to variability in samples as studies showing little or no cognitive deficits tend to exclude participants with unfavourable neurological outcome, greater age ($\geq$70 years) and/or aphasia (Germanò et al., 1998; Haug et al., 2009). Memory (verbal and non-verbal) is the most commonly examined domain, perhaps because of its known effects on everyday life, and studies have reported that it is the most frequently impaired domain post-SAH (Haug et al., 2009; Ravnik et al.,

2006; Madureira, Canhão, Guerreiro & Ferro, 2000; Wong et al., 2009).

Cognitive outcomes and functioning

In a review of the literature, Barker-Collo and Feigin (2006) have reported that neuropsychological functioning (e.g., memory, attention, visual neglect, aphasia, anosognosia) is a predictor for functional outcome after stroke. Although studies have documented that cognitive functioning predicts functional outcome in stroke patients, most of the studies have examined small samples, used brief measures such as MMSE or assessed only specific areas of neuropsychological functioning (Paolucci, Antonucci, Gialloreti, Traballesi & Lubich, 1996; Ozdemir, Birtane, Tabatabaei, Ekukulu, & Kokino, 2001). For example, in a population study (South London Stroke Register) participants (n=645) were assessed for cognitive ability using the MMSE 3-months post stroke and disability was assessed 1, 3 and 4-years post-stroke by Barthel Index (BI) and Frenchay Activity Index (FAI; Patel, Coshall, Rudd, & Wolfe, 2002; Patel et al., 2003). The results indicated that 38% of the stroke survivors were cognitively impaired (MMSE< 24) at 3-months. Case-fatality rates were higher in cognitively impaired subjects at all three time points. Based on BI and FAI scores, subjects who were cognitively impaired at 3-months post-stroke were also more disabled at all three time points. Furthermore, subjects with cognitive impairment had higher rates of being institutionalized, and visual neglect compromised the recovery process.

Jehkonen et al. (2000) followed up 50 patients and reported that visual neglect at the acute stage after stroke is the best predictor of poor functional recovery 1-year after stroke. Robertson, Ridgeway, Greenfield and Parr (1997) noted significant correlations between sustained attention capacity at 2-months and functional outcomes at 2-years post-stroke. A long-term study by van Zandvoort et al. (2005) assessed stroke survivors (n=27) at 20-months using a neuropsychological battery and functional outcome

measures (BI, Modified Rankin Scale [MRS], Short Form-36 [SF-36]). The results showed that the cognitive performance of the patients improved over time, 2/3 were completely independent in ADL (BI), and 86% (MRS) experienced none or very slight functional limitations. However, on the Visual Analogue Scale, most participants reported decreased HRQoL with reduced scores for physical functioning, role limitation due to physical functioning, general health and experienced change in health. Overall, the study did not show any significant relationship between cognitive functioning and HRQoL. However, the study had certain limitations, including selection bias (excluding severely disabled clients), and both BI and MRS showed ceiling effects. Caderfeldt, Gosman-Hedström, Pérez, Sävborg and Tarkowski (2010) assessed 45 elderly (65-91 years) patients 12-months post-stroke to examine their performance of personal activities of daily living (P-ADL) in relation to cognitive impairments measured by MMSE and a battery of neuropsychological tests. The results indicated that those with intact cognition after 12-months improved significantly on P-ADL, whereas those with impaired cognition did not improve.

Ogden et al. (1990) studied a small sample of 16 participants 5-years post SAH and noted that all the participants had "good" neurological recovery (GOS=1) with cognitive impairments ranging from mild to severe. A structured interview revealed that most participants still experienced substantial emotional and psychosocial difficulties including fatigue, and reduced leisure activities and capacity to work. Although the authors used a neuropsychological battery to assess cognitive functioning, no functional outcome measures were administered, so the relationship between cognitive and functional outcomes could not be examined. A later study noted that SAH survivors (n=232) with cognitive impairment at 1-year had worse HRQoL and lesser ability to perform instrumental and basic ADLs (Springer et al., 2009). However, this study

assessed cognitive status with the Telephone Interview for Cognitive Status which does not give a comprehensive picture of neuropsychological functioning and which may be influenced by self-report. A recent study by Haug et al. (2010) examined HRQoL (SF-36, General Health Questionnaire-28 [GHQ-28]) and cognitive functioning (neurpsychological battery) of 26 patients 1-year after severe aneurismal SAH (Hunt and Hess Grade V). The authors noted that participants with moderate to severe cognitive deficits had significantly lower scores on SF-36 domains (physical functioning, role physical, general health and role emotional) and well being (GHQ-28) as compared to population means.

Summary of neuropsychological outcomes

It has been well documented that individuals are likely to experience neuropsychological deficits years after stroke and SAH. Survivors of stroke often have significant global cognitive declines as well as impairments in some specific neuropsychological domains such as memory, language, visouperceptual, psychomotor speed, attention, and information processing speed, and executive functioning. Research shows that variables such as age, gender, injury characteristics such as location are related to the post-stroke neuropsychological functioning. Furthermore, studies have also noted a relationship between neuropsychological and functional outcomes.

Regarding SAH, similar findings have been reported with SAH-survivors experiencing myriad of cognitive problems including mild to severe deficits in memory, language, visouperceptual, psychomotor speed, attention, and information processing speed, and executive functioning as compared to matched controls. Findings indicate that post-SAH neuropsychological functioning is related to the injury characteristics, demographic variables such as age, education, gender, cognitive functioning prior to SAH and functional outcomes post-SAH. Although studies indicate that improvement of

cognitive functioning occurs over the years, time course for their recovery varies for different cognitive domains. Whilst studies have indicated that neuropsychological impairments last up to 5-years post-SAH, so far none of the studies have examined the long-term neuropsychological functioning using detailed neuropsychological test battery.

Mood and emotion

Having reviewed neurological and cognitive outcomes following stroke and SAH, this section explores mood which is another area impacted by stroke that can impact on the wider functional outcomes. In this section studies related to stroke will be reviewed with greater attention to those providing information on SAH.

Emotional problems are a common consequence of stroke that impacts its outcomes and the recovery process (Goodwin & Devanand, 2008; Haacke et al., 2006; Hedlund, Zetterling, Ronne-Engstrom, Ekselius & Carlsson 2010; Kauhanen et al., 1999; Sturm et al., 2004b; Townend et al., 2007). Post-stroke depression (PSD) and anxiety (PSA) have received the most attention (Barker-Collo, 2007; Berg, Palomäki, Lehtihalmes, Lönnqvist & Kaste, 2003; Whyte, Mulsant, Vanderbilt, Dodge & Ganguli, 2004; Wilkinson et al., 1997) and have been reported to be present for years after the acute phase (Chausson et al., 2010; Ogden et al., 1990). Although PSA will be reviewed in this section, as literature on PSA remains scarce, the main focus will be on PSD and post-SAH depression (PSAHD). The following paragraphs begin with the literature regarding the prevalence, and the correlates of these.

Prevalence rates of mood disorders post-stroke vary across studies with reported rates of PSD ranging from 9% to 22.8% at 3-months (Kauhanen et al., 1999; Kim & Choi-Kwon, 2000), 16% at 18-months (Palomaki et al., 1999), and 19% at 3-years (Åström et al., 1993). Prevalence of PSA reportedly ranges from 11-28% at 3-years post-stroke (Åström, 1996; Burvill et al., 1995). In a recent population based study, 25.8% of

stroke survivors had PSD with 3.6% experiencing severe PSD (Chausson et al., 2010). High rates of comorbidity have been reported with 85% of people with PSA also having PSD at 3-years post-stroke (Castillo, Schultz & Robinson, 1995; Castillo, Starkstein, Fedoroff & Price, 1993).

Regarding SAH, studies have shown increased rate of depression as compared to controls (Berry et al., 1997; Tidswell, Dias, Sagar, Mayes & Battersby, 1995), and moderate to severe depression and anxiety is prevalent for a long period post-SAH (Alfieri et al., 2008; Hütter et al., 1995; Powell et al., 2002). Similar to stroke, PSAHD prevalence rates vary at different time periods with 16% (anxiety) and 14% (depression) at 3-months, similar rates (anxiety-17%; depression-8.5%) at 9-months (Powell et al., 2002), and 20% (depression) and 40% (anxiety) at 16-months (Morris, Wilson & Dunn, 2004). A recent long-term study reported that 32% of participants report anxiety and 23% report depression 2 to 4-years post SAH (Visser-Meily, Rhebergsen, Rinkel, van Zandvoort & Post (2009). Discrepancies in prevalence rates may be attributed to differences in sample characteristics, assessment methods, testing time periods, and definitions of depression and anxiety across studies (Hackett, Yapa, Parag & Anderson, 2005). Furthermore, many studies suffer from methodological limitations including selection bias (e.g., excluding the young or old, severe cases, those with cognitive impairments) and assessing at inconsistent time intervals post-SAH. Although numerous studies have examined the presence of post-SAH anxiety and depression none of these are long-term or population based.

The mechanisms of PSD and anxiety remain controversial with studies suggesting a role of demographics, injury characteristics, functional outcomes, and cognitive functioning. Regarding demographic variables, the relationship between age, gender and PSD is unclear with some studies suggesting an association and others not (Berg et al.,

2003; Whyte et al., 2004). Most studies have not found an association between PSD and age (Åström et al., 1993; Van De Port et al., 2007; van Zandvoort et al., 2005; Brodaty, Withall, Altendorf & Sachdev, 2007), though some have reported that frequency of PSD increases with age (Berg et al., 2003; Carota et al., 2005; Kotila, Numminen, Waltimo & Kaste, 1998; Lindén, Blomstrad & Skoog, 2007). For gender, mixed results have also been reported, with some studies reporting no association between gender and PSD (Åström et al., 1993; Kauhanen et al., 1999; Kotila et al., 1998; Lindén et al., 2007) and others reporting higher rates in women (Andersen, Vestergaard, Ingemann-Nielsen & Lauritzen, 1995; Poynter et al., 2009; van De Port et al., 2007). Paradiso and Robinson (1998) reported that as compared to men, women are twice as frequently diagnosed with PSD, have higher frequency of left hemisphere lesions, and prior diagnosis of psychiatric and cognitive impairments; whereas men have more impairment in ADL and social functioning. For SAH, no relationship between age, gender and PSAHD has been noted at 3-months or 6-years (Madureira, et al., 2000; Powell et al, 2004).

Although studies have noted that PSD is associated with lesion location, severity (Åström et al., 1993; Berg et al., 2003; Kotila et al., 1998; van de Port et al., 2007), some studies have suggested that no relationship exists (Brodaty et al., 2007; Carota et al., 2005; Haug et al., 2009). Åström et al. (1993) found that left hemisphere lesion was related to PSD from acute stage until 3-months, however no such relationship was noted 3-years post-stroke. In long-term studies, no association has been found between PSAHD or anxiety and type of SAH (aneurymal vs non-aneurysmal; anterior communicating artery aneurysm vs other) (Hütter et al., 1995), severity of haemorrhage, or time between admission and surgery as assessed by standardised measures (Morris et al., 2004). However, in terms of severity, Kreitschmann-Andermahr et al. (2007) reported that PSAHD was significantly related to GOS scores.

Numerous studies have reported a significant association between PSAHD and cognitive impairments (Andersen et al., 1995; Berg et al., 2003; Mayer et al., 2002) with depressed individuals experiencing more cognitive deficits (House, Dennis, Warlow, Hawton & Molyneux, 1990; Maduriera et al., 2000; Rasquin et al., 2005; Srikanth et al., 2006). A strong correlation has been noted between depression and cognitive deficits for patients with left hemisphere stroke (House et al., 1990). After 12-months, the domains most likely to be impaired in stroke related depression were memory, problem-solving, attention and psychomotor speed (Kauhanen et al., 1999; Rasquin et al., 2005). However, if depression is alleviated by treatment, cognitive functioning improves considerably and this is maintained over 2-years (Narushima, Chan, Kosier & Robinson, 2003), whereas no such improvement was noted in non-depressed patients. Cognitive complaints (mental slowness, memory, attention) are common post-SAH and are strongly associated with depression (Passier et al., 2010). Alfieri et al. (2008) note that depression is associated with reduced attention in SAH survivors at 1-year.

Besides injury characteristics, demographic variables and cognitive functioning, a plethora of studies have confirmed the association between functional impairment and depression post-stroke and SAH (Berg et al., 2003; Brodaty et al., 2007; Carota et al., 2005; Goodwin & Devanand, 2008; Sturm et al., 2004a; Townend et al., 2007; Willey et al., 2010). Literature suggests that post-stroke, depressed patients are more dependent in activities of daily living (ADLs), have significantly greater impairments in physical functioning, are more severely handicapped, and have poorer quality of life as compared to non-depressed patients (Kauhanen et al., 1999; Naess, Lunde, Brogger & Waje-Andreassen, 2010; Sturm et al., 2004a; Strum et al., 2004b; van de Port et al., 2007; van Zandvoort et al., 2005; Visser-Meily et al., 2009; Whyte et al., 2004). Furthermore, remission of depression in the early months post-stroke leads to improved ADLs

(Chemerinski, Robinson & Kosier, 2001); indicating that early treatment of PSD is beneficial to overall physical recovery (Robinson, 2003). On the other hand, Åström et al. (1993) did not find any relationship between ADLs and depression at 3-years post-stroke. Longitudinal examination has shown that depression at 9-months post-SAH is predicted by functioning impairments in the acute stage (Powell et al., 2002). Depression and anxiety after SAH have also been associated with social restriction and inability to return to work (Åström et al., 1993; Morris et al., 2004).

Thus, considerable research documents that emotional problems (i.e., depression and anxiety) are common long-lasting sequelae of stroke and impacts an individual's social life and functioning. Furthermore, a relationship between emotional problems and impaired neuropsychological functioning has also been noted. The next section reviews the literature examining the long-term functional outcomes after stroke, with a focus on SAH.

Long term activity and participation in stroke and SAH survivors:

Although a relationship between impairment, disability, handicap and quality of life has been documented (Hop, Rinkel, Algra & van Gijn, 1998) these relationships are very complex. Among survivors of SAH who make a good functional recovery and are independent in ADLs, many experience psychosocial deficits and emotional changes (Hop et al., 1998; Salmond et al., 2006; Wermer, Kool, Albrecht & Rinkel, 2007).

Commonly used measures to assess activity/disability post-stroke include the Barthel Index (BI; Granger, Dewis, Peters, Sherwood & Barrett, 1979), Frenchay Activity Index (FAI; Holbrook & Skilbeck, 1983), Modified Rankin Scale (MRS; Bamford, Sandercock, Warlow, & Slattery, 1989), Rivermead Mobility Index (RMI; Whiting & Lincoln, 1980), Nottingham 10-point Activities of Daily Living (Ebrahim, Nouri & Barer, 1985), and Northwick Park ADL Index (Sheikh et al., 1979). The most frequently

used measures to assess participation (handicap and HRQoL) include the London Handicap Scale (LHS; Harwood, Rogers, Dickinson & Ebrahim, 1994) and the Short Form-36 (SF-36; Ware & Sherbourne, 1992) and Sickness Impact Profile Scale (SIP; Damiano, 1996). HRQoL measures are typically multidimensional and capture an array of difficulties experienced by stroke survivors including physical functioning, general mental health, and role limitations due to physical or mental health.

While numerous studies have reported that disability and handicap persist for many years post-stroke (Patel et al., 2006), only a few long-term population-based studies have examined these long-term outcomes (Bonita, Solomon & Broad, 1997; Chausson et al., 2010; Dhamoon et al., 2009; Hankey et al., 2002; Taub et al., 1994). An earlier long-term population-based study conducted in Auckland (1991-1992) reported that almost 55% of stroke survivors do not recover completely at 3-years and of these approximately 33% require assistance in at least one activity of self-care. Moreover, men (50%) are more likely to make a complete recovery at 3-years than women (37%; Bonita et al., 1997). In a later 6-year follow up, Hackett, Duncan, Anderson, Broad and Bonita (2000) examined basic ADLs and HRQoL using the Short Form-36 (SF-36). Of 639 stroke survivors (compared with 310 general population controls), 61% had incomplete recovery, 23% were living in institutional care, 42% were dependent in $\geq$1 ADLs, while their perceived mental health was as good as the general population. Compared to controls, stroke survivors scored significantly lower on physical functioning, general health and role limitations due to physical and emotional problems on the SF-36, despite the majority of stroke survivors living at home (77%). In a recent study Chausson et al. (2010) reported that after 5-years, 66.4% stroke survivors were functionally independent, while approximately 25% were completely dependent on family members for basic ADLs. Dhamoon et al. (2009) used the BI to examine functional outcomes of 535

patients in Northern Manhattan and observed an annual decline in functional status (OR= 0.91; 95% CI, 0.84 to 0.99) for those who had a favourable BI (≥95) at 6-months follow-up and the maximum decline occurred 3-years after stroke. While the above provides ample evidence of continuing long-term functional difficulties post-stroke, no separate data on SAH are provided, nor are these examined in relation to other aspects of the ICF, such as neuropsychological outcomes and mood.

In the Perth community stroke study, Hankey et al. (2002) found that of 370 cases of first ever stroke 55% (n=152) survived to 5-years. Of these, 14% were institutionalized and 36% were disabled 5-years post-stroke. The variables predicting death or disability at 5-years included increasing age, recurrent stroke, hemiparesis and baseline disability assessed by BI (score <20/20). Long-term outcomes of stroke were examined in North East Melbourne Stroke Incidence Study (NEMESIS) at 1-and 2-years post-stroke (Sturm et al., 2002; Sturm et al., 2004a; Sturm et al, 2004b). At 1-year post stroke, physical independence and occupation were the most affected domains of handicap and only half the variance in handicap was due to disability. Those who reported complete recovery were much less handicapped as compared to those with incomplete recovery. At 2-years, patients with recurrent stroke had worse HRQoL as compared to those without a further stroke. Independent determinants of HRQoL in survivors included handicap, physical impairment, anxiety and depression, disability, institutionalization, dementia and increased age. At stroke onset, factors such as age, female gender, increased stroke severity, neglect, and low socio-economic status predicted worse HRQoL at 2-years post-stroke. In a 5-year follow-up of the same sample a substantial proportion of survivors (20%) suffered from poor HRQoL and baseline predictors of low HRQoL were increasing age, low socio economic status and increased stroke severity (Paul et al., 2005). Unfortunately, the NEMESIS study, however excluded SAH patients.

In another population base study in South London (1995-1997) stroke survivors were examined at 1-(n=490) and 3-years (n=342) for the levels of disability (BI), handicap (FAI) and HRQoL (SF-36). At 1-year post-stroke 40% of the patients were independent on ADLs which decreased to 34% at 3-years, out of whom 26% were moderately or severely disabled (BI≤14). At both 1-and 3-years approximately 50% of the survivors were handicapped. In terms of activity, 16% were active at 1-year which reduced to 14% at 3-years. On the SF-36, at both time periods, the stroke survivors had lower scores on domains related to physical health as compared to domains related to mental health. This study also examined the relationship between disability, handicap and HRQoL and found a graded positive relationship between all SF-36 domains BI and FAI. However, this study had missing data for dysphasic or confused participants and did not consider the effects of cognitive impairment on disability, handicap or HRQoL (Patel et al., 2006).

In functional outcome studies of SAH survivors, patients often complain of irritability, personality change, loss of interest, emotional disturbances (Meyer et al., 2010; Ljunggren, Sonesson, Säveland & Brandt, 1985; Ogden et al., 1994; Powell et al., 2002) and a diminished capacity for planning and decision making (Salmond et al., 2006). In a retrospective study, Hütter et al. (1995) examined 58 patients with good GOS (GOS=4-5) in terms of their HRQoL at 1-5 years after SAH. It was found that even 1-5 years after SAH, the patients reported substantially reduced HRQoL in terms of motivation, interests, mental capacity, free-time activities, social relationships, concentration, fine-motor coordination and sleep. Furthermore, these patients reported reduced life-satisfaction, and increased emotional lability along with negative job consequences (e.g., job loss, demotion. Similarly, a recent study noted that 53% of patients experienced a favourable outcome after SAH (GOS=5) approximately 5 years (±

2 years) after SAH. However, all the participants scored low on all the SF-36 domains as compared to a cohort of healthy controls. Also, most of the patients with recurrent haemorrhage had significantly lower scores on physical functioning (Scharbrodt et al., 2009). Most of the functional outcome studies have been done on aneurysmal SAH. A recent long-term (mean time=32 months) study on survivors of non-aneurysmal SAH (n=26) reported a good neurological recovery (GOS=4 or 5) and a BI of >90, yet found reduced scores on SF-36 domains of physical functioning, vitality, and general health (Beseoglu, Pannes, Steiger & Hänggi, 2010).

While, considerable research has been done to study the functional outcomes (especially disability and neurological) of SAH survivors, very few medium to long-term population based studies have examined the disability, handicap and health related outcomes in survivors of SAH (Ogden et al., 1997). An international population based study conducted in Australia and New Zealand (1995-1998), reported that many SAH survivors continue to experience reductions in HRQoL (Hackett & Anderson, 2000). The authors noted that out of 432 cases of SAH, 56% were alive at 1-year follow up, with 89% of those living in their own home, though 46% reported incomplete recovery. HRQoL was assessed by SF-36 and as compared to the age and sex-adjusted Australian population norms; HRQoL was significantly lower for SAH cases in areas of role limitations that result from physical problems. Furthermore, participants reported ongoing problems with memory (50%), mood (39%), speech (14%) and ADL (10%). Thus, in addition to having neuropsychological impairments many of the SAH survivors report poor HRQoL (Beseoglu et al., 2010; Haug et al., 2010; Mocco et al., 2006). However these studies have some drawbacks such as follow-up limited to 1 year post-SAH or large variation of follow-up time, small sample size and using only a telephonic interview for assessing cognitive status.

A cross-sectional study examined the effect of psychological symptoms and personality characteristics on HRQoL (assessed by Stroke Specific Quality of Life Scale) of 141 participants 2-4 years post SAH and noted good physical functioning but problems in the emotional and social domains. Reduced HRQoL was strongly related to mood problems, fatigue, cognitive problems, neuroticism and passive coping style (Visser-Meily et al., 2009). However, in this study the authors used only a self-report measure for cognitive functioning and, it was not population-based. Also there was a 2-year range when they were assessed post-SAH. Thus, studies have suggested that severity of post-stroke mood and cognitive deficits has predictive value for functional status and level of supervision required at discharge (Patel et al., 2002; Sacri & Kos, 2002).

Numerous other studies have identified prognostic factors for post-stroke and SAH functional outcomes, including severity of neurological deficits at admission, increasing age, gender, previous stroke, pre-stroke disability, recurrent stroke, urinary continence, level of social support, and presence of hemipresis (Fukuda, Kanda, Kamide, Akutsu, & Sakai, 2009; Hankey et al., 2002; Kwakkel, Wagenaar, Kollen & Lankhorst, 1996; Naess, Nyland, Thomassen, Aarseth & Myhr, 2004; Scharbrodt et al., 2009; Vilkki et al., 2004). However, there is conflicting evidence for the predictive value of these variables. In a meta-analysis of the most frequently examined variables associated with HRQoL, Nobel and Schenk (2010) noted that only physical disability has a notable effect on HRQoL of SAH survivors.

Summary of functional outcomes following stroke and SAH

As noted above, there is ample literature that suggests that disability and handicap are common outcomes after stroke and SAH. Furthermore, in spite of good clinical recovery survivors often report reduced HRQoL. The functional outcomes vary from mild to severe difficulties and may persist for years after the incident of stroke or SAH.

Findings have reported that age, gender, recurrent stroke, and neurological status at the time of admission are related to post-stroke functional outcomes. Furthermore, studies have reported that post-stroke and SAH functional outcomes are related to emotional wellbeing and deficits in cognitive functioning.

However, none of the population based long-term studies examined above have examined the relationship between post-SAH cognitive and functional outcomes. Studying relationship between various functional outcomes may help in planning of rational and cost-effective interventions in the settings of limited resources (Sturm et al., 2002). Furthermore, to date there is no population-based study published on relationships between various long-term neuropsychological impairments (e.g. memory, language, visuoperceptual reasoning, processing speed, and executive functioning) and functional outcomes (in terms of disability, handicap, and quality of life) in survivors of SAH as well as on the frequency and prognostic factors of these. Lastly, there aren't any population-based studies comparing neuropsychological and functional outcomes of SAH survivors with those of controls matched on age, gender, or ethnicity.

PURPOSE

Following from the above, the purpose of this study was to examine the neuropsychological and functional outcomes of SAH survivors. As mentioned earlier, to date none of the population based studies have examined the long-term outcomes of SAH survivors and compared them with controls matched on age, gender and ethnicity. This study includes long-term (5-years) survivors of SAH aged 29- 84 years; and a group of healthy controls from general population matched on age, gender and ethnicity. Five hypotheses are examined in the current study. First, it was hypothesised that SAH-survivors would perform poorly on measures of neuropsychological functioning as compared to the matched controls. Second, it was expected that the SAH-group would

perform poorly on measures of functional outcomes (i.e., disability, handicap and health related quality of life. Thirdly, it was anticipated that the SAH-group would have poorer performance on measures of emotional and psychological well-being. Fourthly, it was hypothesised that there would be a change over time (i.e., from base line to 5-years post-SAH) on functional outcomes and the previous functional outcomes will be related to current functioning. Finally, it was expected that a relationship would exist between neuropsychological and functional outcomes of SAH-survivors.

CHAPTER II

METHOD

CONTEXT OF STUDY

This is a prospective longitudinal study of long term (5-6 years) neuropsychological and functional outcome of SAH survivors. The study sourced the participants and used the existing baseline, 28-day and 6-month follow-up data from the population based stroke incidence study carried out in Auckland [Auckland Regional Community Stroke (ARCOS) study] as this provided a unique opportunity to look at the long-term outcomes in a population based sample. The ARCOS study used a prospective population based register to ascertain all cases of acute new or recurrent stroke that occurred among adults in the "usually resident" population of Auckland during a 12-month period from, March 1, 2002 to February 28, 2003. Methods for ARCOSS are thoroughly described elsewhere (e.g., Anderson et al., 2005; Feigin et al., 2006).

PARTICIPANTS

Figure 3 presents a summary of SAH sample recruitment. Potential participants included survivors of subarachnoid haemorrhage (SAH) (n=96) who were previously enrolled in the ARCOS (2002-2003) study; were alive and had agreed to be contacted for future studies at the 6-month follow up (n=37). Participants were given a Participant Information Sheet (PIS) which provided them with the information and rationale about the study, explanation regarding their role in the study with an opportunity given to them to withdraw from the study at any time. Signed informed consent was obtained from those who agreed to participate (or their representatives) as per regulatory and legal requirements.

As seen in Figure 3, from the 37 SAH participants in ARCOSS who initially agreed to be contacted, 2 refused to take part in the study, 7 did not provide a written

consent and 1 was lost to follow-up. Most of the SAH participants who agreed to participate self-identified as New Zealand Europeans (n= 20, 74 %), while 3 (11%) identified themselves as Maori, 2 (7%) self-identified as Asian, and 2 (7%) self-identified as Pacific islanders. For the purpose of data analysis, due to the small sample size the participants were grouped together as European or 'other'. English was the first language for 23 (85%) of SAH participants. The age range of the participants at the time of the assessment was 29-84 years with the mean age of 62.22 years for the SAH group.

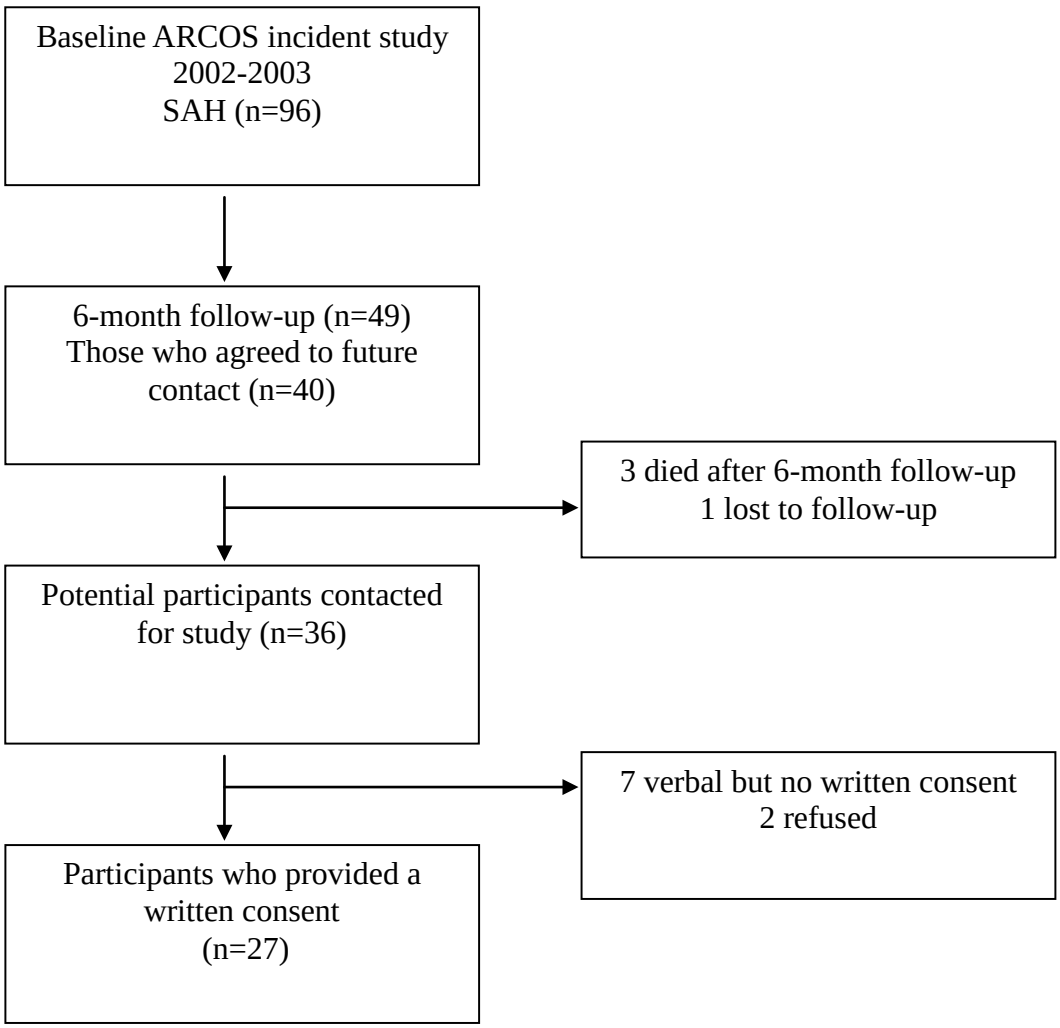

Figure 3

Summary of sample recruitment

Sensitivity analyses were conducted to contrast participants who were included (n=27) and those who were alive but not included (n=9) to ensure demographics of the present sample did not differ from the wider group. The analyses indicated that the groups did not differ in terms of age, gender, ethnicity and baseline BI ($p > .05$). A similar analysis was conducted to determine if the sample (n =27) differed significantly from the 96 SAH cases initially identified in the ARCOS study who were not included in this study (n=69). The analysis indicated that the groups did not differ significantly on age, gender and ethnicity ($p >. 05$). However the groups differed significantly on baseline BI with the excluded participants more dependent than those included in the present study.

Control participants were matched to the SAH sample on age (within 1-year), gender and ethnicity, which have been shown to impact on neuropsychological test performance (Lezak et al., 2004; Strauss, Sherman & Spreen, 2006). Although consideration was given to matching the samples on education as well, matching on four variables would have meant significant extension to recruitment time lines. In order that education could still be considered, information regarding tertiary education was collected. Potential control participants who were extremely unwell; had a neurological history, cognitive disability (i.e. stroke, traumatic brain injury, other neurological condition, mental health diagnosis); or had a history of substance abuse were excluded as these conditions would potentially affect their performance. Unfortunately one participant match could not be found who matched on age, gender and ethnicity so the final control group consisted of 26 participants.

Table 3 shows the demographic details of SAH and control groups. As can be seen from Table 3, the groups were matched on age (within 1-year), gender and ethnicity and English was the first language for equal proportion of participants in each group. For

the SAH group, the assessment time since the SAH incidence ranged from 4.11 years to 6 years with mean years and SD of 5.1 and .53 respectively. The variability in the assessment time was due to difficulty in tracing and contacting some of the participants (i.e., participants moving without leaving a forwarding address, away on a holiday, or busy due to other commitments. As compared to the SAH group, the control group was more likely to be married and living in their own home with others (such as family, friends). SAH group had lesser proportion of participants with tertiary qualifications. The current work situation for the participants varied in both the groups with higher proportion of control group participants employed full time. Both the groups were significantly different on the depression symptomatology where the SAH group a higher proportion (29.6%) met the GDS-SF depression criteria, whereas none of participants in the control group met the GDS-SF criteria of depression.

Although the SAH and control group matched on age, gender and ethnicity in order to check for equivalence across groups, comparisons were made between the groups. Analysis of variance (ANOVA) was performed with SAH and contro group as grouping variable and age as dependent variable. As seen in Table 3 no significant differences were found between both the groups on age. Chi square test were conducted to ensure equivalence between the groups (i.e., SAH and control) for gender, marital status, having English as first language, living arrangements, education and ethnicity. The results indicate that whilst the SAH and control group differed significantly on depression, dwelling place and marital status, no difference between the groups was observed in terms of gender, ethnicity, education, living arrangements or having English as first language.

Table 3

Demographic information for the SAH group and the control group

Variable	SAH group	Control group	p-value
Age Mean years(SD)	62.22(12.57)	62.46 (12.41)	*F(1,51)= 0.759, p= .945*
Gender			
Male (%)	12 (44.4%)	11(42.3%)	$X^2(1)=0.25$,*p*=-.875
Female (%)	15 (55.6%)	15 (57.7%)	
Ethnicity			
European (%)	20(74.1%)	20(76.9%)	
Maori (%)	3(11.1%)	3(11.5%)	$X^2(3)$=-.315, p=.957
Pacific Island (%)	2(7.4%)	1(3.8%)	
Asian (%)	2(7.4%)	2(7.7%)	
Living arrangements			
Alone (%)	10 (37%)	5 (19.2%)	
With others (%)	17 (63%)	21 (80.8%)	
Dwelling place			
Rented accommodation (%)	2 (7.4%)	4 (15.4%)	
Own house (%)	15 (55.6%)	20 (76.9%)	X^2(4)=3.578,p=-.466
Retirement village or institution (%)	1 (3.7%)	2 (7.7%)	
Missing (%)	9 (33.3%)	0	
English first language			
Yes	23(85.2%)	22(84.6%)	X^2(1)=-.003, p= .954
No	4 (14.8%)	4(15.4%)	
Marital Status			
Married (%)	17(63%)	21(80.8%)	
Single (never married, separated,divorced) (%)	1(3.7%)	5 (19.2%)	X^2(2)=5.136,p=0.77
Missing (%)	9(33.3%)	0	
Further qualification after high school?			
Yes	15(55.6%)	18(69.2%)	$X^2(1)=0.27$,p=.870
No	12(44.4%)	8(30.8%)	
Work situation			
Full time paid work (%)	4(14.8%)	13(50%)	
Retired (%)	9(33.3%)	6(23.1%)	
Beneficiary (%)	2(7.4%)	0	
Homemaker (%)	1(3.7%)	2(7.7%)	
Unemployed/redundant (%)	2(7.4%)	0	
Part time paid work (%)	0	5(19.2%)	
Missing	9(33.3%)	0	
Assessment timing (post stroke)			
Mean years (SD)	5.1 (.53)	-	
Geriatric Depression Scale (GDS) Current			
Mean score (SD)	3.58(4.06)	0.96(1.15)	
GDS < 5, N (%)	18(66.7%)	26(100%)	X^2(1)=9.455,p= .002
GDS $\geq$ 5, N (%)	8(29.6%)	0	
Missing N (%)	1(3.7%)	0	
Recurrent Stroke?			
Yes (%)	2(7.4%)	-	
No (%)	24(88.9%)	-	
Missing (%)	1(3.7%)	-	
Complete recovery?[a]			
Yes (%)	14 (51.9%)	-	
No (%)	12(44.9%)	-	
Missing (%)	1(3.7%)	-	

[a]Complete recovery as assessed by Two simple questions item "Do you feel you have made a complete recovery from your stroke?"

MEASURES

Baseline Measures

Base-line assessments from the ARCOS study included participants' information about their age, sex, ethnicity, education, stroke severity (Glasgow Coma Scale; GCS), previous level of activities (Frenchay Activity Index; FAI) prior to SAH and current level of activities (Barthel Index; BI) at the time of stroke. The 28-day assessment included information regarding overall psychological health and wellness (General Health Questionnaire-28; GHQ-28) and disability (Modified Rankin Scale; MRS). At 6-months post-SAH, assessment included administration of FAI, GHQ-28 and information about quality of life (assessed by Short-Form-36; SF-36). Table 4 shows the timings of the data collected at various time periods and the section below describes each of the above mentioned measures as these were re-administered at 5-years follow-up.

5-year Follow-up

Self-administered questionnaires were used for both SAH and control group to obtain demographic information (i.e., age, gender, living arrangements, marital status, education, work situation), information about recurrent stroke, and whether they had developed dementia (See Appendix A). Participants' individual perception of recovery was evaluated by the "Two simple questions" test item "Do you feel you have made a complete recovery from your stroke"? (Dorman, Dennis & Sandercock, 2000). Measures included assessment of impairment, disability, handicap, HRQoL, mood and emotion; and neuropsychological measures related to verbal memory, visual memory, attention, language, visuorperceptual, executive functioning and speed. Appendix A contains copies of all measures used. The section below first reviews measures of impairment, disability, handicap, HRQoL and mood followed by information on measures of cognitive function.

Table 4

Timing of measures for SAH sample

Measures	Time			
	Baseline (2002-03)	28-Day (2002-03)	6-Months (2002-03)	5-years (2007-08)
Demographic Data	X			X
Stroke severity				
-Glasgow Coma Scale	X			
Functional status				
-Frenchay Activity Index	X		X	X
Disability				
-Modified Rankin Scale		X		X
-Barthel Index	X			X
-London Handicap Scale				X
Health Related Quality of life				
-Short Form-36			X	X
Mood and Depression				
-Geriatric Depression Scale-Short form				X
-General Health Questionnaire-28		X	X	X
Hodkinson Mental Test				X
Neuropsychological assessments				X

Note: Neuropsychological assessments included Block Design, Boston Naming Test, California Verbal Learning Test-II, Controlled Oral Word Association, Integrated Visual and Auditory Continuous Performance Test, Logical Memory-I and II, Matrix Reasoning, Rey-Osterrieth Figure, Trails Making Test, Visual Paired Associates I and II, Victoria Stroop Test

Impairment, Disability, Handicap and Health related quality of life measures

Glasgow Coma Scale (GCS; Teasdale & Jennett, 1974). The GCS is a neurological scale to assess the conscious state of a person following injury. The GCS score ranges from 3 to 15 where 3 indicates a deep coma or death and 15 indicates a fully conscious person. The scale comprises three test categories for eye, verbal and motor responses. The scores for these categories range from 1 (does not open eyes) to 4 (opens eyes spontaneously) for eye opening response, 1 (makes no sounds) to 5 (oriented, converses normally) for verbal response and 1(makes no movements) to 6 (obeys commands) for motor response. The GCS total score comprises of the sum of scores in the three categories.

The GCS has been used widely in stroke studies (Broderick, Brott, Duldner & Tomsick, 2003; Hijdra, van Gijn, Nagelkerke, Vermeulen, & van Crevel, 1988; Weir, Bradford & Lees, 2003) and has a high criterion validity as it is valid measure to assess post-stroke level of consciousness and predict functional outcomes (Prasad, 1996). The GCS is also reliable scale, having a high inter-rater reliability (i.e., 96.4% agreement) among experienced users (Rowley & Fielding, 1991; Prasad, 1996; Lindsay, Teasdale & Knill-Jones, 1983).

Modified Rankin Scale (MRS; Bamford et al., 1989). The MRS is a clinician-reported outcome classification scale to evaluate disability after stroke. The MRS was derived from the original Rankin Scale (Rankin, 1957), which consisted of 5 categories to assess stroke severity ranging from "no significant disability" to "severe disability". The MRS consists of 7 categories, ranging from grade 0 (no symptoms at all) to grade 5 (severe handicap), and including the new classification 6 (death). The MRS measures global disability including instrumental activities of daily living (e.g., meal preparation, shopping, handling money) and basic activities of daily living (e.g., walking, dressing, grooming) with emphasis on compromised motor function (Wolfe, Taub, Woodrow & Burney, 1991; De Haan, Limburg, Bossuyt, Van Der & Aaronson, 1995). Based on studies with stroke survivors, good outcome is defined as an MRS score of <3, and, and poor outcome as score between 3 and 6 (Sulter, Steen & De Keyser, 1999).

The MRS is one of the most commonly used outcome measures in stroke research (New & Buchbinder, 2005). It has strong test-retest and inter-rater reliability, (κ = .94 to .99; Wilson et al., 2005) and its construct validity has been affirmed in numerous stroke outcome studies which consistently showed that the location, type and extent of stroke injury are closely related to short and long-term disability on the MRS (Banks & Marotta, 2007; Lai & Duncan, 1999; Nedeltchev et al., 2005). Studies have also reported high construct validity when comparing the MRS to other measures of impairment such as the

NIHSS, Barthel Index (BI) and Glasgow Outcome scale (GOS; Tilley et al., 1996).

Barthel Index (BI; Granger et al., 1979). The BI is a measure of disability originally developed by Mahoney and Barthel (1965) and later modified by Granger et al. (1979). The BI is used to assess the patients' performance for ten activities of daily living (ADLs) associated with personal care and mobility. BI can be self-administered or completed by a clinician and takes approximately 5-10 minutes. With 5 point increments used for BI scoring, the maximum score is 100 indicating that the patient is fully independent in physical functioning. The lowest score is 0 indicating a bed ridden state. The BI can be divided into self care items (feeding, grooming, dressing, and bladder care, and toilet use), and a group related to mobility (mobility on level surfaces, chair/bed transfers, and stair climbing).

The BI is a reliable measure for stroke patients (Sulter et al., 1999). The test-retest reliability of BI is high (weighted $\kappa = 0.98$), as is its inter-rater reliability (weighted $\kappa \geq 0.88$; Wolfe et al., 1991). The BI is also considered a valid prognostic tool and studies have shown that recovery in stroke patients is strongly related to the early BI scores (Huybrechts & Caro, 2007).

Frenchay Activity Index (FAI; Holbrook & Skilbeck, 1983). The FAI is used to assess functional status, focusing on extended activities of daily living. It may be self-administered or administered by interview (Holbrook & Skilbeck, 1983; Turnbull et al., 2000). The FAI comprises 15 multiple choice items covering three domains: domestic chores, leisure/work, and outdoor activities. Each item is related to an activity requiring decision making and organisation (e.g., "in the last three months how often did you undertake preparing meals?"). The participant is required to respond based on the frequency with which he/she undertakes a particular activity (e.g., never=0, under once weekly=1, 1-2 times a week=2, most days=3). The total score ranges from 0 (inactive) to 45(active); and there are three subscale scores (domestic, leisure/ work, and outdoors)

ranging from 0-15. Subscale scores are converted into Sten scores using conversion tables (Holbrook & Skilbeck, 1983). Sten scores facilitate comparison across factors by standardizing both the mean (5.5) and standard deviation (2.0) of the scores. Given the nature of the three factors, scores for males and females are examined separately as women obtain significantly higher scores on the domestic chore factor (t=11.328, $P<$ 0.001), and from males on the leisure/work factor (t= 5.099, $P<0.001$). The FAI is a highly reliable tool for differentiating functional status between pre-stroke, post-stroke and control groups (Schuling, de Haan, Limburg & Goenier, 2008) with reliability of unweighted scores ranging from 0.78 to 0.87. The FAI's construct validity is supported by high correlations with the FAI, BI and Sickness Impact Profile (Schuling et al., 1992). The FAI has been well validated in non-stroke (Trunbull et al., 2000) and stroke populations to assess long-term functional outcomes (Patel et al., 2006)**.**

London Handicap Scale (LHS; Harwood et al., 1994). The LHS is a self-rating scale used to assess functional ability, including in stroke survivors (Sturm et al., 2002) and can be completed in approximately 10 minutes. It measures six domains within the WHO definition of handicap (WHO, 1980): mobility, physical independence, occupation, social integration, orientation (awareness), and economic self-sufficiency. Each domain has six hierarchically arranged descriptions, in increasing order of handicap, and the participant is required to choose the one that best applies to him/her. Thus, handicap within each domain is measured on a 6 point scale, which can have a positive or negative weighting. For example, in physical independence, 'no impairment' has a weighting of 0.102 and the most severe impairment has a rating of -0.061. A final overall score is calculated by adding scores across all 6 domains and adding that score to 0.456. Where, the sum of all “most severe disadvantage values” is -0.456 which when added to 0.456 gives 0.00 and the sum of all “no disadvantage” values is 0.544 which when added to 0.456 gives 1.00. The LHS is well validated in New Zealand stroke survivors (Ackerley,

Gordon, Elston, Crawford & McPherson, 2009), and has an internal reliability of 0.83 (Jenkinson, Mant, Carter, Wade & Winner, 2000); and a high reliability coefficient of 0.91 (Harwood, Gompertz & Ebrahim, 1994).

Short Form 36 (SF-36; Ware & Sherbourne, 1992). The SF-36 is a multipurpose, short health survey to assess HRQoL with 36 multiple choice items that can be administered in 5 to 10 minutes. The survey is suitable for individuals aged ≥14 years and can be self-administered or completed by interview (Anderson, Laubscher & Burns, 1996). The scale assesses eight areas of quality of life: 1) physical functioning (PF); 2) role limitations due to physical health problems (RP); 3) social functioning (SF); 4) bodily pain (BP); 5) general mental health (MH); 6) role limitations due to emotional problems (RE); 7) vitality, energy and fatigue(VT); and 8) general health perceptions (GH). The number of items in each scale varies. Most items are related to participants' functioning in the past one month, and there are a variety of response formats ranging from yes/no response, through three, four, five and six category responses. An example of six category response to the item "In past 4 weeks how much of the time did you feel full of energy," the participant has a choice of 1- None of the time, to 6-All the time.

Scoring involves a two-step process. Items are assigned scores ranging from 0-100, where higher scores indicate better functioning or fewer problems. These are then added and a mean score is calculated for each of the 8 scales. Dimensions such as physical functioning, role physical, bodily pain, and role emotional, measure health status as the absence of disability (e.g., a score of 100 on pain scale = no pain limitations). Other scales such as general health, vitality, and mental health, measure both positive and negative states of health (i.e., a score of ≥50 indicates a positive state of health [not just absence of illness]). The SF-36 also yields two summary scores; the Mental Component Summary (MSC) score, which is based on scores from social functioning, vitality, role emotional and mental health, and the Physical Component Summary (PCS) score, which

is based on scores of physical functioning, bodily pain, role physical and general health. Both these scores have a mean of 50 and a standard deviation of 10.

The SF-36's internal consistency and test-retest reliability are high for each of the 8 subscales (i.e., $\geq$ .80) apart from social functioning which had the median reliability of 0.76 (Ware, Snow, Kosinski & Gandek, 1993). Reliability estimates for the summary scores (i.e., MCS and PCS) was found to be 0.90 (Ware, Kosinski & Keller, 1994). Studies on content validity indicate that it includes 8 of the most frequently measured health concepts (Ware et al., 1993). The SF-36 has been widely used measures of health related quality of life (HRQoL) across stroke populations, including Maori, Pacific and New Zealand European ethnic groups and its reliability and validity is well documented (Scott, Tobias, Sarfati & Haslett, 1999; Scott, Sarfati, Tobias & Haslett, 2000).

Mood and Emotion measures

General Health Questionnaire 28 (GHQ-28; Goldberg, 1978). The GHQ-28 is a tool used to assess overall psychological health and wellness, focusing on two major classes of phenomenon: (i) inability to continue to carry out normal "healthy" functions, and (ii) symptoms of a distressing nature (Goldberg & Williams, 1988, p.5). It is a 28 item self-administered scale designed to detect a range of psychological disorders across four specific subscales: somatic symptoms, anxiety and insomnia, social dysfunction and severe depression. As an example, one item asks 'Have you been getting scared or panicky for no good reason'. Each item has four possible responses, typically ranging from 'not at all', 'no more than usual', rather more than usual' and 'much more than usual'. Participants have to respond in regards to how their health has been, in general, over the past few weeks. Items are scored from 0 to 1 points, using the binary method where responses are scored-0,0,1,1, respectively with a total possible score on the GHQ-28 ranging from minimum, '0' to maximum, '28' (higher score indicating stress). Abnormal mood is defined as a score $\geq$5 (Goldberg & Hillier, 1979).

The GHQ-28 has been translated into 38 languages and has been used in diverse cultural groups (Goldberg & Williams, 1988). The internal consistency reliability for GHQ-28 is high (α=0.92) and the Spearman- Brown split-half reliability was found to be 0.84 (Kılıç et al., 1997). The GHQ-28 has both content validity and construct validity. There is high correlation (*r*= .70) between GHQ-28 scores and a psychiatric assessment (Goldberg & Williams, 1988). The GHQ-28 has a sensitivity of 84% and specificity of 82% (Goldberg & Williams, 1988). The GHQ-28 has been used with New Zealand population to assess post stroke depression (Hackett & Anderson, 2006; Hackett, Hill, Hewison, Anderson & House, 2010).

Geriatric Depression Scale-Short form (GDS-SF; Sheikh & Yesavage, 1986). The GDS is a scale used to assess mood and can be administered to screen for depression and was designed specifically to screen depression in the elderly, however can be administered to individuals aged 17 years and older. As a scale specifically designed for the elderly, it omits many of the somatic items included in other depression questionnaires, but which are common confounds in elderly or ill sample. The scale can be self-administered or the questions can be read to the participant if required. From the 30-item GDS, a 15-item short form (GDS-SF) was developed (Sheikh & Yesavage, 1986), which consists of 15 yes/no questions that can be administered in 5 to 7 minutes. For scoring, 1 point is given to each item with high scores indicating presence of a depressive sign/symptom. If the participant misses any items, scores may be calculated as a proportion according to the ratio of endorsed items to endorsable responses. Scores may range from 0 (no depression) to 15 (severe depression), with normative data available for cut-off values (Strauss, Sherman, & Spreen, 2006). Score ranges for the GDS-SF are as follows 0-4 (normal), 5-8 (mild depression), 8-11 (moderate depression) and 12-15 (severe depression). In this study scores of $\geq$5 will be used as an indicator of possible depression.

The 15-item GDS has Cronbach's alpha values of .71 to .84 (Iglesias, 2004) and split-half reliability of .73 (Jang, Small & Haley, 2001). Inter-item correlations of the GDS-SF range from .32 to .83, with a mean of .56 (Yesavage et al., 1983). Test-retest reliability after an interval of 7-14 days is also high (.84-.85; Mui, Burnette & Chen, 2001). GDS-SF is successfully able to measure changes in symptoms of depression and has been validated in numerous clinical studies to identify severity of depression in elderly populations (Vinkers, Gussekloo, Stek, Westendorp & van der Mast, 2004; Yesavage et al., 1983). GDS-SF has high correlations with other tests measuring depression (e.g., 73-.90, Beck Depression Inventory; Scogin, 1987) indicating good convergent validity. GDS-SF also has low correlations with cognitive screening tests (e.g., Mini Mental State Examination; Feher, Larrabee & Crook, 1992), indicating support for divergent validity.

Tests of cognitive function

To evaluate and quantify the impact of stroke on neuropsychological functioning, a battery of commonly used and validated tests was used to assess the following domains: visual and verbal memory, language, visuoperceptual reasoning, executive functioning (e.g., attention, impulsivity), and response and processing speed. While performance across individual neuropsychological tests was examined, summary scores were also calculated to act as an overall index of neuropsychological impairment in each domain. Using the normative data, all neuropsychological test scores were converted into z-scores, and these z-scores were then averaged to create summary scores. Also included was a screening test for probable dementia.

Hodkinson Abbreviated Mental Test (HAMT; Qureshi & Hodkinson, 1974). The HAMT is an abbreviated mental test of 10 questions derived from the longer Roth Hopkins test (Roth & Hopkins, 1953). The HAMT is a screening tool used to rapidly assess elderly patients for possible dementia and has also been used to assess confusion

and other cognitive impairment (Jitapunkul, Pillay & Ebrahim, 1991). HAMT total scores range from 0-10, and a score of ≤7 is considered indicative of cognitive impairment (Jitapunkul et al., 1991). The HAMT has high reliability (cronbach's α = 0.89) and is a valid tool to differentiate normal control groups from those who are the cognitively impaired (Jitapunkul et al., 1991). The HAMT has been used in stroke studies to assess the cognitive impairment of the survivors (Tuttolomondo et al., 2008).

Verbal Memory

California Verbal Learning Test- Second Edition (CVLT-II; Delis, Kramer, Kaplan & Ober, 2000). The CVLT-II is used to assess verbal learning and memory using list-learning task for individuals aged 16-89 years. The CVLT-II begins with five learning trials for list A, composed of 16 words, four each from four semantic categories (i.e., furniture, vegetables, ways of travelling, and animals). After each trial the participant is asked to recall the list of words and responses are recorded verbatim. The five learning trials are followed by a single trial of 16 new words, the interference list (List-B), comprising four semantic categories (i.e., vegetables, animals, musical instruments and parts of house), which the participants are asked to recall immediately. Thus, lists A and B contain two common (i.e., vegetables, animals), and two different categories. Recall of List B is followed by short delay free-recall and short delay cued-recall of list A where cues are the names of the 4 categories. After a delay of 20 minutes free-recall, cued-recall, and yes/no recognition trials (where the participant is required to recognise 16 words out of 44 words) of List A are administered. It takes approximately 20-minutes to administer CVLT-II (50 minutes including the delay).

For each correct answer on each trial the participant receives a score of 1. Raw scores are converted to z-scores using age and gender appropriate norms provided in the test manual (Delis et al., 2000). Data for the following trials were examined: List A Trial1; List A Trial 5; List B; Short-Delay Free Recall and Cued Recall; Long-Delay Free

Recall and Cued Recall; and Recognition Hits and False Positives.

The CVLT-II has high split-half reliability for normative sample (*r*=.94) and for a mixed clinical sample (*r*=.96; Delis et al., 2000). The test retest reliability coefficients for the standard form ranges from high (.89) for measuring overall level of achievement (e.g., Total Trials 1-5, Short and Long-Delay Free Recall, Total Recognition discrimination), to low (.59) for measuring process/strategy aspects (e.g., Percent Recall, Total Learning slope, Trial 1, Total repetitions). For the alternate forms of the test, reliability coefficients are adequate (.70-.79) for the main CVLT-II overall achievement variables (e.g., Total Trials 1-5, Short and Long-Delay Free Recall, Total Recognition discrimination). The CVLT-II was found to be highly valid in a study of memory performance in a sample with focal frontal lesions (Baldo, Delis, Kramer, & Shimamura, 2002). Jacobs and Donders (2007) examined the criterion validity of CVLT-II, using the test to assess memory impairment in individuals with traumatic head injury, and finding that the test was able to accurately classify 66-71% of control versus moderate to severe brain injury cases.

Logical Memory (LM); subtest Wechsler Memory Scale-III (WMS-III; Wechsler, 1997a). The LM subtest of WMS-III is a measure of memory requiring verbal recall of two short stories presented orally, and assesses immediate and delayed recall of these stories. In LM administration the examiner reads the Story-A once, after which the participant is required to recall it immediately. Story-B is then read twice and the participant immediately recalls the story (1st recall and 2nd recall) after each presentation. During the delayed recall (LM-II) condition, the participant is asked to retell each of the stories approximately 30 minutes after administration of the immediate recall condition. While a recognition trial is also available, due to time constraints this was not included in the test battery.

One point is awarded for each correctly recalled story unit (i.e., specific and literal

information) and thematic unit (i.e., general information), with the unit score for each story ranging from 0-25. Logical memory total recall score are computed by adding the recalls of Story A and B with sum ranging to a maximum of 75 (25 units per recall). LM-II delayed scores are calculated by computing delayed recall story unit (range 0-50).

Split-half internal consistency methodology was used to estimate reliability coefficients for age groups 16 to 89 years for both LM I and II, and test reliability coefficients ranged from .81 to .90 (LM-I) and .71 to .87 (LM-II) (Wechsler, 1997a). Test-retest reliability for LM I and II were calculated with the mean retest interval being 34.6 days, with the stability coefficients calculated for two pooled age groups (16 to 54, 55 to 89 years) and were, r=.77 (16-54 years), r=.77(55-89 years); and LM-II, r=.77 (16-54 years), r=.74 (55-89 years), respectively (Wechsler, 1997a). High correlations between WMS-III auditory memory indexes and other measures of verbal memory (e.g., CVLT; Delis, Kramer, Kaplan, & Ober, 1987; MicroCog Memory Index; Powell et al., 1993), suggest convergent validity.

Visual Memory

Visual Paired Associates (VPA); subtest Wechsler Memory Scale-Revised (WMS-R; Wechsler, 1987). The WMS-R subtest, visual paired associates (VPA), is a measure of both immediate and delayed visual memory and learning over trials. This subtest begins with a practice task where the participant is shown two abstract line drawings, each paired with a different colour. The participant is asked to remember the colour that goes with each figure. They are then shown the figures in a different order and asked to indicate the appropriate colour that is associated with each figure from a display of 3 possible colours. After the practice task, the test VPA-I begins with presentation of six figures, each paired with a different colour, and participants are asked to remember each colour and design pair. They are then shown the designs in a different order without their colours and asked to indicate the correct colour (from an array of 6), that went with

each figure. They can respond verbally or by pointing to the colour. If they answer incorrectly, the examiner points to the correct colour. The set of six designs is presented in this manner at least three times. If the examinee answers all six items correctly on or before the third trial, the test is discontinued. If the participant does not give 6 correct answers on any of the first three trials, the examiner presents a 4th learning trial, and if necessary a 5th and 6th trial until all 6 items are correct; or until 6th trial, whichever comes first. The examiner provides corrective feedback on all learning trials. For each correct response 1 point is scored. An immediate recall score is obtained by summing correct responses across first three trials (range=0 to 18). A delayed recall trial (VPA-II) is administered approximately 30 minutes later, in which the participant is shown the six figures and asked to indicate (from the array of 6 colours) which colour goes with each figure. For each correct response one point is scored (range 0 to 6). No corrective feedback is given during the delayed recall trial. Immediate and delayed raw scores were converted to z-scores using the means and standard deviations by age from the standardisation sample of the WMS-R (Wechsler, 1987). The standardization sample does not include those aged 25-34 or 45-54. When participants' ages fell in these ranges the closest age group to their age was used.

Reliability coefficients for VPA-I, range from .52 to .68 with an average of .58 (Wechsler, 1987). Coefficients for VPA-II range from .31 to .69 with an average of .58 (Wechsler, 1987). Studies suggest that the VPA subtest can be encoded into memory using verbal strategies (Leonberger, Nicks, Larrabee & Goldfader, 1992; Loring, 1989). According to the manual, to reduce the use of verbal strategies, the examiner should not verbalise colour names either when presenting trials or giving corrective feedback. As compared to other visual memory tests, VPA was selected as it has learning trials and does not require a physical response. Tests from WMS-III targeting visual memory (i.e., Faces, Family pictures and Visual Reproduction) were not used as the Faces subtest

provides for assessment of recognition memory but not free recall or learning over trials; and Family Pictures is very complex, involving remembering characters, their location and their activities from four scenes under a time limit. This was considered to be too demanding/complex for many of our participants.

Rey-Osterrieth Complex Figure (ROCF; Meyers & Meyers, 1995). The ROCF is used to assess visual-spatial constructional ability and visual memory and can be administered to individuals aged 6 to 93 years. The ROCF is a detailed geometrical design, which the participant first copies with the design being exposed, for up to 5 minutes. The copy trial is followed by free recall trials conducted 3-minutes and 30-minutes later, in which the participant is asked to reproduce the design from memory. A recognition trial is also included where the participant is presented with 24 geometrical figures, some of which are part of the original ROCF, and others which are not, and is asked to identify those that were part of the original design. Scoring the recognition condition allocates one point for each of the figures correctly identified as either part or not part of the original figure. For copy and recall trials, the design is divided into 18 elements, each of which are scored 0.5, 1, or 2 with the highest possible score being 36. Points are awarded for awarded for accuracy and location of each element of the design produced. That is, up to two points are given for each element; 1 point for an accurately drawn element, and 1 for being accurately placed in relation to other elements of the figure. No points are given if the element is absent or not recognizable.

For adults, split-half and coefficient alpha reliabilities are high (>.60 for copy, >.80 for recall conditions) (Berry, Allen & Schmitt, 1991; Fasteneau, Bennett & Denburg, 1996). Test-retest reliability at 6-months is also high (Immediate recall, r =.76; Delayed recall, r=.89; Recognition Total correct, r=.87; Meyers & Meyers, 1995). High correlations between the ROCF and other tests of memory and constructional abilities (e.g., Form Discrimination, Hooper, Token Test) are reported (Meyers & Meyers, 1995).

The ROCF is sensitive to neurological disorders which affect memory and executive function, such as anterior communicating artery aneurysm (Kixmiller, Verfaellie, Mather & Cermak, 2000).

Language

Boston Naming Test (BNT; Kaplan, Goodglass & Weintraub, 2001). The BNT is used to assess ability to name visually presented pictures of objects, and can be administered to individuals ≥18 years and older. The test takes approximately 10-20 minutes to complete; and consists of 60 line drawings of objects of increasing difficulty; ranging from simple, high frequency objects (e.g., chair) to rare objects (e.g., abacus). Full credit (1-point) is given if the item is correctly named within 20 seconds. If the participant misperceives the item or fails to provide a response within the 20 seconds, a stimulus cue is provided (e.g., "it is a piece of furniture") and an additional 20 seconds is given to name the picture correctly. If the participant provides the correct answer, full credit (1-point) is obtained. If the participant is still unable to name the picture correctly, a 'phonemic' cue is offered (i.e., "it starts with ch" the first phoneme of the name). No point is given for items where phonemic cues are presented Item administration for adults begins with item 30, and full credit is given for items 1 to 30 if items 30 to 38 are answered correctly. If any of these items is failed, items are administered in reverse order from item 29 until 8 consecutive items are answered correctly without any cues; and then administration resumes in a forward direction. The test is discontinued if the participant makes eight consecutive errors. A total score is calculated by adding the sum of spontaneous correct items, and number of correct items after stimulus cue, and can range up to a maximum of 60.

The BNT has high internal consistency (α=.90) and test-test reliability, with coefficients ranging from (r=.62 to.92) (Graves, Bezeau, Fogarty & Blair, 2004; Mitrushina & Satz, 1995). Swarie, Chelune, Naugle and Luders (1996) found test retest

reliability of .94 when adults with intractable epilepsy were retested after 8 months. High correlations have been found between BNT and other tests measuring language abilities (*r*=.76 to .86; Visual Naming Test of the Multilingual Aphasia Examination) and intelligence (Schefft, Testa, Dulay, Privitera & Yeh, 2003). The BNT has been used with stoke survivors, where patients performed poorly as compared to controls (Cao, Ferrari, Patella, Marra & Rasura, 2007).

Controlled Oral Word Association (COWA; Spreen & Benton, 1977). The COWA assesses fluency of speech, where the participant orally generates as many words as possible within 60 seconds, for each of 3 letters and one semantic category. This rapid and organized word retrieval ability is sensitive to brain dysfunction (Lezak et al., 2004). All responses are written down verbatim, and the test takes approximately 5 minutes to administer. The test can be administered to individual aged 7 to 95 years (Strauss et al., 2006).

For phonemic fluency, a total score is generated as the sum of all the words produced for across three letters (F, A, and S); while the sum of all correct words for the category "animals" is scored separately. For the scoring of phonemic category, slang terms and foreign words that a part of standard English (e.g., "soirée") are deemed correct. Proper names, incorrect words and variations or repetitions of the same word are excluded from the scoring. For scoring of the animal category, names of extinct, imaginary or magic animals are considered correct (e.g., unicorn, moa); whereas, given names such as "Donald" and "Mickey" are not counted as correct.

The reliability (i.e., internal consistency) for F, A, and S is high (r=.83). In healthy adults, test-retest reliability coefficients are also high (> .70) for both letter and semantic fluency after an interval of more than five years (Tombaugh, Kozak & Rees, 1999; Ross, 2003). Correlations between phonemic fluency tests (e.g., FAS and CFL) are high (.85 to.94) when used in different settings and groups (Cohen & Stanczak, 2000; Troyer,

2000). Correlations between forms using different semantic categories (e.g., animals and clothing, animals and food) are also moderately high (.66-.71; Delis, Kaplan & Kramer, 2001; Riva, Nichelli & Devoti, 2000). The COWA has been successfully used to measure performance of moderately aphasic stroke survivors over many different time intervals (3, 6, 9, 12, & 18-months) (Sarno, Postman, Cho & Norman, 2005).

Visuoperceptual functioning

Block Design (BD; subtest Wechsler Adult Intelligence Scale-III (WAIS-III; Wechsler, 1997b). The BD of the WAIS-III is used to assess nonverbal fluid reasoning and visual motor integration and takes approximately 20 minutes to administer. During BD the participant is presented with blocks; each of which has two red sides, two white sides, and two sides that are half red and half white. The participant is required to reproduce designs from a picture or a model using these blocks. The designs get harder over trials, starting with simple two-block designs, and finishing with complex nine-bock designs. The participant is presented with a total of 14 designs, each of which must be replicated as quickly as possible within a specified time limit. The test is discontinued if the participant receives 0 points on three consecutive trials. Designs 1 to 6 have two trials each, and the designs are considered failed only if the participant receives 0 on both the trials of each design. Designs 1 to 6 may receive a score of 2 (successful completion within time limit on trial 1), 1 (successful completion within time limit on trial 2), or 0 (fails both trials). For designs 7-14 a minimum of 4 points is awarded on successful completion of the design within the time limit and the examinee can receive 1-3 bonus points for quicker completion. Thus, as a timed task, BD can be negatively impacted by slow response. A maximum raw score of 68 may be obtained, which is then converted into a z-score using available age and gender related norms (Wechsler, 1997b).

Reliability coefficients for BD were found to be in the range of .76 to .90, while test re-test reliability for different age groups ranged from r=.77 to r=.86. High and

statistically significant correlations (r=.80) were found between WAIS-III, BD and Wechsler Intelligence Scale for Children (WISC-III) BD, indicating high criterion-related validity (Wechsler, 1997b).

Matrix Reasoning (MR), subtest Wechsler Adult Intelligence Scale-III (WAIS-III; Wechsler, 1997b). The MR is a subtest of WAIS-III used to assess nonverbal fluid reasoning, spatial processing and attentiveness to visual detail and takes about 20-30 minutes to administer. MR is an untimed test composed of 26 items, which requires four types of non-verbal reasoning: pattern completion, classification, analogy, and serial reasoning. On each item the participant is presented with an array or matrix which has a missing section. The participant is asked to look at five response options and choose the option (by pointing or identifying the number) that completes the array/matrix. The participant is presented with three sample trials to help them understand the instructions for the test. If the participant gives an incorrect response to a sample item, the correct way to solve the problem is illustrated. No corrective feedback is given during the actual test.

A score of 1 is given for each correct response on test items. The test starts from item 4 and if the participant receives a score of 1 on items 4 and 5, full credit is given for items 1-3. If the participant scores 0 on either item 4 or 5, items 1 to 3 are administered in reverse order until the participant receives a perfect score on two consecutive items. The test is discontinued after four consecutive scores of 0 or four scores of 0 on five consecutive items.

The split-half reliability of MR is high, with coefficients ranging from .84-.91, and test-retest reliability coefficients ranging from .70 to .78. Criterion related validity is reportedly high (r=.81) when correlations were calculated with Raven's Standard Progressive Matrices (Wechsler, 1997b). MR has been used in stroke studies to assess visuoperceptual functioning (Alfieri et al., 2008).

Executive Function and Speed of Processing

Trail Making Test (TMT; Partington & Leiter, 1949). The TMT is used to assess speed for tasks requiring attention, sequencing, mental flexibility, visual search and motor function in individuals aged 15 to 89 years. The TMT consists of two parts; Part A consists of encircled numbers (1-25) and part B consists of encircled numbers (1-13) and letters (A-L). For Part A, the participant is required to join the numbers in order (i.e., 1-2-3-4, etc.) and for part B must join number and letters in alternate order (i.e., 1-A-2-B, etc.) with a pencil as quickly as possible and without lifting the pencil. Before administration, participants are provided with 8-item practice sessions for Parts A and B, and the test is discontinued if the participant is unable to complete the practice exercise. Trail A therefore is often used as indication of baseline speed for scanning task while B requires this plus added executive ability to alternate between sets. During administration, if the participant makes an error, the examiner points out the mistake and asks the participant to proceed from the point where the mistake occurred. Performance is measured as the time in seconds taken to complete Part A and Part B, with timing continuing even if errors occur. Raw scores were converted to z-scores using available age and education related normative data (Strauss et al., 2006).

In a study of 384 healthy participants retested after 11-months, test-retest reliability coefficients were reported moderate for Trail A (r=.79) and high for Trail B (r=.89; Dikmen, Heaton, Grant & Temkin, 1999). In clinical samples moderate to high reliability coefficients are reported (i.e., Trail A 69 to .94; Trail B .66 to .86; Goldstein & Watson, 1989). The TMT has been used to test visual search, scanning, and executive functioning (Trail B) indicating a high construct validity (Kortte, Horner & Windham, 2002), and has been found to be a useful tool in assessing neurological functioning of adults and children (Reitan & Wolfson, 2004). The TMT has also been used post-stroke to assess executive functioning (TMT B) and speed processing (TMT A; Winkens, Van

Heugten, Wade, Habets & Fasotti, 2009).

Victoria Stroop Test (Regard, 1981). The Stroop test is a measure of cognitive speed and flexibility and can be administered to those aged 18 to 94 years. The test consists of three cards with six rows of four items (i.e., 24 items total) on each card. Part D (dots), consists of coloured dots and the participant is asked to name the colour of each dot, in order, as quickly as possible. In Part W (words), the participant is required to name the colours of each of the 24 words printed on the card (i.e., when, hard, over, then). In Part C (colour) the coloured stimuli are colour names (i.e., blue, green, red, and yellow) and the ink in which each colour name is printed does not match with the name of the colour name printed. The participant must name the colour of the ink word is printed in as fast as possible (i.e., if "red" is written in blue ink, the correct response is "blue"), and must refrain from reading the words. This version of the test was chosen for its quick administration and its ability to differentiate between conditions. For example, part D is used to indicate of baseline cognitive speed with performance hindered by general slowness. Part W introduces one aspect of interference, and performance on Part C can therefore be judged in relation to other conditions. An index of interference (Part C/Part D) is recommended by Graf et al. (1995) because it is sensitive to age-related slowing; giving an indication of whether the interference is normal for a particular age group. Scoring consists of time taken in seconds to complete each task, along with the number of errors made. The ratio of C/D is also used. Spontaneous self-corrections are scored as correct. Raw scores were converted to z-scores using age-related normative data (Strauss et al., 2006).

Test-retest reliability coefficients of .90 (Part D), .83 (Part W) and .91 (Part C), are reported with a one month interval (Strauss et al., 2006). The Victoria Stroop Test is valid instrument and has been widely used as a measure of selective attention, cognitive flexibility and processing speed with people suffering from neurological damage (Strauss

et al., 2006; Stuss, Floden, Alexander, Levine & Katz, 2001) and to assess executive functioning post-stroke (Barker-Collo, 2007).

Integrated Visual and Auditory Continuous Performance Test (IVA-CPT; Sandford & Turner, 2004). The IVA-CPT is a computerised test used to measure sustained attention and response control and can be administered to individuals aged 6 to 99 years. It is easy to administer, and takes 13-minute with instructions presented in a female voice and visually by computer. The test consists of random presentation of two stimuli (i.e., the numbers "1" and "2") presented visually and orally. The participants must click the mouse when they see or hear the number "1" (target stimulus) and not click when they see or hear the number "2" (distractor stimulus). The test begins with two 1 minute "warm up" sessions with presentation of visual and auditory targets to establish baseline reaction time; followed by a 1.5 minute practice with both targets and distracters presented. The practice session consists of 32 trials and the computer provides feedback ("whoops") if the participant makes an error. The practice session reduces practice effects and allows the examiner to know if the participant has understood the test. The test itself consists of 500 trials divided into 5 sets of 100 trials, with each trial lasting 1.5 seconds. During the test, both stimuli are presented pseudorandomly, with varied target frequency. During the first 50 trials of each block, 84% of trials are presentation of target ('1'), and 16 % non targets ('2'). During the next 50 trials, 16% trials are targets ('1'), and 84% are non targets ('2'). With "rare" blocks increasing chances of omission/attention errors and common blocks increasing chances of commission/impulsive errors. The total administration for the IVA-CPT is approximately 20 minutes.

The IVA-CPT automatically collects and scores all data in relation to age appropriate norms using quotient scores. Quotient scores have a mean of 100 and standard deviation of 15 and can easily be interpreted, and allow comparison between

scales. These were converted to z-scores for the purposes of analyses. The IVA- CPT analysis provides six global composite scores and 22 other scales. For the purpose of this study, IVA-CPT scales assessing attention and impulsivity were examined: (1) "Prudence" scales provide measure of impulsivity and response inhibition, and reflects errors of commission. A high score is indicative of low impulsivity, while high scores are associated with a person who is cautious, thoughtful and careful. (2) "Vigilance" scales measure attention, and indicate when an individual fails to click the mouse when the target is presented. High scores on the vigilance scale indicate that the person is watchful, alert and attentive, with low vigilance scores indicating problems with discriminatory attention, staying on task, and/or being alert. Separate prudence and vigilance scores are provided for visual and auditory modalities.

Though the IVA-CPT was originally designed to assist in diagnosis and differentiating among subtypes of Attention Deficit Hyperactivity Disorder, it has been used to assess attention and vigilance in stroke patients (Barker-Collo, 2007). Test-retest reliability (intervals 1-4 weeks) are adequate for attention quotients (i.e., r=.66-.75) and low for response control scores (i.e., r=.37-.41). In the current study, only scales with high reliability (i.e., prudence and vigilance, with reliabilities 0.64, 0.61 and 0.32, 0.71 respectively) were used (Strauss et al., 2006).

PROCEDURE

For this study, ethical approval was obtained from the Multiregional Ethics Committee (SAH sample) and from the University of Auckland Human Participants Ethics Committee (control sample). The procedure for the SAH group is described below first followed by the procedure for the control group.

SAH group

For the SAH group, participants from the ARCOS study who had experienced a SAH in 2002-2003 (n=87) and had previously agreed to participate in future research

were contacted (n=37) via telephone. For the participant who could not be contacted based on the previously recorded contact details, an exhaustive effort was made to locate him/her using telephone directories, general practitioner records, electoral rolls and via contacting relatives using details on existing research forms. Once located potential participants were sent a cover letter inviting them to participate in the study along with a participant information sheet (PIS; Appendix B) explaining the purpose of the study, a consent form (CF) and a freepost envelope addressed to the researchers (Appendix C). Out of the 37 participants, 36 (97.3%) were contacted for the study. Out of the 36 participants contacted, 27 (75%) gave written consent to participate in the study. For those without English as first language, interpreters were used in all assessments.

Following receipt of the consent form, the participants were interviewed over the telephone to complete questionnaires (Information about their recovery from "two simple questions test", HAMT, BI, SF-36 and FAI) which took approximately 15-20 minutes**.** Administration was split into telephone, interview, mail-out questionnaires, face to face interviews in order to spread burden expressed by participants. A summary of measures and how they were administered appears in Table 5. After the telephone interview the participants were sent the following structured self-administered questionnaires via mail: (i.e., GHQ-28, GDS-SF and LHS), which was followed by a telephone call to arrange a day and time for a face-to-face interview to complete MRS and administer the battery of neuropsychological tests. Where the participant was too unwell (n=2) to complete the questionnaires, the data was collected using a proxy (a paid or unpaid caregiver). Those who agreed to participate in a face-to-face interview were then assessed at their usual place of residence by the researcher. Before the start of the assessment time was taken to establish rapport. As highlighted by Lezak et al. (2004), initial rapport is imperative to the assessment session, considering that increased anxiety is likely to impact negatively on the test performance. Considering that the assessments were carried out at the

participants' usual place of residence, all steps were taken to ensure optimum testing environment (i.e., adequate lighting, reducing distractions such as noise, presence of other people in the room, and turning off the cells phones). Participants were also reminded to use any hearing aids or glasses they might need. They were also asked to advise the researcher if they needed any breaks during the testing. The neuropsychological test battery was designed to be completed in approximately 90-minutes, however for those participants who felt fatigued (based on self-report after checks made by researcher), the tests were completed over two sessions. For 16 (59.3%) participants, the neuropsychological assessments were finished in one session, 8 (29.6%) had to be completed in two sessions due to participants feeling fatigued. Three participants only completed the self-administered questionnaires, of whom 1(3.7%) had moved out outside the greater Auckland and 2 (7.4%) refused the face to face interview for the neuropsychological testing. Thus, out of 27 potential participants 24 completed the neuropsychological assessment (see Table 5).

Table 5

SAH participants who completed neuropsychological assessments (n=23)

Measures	Number completed	Number not completed	Reason				
			Poor Eye sight	Difficulty understanding instructions	Hemiplegia	Englsih not first language	Refusal
TMT	20	3	1	1	1		
VST	22	1	1				
CVLT-II	20	3				3	
ROCF	21	2			2		
COWA	20	3				3	
LM	20	3				3	
BNT	20	3				3	
VPA	23	0					1
BD	21	2			2		
IVA-CPT	22	1					1

BD= Block Design; BNT= Boston Naming Test; BT= Bell's Test; COWA=Controlled Oral Word Association; CVLT-II=California Verbal Learning Test-II; IVA-CPT= Integrated Visual and Auditory Continuous Performance Test; LM= Logical Memory; ROCF= Rey-Osterrieth Figure; TMT= Trails Making Test; VST= Victoria Stroop Test

Neuropsychological assessments requiring verbal output (e.g., CVLT-II, LM, BNT, COWA) were not administered for people with expressive aphasia. In cases where the aphasia was quite mild, the BNT which is designed to quantify mild aphasia was attempted, subject to participants' willingness and level of frustration experienced. These tests were also omitted for people who did not have English as their first language. For all other assessments interpreters were used. It is important to note that dysphasia does not necessarily mean presence of alexia or agraphia; therefore many dysphasic subjects were still able to complete questionnaires. Where receptive aphasia was suspected, standard administration protocol was used which involves presentation of practice items (for all assessments except the ROCF and COWA). Thus, ability to complete each test was determined by ability to demonstrate understanding on practice tests. Following the assessment of GDS-SF, any mood disorder noted was reported to the study supervisor. The participants were provided with a pamphlet about depression, and were advised to contact their general practitioner.

Furthermore tests requiring physical responses (e.g., ROCF, BD, Trails A and B) were not administered for participants where hemiplegia was likely to impact their performance. For those participants who had difficulties with their vision (e.g., poor eyesight or colour blindness), tasks such as, ROCF, STROOP, VPA were omitted. Furthermore where receptive aphasia was suspected, standard administration protocols were observed, which for all assessments except the ROCF, COWA, CVLT-II, LM, involves presentation of the practice items. An individuals' ability to participate was determined by his/her ability to perform on practice items. If it became apparent that the participant did not understand the nature of the task, it was discontinued, as the data collected would not necessarily be a valid indication of the ability level.

At the conclusion of the face-to-face interview, information was also gathered on general medical status, further strokes (since the stroke which occurred 5-years ago) and

reported dementia, which were likely to impact on performance. For recurrent events (including possible events), additional information was also gathered from medical or nursing home records, and if needed from treating doctors, including verification of further stroke events and their classification.

Control group

The control group included, healthy New Zealand participants from the general community who were matched to the SAH group on age, gender and ethnicity. Potential participants were recruited in an number of ways including the snowballing technique and recruitment from the community organisations such as Grey Power and Age Concern (an agency that provides volunteer/visitor services to the elderly) to ensure adequate coverage of the upper age range as well as through PIS (with researcher's contact details) posted in churches, libraries, retirement homes and medical services (see Appendix D).

Where requested (and where practicable) the researchers also conducted brief presentations about the research to the community agencies involved, and their patrons. Where presentations were made, PISs and consent forms (see Appendix E) were made available with free post envelopes addressed to the researcher in which signed consent can be returned. The interested parties were then contacted via telephone or email using the contact details requested on the consent form. Potential participants who matched those to the SAH group were also contacted using snowballing technique where researchers' contacts approached the interested participants whereby potential participants were provided with written information about the study by individuals other than the researcher. Thus, the interested participants were sent a PIS, consent form and a free post envelop addressed to the researcher. Following the receipt of the consent form, the assessment procedure for the control group was same as that for the SAH group.

Table 6

Data collection techniques for SAH and Control Group

Measures	By Telephone	By Mail	Face to face interview
Verbal consent	X		
Demographic Data	X		
Complete recovery?[a]	X		
Functional status -Frenchay Activity Index	X		
Disability -Modified Rankin Scale -Barthel Index	X		X
Handicap -London Handicap Scale		X	
Health Related Quality of life -Short Form 36 Questionnaire	X		
Mood and Depression -15 item Geriatric Depression Scale -General Health Questionnaire		X X	
Hodkinson Abbreviated Mental Test	X		
Neuropsychological assessments -Verbal Memory (CVLT-II, LM-I and II) -Visual Memory (VPA, ROCF) -Language (BNT, COWA) -Attention & Executive function (TMT A/B, VST, IVA-CPT) -Visuoperceptual Ability (BD, MR)			X
Clinical Information - recurrent stroke -diagnosis of dementia			X

BD= Block Design; BNT= Boston Naming Test; BT= Bell's Test; COWA=Controlled Oral Word Association; CVLT-II=California Verbal Learning Test-II; IVA-CPT= Integrated Visual and Auditory Continuous Performance Test; LM= Logical Memory; MR=Matrix Reasoning; ROCF= Rey-Osterrieth Figure; TMT= Trails Making Test; VPA=Visual Paired Associates; VST= Victoria Stroop Test

[a]Complete recovery as assessed by Two simple questions item "Do you feel you have made a complete recovery from your stroke?"

After the administration of the tests, all the neuropsychological tests were converted into z-scores based on normative data from Mayo's Older Adults Normative Studies (MOANS; Harris, Ivnik & Smith, 2002; Lucas et al., 1998a; Lucas et al., 1998b; Steinberg, Bieliauskas, Smith & Ivnik, 2005; Steinberg, Bieliauskas, Smith, Ivnik &

Malec, 2005; Steinberg, Bieliauskas, Smith, Langellotti & Ivnik, 2005), Strauss et al. (2006) and test manuals. Overall neuropsychological impairment was calculated by counting the number of z-scores from the neuropsychological tests that were ≤ -2.00 (BD, BNT, COWA, CVLT-II, IVA-CPT, LM, MR, ROCF, TMT, VPA, VST). If a neuropsychological test was not conducted due to medical problems such as aphasia, hemiplegia, blindness, deaf etc, then their test score was counted to have a z-score of ≤ 2 in that domain. Domain scores were calculated where the z-scores for the following neuropsychological tests were averaged: verbal memory (CVLT-II long delay free recall, LM II), visual memory (VPA II, ROCF 30-minute delayed recall), language (BNT, COWA words), visuoperceptual functioning (BD, MR), executive function (IVA-CPT overall attention), information processing speed (Trails A, Stroop dots).

After the tests and measures were scored, raw scores, z-scores, domain scores and standard scores were entered into PSAW 17.0 file for analysis. Each participant was given a code number and all identifying information was removed from the file.

CHAPTER-III

RESULTS

OVERVIEW

The analyses for this research are presented in a number of sections. The first section consists of the preliminary analyses which includes: 1) Inspection of the data set (i.e., accuracy of input and managing missing data), and 2) analyses to test the assumptions related to the analyses performed. In the second section, means, standard deviations and frequencies are presented to describe the overall performance of both SAH and control groups across measures. Following this, one-way ANOVA is used to examine the measures of neuropsychological and general functioning on which the survivors of SAH differ from matched controls. Section three considers relationships between baseline and 6-month assessments and current functioning. This includes: a) examination of change over time for those measures where longitudinal data are available; b) correlation analyses to determine what demographic factors (e.g., age, gender, education, ethnicity) and test results at 6-months and injury characteristics (e.g., SAH severity), are most related to current functioning (i.e., disability, handicap, quality of life, mood, and neuropsychological outcomes). In section four, interrelationships amongst current measures of functioning are considered. This includes correlational analyses to examine the relationship between areas of neuropsychological deficit, functional abilities/quality of life, and mood.

Section 1: Preliminary Analysis

Preliminary analyses included inspection of the data set (i.e., accuracy of input, management of missing data) and analyses of testing assumptions (i.e., normality, linearity and homogeneity of variance). The variables were examined separately for the SAH and control groups.

Inspection of the data set

Missing data: A moderate amount of data was missing in the data set that needed to be dealt with. Tabachnik and Fidell (2007) have recommended various ways of handling the missing data including substituting means and deleting cases or variables. A number of participants from the SAH group did not complete the following functional outcome measures: LHS (n=9, 33%), SF-36 (n=1, 3%), GHQ-28 (n=9, 33%), FAI (n=1, 3%), BI (n=1, 3%), GDS-SF (n=7, 25%) and MRS (n=3, 11%). Furthermore some participants did not complete the full neuropsychological measures due to refusal (n=4), hemiplegia (n=2), visual problems (n=2), difficulty understanding instructions (n=1) and English not being the first language (n=3). Where the whole measure was not completed, it was left out in the analyses. Thus, by selecting the *exclude cases pairwise option* for analyses, cases were excluded if they were missing the data required for the specific analyses. However, the cases were included in the analyses for which they had the necessary information because the *listwise* (casewise) deletion has the disadvantage of unnecessary loss of a large amount of data. All functional outcomes data was complete for the control group, however four control participants did not complete the language tasks in the neuropsychological assessment because English was not their first language.

For 5-year, post-SAH survivors, there were two cases with single missing values on functional outcome measures (GHQ-28 and FAI). A small amount of data was also missing for GHQ-28 (2 values for 2 cases), FAI (3 values for 3 cases) and SF-36 (1 value)

from the 28-day and 6-month assessments. Since the proportion of missing values was very small, missing values were substituted with the mean for the missing item's corresponding subscale for the individual (e.g., GHQ-28 and SF-36) prior to the analyses. Where the measure did not have a subscale (e.g., FAI), missing value was substituted with the mean score of the measure. However this method was used only where up to two items were missing per scale. In this data set none of the cases had more than two missing values per measure.

Testing Assumptions

Normality: It is important to screen the data for normality prior to a multivariate analysis (Tabachnik & Fidell, 2007). Normal distribution values of skewness and kurtosis are zero and normality is considered violated when the skewness and kurtosis values differ significantly from zero. The Kolmogorov-Smirnov test is used to examine normality and when tests are not significant ($p<.05$) it means that our distributions is not significantly different from the normal distribution and the assumption of normality has not been violated.

Tests were conducted using the aforementioned criteria for each of the neuropsychological measures: IVA-CPT (full attention, auditory attention, visual attention, auditory impulsivity and visual impulsivity), Trails A/B, Stroop test (Dots, colours, words, colours to dots ratio), LM I and II, CVLT-II (Short delay free and cued recall, long delay free and cued recall, recognition hits, false positives), ROCF (3-minute recall, delayed recall and recognition), VPA I and I, BD, MR, COWA (FAS and Animals), BNT and domain scores (verbal memory, visual memory, language, visuoperceptual functioning, executive functioning/attention, information processing speed and overall neuropsychological impairment. Functional measures included: LHS, SF-36 (MCS, PCS, PF, RP, BP, Vitality, GH, SF, RE, MH), MRS, FAI, BI, GDS-SF and GHQ-28 (total score,

Somatic Symptoms, Anxiety and insomnia, Social Dysfunction, Symptoms of depression). The tests were conducted separately for both the groups (SAH and control). As can be seen in Table 7, out of 43 neuropsychological variables, 16 and 21 variables violated the normality assumption for the SAH and control group respectively. For the functional outcome measures, 12 and 19 out of 20 variables violated the assumptions for the SAH and control group, respectively. In order to address the non-normality, data transformations of the variables was computed using methods such as square root, logarithmic and inverse transformations (Tabachnik & Fidell, 2007). However these data transformations improved normality for only nine neuropsychological variables (i.e., ROCF [copy, short delay recall], VPA II, BNT, COWA word, CVLT-II [List B, false positive], IVA-CPT [auditory attention, visual impulsivity]) while all the functional outcome measures remained non normal. In order to maintain interpretability, it was therefore decided to use the existing variables without transformations but to interpret the results with caution. Furthermore, the analyses used in the research (e.g., ANOVA, regression) are relatively robust to violations of normality, particularly with reasonably equal sample sizes which is the case in this study (Tabachnik & Fidell, 2007).

Linearity refers to the assumption that there is a straight line relationship between two variables and is assessed by inspection of bivariate scatter plots (Tabachnik & Fidell, 2007). Scatterplots were created for all the aforementioned neuropsychological and functional outcome measures. No evidence of violations to the assumption of linearity was found.

Homogeneity of variance refers to the assumption that the spread or dispersion of scores for each group is similar and that the means of samples are obtained from populations of equal variance (Tabachnik & Fidell, 2007). The Levene's Test of equality of variance was used to assess the assumption that the variances are equal in two groups.

Table 7

Neuropsychological and functional measures violating the assumptions of Normality across groups

SAH	CONTROL
Neuropsychological Measures	
RCF (short delay recall, long delay recall)	*TMT A*
VPA I	*RCF (short delay recall, long delay recall)*
BNT	*VPA II*
CVLT II (Trial 5, short delay cued, long delay recognition hits, false positive)	*MR*
IVA-CPT (Auditory attention, visual impulsivity, auditory impulsivity)	*LM (immediate and delayed recall)*
Overall neuropsychological impairment	*COWA (FAS and Animal)*
Language	*CVLT II (Trial5, List B, Short delay cued, long delay cued, false positive)*
Executive functioning	*IVA-CPT (visual attention, full scale response impulsivity, visual impulsivity, auditory impulsivity)*
	Overall Neuropsychological Impairment
	Executive Functioning
Functional Measures	
MRS	*MRS*
FAI	*FAI*
BI	*BI*
SF-36 (RP, GH, SF, RE)	*SF-36 (PF, RP, BP, VT, GH, SF, RE, MH, PCS)*
GHQ-28 (Total score, Somatic Symptoms, Anxiety and insomnia, Social Dysfunction, Symptoms of Depression)	*GHQ-28 (Total score, Somatic Symptoms, Anxiety and insomnia, Social Dysfunction, Symptoms of depression)*
	GDS-SF
	LHS

BI=Barthel Index; BNT=Boston Naming Test; BP=Body Pain; COWA=Controlled Oral Word Association; CVLT II=California Verbal Learning Test (2nd Edition); FAI=Franchay Activity Scale; GDS-SF=Geriatric Depression scale-short form; GH=General Health; GHQ-28=General Health Questionnaire; IVA-CPT=Integrated Visual and Auditory Continuous Performance test; LHS=London Handicap Scale; LM=Logical Memory; MH= Mental Health; MR=Matrix Reasoning; MRS= Modified Rankin Scale; PCS=Physical Component Score; PF=Physical Functioning; RE=Role Emotional; ROCF=Rey Osterreith Complex Figure; RP=Role Physical; SF=Social Functioning; SF-36=Short Form-36; TMT=Trail Making Test; VPA=Visual Paired Associates; VT=Vitality

If these tests are significant, it suggests that there is a difference between variances for the groups on the dependent variables and the assumption of homogeneity of variance has been violated (Pallant, 2005). The Levene's test was used to screen for homogeneity of variance on the aforementioned neuropsychological and functional variables. Levene's test was significant ($p<.05$) for the following neuropsychological scales: Bells test, ROCF copy, LM immediate recall, and visual impulsivity, suggesting that the assumption of homogeneity of variance has been violated. On the functional measures, Levene's test was significant ($p< .05$) for the following variables: GDS-SF, FAI, BI, LHS and SF-36 (PF, RP, BP, GH, SF, RE, PCS, MCS) suggesting violation of the assumption of homogeneity for these measures. However the analyses used in this study are reasonably robust to violation of homogeneity of variance if the group sizes are reasonably similar as is the case in this study (Pallant, 2005; Tabachnik & Fidell, 2007). In addition, it is expected that the clinical samples will show greater variability than healthy controls (Hadjivassiliou et al., 2001; Mavaddat et al., 1999) in their performance, and this was supported by the data.

Section 2: Overall Performance and Group Comparison

This section examines the overall performance of both SAH and control groups across measures. Tables 8 and 9 present the means and standard deviations of SAH and control groups for performance across neuropsychological and functional measures including neuropsychological domain scores. To compare the two groups two one way ANOVAs were run with the group (i.e., control and SAH) as the grouping variable. For the first ANOVA all the neuropsychological variables listed in Table 8 were entered into the analyses. Table 9 lists all the dependent variables for the second ANOVA. As can be seen in Table 8, those who had experienced an SAH were significantly more impaired on neuropsychological functioning and performed significantly worse than those in the

Table 8

Performance across neuropsychological functioning (z-scores) for SAH and control group with significance of difference

Measures	*SAH group (N=27)*		*Control Group (N=26)*		*Differences between Groups*	*Effect Size (eta²)*
	Mean	*SD*	*Mean*	*SD*		
Executive Domain Score	-1.40	1.91	-0.30	1.38	*F(1,43)=4.97, p=**.031***	*0.104*
IVA-CPT-Full Attention	-1.40	1.91	-0.30	1.38	*F(1,43)=4.97, p= **.031***	*0.104*
Auditory Attention	-1.18	1.63	-0.07	-0.07	*F(1,43)=6.97, p=**.012***	*0.140*
Visual Attention	-1.43	2.33	-0.63	1.40	*F(1,43)=2.01, p=.164*	*0.045*
Auditory Impulsivity	-0.54	2.66	0.12	1.98	*F(1,43)=0.91, p=.346*	*0.021*
Visual impulsivity	-0.28	1.54	0.77	0.73	*F(1,43)=9.13, p=**.004***	*0.175*
Trails B	-0.72	1.03	0.54	0.73	*F(1,43)=23.11,p<**.001***	*0.350*
Stroop Test -Words	-1.62	1.85	0.03	1.22	*F1,45)=13.44,p=**.001***	*0.230*
Colour Names	-0.49	1.16	0.50	0.72	*F(1,45)=12.87,p=**.001***	*0.222*
Colour/Dot	0.17	1.05	2.25	0.56	*F(1,42)=68.97, P< **.001***	*0.621*
Information Processing Domain	-1.09	2.50	0.54	0.96	*F(1,45)=9.45,p=**.004***	*0.173*
Stroop Dot Trial	-1.11	2.69	0.40	1.26	*F(1,45)= 6.52, p=**.014***	*0.127*
Trails A	-0.56	1.05	0.69	0.79	*F(1, 43)=20.74, p<**.001***	*0.325*
Verbal Memory Domain Score	-0.05	1.25	1.02	0.73	*F(1,39)=11.82, p=**.001***	*0.232*
Logical Memory-I	-0.12	1.28	1.00	0.67	*F(1, 39)=12.96, p=**.001***	*0.250*
II	0.23	1.24	1.36	0.78	*F (1,39)=12.76, p=**.001***	*0.246*
CVLT-II - Short Delay Free	-0.26	1.21	0.48	1.27	*F(1,39)= 3.64, p=.064*	*0.085*
Short Delay Cued	-0.39	1.42	0.25	1.28	*F(1,39)= 2.34, p=.134*	*0.056*
Long Delay Free	-0.34	1.40	0.68	0.87	*F(1,39)= 8.13, p=**.007***	*0.172*
Long Delay Cued	-0.42	1.51	0.18	1.20	*F(1,39)= 2.02, p=.163*	*0.049*
Recognition Hits	-0.61	1.81	0.07	1.20	*F(1,39)= 2.00, p=.165*	*0.049*
False Positives	-0.13	1.21	-0.12	1.18	*F(1,39)= 0.002 p= .968*	*0.001*
Visual Memory Domain Score	-0.14	1.03	0.55	0.55	*F(1,46)= 7.18, p= **.010***	*0.135*
ROCF Copy	-2.63	3.10	-1.36	1.53	*F(1,44)=3.31, p =.076*	*0.070*
3-minute recall	0.26	1.87	0.58	1.40	*F(1,44)=0.44, p= .513*	*0.009*
Delayed recall	-0.18	1.48	0.62	1.41	*F(1,44)=3.47, p=.069*	*0.073*
Recognition	-0.84	1.92	0.12	0.98	*F(1,44)=4.93, p=**.032***	*0.101*
VPA Learning Trials	-0.02	0.92	0.71	0.82	*F(1,45)=8.25, p=**.006***	*0.155*
Delayed recall	0.01	0.84	0.49	0.36	*F(1,45)=6.88, p=**.012***	*0.133*
Visuoperceptual Domain Score	-.046	1.00	0.84	0.77	*F(1,46)=11.99, p=**.001***	*0.207*
Block Design	0.23	1.41	0.88	0.89	*F(1,44)=3.67, p=.062*	*0.077*
Matrix reasoning	-0.21	1.01	0.80	0.91	*F(1,46)=13.21, p=**.001***	*0.223*
Language Domain Score	-0.82	1.49	0.52	0.65	*F(1,39)=14.55, p<**.001***	*0.272*
COWA FAS	-0.72	0.74	0.46	1.00	*F(1,39)=15.82, p<**.001***	*0.288*
Animals	-0.12	1.22	1.00	1.06	*F(1,39)=9.84, p=**.003***	*0.202*
Boston Naming Test	-0.89	2.71	0.58	0.74	*F(1,39)=5.99, p=**.019***	*0.133*
Overall Impairment	2.23	2.62	0.58	0.95	*F(1,45)= 8.983, p=**.004***	*0.163*

COWA=Controlled Oral Word Association Test; CVLT II=California Verbal Learning Test (2nd Edition); IVA-CPT=Integrated Visual and Auditory Continuous Performance Test; ROCF=Rey Osterreith Complex Figure; VPA=Visual Paired Associates

matched control group on all domain scores. In terms of overall impairment scores, test means did not change significantly when inability to complete tasks due to cognitive impairment did not result in -2 being substituted for the missing value. The ANOVA showed that the means were significantly different and the effect size ranged from small to large (κ= .001 to 0.621; Cohen, 1988) indicating that the difference between the groups varied from small to large. Within the domains, they performed significantly worse on all measures within the language and information processing domains. However, in the visual memory domain, the SAH group performed significantly worse on only ROCF recognition and visual paired associates, learning and delayed recall with no significant differences on other tasks such as ROCF copy, short delay or long delay recall. Within the verbal memory domain, the SAH group performed significantly worse on the Logical Memory task (immediate and delayed recall) and CVLT long delay free recall trials of these tests. In the visuoperceptual domain, the SAH group performed significantly worse on the matrix reasoning measure however, did not differ significantly on the block design task. Within the executive domain, while the SAH group performed significantly worse on IVA-CPT full attention task, which makes up the executive domain, no significant difference was noted on the other tasks of IVA-CPT such as visual attention and auditory impulsivity. However, the SAH group performed significantly worse on all the remaining executive functioning tasks.

As seen in Table 9, the SAH group had worse performance on all functional outcome measures, with significantly lower scores on all the measures of disability and handicap. Regarding HRQoL, the SAH group scored significantly lower than the control group on the MCS and PCS of the SF-36. However, within this measure, the SAH and control groups differed significantly only on subscales of physical functioning and social functioning with no differences on other subscales (i.e., role limitations due to physical

Table 9

Performance across functional measures for SAH and control groups and significance of difference between the groups

Measures	*SAH group (N=27)*		*Control Group (N=26)*		*Differences between groups*	*Effect Size (eta²)*
	Mean	*SD*	*Mean*	*SD*		
Handicap						
LHS	*0.74*	*0.24*	*0.97*	*0.06*	*F(1,42)=22.91, p<**.001***	*0.352*
Quality of Life						
SF-36						
MCS	72.52	18.53	80.50	6.63	*F(1,50)=4.27, p= **.044***	*0.078*
PCS	71.18	25.11	81.90	12.82	*F(1,50)=3.76, p= .058*	*0.070*
PF	60.58	38.51	82.31	18.77	*F(1,50)=6.69, p= **.013***	*0.118*
RP	75.00	42.43	84.62	28.36	*F(1,50)= 0.92, p= .341*	*0.018*
BP	76.63	24.20	86.06	17.12	*F(1,50)=2.63, p= .111*	*0.050*
VT	59.81	12.69	63.27	11.04	*F(1,50)=1.10, P= .299*	*0.022*
GH	72.50	26.01	74.62	16.61	*F(1,50)=0.12, p= .728*	*0.002*
SF	79.81	27.40	94.71	8.78	*F(1,50)=6.97, p= **.011***	*0.122*
RE	84.61	36.79	92.31	21.72	*F(1,50)=0.84, p= .363*	*0.017*
MH	65.85	13.91	71.70	9.73	*F(1,50)=3.08, p= .085*	*0.058*
Disability						
MRS	0.83	1.66	0.00	0.00	*F(1,48)= 8.57, p= **.014***	*0.120*
FAI	28.69	11.89	40.46	4.39	*F(1,50)=22.42, p< **.001***	*0.310*
BI	88.65	25.87	100.00	0.00	*F(1,50)=5.00, p= **.030***	*0.091*
GCS	12.46	4.197				
Mood						
GDS-SF	3.58	4.06	0.96	1.15	*F(1,50)=9.98, p= **.003***	*0.166*
GHQ-28						
Total	1.56	1.50	1.46	2.71	*F(1,42)= 0.02, p= .895*	*0.001*
Somatic Symptoms	0.56	0.70	0.69	1.49	*F(1,42)=0.13, p= .720*	*0.003*
Anxiety and Insomnia	0.56	0.92	0.53	1.50	*F(1,42)=0.00, p= .966*	*0.025*
Social Dysfunction	0.44	0.78	0.23	0.59	*F(1,42)=1.07, p= .307*	*0.025*
Symptoms of Depression	0.00	0.00	0.00	0.00	*F(1,42)=.000, p > .05*	*0.000*

BI=Barthel Index; BP=Body Pain; FAI=Frenchay Activity Index; GCS=Glasgow Coma Scale; LHS=London Handicap Scale; MCS=Mental Component Score; MH=Mental Health; MRS=Modified Rankin Scale; PCS=Physical Component Score; PF=Physical Functioning; RP=Role Physical; GDS-SF=Geriatric Depression Scale-Short form; GH=General Health; GHQ-28=General Health Questionnaire; RE=Role Emotional; SF-36=Short Form-36; SF=Social Functioning; VT=Vitality

functioning, body pain, vitality, general health, mental health and role limitations due to mental health). An ANOVA with group as the grouping variable and LHS, SF-36, MRS, FAI, BI, GDS-SF and GHQ-28 (listed in Table 9) as dependent variables showed that the two groups were significantly different on means and the effect sizes ranged from small to large (κ=0.001 to 0.352), which suggests that the difference between the groups varies from small to large. On the measures of mood and emotion, the SAH group scored

significantly lower on the measure of GDS-SF, however did not differ significantly on the GHQ-28 total as well as the subscales. Although the SAH group had significantly lower scores on the GDS-SF, no differences were noted on the symptoms of depression subscale of the GHQ-28. The GDS-SF items that contributed to a significance difference between the groups included satisfaction with life, dropped activities, feeling that life is empty, feeling helpless, wanting to stay at home and experiencing memory problems. However, GHQ-28 subscale of depression does not include these items which may explain why the difference between the groups is not significant on the symptoms of depression subscale.

Section 3: Change over time

This section examines the relationships between baseline and 6-month assessments and current (5-year) functioning. These changes are presented for the SAH-group only as the controls were only assessed at the 5-year follow-up.

Change Over Time

Changes from baseline, 28-day and 6-month assessment to the present 5-year follow-up are examined for the SAH group. Not all measures were administered at each assessment. Those which were administered during at least 2 assessments include the BI, FAI, MRS, SF-36, and GHQ-28.

Table 10

Change over time for SAH group on measures of disability

Measures	Baseline		28-day		6-month		5-years		Change over time
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	significant levels
BI	72.22	39.28	------		-------	--------	88.65	25.87	F(1,25)=180.09,p< .001
FAI	33.46	5.51	------		26.55	11.08	28.69	11.89	F(1,24)=349.42,p< .001
MRS	------	------	1.89	1.62	-------	-------	0.83	1.66	F(1,23)=23.12,p< .001

BI=Barthel Index; FAI=Frenchay Activity Scale; MRS=Modified Rankin Scale

Table 10 presents the means at relevant time frames for the BI, FAI, and MRS,

for the SAH participants as well as the significance of change over time. These analyses of change over time were conducted using repeated measures within-subjects comparisons. As can be seen in Table 10, the BI improved significantly from baseline to 5-year follow-up, as did overall levels of disability as measured by the FAI, which worsened from baseline to 6-months, though it showed a moderate improvement in functioning between 6-months and 5-years. In contrast, the MRS showed a significant decrease in levels of disability from 28-days to 5-years.

Table 11

Change over time for SAH group on the SF-36 measure of quality of life and its subscales

Measures SF-36	6-months		5-years		Change over time Significant values
	Mean	SD	Mean	SD	
MCS	77.08	22.41	72.52	18.53	F(1,20)=281.17,p<.001
PCS	70.20	24.55	71.18	25.11	F(1,20)=168.28,p< .001
PF	71.36	33.88	60.58	38.51	F(1,20)=88.92,p<.001
RP	67.05	38.87	75.00	42.43	F(1,20)=72.07,p< .001
BP	79.89	28.40	76.63	24.20	F(1,20)=227.81,p<.001
VT	66.14	23.10	59.81	12.69	F(1,20)=322.54,p<.001
GH	62.50	12.42	72.50	26.00	F(1,20)=370.28,p<.001
SF	81.82	28.02	79.81	27.40	F(1,20)=197.96,p<.001
RE	77.27	37.64	84.61	36.80	F(1,20)=138.16,p<.001
MH	83.09	14.02	65.85	13.91	F(1,20)=23.464,p< .001

BP=Body Pain; GH=General Health; MCS=Mental Component Score; MH=Mental Health; PCS=Physical component score; PF=Physical functioning; RE=Role emotional; RP=Role Physical; SF=Social functioning; VT=Vitality

Table 11 shows the change in HRQoL from 6-months to 5-years post-SAH. As seen in the table, the scores of SF-36 changed significantly over time. Within this measure, scores improved for the PCS, indicating better physical functioning. In contrast, scores reduced significantly on the MCS suggesting a worsening of mental functioning. Regarding SF-36 subscales, the scores decreased significantly on PF, BP, VT SF, and MH suggesting reduced functioning in the areas of physical functioning,

body pain, vitality, social functioning and mental health. On the other hand, a significant increase in the scores of subscales such as RP, GH and RE suggested better general health and fewer role limitations due to physical and emotional functioning.

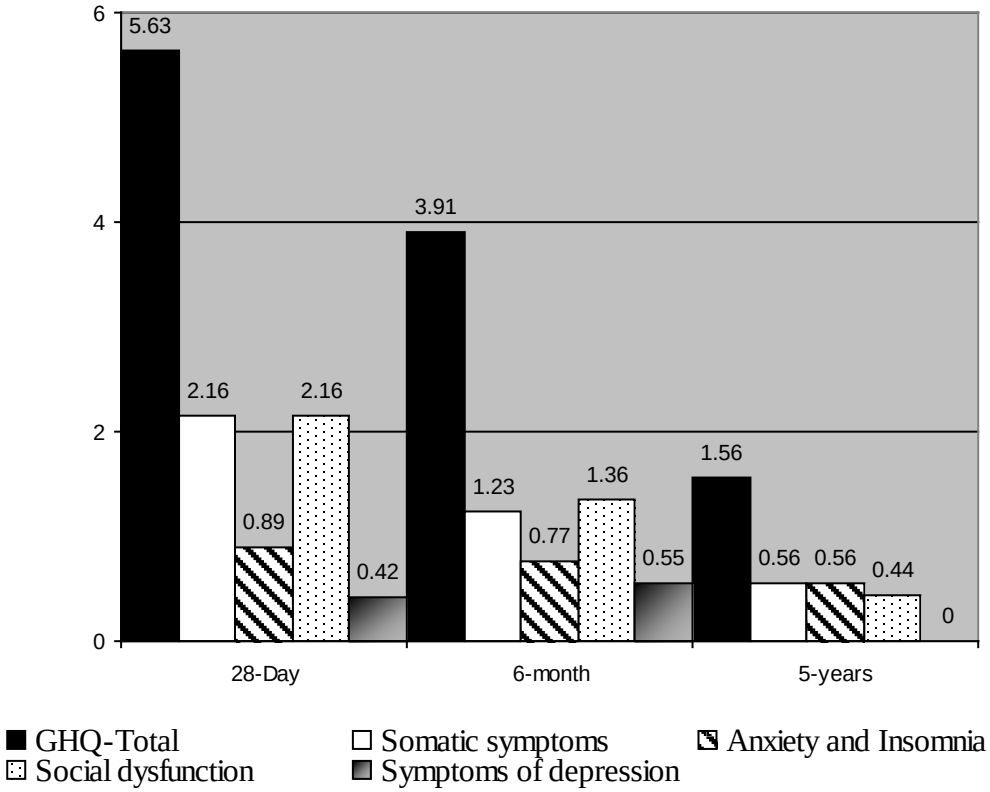

Figure 4

Changes over time for SAH group on the General Health Questionnaire (GHQ-28) total and scale scores

Figure 4 presents total scores obtained on the GHQ-28 at 28-days, 6-months and 5-years. When within subjects repeated measures ANOVAs were conducted, GHQ-28 total scores reduced significantly across the three time periods [F (1,11)=23.90, p= .001]. Within this measure scores reduced significantly for the subscales of somatic symptoms [F(1,11)=17.91,p=.001], anxiety and insomnia [F(1,11)=11.88,p=.005] and social dysfunction [F(1,11)=29.68,p=.001]; while no significant change occurred on the symptoms of depression subscale [F(1,11)=1.80, p>.05]. The results indicate that anxiety and insomnia seemed to have changed very little as compared to other subscales of GHQ-28.

Impact of baseline demographics and current functional and cognitive outcomes

In order to determine the impact of demographic factors and test results at 6-months to current functioning (i.e., disability, handicap, quality of life, mood, and neuropsychological functioning) ANOVAs were conducted between demographic factors (i.e., age, gender, ethnicity, education), injury characteristics (i.e., SAH severity assessed using GCS), and current performance on neuropsychological measures (i.e., verbal memory, visual memory, information processing, visuoperceptual functioning, language, executive functioning, overall impairment), mood (GHQ-28 and GDS-SF), and functional outcomes of SAH survivors. Functional outcomes of interest include: quality of life (SF-36); disability (MRS, FAI, and BI); and handicap (LHS).

This was followed by examining the relationship between test results at baseline and 6-months (MRS, FAI, GHQ-28, SF-36) and current functioning. Current functioning included: disability/handicap (MRS, BI, FAI), quality of life (SF-36 MCS and PCS) and neuropsychological outcomes (verbal memory, visual memory, information processing, visuoperceptual functioning, language, executive functioning, overall impairment). For simplicity of presentation, these will be examined as correlations with demographic and stroke characteristics first, followed by those with baseline functioning and current functioning. Using Pearson's bivariate correlations, the results indicate that age and GCS score at time of stroke were not significantly related to any of the outcomes of interest ($p > .01$).

To examine the relationship between demographic variables (i.e., gender, ethnicity, and education), recurrent stroke and functional outcomes, one way ANOVAs were conducted with the demographic variables and recurrent stroke as the group variables for each ANOVA and current functioning as dependent variables. Current functioning included: disability/handicap (MRS, BI, FAI), quality of life (SF-36 MCS

and PCS), mood (GHQ-28, GDS-SF) and neuropsychological outcomes (verbal memory, visual memory, information processing, visuoperceptual functioning, language, executive functioning, overall impairment). The results indicate that those who had a recurrent stroke did not differ significantly on any of the outcome measures as compared to those who reported having a recurrent stroke suggesting that recurrent stroke was not related to functioning of 5-years post-SAH survivors. Regarding gender, women did not significantly differ from men on the neuropsychological functioning outcomes. For functional outcomes, females had worse scores on HRQoL domains as compared to men with significantly lower scores than the males on only the SF-36 MCS [$F(1,24)= 4.521$, $p= .044$], which indicated that women had worse outcomes regarding emotional wellbeing. No significant differences were noted on any other functional outcome measures.

For ethnicity, the results suggest that the two groups (i.e., European and others) differed significantly on the somatic symptoms subscale of GHQ-28 [$F(1,16)= 8.260$, $p=.011$] with worse scores for 'others' group. Within the neuropsychological functioning area the groups differed significantly on visuoperceptual [$F(1,20)=12.04$, $p=.002$] and information processing domains [$F(1,19)=5.231$, $p=.034$] with European group performing better.

Table 12 presents bivariate correlations among the measures at different times. As seen in Table 12, higher scores on MRS (more disability) assessed at 28-days post SAH correlated with worse MRS, BI, FAI, both MCS and PCS of the SF-36, and GHQ-28 total, and somatic symptoms at 5-years. Better performance on FAI at 6-months correlated significantly with improved BI, FAI, MRS and SF-36 (PCS and MCS) at 5-years. Furthermore, higher scores on GHQ-28 total at 6-months (abnormal mood) correlated significantly with increased social dysfunction (GHQ-28) and poor MCS and

PCS (SF-36) at 5-years. Within the GHQ-28 subscales, increased somatic symptoms and social dysfunction correlated significantly with increased social dysfunction (GHQ-28) and poor PCS and MCS at 5-years. Increased symptoms of depression (GHQ-28) only correlated significantly with poor MCS and PCS (SF-36) at 5-years and with none of the other measures. No significant correlations emerged between anxiety and insomnia (GHQ-28) at 6-months and other functional measures at 5-years. At 6-months a better performance on MCS (SF-36) was significantly related to better performance on BI, MRS, FAI, GHQ-28 somatic symptoms, MCS and PCS at 5-years. Lastly higher score on PCS (SF-36) at 6-months was significantly correlated with better BI, MRS, FAI, GHQ-28 total and somatic symptoms, MCS and PCS at 5-years.

Table 12

Correlations of Functional Outcomes over time

	5-year Function measures								
	BI	FAI	MRS	GHQ-28				SF-36	
				Total	SS	AI	DS	PCS	MCS
28-Day									
MRS	-.708**	-.869**	.743**	.503*	.480*	ns	ns	-.592**	-.778**
6-Month									
FAI	.778**	.833**	-.747**	ns	ns	ns	ns	.568**	.544**
GHQ-28 -Total	ns	ns	ns	ns	ns	ns	.661**	-.639**	-.546*
-Somatic Symptoms	ns	ns	ns	ns	ns	ns	.661**	-.676**	-.543*
-Anxiety and Insomnia	ns	ns	ns	ns	ns	ns	ns	ns	ns
-Social Dysfunction	ns	ns	ns	ns	ns	ns	.765**	-.461*	-.469*
-Symptoms of Depression	ns	ns	ns	ns	ns	ns	ns	-.593*	-.481*
SF-36-MCS	.740**	.737**	-.704**	ns	-.614*	ns	ns	.935**	.834**
-PCS	.693**	.838**	-.738**	-.515*	-.676**	ns	ns	.891**	.903**

AI=Anxiety and Insomnia; FAI=Franchay Activity Index; MCS=Mental Component Score; MRS=Modified Rankin Scale; PCS=Physical Component Score; GHQ-28=General Health Questionnaire-28; SF-36=Short Form-36; SS=Somatic Symptoms; SD=Social Dysfunctions

Section 4: Relationship between neuropsychological deficit, mood and functional outcomes/quality of life at 5-years

In this section, a series of bivariate correlation matrices were generated to examine the degree and direction of the relationships between areas of neuropsychological deficit and functional abilities/quality of life and mood at 5-years post-SAH. These analyses were conducted for the SAH group only. Due to the number of correlations being generated and the small size of the sample a conservative level of significance was used ($p < .01$). Those significant at the $p < .05$ level are identified, but interpreted with caution.

Relationship between neuropsychological functioning and functional outcomes

To examine the relationships between neuropsychological impairment (i.e., verbal memory, visual memory, information processing, visuoperceptual functioning, language, executive functioning, overall neuropsychological impairment) and functional outcomes (i.e., disability/handicap [MRS, BI, FAI], quality of life [SF-36 MCS and PCS]), Pearson's bivariate correlations were generated and are presented in Table 13. These correlations indicate that overall neuropsychological impairment was significantly related to all disability outcomes (BI, MRS, FAI), handicap (LHS) and HRQoL (SF-36). Within the neuropsychological domain scores, no significant relationship was noted amongst verbal memory and functional outcomes. However, poorer performance on visual memory was significantly related to greater disability (FAI) and reduced HRQoL (SF-36), though no significant relationship was noted to handicap. Worse performance on the language domain was also significantly related to worse outcomes on the BI, MRS, LHS and SF-36. Performance on visuoperceptual abilities was significantly related to greater disability (FAI) and poorer HRQoL (SF-36). Regarding executive functioning, the only significant relationships noted were with HRQoL (MCS, RE, MH). Although a significant relationship was found between information processing and disability (MRS,

FAI, BI) and HRQoL (SF-36); the relationship with handicap (LHS) was not significant. Lastly, neither body pain nor vitality was significantly related to any of the neuropsychological domains.

Table 13

Correlations between Neuropsychological functioning and Functional outcomes at 5-years

	Overall NP[a] impairment	Verbal Memory	Visual Memory	Language	Visuoperceptual	Executive functioning	Information processing
BI	-.540*	ns	ns	.463*	ns	ns	.831**
MRS	.535*	ns	ns	-.493*	ns	ns	-.774**
FAI	-.666**	ns	.460*	ns	.564**	ns	.464*
LHS	-.708**	ns	ns	.797**	ns	ns	ns
SF-36(MCS)	-.679**	ns	.562**	.549*	.462*	.590**	.584**
SF-36(PCS)	-.633**	ns	.463*	.594**	.505*	ns	.482*
PF	-.575**	ns	ns	.545*	.472*	ns	.528*
RP	-.554**	ns	.520*	.664**	ns	ns	ns
BP	ns	ns	ns	ns	ns	ns	ns
VT	ns	ns	ns	ns	ns	ns	ns
GH	-.470*	ns	ns	ns	.630**	ns	ns
SF	-.652**	ns	.570**	.603**	ns	ns	.599**
RE	-.599**	ns	.510*	ns	ns	.741**	.612**
MH	-.544*	ns	.447*	ns	.536*	.508*	ns

AI= Anxiety and Insomnia; BP=Body Pain; DS=Depression Symptoms; FAI=Frenchay Activity Index; GH=General Health; LHS=London Handicap Scale; MCS=Mental Component Score; MH=Mental Health; MRS=Modified Rankin Scale; PCS=Physical Component Score; PF=Physical Functioning; RP=Role Physical; SD=Social Dysfunction; SF-36=Short Form 36; SF=Social Functioning; SS=Somatic symptoms; RE= Role emotional; VT=Vitality

[a] Overall Neuropsychological Impairment.

ns $p > .05$

* $p < .05$

** $p < .01$

Relationship amongst 5-year functional outcomes

It is important to establish whether functional outcomes have expected interrelationships. Therefore correlations were generated amongst measures of disability (MRS, FAI, BI), handicap (LHS) and HRQoL (SF-36) and are presented in Table 14. For SF-36 it was decided to include only the component scores (MCS and PCS) as generating correlations for the subscales would have resulted in too many correlations, reducing power to detect real effects. Moreover the relationships between subscales are well established (Ware & Sherbourne, 1992).

Table 14

Correlations amongst measures of disability, handicap and quality of life at 5-years post SAH

	LHS	MRS	FAI	BI	MCS
MRS	-.681**				
FAI	.612**	-.663**			
BI	.708**	-.874**	.702**		
MCS	.651**	-.498*	.494*	.586**	
PCS	.701**	-.549**	.732**	.534**	.713**

LHS=London Handicap Scale; MRS=Modified Rankin Scale; BI=Barthel Index; FAI= Frenchay Activity Index; MCS=Mental component Score; PCS=Physical Component Score
*p< .05
**p< .01

As seen in Table 14, increased handicap (LHS) was significantly related to increased disability. Similarly, a significant relationship was noted between LHS and SF-36 component scores. As expected the highest correlation was found between the two most similar measures of overall disability (BI and MRS). Whereas less related construct such as the SF-36 MCS had weaker, though still significant, relationships to these more

physically based measures of disability. However, the SF-36 MCS had a marginally stronger relationship to the BI than the SF-36 Physical component score, which was unexpected.

Relationship between neuropsychological outcomes

A series of bivariate correlations were generated to examine the relationship amongst neuropsychological domain scores, as presented in Table 15. As expected, overall neuropsychological impairment was significantly related to impairment across neuropsychological domains. Visual memory was significantly related to all other domains, however, verbal memory was only significantly related to visual memory and language. Language was significantly related to all the domains except executive functioning. No significant, relationship was seen amongst visuoperceptual and executive functioning (p> .05).

Table 15

Correlations amongst neuropsychological domains at 5-years post SAH

	Overall NP[a] impairment	Verbal memory	Visual Memory	Language	Visuoperceptual	Executive functioning
Verbal Memory	-.542*					
Visual Memory	-.754**	.576**				
Language	-.837**	.593**	.552*			
Visuoperceptual	-.579**	ns	.443*	.479*		
Executive functioning	-.652**	ns	.526*	ns	ns	
Information processing	-.764**	ns	.541*	.602**	.482*	.625**

[a] Overall Neuropsychological Impairment.
ns p > .05
*p< .05
**p< .01

Relationship between mood and neuropsychological and functional outcomes

A series of bivariate correlations were generated to examine the relationship between neuropsychological outcomes (i.e., verbal memory, visual memory, information procession, visuoperceptual functioning, language, executive functioning, overall neuropsychological impairment) and mood (GDS-SF and GHQ-28). As seen in Table 16, all functional outcomes were significantly related to depression as measured by GDS-SF. Whilst GHQ-28 total was significantly related to only LHS, the relationship with other functional outcomes measures was insignificant. Within the GHQ-28 scaled scores, only somatic symptoms was related to disability (MRS, FAI, BI), handicap (LHS) and SF-36 component scores (MCS and PCS). None of the correlations were significant between functional outcomes and GHQ-28 subscales of social functioning and anxiety (p> .05). Correlations were not generated for the depression scale of GHQ-28 as all the participants scored '0' on this subscale.

Table 16

Correlations between functional outcomes and mood at 5-years post SAH

	GDS-SF	GHQ-28 Total	GHQ-28 Somatic symptoms
LHS	-.712**	-.521**	-.639**
MRS	.667**	ns	.509*
FAI	-.680**	ns	-.499*
BI	-.664**	ns	-.500*
MCS	-.707**	ns	-.545*
PCS	-.705**	ns	-.537*

BI=Barthel Index; FAI=Franchay Activity Index; GDS-SF=Geriatric Depression Scale-Short form; GHQ-28=General Health Questionnaire; LHS=London Handicap Scale; MCS=Mental Component Score; MRS=Modified Rankin Scale; PCS=Physical Component Score;
ns p> .05
*p< .05
**p< .01

Relationships between neuropsychological outcomes (i.e., overall neuropsychological impairment, verbal memory, visual memory, executive functioning, processing speed, visuoperceptual abilities, language) and mood (GHQ-28 and GDS-SF) are presented in Table 17. Depression (GDS-SF) was only related to overall neuropsychological impairment, visuoperceptual, and executive functioning. On the other hand, GHQ-28 total and anxiety subscale did not have any significant relationship with any of the neuropsychological outcomes. Furthermore no correlations were generated for the depression subscale as all the participants scored '0' on this subscale. The only significant relationship was noted between somatic symptoms and information processing and social dysfunction and verbal memory and executive functioning.

Finally, correlations were also generated between the mood measures (GHQ-28 and GDS-SF). However no significant relationship emerged between GDS-SF and GHQ-28 (total and subscales).

Table 17

Correlations between neuropsychological domain scores and mood scores at 5-years post-SAH

	GDS-SF	GHQ-28 Somatic Symptoms	GHQ-28 Social Dysfunction
Overall Impairment	.504*	ns	ns
Verbal Memory	ns	ns	-.705*
Visual Memory	ns	ns	ns
Language	ns	ns	ns
Visuoperceptual	-.505*	ns	ns
Executive functioning	-.466*	ns	-.831**
Information processing	ns	-.581*	ns

GDS-SF=Geriatric Depression Scale-Short Form; GHQ-28=General Health Questionnaire-28
ns $p > .05$
* $p < .05$
** $p < .01$

Summary of inter-relationships

While consideration was given to the possibility of conducting regression analyses to examine what neuropsychological factors best predict functional outcomes at 5-years post-SAH, the small sample size suggests that this would be inappropriate (Tabachnik & Fidell, 2001). However, in summarising the above correlations, it can be seen that the reduced handicap on the LHS was significantly related to better overall neuropsychological functioning and even more so to better performance in the language domain. It was also significantly related to reduced depression scores on the GDS-SF, and better overall and somatic health (GHQ-28). Of note, Language performance was not related to GHQ-28 total or somatic scores, nor to GDS-SF; suggesting that this area of neuropsychological functioning may have an independent relationship to LHS.

Based on the above, it can also be seen that better activity levels (FAI) was significantly related to better overall neuropsychological functioning and visuoperceptual ability; and also had weaker although significant relationship with visual memory and information processing. FAI was also significantly related to reduced depression scores on GDS-SF, and better somatic health (GHQ-28). However, visual memory was not significantly related to either GDS-SF or to GHQ-total and somatic symptoms thus indicating that visual memory may have an independent relationship with FAI.

Regarding MRS, the correlations show that MRS has the strongest relationship with information processing followed by significant but a slightly weaker relationship with overall neuropsychological functioning and language. MRS is also significantly related to depression (GDS-SF) and somatic symptoms (GHQ-28). As mentioned earlier language is not significantly related to any of the mood measures (GDS-SF and GHQ-28), suggesting that it may have an independent relationship with MRS.

For MCS of the HRQoL measure used (SF-36), a significant relationship was

noted with better overall neuropsychological functioning, executive functioning and information processing. Although a significant relationship was also seen with the visual memory, language and visuoperceptual domains, this relationship was weaker as compared to the above mentioned relationships. Furthermore MCS was also significantly related to reduced depression scores on GDS-SF and somatic symptoms (GHQ-28).

Regarding the physical component score (PCS) of SF-36, a significant relationship was seen with better overall neuropsychological functioning and language and to a lesser degree with visual memory, visuperceptual and information processing domains. Reduced depression (GDS-SF) and somatic symptoms (GHQ-28) were also related to physical component of health related quality of life. As noted earlier language and visual memory was not related to any of the mood measures thus suggesting that these domains may have an independent relationship with both the physical and mental component scores of SF-36.

CHAPTER IV

DISCUSSION

This study examined the long-term (5-6 years) functional and neuropsychological outcomes of subarachnoid haemorrhage (SAH) survivors when compared to a healthy control group matched on age, gender and ethnicity. Specific aims were to identify measures on which the neuropsychological, general functioning (e.g., quality of life, disability) and mood of SAH survivors differ from matched controls; to explore the relationships between baseline, 28-day, 6-month assessments and current functioning of the SAH group; and to examine the relationships between areas of neuropsychological deficit, mood and functional abilities/quality of life for SAH survivors. Each of these is examined, in turn, below.

OVERALL PERFORMANCE AND GROUP COMPARISON

The results indicate that 5-year SAH-survivors have impaired overall neuropsychological functioning, with significant deficits in executive functioning, information processing, verbal and visual memory, abstract visuoperceptual problem solving and language when compared to matched controls. The SAH group also had poorer functional outcomes with significantly worse performance on all measures of disability and handicap, and selected areas of HRQoL. Reduced HRQoL was evident in areas relating to mental health (i.e., mental component score [MCS]), and physical and social functioning. Regarding mood and emotion, while the SAH group was significantly more depressed as compared to the controls, they did not differ significantly on a measure of general health and well being (i.e., GHQ-28). Each of these is discussed in relation to the literature below. It must be acknowledged prior to this discussion that a

large number of between group comparisons were conducted while this could then increase the likelihood of errors. The large size of effects found suggests that difference between group identified as significant are in fact true differences.

Neuropsychological functioning

As compared to the controls, the SAH group had worse overall neuropsychological impairment and showed deficits across the assessed areas. Current findings are consistent with the previous research that reported that SAH survivors experience cognitive deficits as compared to controls (Hadjivassiliou et al., 2001; Mavaddat et al., 1999; Powell et al., 2004; Ravnik et al., 2006; Samra et al., 2007). These previous studies were not population-based, included only participants with a favourable outcome (GOS=4-5) and testing time post-SAH ranged from 1-year to 41-months. Moreover these studies used computerised batteries, a limited number of tests, or did not assess all the domains that we assessed in our study (Mavaddat et al., 1999; Ravnik et al., 2006; Samra et al., 2007). While the mean scores of the SAH group on the all the neuropsychological tests were lower than that of controls, these differences were not significant on some measures of executive functioning (i.e., IVA-CPT visual attention and auditory impulsivity), verbal memory (i.e., CVLT-II short delay free and cued recall, long delay cued recall, recognition hits and false positives), visual memory (i.e., ROCF copy, and recalls) and visuoperceptual ability (i.e., block design). The results of present study are similar to findings by Ogden et al. (1990) who reported that at 5-years, SAH survivors had mild to severe impairments in the domains of memory, visuospatial, and executive functioning. In contrast, the present study also found that SAH survivors had impaired information processing speed and language. However, Ogden et al. (1990) had a small sample (n=16) which excluded people who did not speak English and did not compare the SAH-group to matched controls. Moreover, they used only one test each for

information processing and language. Their results differ from the present findings because of the different measures used as the measures used in the present study are more sensitive to cognitive deficits.

In the wider stroke literature, as compared to controls, stroke-survivors have been found to be more impaired across cognitive domains (Rasquin et al., 2005; Srikanth et al., 2006), and are more at risk of developing dementia (Desmond et al., 2002; Srikanth et al., 2006). However, in this study none of the participants reported having a dementia diagnosis, and only one participant met dementia cut off on HAMT. This could be because SAH-survivors are usually much younger as compared to stroke survivors, with more than half of SAH-survivors usually being under 55 years of age (The ACROSS Group, 2000). Thus, consistent to previous research in stroke and SAH-survivors, the current findings indicate that even 5-years after SAH, long-term cognitive impairments exist across various cognitive domains.

In terms of alternative explanations for poorer cognitive performance in the SAH group, while worse performance in the SAH group may have been due to lesser tertiary education in SAH participants as compared to controls, correlations between education and current neuropsychological functioning were not significant, suggesting this was not the case. Another alternative is the possibility of some additional process (e.g., a further stroke, degenerative process) contributing to between group differences. However, only 2 of our participants reported having a recurrent stroke and none of them reported having dementia, with only one meeting criteria for this on the HAMT. Out of these three participants, two did not complete neuropsychological tests. Given this, it is unlikely that these factors impacted significantly on the current findings.

Disability and Handicap

The SAH group had poorer performance on all the functional outcome measures when compared to controls with significantly poorer performance on the measures of disability and handicap. Although research examining long-term disability and handicap is limited for SAH survivors, studies of long-term stroke survivors more generally have yielded similar results. For example, Haug et al. (2010) reported that approximately 39% of the SAH survivors were disabled at 1-year post-SAH. Similarly, Hankey et al's. (2002) population based study reported that at 5-years post-stroke approximately 1/3 survivors remain disabled. These studies did not compare stroke survivors to matched controls. Regarding handicap, current results are similar to a previous stroke study where handicap (measured by LHS) increased at 3 and 12-months post-stroke as compared to that prior to stroke (Sturm et al., 2002); though this previous study excluded SAH-survivors. Thus, findings of the current study are in line with those reported by population-based studies examining long-term post-stroke functional outcomes; in that disability and handicap remain prevalent many years after stroke (Chausson et al., 2010; Dhamoon et al., 2009; Hackett et al., 2000). Even at 6-years post-stroke, individuals are commonly not fully recovered and are dependent on others for at least one activity of daily living (Bonita et al., 1997; Hankey et al., 2002).

Quality of life

The SAH-group reported worse HRQoL across all domains of the SF-36 as compared to the controls, though only significantly so for MCS, physical functioning and social functioning subscales. While current findings are similar to others who found that patients with SAH had significantly lower scores on all SF-36 domains at 5-years post-SAH as compared to controls (Scharbrodt et al., 2009), they suggested more restricted areas of difficulty. However, in Scharbrodt et al's (2009) study was not population based,

the study controls were not matched and the SAH-survivors were compared to general population norms. In the current study, SAH-survivors experienced the most significant problems on the MCS and subscales of physical and social functioning. Similar to these findings, a number of long-term studies have found that post-SAH, individuals experience reduced HRQoL; with difficulties in the domains of emotional and physical health which impact their social functioning (Beseoglu et al., 2010; Hackett & Anderson, 2000; Visser-Meily et al., 2009). However, these studies have also reported problems in general health, body pain, vitality, and in role limitations due to physical and emotional health at 1-year to 32-months post-SAH (Beseoglu et al., 2010; Haug et al., 2009; Haug et al., 2010). It is possible that at 5-years although problems in these areas may still exist, these may be underreported by the SAH group or may resolve more over time than those in emotional and social areas. Another study by Hütter et al. (1995) used a self-rating scale to measure HRQoL among SAH-survivors up to 5-years post-SAH and found overall reduced HRQoL including reduced motivation, interests, mental capacity, pleasurable activities, social relationships, fine motor coordination, concentration and sleep. Although some of the affected areas are similar to the ones found in the present study (i.e., mental component score, physical functioning and social functioning), difference in other areas may be because the measures used in the two studies differed. Further, the time span for assessment varied in this study from 1-to 5-years, thus time post-SAH is likely to have complicated the findings, not providing an accurate picture.

Mood

Consistent with the previous research, the SAH group in the current study performed poorly on the primary measure of mood (GDS-SF) as compared to controls reporting significantly more depressive symptomatology than controls (Berry et al., 1997; Tidswell et al., 1995; Visser-Meily et al., 2009). In the present study approximately 30%

of the SAH participants met the GDS-SF cut-off (≥5) for probable depression; whereas Lindberg (1995) reported that at 7-years post-SAH 22% were classified as depressed using the Zung's Self-Rating Depression Inventory. However, contrary to previous findings no significant differences between the groups were observed in this study on overall wellness and psychological health as measured by the GHQ-28. The results indicate that although the means of the two groups on GHQ-28 were not significantly different, there was a large spread of scores within both groups, which probably contributed to this overlap between groups. In relation to the depression subscale of GHQ-28, this scale encompasses items relating to severe depression (e.g., self-harm and suicidal ideation). The participants in our study did not endorse any such items and therefore both the groups scored '0' on this subscale; indicating that none of the participants in either group were severely depressed even though depression was evident to a lesser degree as per GDS-SF scores. Previous studies have reported that long-term SAH-survivors complain of fatigue, anxiety, irritability, and problems with sleep (Berry et al., 1997; Hütter et al., 1995; Wermer et al., 2007) and these areas are covered by the GDS-SF and the Zung Depression Scale. The presence of such difficulties is further supported by lower scores on the vitality subscale of SF-36, which covers the aspects related to fatigue. Thus depression scales which allow for greater range of difficulties (GDS-SF and Zung Depression Scale) show consistency in findings, with the discrepancy between this and GHQ-28 depression due to the restricted nature of this task.

Although previous research has noted that anxiety post stroke and SAH is common (Barker-Collo, 2007; Powell et al., 2002; Visser-Meily et al., 2009), in our study no significant differences on the GHQ-28 anxiety subscale were noted between the SAH and control groups. Similar to the above discussion of depression, an explanation thus could be the limitation of GHQ-28 as it may not be sensitive or generic enough to assess

the psychological domains of mental health such as mild to moderate anxiety. For example, the GHQ-28 items relating to anxiety and insomnia have limited number of items (n=7) covering mostly emotional items (e.g., feel under strain, scared, panicky, nervous) and does not cover the somatic items related to anxiety. Thus, it may be more appropriate to use Beck Anxiety Inventory (Beck, Steer, Ball, & Ranieri, 1996) or Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) for this sample as the items in these tests cover wider aspects related to anxiety (i.e., both emotional and physical symptoms) and also assess levels of anxiety (i.e., mild to severe). A surprising finding was that although the groups were significantly different on the social functioning subscale of SF-36, they were did not differ significantly on social dysfunction subscale (GHQ-28). As with depression and anxiety this is likely to reflect that the two tests measure different things. The items on the GHQ-28 are more related to individual tasks that are related to social functioning (e.g., taking longer over things, being occupied, doing things well). Whereas the SF-36 directly measures the extent and amount of time to which the physical and emotional health interferes with the social activities.

Considering the above findings, it can be concluded that GHQ-28 may not be very suitable for this population as it does not capture the full breadth of depression and anxiety symptoms as compared other scales such as GDS-SF do or perhaps (HADS). A study using the HADS reported that at 8.9 years (mean testing time) SAH-survivors had significantly higher scores on depression but not on anxiety as compared to population norms (Wermer et al., 2007).

CHANGE OVER TIME

As discussed earlier, whilst the SAH group had significantly lower scores on disability measures as compared to controls, significant change over time (i.e. from

baseline to 5-years post-SAH) was noted with reduced levels of disability. These results support previous research where functional status of SAH-survivors improved over time (i.e., from onset to 28-months) (Dombovy, Drew-Cates & Serdans, 1998; Mocco et al., 2006). Hop, Rinkel and Algra (2001) also reported an improvement in functioning with 50% of participants having reduced disability (measured by MRS) at 18-months compared to 4-months post-SAH. However, in their study part of the initial sample was lost to follow-up and patients with cognitive impairments were excluded. This may have affected the results, as functioning is more likely to be reduced in participants with cognitive deficits. Similar findings were reported in a study of stroke-survivors with improved BI scores from baseline to 77.4 months post-stroke (Pan, Wu, Lee & Chen, 2007). In the current study 44.9% (n=12) of SAH-survivors stated they had not recovered completely at 5-years, which is similar to previous population-based findings where 46% of the participants had not recovered completely from the SAH at 1-year post-SAH (Hackett et al., 2000).

In this study, scores on FAI were higher (indicating less disability) at baseline as compared to those at 6-months or 5-years post-SAH. However, this is only because the participants' scores at baseline reflected their activity levels 3-months before SAH. Thus, as compared to the activity level prior to SAH, their overall activity level has reduced significantly at 5-years post-SAH. By contrast, it is important to note that significant improvements from 6-month to 5-year post were noted. Similarly, in other stroke studies ADL capacity has been found to improve gradually over time, with 76% stroke-survivors reporting independence in ADLs at 2-years as compared to 55% at the acute stage (Ahlsiö, Britton, Murray, & Theorell, 1984).

Although studies have examined long-term HRQoL post-SAH, this is to the researcher's knowledge the first population-based follow-up study that compared the

HRQoL at the acute stage (28-day) post-SAH to 5-years post-SAH. The results of this study replicate those of an earlier study where participants experienced improvements in physical functioning and physical role but impaired overall HRQoL at 5-years as compared to 3-months post-SAH (Scharbrodt et al., 2009). As this other study excluded cases with severe SAH, which was not the case here, their findings are likely to have been more restricted and less generalisable. While the SAH survivors reported significant improvement in overall physical functioning (PCS), they reported worsening of the overall mental health functioning (MCS) and reduced functioning specific to the areas of physical functioning, body pain, vitality, social functioning and mental health. A longitudinal study conducted at 1-week, 6-months and 2-years post-stroke found that over time HRQoL in participants deteriorated progressively with increased disability (Ahlsiö et al., 1984). Similarly, a study reported HRQoL assessed by Visual Analogue Scale (VAS) and in all domains of SF-36 improved at 18-months as compared to that reported at 4-months post-SAH, particularly in participants who had improvements in disability as measured by MRS (Hop, Rinkel, Algra & Van Gijn, 2001), which is similar to the current findings. Although Hop et al. (2001) reported that VAS is a useful measure to assess change in HRQoL; they concluded that increased SF-36 scores reflected reduced disability and therefore it is a better measure of HRQoL. For stroke survivors, findings are similar to the current SAH study where the stroke survivors reported reduced HRQoL at 24-months on the VAS and SF-36 when compared to the normative data (van Zandvoort et al., 2005). This study also excluded patients with poor functional outcomes at the acute stage which might have impacted the findings and reduced their generalisability. Another study by Mocco et al. (2006) reported that a significant improvement in the physical and psychosocial scores occurred between 3-and 12-months post-SAH. A long-term study examined the HRQoL using VAS and found

that, as compared to their HRQoL prior to SAH, 14% had increased, 38% had decreased, and 48% reported unaltered HRQoL at 7-years (mean time) post-SAH (Lindberg, 1995).

To summarise the findings of HRQoL, the current findings indicate that SAH-survivors participants have reduced HRQoL even at 5-years post SAH. Although the participants in this study have improvements in certain areas such as overall physical functioning domain, general health and role limitations due to the physical and emotional health, their emotional health had declined significantly. This could be that after 5-years whilst they have come to terms with their SAH experience and their physical problems, they still have reduced energy and vitality, are experiencing more body pain; and problems in physical and social functioning, which perhaps impacts their mental health. These results replicate previous findings that stroke and SAH survivors experience declines in HRQoL (Haug et al., 2010; Kreitschmann-Andermahr et al., 2007; Mayer et al., 2002; Vilkki et al., 2004) however these studies were not population based, were done at shorter durations post-SAH or stroke, excluded severe cases and used limited measures (e.g., checklists or interview) to examine HRQoL.

The well-being of the SAH group (i.e., GHQ-28) improved significantly over time with significant reductions of scores in the areas of somatic symptoms, anxiety and insomnia and social dysfunction. In another long-term study, Wermer et al. (2007) used a semi-structured questionnaire and found that at 8.9 years (mean follow up time), SAH-survivors reported increased agitation, apathy, emotionality with positive overall change in personality. Similarly, Bosworth, Horner, Edwards, & Matchar (2000) also noted that stroke patients evaluated their health status as slightly improved at 6-months, followed by a slight decline at 12-months which is often impacted by their physical functioning, psychological health and their marital and living situation (i.e., living alone, with others, institutionalized) which is not consistent with current findings which found that

wellbeing improves over time. This difference in findings could be due to the variation in the assessment timing in the above mentioned studies. It is possible that whilst the wellbeing of survivors is poorer than the controls at 5-years post-SAH, it has improved over time and participants feel relatively better as compared to how they felt at 6-months post-SAH. Consistent with previous SAH and stroke research, whilst the scores on the depression subscale reduced over time in the current study, the scores did not reach a significant level (Alfeiri et al., 2008; Rasquin et al., 2005). However, it is important to note that in the current study, none of the participants endorsed any items related to the GHQ-28 depression subscale. It is possible that SAH-survivors may have been more hopeless/suicidal at 6-months post-SAH when they endorsed the depression symptoms as they were still coming to terms with their SAH experience; whereas at 5-years post-SAH, some acceptance of the experience and coping with its outcomes is likely to have occurred. Another possibility for reduced depression at 5-years post SAH is that survivors are functionally less impaired so have less concerns to be depressed about. Similar to this study, a stroke study also reported that although the prevalence of depressive symptoms reduced over time (from 3-months to 7-months), there was no significant difference in the prevalence of depressive symptoms at baseline and follow-up (Nys et al., 2006). Previous longitudinal studies on stroke survivors done at various time points also reported survivors experiencing depression for a long time after stroke (Berg et al., 2003). As stated earlier, in the current study, approximately 30% of the SAH-survivors met the GDS-SF criteria of depression. Similarly, Carota et al. (2005) have also reported reduced rates of depression of 34% at 3-months to 24% at 12-months post-stroke. In the current study, because GDS-SF was administered only at 5-years, therefore the change in depression rates as per GDS-SF could not be examined. To summarize, overall findings are in line with the previous research that although depression reduces over time, it is still

prevalent in survivors at 5-years post SAH.

FACTORS ASSOCIATED WITH FUNCTIONAL OUTCOMES AT 5-YEARS

Having examined the mood, neuropsychological and functional outcomes, and change from baseline to functioning at 5-years of SAH-survivors, this section discusses the factors (i.e., demographics, baseline functioning, mood and neuropsychological functioning) that were related to the functional outcomes at 5-years post-SAH.

Baseline demographics and current functional outcomes

The results indicate that demographic factors (i.e., age, education), recurrent stroke and injury characteristics were not significantly related to 5-year functioning. In current study the participants with tertiary education (approximately 55%) did not differ significantly from those without tertiary education on any of the functional and neuropsychological outcomes. The current results are similar to previous findings which reported that functional or cognitive outcomes for SAH were not related to any demographic characteristics including as age and education (Dombovy et al., 1998). The above mentioned study also had a small sample and used only one measure (Functional Independence Measure) to assess functional outcome at 28-months post-SAH. Some studies that reported a relationship between age and long-term HRQoL consisted of small sample size; a number of participants lost to follow-up, and excluded patients due to aphasia (Haacke et al., 2006; Paul et al., 2005). In contrast to current findings, the literature points towards a relationship between recurrent-SAH and poorer physical functioning assessed by SF-36 (Scharbrodt et al., 2009). In regards to ethnicity, the two groups (i.e., European and others) in the current study differed significantly on somatic symptoms subscale (GHQ-28), visuoperceptual and information processing domain. Studies on stroke-survivors beyond SAH also report a relationship between ethnicity and functional and cognitive outcomes (Dhamoon et al., 2009; Hankey et al., 2002; Patel et

al., 2003). However, these studies assessed survivors at 6-months post stroke using screening tests (i.e., MMSE) for measuring cognition, assessed only disability (i.e., BI and FAI) and did not examine the HRQoL. Thus the current study stretches beyond these studies by providing more comprehensive information about the long-term outcomes after SAH using a battery of neuropsychological tests along with assessing disability, handicap and HRQoL.

The present study found that SAH severity (i.e., GCS) was not related to long-term neuropsychological and functional outcomes. This was an unexpected outcome of the study given that the literature has highlighted the relationship between initial injury characteristics and functional outcomes (Säveland et al., 1986; Sarrafzadeh, Haux, Kücler, Lanksch & Unterberg, 2004). These studies had short term follow-up up post-SAH and lacked detailed measures for outcome assessment (e.g., used only disability measures or clinical interview). Thus, the present study extends these findings. Similarly, studies on stroke survivors have suggested a relationship between long-term disability (MRS BI), handicap (LHS) and initial stroke severity but these studies excluded cases with SAH (Dhamoon, et al., 2009; Paul et al., 2005; Sturm et al., 2004a; Vibo, Kõrv & Roose, 2007). In the current study, the lack of relationship between injury characteristics (GCS) and current functioning could also be a reflection of the small number of participants. It is also possible that our results differ to those reported at 1-year post-SAH because at 5-years, improvement in functional outcomes could be contributing to the non-significant relationship between GCS and functional outcomes. Another explanation is that the participants with poorer outcomes may have died (n=3) during the 5-year follow-up period. Indeed, as indicated by the sensitivity analysis, those from the initial incidence sample who were not included in the current study (n=69) were significantly more dependent at baseline (as assessed by BI) than those who participated

in the current study.

Regarding gender, although women had poorer scores on disability and handicap, these differences were not significant. However, a possible trend for women to experience worse mental health difficulties (MCS) on the SF-36 as compared to men was noted. Whilst studies have noted that women experience worse functional outcomes in terms of disability and handicap post-stroke, the current findings did not suggest this (Fukuda, et al., 2009; Hackett et al., 2000; Sturm et al., 2002; Sturm et al., 2004a; Vibo et al., 2007). It is noteworthy that the current study's small sample size limited its power to detect differences between genders.

Where HRQoL is concerned, the present results replicate the same pattern as previous findings of SAH and stroke survivors (Scharbrodt et al., 2009; Vibo et al., 2007) where women reported poorer HRQoL in regards to mental health as compared to men. A previous New Zealand study also reported that as compared to men, women generally scored lower on all domains of HRQoL (SF-36) with particularly low scores (<50) on physical functioning at 6-years post stroke (Hackett et al., 2000). These women were older (≥75 years), resided in institutions and experienced limitations in their daily activities; and quality of life was assessed via the SF-12, a shorter version of SF-36, which might have affected the overall outcomes. A trend for women to be experiencing poorer HRQoL in the current study may have been due to their older age (mean age 65.1 years for women as compared to 58.5 years for men) and their perception of complete recovery (60% of women reported incomplete recovery as compared to 25% of men) which is consistent with literature that even in general population more women tend to be depressed as compared to men (Angst et al., 2002). It is also possible that although women and men recover similarly but women perceive their recovery as worse which could be linked to their feeling more depressed. In the current study, higher number of

women experiencing emotional problems such as depression as compared to men (40% versus 16.7%) could also be impacting on poorer HRQoL. This is consistent with previous findings that report that depression is an important determinant of poor HRQoL at 4-years post stroke (Haacke et al., 2006).

Baseline functioning and current functional outcomes

Of interest to this study was whether factors assessed at the time of SAH were related to functional outcomes at 5-years. Indeed functioning at baseline and 6-months were significantly related to current functioning. These results replicate those of previous studies which report that disability, HRQoL and emotional status in the acute stage after SAH are related to the long-term functioning of SAH and stroke survivors (Herrmann, Black, Lawrence, Szekely & Szalai, 1998; Al-Khindi, Macdonald & Schweizer, 2010).

Thommessen, Bautz-Holter and Laake, (1999) identified poor functioning in the acute phase post-stroke, including problems in ADLs particularly urinary incontinence (assessed by BI), increased the probability of living in an institution at 12-months. However, they examined only elderly (mean age 78.4 years) stroke survivors and excluded SAH cases. Within the wider stroke literature it has been suggested that initial functioning and baseline anxiety or depression are prognostically important factors for long-term HRQoL outcomes post-stroke (Ahlsiö, Britton, Murray & Theorell, 1984; Nys et al., 2006). Regarding functioning, baseline disability (defined by BI) has been shown to predict long term disability at 5-years post-stroke (Hankey et al., 2002). A review of literature has similarly suggested that ADLs at the time of stroke predict functional recovery after stroke (Kwakkel, Wagnaar, Kollen, & Lankhorst, 1996) and our findings replicate these findings. Thus, the current study highlights the importance of baseline functioning that can help in predicting the functional outcomes at a later stage. Clinically the findings of this study are particularly important given that appropriate treatment (e.g.,

therapeutic or pharmacological interventions such as antidepressants) may be rendered at a much earlier phase to alleviate mood problems that can impact the HRQoL at a later stage in the longer-term. It also points to the need to be more diligent in ensuring these are addressed more in females where perceived recovery is worse than in males, despite similar levels of disability and handicap.

Relationships amongst disability, handicap and HRQoL

The literature has pointed towards a significant relationship between disability, handicap and HRQoL after stroke or SAH (Ahlsiö et al., 1984; Haacke et al., 2006; Kwon, Hartzema, Duncan & Lai, 2004; Noble & Schenk, 2009; Sturm et al., 2004a; Sturm et al., 2004b; Thommessen et al., 1999). This has been replicated in the current study where all the functional outcome measures correlated with each other. Consistent with current results, Patel et al. (2006) reported that correlations between disability and HRQoL domains are significant at both 1-and 3-years after stroke. Stroke survivors who require greater assistance with ADLs report significantly worse well being (i.e., sense of personal growth, social relations) as compared to those who were functionally independent (Clarke, Marshall, Black & Colantonio, 2002). It is important to note that in the current study, the degree of relationship varied amongst functional outcomes suggesting that these relationships are complex. This was also evident in a study which reported that individuals who gain functional independence at a mean of 10-years post-SAH continue to experience psychosocial consequences such as unemployment, working part time and having marital problems due to SAH, or experience emotional problems such as depression and anxiety (Wermer et al., 2007). Similarly, in a study of stroke patients, although 30% of the survivors were independent on ADLs (based on BI), they reported incomplete recovery when asked “have you made a complete recovery from your stroke”, and were also more handicapped than those who reported full recovery.

Thus, independence in ADLs is not equivalent to full recovery (Sturm et al., 2002).

Cognitive functioning, mood and functional outcomes

An additional area of interest to the present study was whether 5-year mood and cognitive functioning were related to functional outcomes at 5-years. The findings indicate a large number of significant relationships between handicap, disability, HRQoL and current neuropsychological functioning. A number of prior studies have also reported that participants with poor cognitive functioning experiencing worse functional outcomes such as HRQoL and disability as compared to those with good cognitive outcomes (Haug, 2010; Mayer et al., 2002; Springer et al., 2009). These prior studies only examined the outcomes up to 1-year post-SAH, used limited measures and excluded participants with severe neurological deficits including aphasia or with ruptured arteriovenous malformation. Whereas the present study included all SAH-survivors (i.e., all types of SAH, with poor neurological outcomes), who consented were included, and assessments included a battery of neuropsychological and functional tests. Thus the current findings extend the previous research.

In the present study, no significant relationship was noted between body pain, vitality and neuropsychological functioning which is contrary to the previous findings, which suggest that pain and fatigue have an impact on cognitive functioning (Lezak et al., 2004). The reason for this lack of association could potentially be because those SAH survivors, who refused to participate in the study, did so as they were experiencing fatigue or pain.

Furthermore, in the current study the only cognitive domain that did not have a significant relationship with any of the functional outcomes was verbal memory, which is an unexpected finding as many other previous studies have shown that impairments in verbal memory are related to significant limitations in daily functioning for SAH-

survivors (Haug, 2010). A possible explanation for this difference is that in the current study verbal tests were omitted for people who did have English as their first language (n=3), reducing the sample size. Thus, this may not provide a true picture of verbal memory or other language abilities.

With regards to handicap, in this study, overall neuropsychological impairment and language emerged as the domains association with reduced handicap in SAH-survivors. These findings vary from that of a stroke study which did not find any relationship between handicap and cognitive decline (Sturm et al., 2004a). However, this later study did not include formal testing of cognition and the results were based on reporting of "known history of dementia" which does not really yield a genuine profile of the cognitive functioning. On the other hand the present study assessed the neuropsychological functioning in detail thus giving a clearer picture.

The results of the present study suggest that disability was related to overall neuropsychological impairment, with visuoperceptual abilities, information processing, language and visual memory being independently related to the disability measures (MRS and FAI). Similarly, several earlier studies for SAH and stroke survivors have reported that global cognitive deficits are associated with reduced disability (Dombovy et al., 1998; Passier et al., 2009; Springer et al., 2009; Thommessen et al., 1999). These studies differed from the current study as they were relatively short-term, lost a large number of participants at follow-up and used a telephone interview or cognitive screen such as MMSE to assess cognition rather than a detailed and comprehensive neuropsychological test battery. Only a few studies have examined the relationship between specific cognitive domains and recovery. For example, Vilkki, Holst, Öhman, Serro and Heiskanen (1990) found that incomplete recovery a 1-year was associated with poor verbal memory and cognitive inflexibility in SAH patients. The authors however

did not administer a test to assess disability and the recovery was based on the scores of GOS. As with SAH studies, literature for stroke survivors also indicate that people with cognitive impairments have reduced performance on ADLs (Caderfeldt et al., 2010; Cao et al., 2007; Patel et al., 2003; Patel et al., 2002). However, these studies were not population based and used screening tools such as MMSE, with only a few researchers using detailed neuropsychological tests to examine cognitive functioning. In contrast to many previous studies, the present study used an extensive neuropsychological battery to examine cognitive outcomes and also used a variety of functional outcome measures. Within the specific cognitive domains, whilst a strong relationship was seen between visuoperceptual abilities and disability, language and visual memory emerged as having independent relationships with disability and handicap measures. Although in our study we did not find a relationship between executive functioning and disability, an earlier stroke study reported that impaired executive functioning had a clear relationship with basic and complex ADLs (Pohjasvaara et al., 2002). The results of this study may have differed as the authors used a wide range of tests related to executive functioning to specifically explore the relationship between disability and executive functioning. Furthermore, this study used stroke sample, excluded SAH cases and tested the survivors at 4-months post-stroke. The current study suggested that attention is not significantly related to disability and handicapped (BI, MRS, FAI, LHS) which is contrary to previous research by McDowd, Filion, Pohl, Richards and Stiers (2003) that highlighted the role of attention in functional outcomes. The authors used a number of neuropsychological tests specifically assessing attention abilities found that intact attention abilities are associated with positive physical functioning. The present findings differed from the above stated study because in the present study a collapsed domain score was used for attention abilities, so it was not possible to examine specific types or tests of attention which might

have affected the relationship with functional outcomes.

Regarding HRQoL, relationships were seen between various HRQoL domains and neuropsychological impairment. An earlier study by Vilkki et al. (1990) also found that cognitive inflexibility, memory deficits and processing speed were related to problems in social relationship and poor working capacity 1-year after SAH which is similar to current findings. Additionally, the present study found that overall cognitive impairment, language and attention are also significantly related to HRQoL. Vilkki et al. (1990) did not use any test for assessing language nor did they use any measure to explore HRQoL. They instead relied on a checklist to explore aspects related to work and social activity thus not capturing wider aspects of HRQoL, something that was done in the present study by using SF-36. In the current study, a strong relationship was also noted between social dysfunction on the GHQ-28, the SF-36 MCS and executive functioning. This implies that people who have problems with attention and impulse control experience social difficulties which could impact their HRQoL. A long-term study also reported that stroke survivors with cognitive deficits report reduced well-being and mental health with lower sense of purpose in life when compared to those with better cognitive functioning (Clarke et al., 2002); however this study only assessed cognition via MMSE and lost almost 40% of the participants due to death, dementia, refusal and severe cognitive problems. Similar to the current findings, Nys et al. (2006) found that cognitive disorders at baseline; including visual perceptual/construction, visual memory and executive functioning, were the strongest risk factors for reduced HRQoL at 6-months post-stroke. However, these authors excluded those with SAH, those aged >85 years and those with recurrent stroke, psychiatric history, pre-existing dementia and dependence on ADLs, limiting the representative of their sample. Unfortunately measures of cognition were not administered prior to the 5-year follow-up in the present

sample, so change in these over time could not be examined. Overall, the results of this study are in agreement with those published previously which noted that individuals with impaired cognition have worse HRQoL post-SAH as compared to those who are cognitively intact (Haacke et al., 2006; Scott, Eccles, Molyneux, Kerr, Rothwell & Carpenter, 2010; Springer et al., 2009; Visser-Meily, 2009). These studies had certain limitations such as inconsistent follow-up periods with the time of cognitive evaluation varying between and within studies; exclusion of participants with a history of dementia; a lack of detailed neuropsychological assessment and/or use of a telephone interview or self-report questionnaire to assess cognition.

Mood and functional outcomes

Regarding mood, the results of this study indicate that poor long-term functional outcomes such as disability and HRQoL are significantly related to depressed mood and somatic symptoms. Handicap was also significantly related to a general sense of well being (GHQ-28). Both the physical and mental aspects of HRQoL were significantly related to depression, though this was weaker for somatic symptoms as measured by GHQ-28. Finding that depression, anxiety, fatigue, and sleeplessness in relation to reduced HRQoL is in accordance with previous SAH and stroke studies (Ahlsiö et al., 1984; Visser-Meily, 2009; Wermer et al., 2007). However prior studies used inconsistent time periods (2-4 years) to assess the participants and excluded those who with language limitations (e.g., unable to speak Dutch) or who had a reduced life expectancy. For example, previous work has shown that even 4-years after the incidence, a substantial number of patients experience depressive episodes and subsequently a considerably reduced HRQoL (Haacke et al., 2006). Similarly, a previous study that examined the HRQoL of SAH-survivors at 9-months found that of three scales on the Brain Injury Community Rehabilitation Outcome-39 scale (mobility, self-organization, and productive

employment), the only significant association was found between mood and self-organization (e.g., event planning; Powell et al., 2002). As a causal relationship between mood and HRQoL was not examined in the current study, it could be hypothesized that due to low mood people may reduce their participation in activities and become dependent on others; while alternatively it could be seen that reduced activity could lead to isolation and lowering of mood. Teasing apart the direction of these causal relationships could be a focus for future research.

Studies have also reported that depression is significantly associated with increased disability especially within the domains of domestic, social, occupation and leisure activities in long-term SAH-survivors (Lindberg, 1995). The main drawback for this study was that the testing time post-SAH was inconsistent ranging from 3-to 14-years. Our data is consistent with previous studies of stroke, where depression has been associated with poorer functional outcomes in terms of problems with ADLs at various stages (i.e., 6, 12 and 18-months) of post-stroke recovery (Berg et al., 2003; Naess et al., 2010). In the ARCOS study 27% of the stroke-survivors had abnormal mood on the GHQ-28 at 6-months, and the predicting variables included premorbid dependency on ADLs and requiring help in ADLs immediately post-stroke (Hackett & Anderson, 2006). Unfortunately, this study did not use a specific measure for depression or anxiety but relied upon the GHQ-28 subscale, which as previously stated, has limitations. However, despite the lack of range on GHQ-28, the relationship existed, which suggests that the participants were more depressed and dependent on ADLs at 6-months post-SAH. Similar outcomes have been noted indicating that limitations in ADLs and handicap predict long-term post-stroke depression (Goodwin & Devanand, 2008; Herrmann et al., 1998; van De Port et al., 2007). Inversely, it has also been found that depression in the acute stage is associated with poor health outcomes at 12-months (Bosworth, et al.,

2000). Unfortunately, these studies were not population-based and excluded patients with aphasia or poor functional outcomes in the acute stage, and associations between depression and functional outcomes were not provided for different stroke subtypes. Similar to the relationship between disability and depression, mood problems such as depression and anxiety have previously been identified as independent predictors of post-stroke handicap, although this study excluded SAH cases (Sturm et al., 2004b), an association that was supported in the current study. Based on the results of the current study, it is hypothesized that the relationship between mood and functional outcomes can result in a vicious cycle where people experience depression as they become socially isolated due to disability and handicap following SAH, which limits their ability to visit family and friends and pursue leisure activities. These limitations further impact their HRQoL and perhaps create a sense of loneliness and low mood and vice versa where low mood may impact the HRQoL.

Anxiety is a common occurrence in SAH and stroke survivors and often impacts their long-term functional outcomes (Åström, 1996; Burvill et al., 1995; Morris et al., 2004) including dependence on ADLs, limitations in socialization and occupation. However, this finding was not supported in the current study. As discussed earlier the lack of relationship between anxiety and functional outcomes in the current study could be attributed to the anxiety measure used (i.e., GHQ-28) which may be deemed inappropriate to measure anxiety in the current sample.

In considering the above, it is important to note that the literature links mood and cognitive functioning in stroke and SAH survivors (Berg et al., 2003; Brodaty et al., 2007; Madureira et al., 2000; Passier et al., 2010; Rasquin et al., 2005). In the current study it was noted that individuals with overall cognitive impairment and difficulties within the domains of visuoperceptual abilities, executive functioning/attention and

information processing tended to be depressed (GDS-SF). Despite the limitations of GHQ-28 stated earlier, it does measure somatic problems, which was found to be related to information processing domain. Although these relationships in the present study were weak, they replicate the findings of previous SAH research where depression and cognitive deficits co occur (Alfieri et al., 2008; Madureira et al., 2000; Mayer et al., 2002). Regarding domain specific deficits, our results are different to those reported by Mavaddat et al. (1999) who linked depression in SAH participants with impaired performance on tests of language and spatial memory. Their study was different to the current study in that instead of using neuropsychological tests to study cognitive profile, they used a computerized test battery (CANTAB), and limited their sample to participants with a favourable outcome (GOS=4-5) and with a large variation in timing of follow up (i.e., 6-24 months). As discussed earlier, cognitive profiles can change over time, therefore Mavaddat et al.'s findings can not be considered conclusive as they assessed functioning till 24-months only. Although executive functioning was related to depression in the SAH-participants in current study, the relationship was weak. Similarly, Pohjasvaara et al. (2002) found a relationship between executive functioning and depression as assessed by the BDI, but not through clinical psychiatric evaluation in stroke patients. The authors examined the participants at 4-months post stroke and used an extensive range of executive functioning tests. In contrast, in the present study only a collapsed domain score was used to indicate executive functioning which could be why the relationship emerged as weak. Inclusion of somatic items in the BDI (e.g., sleep, fatigue, appetite) is also likely to skew findings post-stroke as individuals are likely to experience post-stroke somatic difficulties. Vilkki et al. (1990) reported that although cognitive performance and anxiety and depression were unrelated at 1-year after SAH, patients frequently complained of experiencing these mood problems. Vilkki and

colleagues (1990) did not use any test to examine visuoperceptual abilities and no formal tests were administered to measure depression and the emotional status was assessed using a symptom checklist and clinical interview which could be why their results differ from this study. A study using extensive neuropsychological battery found that reduced cognitive speed predicted anxiety at 3-months post-stroke (Barker-Collo, 2007). Thus, as discussed earlier, studies have produced mixed results regarding the association between depression/anxiety and cognitive impairment with some showing no relationship with global or specific cognitive deficits whereas others suggest a correlation. Unlike the current study which is population based and used a battery of cognitive tests, the previous studies were not long-term, excluded severe cases and did not use a neuropsychological battery to examine cognitive functioning but relied on screening tests or limited the abilities that were assessed. It is possible that the present study yielded weaker relationships because, although people were experiencing cognitive deficits, these problems were not severe enough to impact on their mood to a great extent. Another possible reason could be that at 5-years post-SAH, they are possibly experiencing mild depression which could explain a weak relationship with cognitive functioning. In the current study the severity of depression was not assessed and a separate measure for anxiety was not used, which could have possibly clarified this relationship. Furthermore, as opposed to previous studies that used cognitive screens, using a battery of neuropsychological tests in the current study also did not show a relationship between mood and cognitive functioning. This could be because as discussed previously GHQ-28 is perhaps not an appropriate measure to assess anxiety and depression in this population. As compared to GHQ-28, although GDS-SF is a relevant measure to assess depression, it is specifically to assess depression in elderly; whereas SAH-survivors are often younger in age. Considering this, it might be more appropriate to use BDI (Beck, Steer, Ball &

Ranieri, 2006) which is more suitable for people ≥17 years or HADS (Zigmond & Snaith, 1983) as it assesses both anxiety and depression problems.

No relationship was noted between cognitive functioning and the depression subscale for GHQ-28. As discussed earlier this could be because GHQ-28 is not an appropriate measure to assess depression in this population. The results also suggest that people have problems in social functioning which is related largely to deficits in executive functioning and to some extent impairments in verbal memory and information processing. Thus it can be concluded that individuals feel depressed as they experience difficulties in social interactions and participation in activities owing to deficits in executive functioning/attention and verbal memory. Current results show similar patterns to those of Vilkki et al. (1990) who reported that poor cognitive performance, particularly deficits in memory and cognitive flexibility, were related to difficulties in social relations in patients at 1-year post-SAH. Similarly, Rasquin et al., (2005) have linked post-stroke cognitive decline to psychiatric symptoms such as depression and somatization suggesting that patients with mood disorders had impaired executive functioning and global cognitive functioning (measured by CAMCOG) from 1 to 6-months after stroke. The authors also reported that patients with vascular dementia reported more mood problems as compared to those with mild or no cognitive deficits, which differs from this study as none of the participants reported experiencing dementia. Similarly other stroke studies have reported that people with depression tend to be cognitively impaired (as assessed by MMSE) for a long time (up to 2-years) post-stroke (House et al., 1990) and remission in depression was associated with improved cognition (Narushima et al., 2003).

Although the current results do not throw light on the causal relationship between cognitive deficits and mood problems, based on above it can be summarized that this

relationship could be occurring at various levels. It is possible that mood problems can give rise to cognitive deficits or vice versa where cognitive deficits impact the mood (Robinson, 1998). It could also be speculated that both cognitive deficits and mood problems could be a direct consequence of the brain damage caused by SAH (Kreitschmann-Andermahr et al., 2007).

CLINICAL IMPLICATIONS

This is the first population-based study to examine the neuropsychological and functional outcomes of 5-year SAH survivors in New Zealand. The results of the study suggest that an average SAH survivor experiences difficulties in the following areas of functioning: increased handicap, disability, and poor health related quality of life. They also experience problems across cognitive domains, including executive functioning, information processing, memory, language, and visuoperceptual abilities. Furthermore, although SAH survivors are more depressed than controls, they are not suicidal. The above findings point to the need for assessing long-term physical functioning, mood and administering a comprehensive battery of neuropsychological tests for SAH-survivors so that clinicians can design appropriate rehabilitation to address their needs.

This study indicates that baseline functioning is significantly related to long-term outcomes, therefore acute stage treatments should be considered valuable as this may assist the survivors gain functional abilities to increase their activity and participation at a later stage. It is suggested that as part of the treatment survivors should be given some strategies to help them cope with their illness and trauma. Furthermore, clinicians should be aware that functional impairments persists for years after SAH so the rehabilitation interventions should focus at the long-term needs of the SAH-survivors. Poor HRQoL in the domains of mental health, physical functioning and social functioning has been identified in the present study which should be specifically considered when planning

treatment. For this, more strategies with an aim at increasing leisure activities and social interactions should be used.

The findings indicate that women are generally more impaired than men with significantly worse quality of life in relation to mental health and also tend to be more depressed. It is therefore recommended that interventions should particularly target women because they tend to perceive their recovery as worse which impacts their emotional health. For example, while providing rehabilitation, women's emotional state should be regularly monitored and mood problems such as depression should be addressed at the earliest via psychoeducation, therapy or medication as appropriate.

Based on the findings of this study, GHQ-28 may not be considered an appropriate tool to assess mood problems in SAH-survivors as it did not significantly differentiate the SAH and control group on anxiety and depression. Although GDS-SF did differentiate between the SAH and control group, it might not be suitable for a younger population as people often experience SAH at a younger age as compared to other strokes (The ACROSS Group, 2000). As stated earlier, using an alternate test such as BDI is suggested, which may be more suitable to assess depression in this population. Regarding anxiety, whilst literature points towards people experiencing post-SAH anxiety (Barker-Collo, 2007; Visser-Meily et al., 2009), GHQ-28 not identify this issue. It is therefore suggested that instead of GHQ-28 another scale such as HADS might be more appropriate as it can identify both anxiety and depression problems in SAH population.

The study identified that cognitive factors are related to wider functional outcomes and therefore, as stated earlier clinicians should use a comprehensive battery of tests to assess neuropsychological functioning post-SAH. Specifically, language and visual memory deficits may play a role in wider functioning at 5-years. It is therefore emphasized that cognitive assessments should specifically include tests related to

language and memory. Furthermore interventions should aim to address these deficits. For example, speech and language therapists should be involved as part of the rehabilitation process.

LIMITATIONS

There are certain limitations of the current study that should be kept in mind during interpretation and when considering generalisability of the results. One limitation of this study was that participants were lost to follow-up, which is a common occurrence in long-term studies (Srikanth et al., 2006). Of the original 96 participants, at 6-months post-SAH, a large number (n=48) of participants were lost to follow-up (see page 52) at 5-years. Of the 37 were contactable at the start of the study, 73% participated in the study (n=27). Thus, the sample may not be truely representative of the SAH-population. Additionally, small sample size restricted the power of the statistical analyses to examine relationships among variables and the predictive value of variables that could impact on the current functioning. Thus, although this study was population-based, the findings may not be generalised to larger populations. However, considering this is the only identified population based study examining the long-term neuropsychological and functional outcomes of SAH-survivors via detailed neuropsychological and functional assessments; the results are of use for clinicians in better understanding long-term recovery, and planning appropriate interventions. For example, language and visual memory deficits seem to play the most important role in the functioning than other domains such as verbal memory; therefore these domains should be the focus for the clinicians.

Selection of the neuropsychological tests is another limitation of this study. Studies show that SAH-survivors experience cognitive deficits across domains, which range from mild to severe impairments (Haug et al., 2010). Hence, in selecting

neuropsychological tests consideration was given to including those that could be administered to participants with intact cognition or mild impairments as well as to those who suffered more severe cognitive impairments. As a result, tests used in the current study were neither too difficult nor too easy for the participants. Thus, only two participants did not complete the neuropsychological assessments due to severe impairment. Therefore, whilst a lot of information was gathered via in-depth neuropsychological assessment and the findings go beyond the limited screening of most studies, it cannot be considered as a full neuropsychological assessment and exclusion of the 2 most severe cases suggest the findings are an underestimate.

In long-term studies, other factors such as dementia, a recurrent stroke and type of treatment may impact on functional and neuropsychological performance (Scharbrodt et al., 2008; Mavaddat et al., 1999). In the present study, none of the participants reported having received a diagnosis of dementia, 1 participant had <6 score on HMT and only 2 participated reported experiencing a recurrent stroke, which reduces the likelihood of this kind of a issue. It is also possible that the participants were unaware of their condition and some of them could have dementia or experienced a “silent stroke” which was not diagnosed. This study also did not examine the impact of different treatment types on cognitive or functional outcomes.

In this study interpreters were used for participants who were not fluent in English. However for both SAH and control group, language and verbal tests were not administered (n=7) because it would not have given a true picture of their language and verbal abilities. Furthermore for SAH group, some participants did not undergo all the neuropsychological tests due to hemiplegia (n=2), refusal (n=4), visual problems (n=2) and difficulty understanding instructions (n=1). It is possible that this could also have impacted the overall findings for the study.

In the current study premorbid abilities of the participants were not assessed, which would have provided a clearer picture of their abilities prior to SAH. It would have been good to covary if premorbid abilities were assessed. Perhaps it would have increased or reduced the size of effects on differences between groups. Thus, although SAH survivors are more impaired than matched controls, it is possible that this may not reflect declines for the individual. Furthermore, whilst the control group was matched on age, gender and ethnicity, sample was not matched on education as it would have been very time consuming to match the sample on this variable as well. Information on education was gathered and it was not related to functional and neuropsychological outcomes, it would have been ideal to match the sample on education in order for them to be more comparable and be more certain that this did not impact findings.

The final limitation of this study is that the natural history of the recovery process could not be examined for all variables of interest. For example, the neuropsychological assessments were done only at 5-years post-SAH and not at baseline, therefore change over time and relationship between initial neuropsychological functioning to 5-year functioning could not be examined. Similarly, not all the functional measures were administered at each time frame (refer to page 56), which made it difficult to measure change over time for each functional outcome.

Due to there being very limited long-term outcome studies following SAH, it is important for more research to be done in this area. Given the above limitations, future studies would include a larger sample perhaps extending into other regions of New Zealand. Future studies would look at repeated neuropsychological and functional assessments at various time points using both shorter and longer durations to ascertain a more focused picture of the patterns of change and clinical improvement after SAH (e.g., from baseline up to 10-years). For example, it would helpful to identify cognitive deficits

at an earlier stage in order to assist rehabilitation and interventions at an appropriate time. It is worth noting that funding has been approved for another stroke incidence study with follow-ups at different time points which encompass computerised neuropsychological test battery. It would also be useful to get some information about the health related quality of life and emotional functioning of the caregivers of the SAH-survivors as caregivers of SAH patients tend to experience many adverse health effects (Hop et al., 1998).

It is also suggested that different mood measures be used in future studies as the GHQ-28 did not cover wider aspects of anxiety and depression. Although GDS-SF differentiated between the groups, it could be suggested that another depression measure (e.g., HADS) should be considered as SAH often impacts a younger group as compared to other stroke types. Moreover, a scale such as the HADS can assess depression as well as anxiety which is also an important emotional outcome post-SAH (Visser-Meily et al., 2009). Finally, it would be useful to gather qualitative information via interview regarding the participants daily functioning, HRQoL and the impact of neuropsychological impairments on their daily life to give a detailed account of their personal experience post-SAH, something which formal tests do not cover. As per the ICF model (WHO, 2001), future research should also look at examining the role of personality and environment on the functional outcomes following SAH.

STRENGTHS

Whist a number of limitations of this study have been noted, it is important to note its considerable strengths. Although there has been some previous research about neuropsychological and functional outcomes following SAH, majority of studies have focused on outcomes up to 1-year. As mentioned earlier, this was a population-based 5-year study examining SAH-outcomes. While some potential participants were lost to

follow-up, exhaustive efforts were made to locate potential participants (e.g., using previously recorded contact details, telephone directories, general practitioner records, electoral rolls and via contacting relatives using details on existing research forms) and only one participant was not contacted due to change of address. In this study we used interpreters where English was not the first language of the participants to enable their participation in the assessments which would have otherwise reduced the sample size even more. Furthermore, an extensive neuropsychological battery was used to assess various abilities; and within each ability, two to three tests were used. Commonly used standard neuropsychological tests were used so the findings could be useful to the clinicians. We also tried to tap into each of the aspects of the ICF (WHO, 2001) using a range of tests to assess functional and neuropsychological outcomes to understand the SAH recovery in a comprehensive manner.

CONCLUSION

This study highlights that even 5-years post-SAH, survivors have a myriad of difficulties in activity and participation and experience deficits across areas of cognitive functioning as compared to matched controls. Generally, the results suggest that functional outcomes share complex relationships with mood and cognition and that these themselves are related. Several variables may be associated with the long-term functional outcomes post-SAH which include baseline functioning and current mood with females experiencing worse health related quality of life. Furthermore, although the SAH-survivors have cognitive deficits in various domains which impact their functioning, language and visual memory emerged as independent factors associated with their current functioning. Thus, the current findings suggest that post-SAH assessments should include assessments for these deficits and the same should be addressed via appropriate rehabilitation interventions.

Appendix A:
Measures Used

DEMOGRAPHIC INFORMATION

Date of assessment_____
Title: ___ First name(s): _____ Last name:_____
Date of Birth: _____
Sex: Male _____ Female _____
Which ethnic Group do you belong to?

○ NZ Maori ○ NZ European
○ Other European ○ Samoan
○ Cook Island ○ Tongan
○ Niuean ○ Chinese
○ Indian ○ Other (if other please specify _____)

What is your current marital status? **(tick one only)**

O Married, civil union, or living with partner
O Separated, divorced or widowed
O Never married (single)

Do you live alone?

O Yes
O No

If **No**:

O Living with family or partner _____**Yes** ____ **No**
O Living with others _____ **Yes** ____ **No**

What is your usual dwelling place? **(tick one only)**

O Rented ○ Own home
O Family or friend's home ○ Retirement village or similar
O Rest home ○ Private hospital
O Boarding house ○ Other (if other please specify _____)

Which of the following is your current work situation? **(tick one only)**

O Full time paid work ○ Part time paid work
O Retired ○ Unemployed or redundant
O Beneficiary ○ Homemaker
O Other (if other please specify _____)

Do you feel you have made a complete recovery from your stroke?

○ Yes ○ No

Have you had a recurrent stroke/SAH?

○ Yes ○ No

At what age (in years) did you first leave school? _____
After leaving school did you acquire any further qualifications? _____**Yes** _____ **No**
If yes: (please specify)

O Degree (e.g., BSc, MA, PhD etc.) _____
O Diploma (e.g., teaching, nursing, etc.)_____
O Certificate (trade, apprenticeship, etc.) _____
O Other (please specify) _____

HODKINSON MENTAL TEST (HMT)

Score one point for each question answered correctly

Score

1. _____ Age of patient
2. _____ Time (to nearest hour)
3. _____ Address given, for recall at end of test: 42 West street
4. _____ Name of Hospital (or area of town if at home)
5. _____ Year
6. _____ Date of birth of participant
7. _____ Month
8. _____ Years of first World War
9. _____ Name of current New Zealand Prime Minister
10. _____ Count backwards from 20-1 (no errors allowed, but may correct self)

Total _____

MODIFIED RANKIN SCALE

How would you grade the participant's level of disability and need for assistance? **(tick one only)**

- O 0- No symptoms at all
- O 1- No significant disability despite symptoms; able to carry out all usual duties and activities
- O 2- Slight disability; unable to carry out all previous activities, but able to look after own affairs without assistance
- O 3- Moderate disability; requiring some help, but able to walk without assistance
- O 4- Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance
- O 5- Severe disability; bedridden, incontinent and requiring constant nursing care and attention

GERIATRIC DEPRESSION SCALE

These questions are about your mood (tick **yes or no** only once on each line)

	YES	NO	
1.	_____	_____	Are you basically satisfied with your life?
2.	_____	_____	Have you dropped many of your activities and interests?
3.	_____	_____	Do you feel that your life is empty?
4.	_____	_____	Do you often get bored?
5.	_____	_____	Are you in good spirits most of the time?
6.	_____	_____	Are you afraid that something bad is going to happen to you?
7.	_____	_____	Do you feel happy most of the time?
8.	_____	_____	Do you often feel helpless?
9.	_____	_____	Do you prefer to stay at home, rather than going out and doing new things?
10.	_____	_____	Do you feel you have more problems with memory than most?
11.	_____	_____	Do you think it is wonderful to be alive now?
12.	_____	_____	Do you feel pretty worthless the way you are now?
13.	_____	_____	Do you feel full of energy?
14.	_____	_____	Do you feel that your situation is hopeless?
15.	_____	_____	Do you think that most people are better off than you are?

SF-36 HEALTH SURVEY

1. In general, would you say your health is: (tick ONE only)

Excellent	Very good	Good	Fair	Poor

2. **Compared to one year ago**, how would you rate your health in general **now**? (tick ONE only)

Much better now than one year ago	Somewhat better now than one year ago	About the same as one year ago	Somewhat worse now than one year ago	Much worse now than one year ago

3. The following questions are about activities you might do during a typical day. Does your **health now limit you** in these activities? If so, how much?

	ACTIVITIES	Yes, limited a lot	Yes, limited a little	No, not limited at all
i	**Vigorous activities**, such as running, lifting heavy objects, participating in strenuous sports			
ii	**Moderate activities**, such as moving a table, pushing a vacuum cleaner, bowling, or playing golf			
iii	Lifting or carrying groceries			
iv	Climbing **several** flights of stairs			
v	Climbing **one** flight of stairs			
vi	Bending, kneeling or stooping			
vii	Walking **more than one kilometre**			
viii	Walking **half a kilometre**			
ix	Walking **100 metres**			
x	Bathing or dressing yourself			

4. During the **past 4 weeks**, have you had any of the following problems with our work or other regular daily activities **as a result of your physical health**? (tick ONE on each line)

	Yes	No	
i			Cut down on the amount of time you spent on work or other activities
ii			Accomplished less than you would like
iii			Were limited to the kind of work or other activities
iv			Had difficulty performing the work or other activities (for example, it took extra effort)

5. During the **past 4 weeks**, have you had any of the following problems with your work or other regular daily activities **as a result of any emotional problems** (such as feeling depressed or anxious)? (tick ONE on each line)

	Yes	No	
i			Cut down on the amount of time you spent on work or other activities
ii			Accomplished less than you would like
iii			Didn't do work or other activities as carefully as usual

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? (tick ONE only)

Not at all	Slightly	Moderately	Quite a bit	Extremely

7. How much **bodily** pain have you had during the **past 4 weeks**?

None	Very mild	Mild	Moderate	Severe	Very Severe

8. During the **past 4 weeks**, how much did **pain** interfere with your normal work (including both work outside the home and housework? (tick ONE only)

Not at all	A little bit	Moderately	Quite a bit	Extremely

9. These questions are about how you feel and how things have been with you **during the past 4 weeks**. For each question, please give the one answer that comes closest to the way you have been feeling. How much of the time during the **past 4 weeks**: (tick ONE on each line)

		All of the time	Most of the time	A good bit of the time	Some of the time	A little of the time	None of the time
i	Did you feel full of life?						
ii	Have you been a very nervous person?						
iii	Have you felt so down in the dumps that nothing could cheer you up?						
iv	Have you felt calm and peaceful?						
v	Did you have a lot of energy?						
vi	Have you felt down?						
vii	Did you feel worn out?						
viii	Have you been a happy person?						
ix	Did you feel tired?						

10. During the **past 4 weeks**, how much of the time has your **physical health or emotional problems** interfered with your social activities (like visiting with friends, relatives, etc.)? (tick ONE only)

All of the time	Most of the time	Some of the time	A little of the time	None of the time

11. How TRUE or FALSE is **each** of the following statements for you?

		Definitely true	Mostly true	Don't know	Mostly false	Definitely false
i	I seem to get sick a little easier than other people					
ii	I am as healthy as anybody I know					
iii	I expect my health to get worse					
iv	My health is excellent					

GENERAL HEALTH QUESTIONNAIRE

We would like to know if you have any medical complaints, and how your health has been in general, over the past few weeks. Please answer ALL questions on the following pages simply by ticking one block for the answer which you think most nearly applies to you. Remember that we want to know about present and recent complaints, not those you have had in the past.

Have you recently….

(tick one block only)

		Better than usual	**Same as usual**	**Worse than usual**	**Much worse than usual**
1	Been feeling perfectly well and in good health?				

		Not at all	**No more than usual**	**Rather more than usual**	**Much more than usual**
2	Been feeling in need of a good tonic?				
3	Been feeling run down and out of sorts?				
4	Felt that you are ill?				
5	Been getting any pains in your head?				
6	Been getting a feeling of tightness or pressure in your head?				
7	Been having hot or cold spells?				
8	Lost much sleep over worry?				
9	Had difficulty in staying asleep?				
10	Felt constantly under strain?				
11	Been getting edgy and bad tempered?				
12	Been getting scared or panicky for no good reason?				
13	Found everything getting on top of you?				
14	Been feeling nervous and uptight?				

(tick one only)

		More so than usual	**Same as usual**	**Rather less than usual**	**Much less than usual**
15	Been managing to keep yourself busy and occupied?				

(tick one only)

		Quicker than usual	**Same as usual**	**Longer than usual**	**Much longer**
16	Been taking longer over the things you do?				

(tick one only)

		Better than most	**About the same**	**Less well than usual**	**Much less well**
17	Felt on the whole you were doing things well?				

(tick one only)

		More satisfied	**About the same**	**Less satisfied**	**Much less satisfied**
18	Been satisfied with the way you've carried out your tasks?				

(tick one only)

		More so than usual	Same as usual	Rather less than usual	Much less than usual
19	Felt that you were playing a useful part in things?				
20	Felt capable of making decisions about things?				
21	Been able to enjoy your normal day-to-day things?				

(tick one only)

		Not at all	No more than usual	Rather more than usual	Much more than usual
22	Been thinking of yourself as a worthless person?				
23	Felt that life is entirely hopeless?				
24	Felt that life isn't worth living?				

(tick one only)

		Definitely not	I don't Think so	Has crossed my mind	Definitely have
25	Thought of the possibility that you might do away with yourself?				

(tick one only)

		Not at all	No more than usual	Rather more than usual	Much more usual
26	Found at times you couldn't do anything because your nerves were too bad?				
27	Found yourself wishing you were dead and away from it all?				

(tick one only)

		Definitely not	I don't think so	Has crossed my mind	Definitely have
28	Found that the idea of taking your own life kept coming into your mind?				

LONDON HANDICAP SCALE

This questionnaire asks six questions about your everyday life. Please answer each question. Read the instructions in each question and then answer by ticking the box next to the sentence which describes you best. When answering the questions, it may help to think about things you have done over the past week. Compare what you can do with what someone like you who is in good health can do.

1. **Getting Around**

Think about how you normally get from one place to another, using any help, aids or means of transport that you normally have available.

Does your health stop you from getting around? (tick one box only)

	Not at all	You go everywhere you want to, no matter how far away
	Very slightly	You go most places you want to, but not all
	Quite a lot	You get out of the house, but not far away from it
	Very much	You don't go outside, but you can move around from room to room indoors
	Almost completely	You are confined to a single room, but can move around in it.
	Completely	You are confined to a bed or a chair. You cannot move around at all. There is no one to move you

2. **Looking after yourself**
   Think about things like housework, shopping, looking after money, cooking, laundry, getting dressed, washing, shaving and using the toilet.

Does your health stop you looking after yourself? (tick one box only)

	Not al all	You can do everything yourself
	Very slightly	Now and then you need a little help
	Quite a lot	You need help with some tasks (such as heavy housework or shopping), but not more than once a day
	Very much	You can do some things, but you need help more than once a day. You can be left alone safely for a few hours.
	Almost completely	You need help to be available all the time. You cannot be left alone safely.
	Completely	You need help with everything. You need constant attention, day and night

3. **Work and leisure**
   Think about things like work (paid or not), housework, gardening, sports, hobbies, going out with friends, travelling, reading, looking after children, watching television and going on holidays.

Does your health limit your work or leisure activities? (tick one box only)

	Not al all	You can do everything you want to
	Very slightly	You can do almost all the things you want to do
	Quite a lot	You find something to do almost all the time, but cannot do some things for as long as you would like
	Very much	You are unable to do a lot of things, but can find something to do some of the time
	Almost completely	You are unable to do most things, but can find something to do some of the time
	Completely	You sit all day doing nothing. You cannot keep yourself busy or take part in any activities

4. **Getting On With People**
   Think about family, friends and people you might meet during a normal day.

Does your health stop you understanding the world around you? (tick one box only)

	Not at all	You get on well with people, see everyone you want to see, and meet with new people
	Very slightly	You get on well with people, but your social life is slightly limited
	Quite a lot	You are fine with people you know well, but you feel uncomfortable with strangers
	Very much	You are fine with people you know well, but you have few friends and little contact with neighbours. Dealing with strangers is very hard
	Almost completely	Apart from the person who looks after you, you see no one. You have no friends and no visitors
	Completely	You don't get on with anyone, not even people who look after you

5. **Awareness of Your Surroundings**

Think about taking in and understanding the world around you, and finding your way around in it

Does your health stop you understanding the world around you? (tick one box only)

	Not at all	You fully understand the world around you. You see hear, speak and think clearly, and your memory is good
	Very Slightly	You have problems with hearing, speaking, seeing or your memory, but these do not stop you doing most things
	Quite a lot	You have problems with hearing, speaking, seeing or your memory, which make life difficult a lot of the time. But, you understand what is going on
	Very much	You have great difficulty understanding what is going on
	Almost completely	You are unable to tell where you are or what day it is. You cannot look after yourself at all
	Completely	You are unconscious, completely unaware of anything going on around you.

6. **Affording the Things You Need**

Think about whether health problems have lead to any extra expenses, or have caused you to earn less than you would if you were healthy

Are you able to afford the things you need? (tick one box only)

	Yes, easily	You can afford everything you need. You have easily enough money to buy modern labour-saving devices, and anything you may need because of ill-health
	Fairly, easily	You have just about enough money. It is fairly easy to cope with expenses caused by ill-health
	Just about	You are less well-off than other people like you; however, with sacrifices you can get by without help
	Not really	You only have enough money to meet your basic needs
	No	You are dependent on government benefits, or money from other people or charities. You cannot afford things you need
	Absolutely not	You have no money at all and no government benefits. You are totally dependent on charity for your most basic needs

FRENCHAY ACTIVITY INDEX

These questions relate to your usual level of activities.

In the last **three months,** how often did you undertake:

		Never	**Under once weekly**	**1-2 times a week**	**Most days**
1	Preparing meals				
2	Washing up				

		Never	**1-2 times in 3 months**	**3-12 times in 3 months**	**At least weekly**
3	Washing clothes				
4	Light housework				
5	Heavy housework				
6	Local shopping				
7	Social outings				
8	Walking outside>15 mins				
9	Actively pursuing hobby				
10	Driving car/bus travel				

In the last **six months**, how often did you undertake:

		Never	1-2 times mont	3-12 times months	At least weekly
11	Outings/car rides				

		None	Light	Moderate	All necessary
12	Gardening				
13	Household/car maintenance				

		None	1 in 6 months	Less than 1 a fortnight	Over 1 a fortnight
14	Reading books				

		None	Up to 10 hours/week	10-30 hours/week	Over 30 hours/week
15	Gainful work				

BARTHEL (ADL) INDEX

The next series of questions relate to your current level of activity: **(tick one box only)**

1. Feeding

	Independent: Able to use any necessary device; feeds in a reasonable time; able to cut up food, use condiments, spread butter etc. on his/her own. Food may be placed within reach
	Needs help: eg with cutting or spread butter
	Dependent: needs to be fed

2. Bathing

	Independent: Able to wash self all over; may be by using shower, a full bath or standing and sponging all over. Includes getting into and out of bath, or shower room
	Dependent: Needs some help with personal care

3. Grooming

	Independent: Doing all personal activities, eg. Washing hands and face, combing hair. Includes shaving and teeth. Not needing any help
	Dependent: Needs some help with personal care

4. Dressing

	Independent: Able to dress, includes (buttons, zip, laces), getting clothes out of closet/ Drawers. No help needed at all, may use rail for stabalizing.
	Needs help: Needs minor help verbal or physical managing clothes and balancing
	Dependent: Unable to dress without major assistance

5. Bowels

	Continent: If needs enema, suppository, must manage him/herself
	Occasional accident: Maximum once per 24 hours; needs help with device.
	Incontinent or catheterized and unable to manage

6. Bladder

	Continent: Able to use any device (e.g. catheter) if necessary
	Occasional accident: Maximum once per 24 hours; needs help with device
	Incontinent or catheterized and unable to manage

7. Toilet

	Independent: Able to handle clothes, wipe self, flush toilet, empty commode completely unaided. Able to get on and off alone.
	Needs help: Able to manage with minor help balancing, handling clothes or toilet paper. However, still ale to use toilet
	Dependent: Unable to manage without major assistance

8. Chair/Bed Transfers

	Independent: No help; includes locking wheelchair if necessary.
	Minimal help: Includes verbal supervision and minor physical help such as might be given by a not very strong spouse.
	Major help: Able to sit unaided, but needs much help (two people).
	Dependent: Needs hoist or complete lift by two people. Unable to sit.

9. Mobility on level surfaces

	Independent: May use any aid; speed is not important. Able to mobilise about house.
	Needs help: Verbal or physical supervision, including help up into walking frame or other help standing.
	Independent in wheelchair: Must be able to negotiate corners alone.
	Immobile: Including being wheeled by another.

10. Stairs

	Independent: Must carry walking aid if used
	Needs help: Physical or verbal supervision
	Unable: Needs lift (elevator), or cannot negotiate stairs

Appendix B:
Participant Information Sheet-SAH group

Participant Information Sheet

Project title: ASTRO: Auckland **Str**oke **O**utcomes Study
Researcher Name: Associate Professor Valery Feigin

An invitation

You are invited to take part in a research study because you had a stroke about 5 years ago and participated in the ARCOS III study (Auckland Regional Community Stroke 2002-2003 study). This study is a follow-up of stroke survivors who initially participated in the ARCOS III study. By conducting this follow-up study we hope to determine long-term impact of stroke on stroke survivors and carers, which is important for improving health care organisation and reducing stroke burden on stroke families and the society. We also hope to learn new information about the effects of stroke on your various cognitive functions (e.g. memory, language) and mood. By conducting interviews we hope to find out about life after stroke, what costs, both in financial terms and personal terms have occurred and what impact this has had on your family. This study is coordinated by the Clinical Trials Research Unit at the University of Auckland.

Your participation is entirely voluntary (your choice). You do not have to take part in this study. If you choose not to take part, any care or treatment that you are currently receiving will not be affected. If you do agree to take part, you are free to withdraw from the study at any time, without having to give a reason. Withdrawing at any time will in no way affect your future health care. To help you make your decision please read this information brochure. You may take as much time as you like to consider whether or not to take part. If you require an interpreter this can be arranged.

What are the aims of this study?

The main aim of the study is to:

- Determine the broad long-term (5 to 6 years) impact of stroke by documenting very carefully the frequency, characteristics and effects on all people who participated in ARCOS III study (from March 2002 to February 2003).

The study also aims to find out what the effects of stroke are on:

- Changes in thinking (cognitive function)
- Disability, handicap and physical function
- Mood and emotions
- Quality of life and daily activities

- Your family and work
- Your role within your community
- Your perception of how life is for you
- Direct and indirect costs associated with stroke

What types of people can be in the study?

All people who were part of the ARCOS III 2002-2003 study and who had a stroke about five years ago while living in Auckland are able to participate in the study. If the person who has had the stroke is unable to consent to participate in the study, we ask a representative (you) to take part in the study.

How many people will be in the study?

We estimate about 700 people will be involved in this study.

What happens if I do decide to take part?

If you decide you would like to take part, your participation would be for a short time. A research nurse and neuropsychologists, who have been specially trained for this project, will interview you over the telephone and in your own home or place of residence. You will be asked to discuss what it has been like to have had a stroke, what kind of services you have had contact with, who helped you the most after your stroke, what it has been like for your family to have been with you during this period, and what are your hopes and fears for the future. You will also be asked questions about your recovery, mood, treatments, care and services that you have received after your stroke in 2002-2003. Should you have any major illnesses over the last 5 years (e.g. recurrent stroke, heart attack, diabetes etc), your medical notes will be reviewed/accessed by the research nurse, if necessary.

How many interviews/procedures will there be?

Approximately two weeks after you receive this Information Sheet and the Consent Form, you will be contacted by a research nurse over the telephone. You will have an opportunity to ask additional questions about the study, and if you agree to participate in the study you will be interviewed. There will only be two interviews - one telephone interview by a research nurse and one face-to-face interview by a study neuropsychologist. We would also like to speak with your family members and will ask them if they are willing to have a telephone interview with the interviewer and fill out one questionnaire which will be mailed out to them. We would like to double-check some information with you after the interview if necessary. The telephone interview will take about 30 minutes, and the face-to-face interview will take about two and a half hours. You will also be asked to complete one questionnaire which will be mailed out to you, to assess various aspects of your health and recovery. Completing these questionnaires will take approximately 30 minutes. After the interview we will arrange a day and time for the face-to-face interview. Should you feel tired during the face-to-face interview, you will be offered a break.

What is the time-span for the study?

The study is expected to start on 1 November 2006 and will continue until 1 November 2009.

The risks and benefits of the study

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. Your usual medical care will not be affected in any way by participating in the 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 interests to continue. Similarly your doctor may at any time provide you with any other treatment he/she considers necessary.

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. You will also be given results of your blood pressure and pulse measurements. The results obtained from your participation may help others with this condition in the future.

Compensation

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.

Confidentiality

The study files, any notes taken and all other information that you provide will remain strictly confidential. No material that could personally identify you will be used in any reports on this study. Upon completion of the study your records will be stored for 10 years in a secure place at the central coordinating centre in Auckland. All computer records will be password protected. All future use of the information collected will be strictly controlled in accordance with the Privacy Act.

Your rights

If you have any queries or concerns regarding your rights as a participant in this research study, you can contact an independent Health and Disability Consumer Advocate. This is a free service provided under the Health and Disability Act: Telephone (NZ wide) 0800 555 050. Free fax 0800 2787 7678 (0800 SUPPORT) email advocacy@hdc.org.nz.

Finally

This study has received Ethical Approval from the Northern X Regional Ethics Committee 4 October 2006. If you would like some more information about the study please feel free to contact the ASTRO Study Manager Elizabeth Glen at the Clinical Trials Research Unit, Faculty of Medicine and Health Sciences, University of Auckland on 373 7599 extension 84662.

Study Investigators

The principal investigator for this study is:

Associate Professor Valery Feigin, MD, PhD
Clinical Trials Research Unit
Faculty of Medicine & Health Sciences
School of Population Health
University of Auckland
Private Bag 92019
Auckland
Tel: (09) 373 7599 ext. 84728

Please keep this brochure for your information.
Thank you for reading about this study.

Appendix C:
Consent Form-SAH group

CONSENT FORM

for Stroke Survivors

Project title: ASTRO: Auckland **Str**oke **O**utcomes Study
Researcher Name: Associate Professor Valery Feigin

REQUEST FOR INTERPRETER			
English	I wish to have an interpreter.	Yes	No
Maori	E hiahia ana ahau ki tetahi kaiwhakamaori/kaiwhaka pakeha korero.	Ae	Kao
Samoan	Oute mana'o ia iai se fa'amatala upu.	Ioe	Leai
Tongan	Oku ou fiema'u ha fakatonulea.	Io	Ikai
Cook Island	Ka inangaro au i tetai tangata uri reo.	Ae	Kare
Niuean	Fia manako au ke fakaaoga e taha tagata fakahokohoko kupu.	E	Nakai

1. I have read/had explained to me, and understand, the Information Sheet dated 4 October 2006 for stroke survivors, their families, representatives and carers taking part in the ASTRO study. I have had the opportunity to discuss this study with the investigator. I am satisfied with the answers I have been given.
2. I understand that taking part in this study is voluntary (my choice). I realise the study involves an interview with medical and lifestyle questions, that I may choose not to answer any questions or withdraw from the study at any time and this will in no way affect my future health care.
3. I agree to a member of the research team approved by the Northern X Regional Ethics Committee to reviewing my relevant medical records for the sole purpose of checking the accuracy of the information recorded for the study.
4. I give my approval to the researchers to access information regarding my health history and health services usage.
5. I understand that my participation in this study is confidential and that no material that could identify me will be used in any reports on this study.
6. I understand the compensation provisions for this study.
7. I have had time to consider whether I and/or my family member should take part.

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

I am indicating my approval (or otherwise) for the following:

I wish to receive a copy of the results. I understand that there may be a significant delay between data collection and the publication of the study results.	Yes / No
To contact my GP if required for permission to access my medical records	Yes / No
I give my approval to the researchers to approach my family caregiver for their participation in the study	Yes / No

I _____ hereby consent to take part in this research.

OR

I am a representative of _____ (the participant), being a person who is lawfully acting on the participant's behalf or in his or her interests. My relationship to the participant is _____. I agree to health information about the participant being disclosed for the purposes of this research. I also agree to participate in this research.

(Please draw a line through the statement above that is not relevant).

Signature
(or representative)..... Signature of witness……

Date: ..... Name of witness……

Project explained by..... Project role ……

Signature ..... Date ……

Note: A copy of the consent form to be retained by participant and a copy to be placed in the medical file.

Approved by the Northern X Regional Ethics Committee

Appendix D:
Participant Information Sheet-Control group

DEPARTMENT OF PSYCHOLOGY

THE UNIVERSITY OF AUCKLAND
NEW ZEALAND
Te Whare Wānanga o Tāmaki Makaurau
Building 721, Tamaki Campus
261 Morrin Road, Glen Innes
Auckland, New Zealand
Telephone 64 9 373 7599
Facsimile 64 9 373 7000
The University of Auckland
Private Bag 92019
Auckland, New Zealand

PARTICIPATION INFORMATION SHEET

Title: Cognitive abilities and general well being of New Zealand adults
Researcher: Navjot Chahal

Dear potential participant,

My name is Navjot Chahal and I am a post graduate student enrolled in the Doctor of Clinical Psychology at the University of Auckland. I am the lead investigator for a research project being conducted in the Department of Psychology. The research is partially funded by Research Expenses Funding for Doctoral Students, Department of Psychology and aims to examine the neuropsychological and functional outcomes of healthy New Zealand adults.

You are invited to participate in a study about cognitive abilities and general wellbeing. Cognitive abilities include performance on tests of memory, attention, language and visual problem solving. I am asking individuals who speak English and live in New Zealand to participate in individual assessment sessions that include tests of these abilities. Participant will participate in one assessment session of up to 150 minutes. Assessments will take place in accessible facilities within the University of Auckland or in your home. The assessment will be conducted by me, or by another postgraduate student (Helen Vykopal) also enrolled in the Doctor of Clinical Psychology.

You are under no obligation to participate. Your participation is **voluntary** and you may withdraw at any time. Your name and identifying information will not be associated with published results.

The tests to be administered are commonly used by clinicians in the field. As such participation in this study could influence how you perform if you are referred for a neuropsychological assessment following participation in the study. Therefore, all the test data will be held in secure storage on university premises for a period of 10 years, and if you are referred for neuropsychological assessment during this period, a summary of your test results can be made available following written request from your GP. After the 10 years has elapsed all data will be shredded.

Participants can withdraw their information from the study by contacting the researcher at any time before May 1st 2009. There are no risks associated with the study though people often find the tasks involved slightly boring, and it is expected that you may find some of the items difficult. Participants will be given a petrol voucher in case they have to travel for assessment or a nominal gift of approximately $ 5 on participation.

If you would like to participate please complete the attached consent form and return in the freepost envelope provided <u>OR</u> contact me using the information below to

obtain further information and a consent form.

Thank you for your time and help in making this study possible. If you have any questions or would like to discuss participation, please contact me at the address/ phone number below:

Contact persons:
Navjot Chahal,
Department of Psychology
The University of Auckland
Private Bag 92019, Auckland, New Zealand
(09) 3737599 ext. (84990)
Email- ncha127@ ec.auckland.ac.nz

The head of the department 2007 is:
Fred Seymour
Department of Psychology
The University of Auckland
Private Bag 92019
Auckland, New Zealand
(09) 373 7599 ext (88516)

If you have any concerns of an ethical nature you can contact: Chair, The University of Auckland Human Participants Ethics Committee, Office of Vice Chancellor, University of Auckland, Private Bag 92019, Auckland. Telephone (09) 373 7599 ext. 87830

Approved by University of Auckland Human Participants Ethics Committee on 20th June for a period of 3 years from June 2007 to June 2010 Reference: 2007/206.

Appendix E:
Consent Form-Control group

DEPARTMENT OF PSYCHOLOGY

THE UNIVERSITY OF AUCKLAND
NEW ZEALAND
Te Whare Wānanga o Tāmaki Makaurau
Building 721, Tamaki Campus
261 Morrin Road, Glen Innes
Auckland, New Zealand
Telephone 64 9 373 7599
Facsimile 64 9 373 7000
The University of Auckland
Private Bag 92019
Auckland, New Zealand

CONSENT FORM

THIS CONSENT FORM WILL BE HELD FOR A PERIOD OF SIX YEARS

Title: Cognitive abilities and general well being of New Zealand adults
Researcher: Navjot Chahal

I have been given and have understood an explanation of this research project. I have had the opportunity to ask questions and have them answered.

I understand that I may withdraw myself or any information traceable to me at any time up to May 1st 2009 without giving a reason.

I understand that all information collected will be held in secure storage on University premises for a period of 10 years.

I understand that, with my permission, the information collected will be available to my GP.

- I agree to take part in this research
- I understand that participation will involve one session lasting up to two and a half hours.

Signed:_____ Date:_____
(Signature)

Name:_____
(Please Print)
Contact Phone(s):_____

I wish to receive a petrol voucher/ nominal gift of approximately $5 for my participation in the assessment session.

YES ☐ NO ☐

Approved by the University of Auckland Human Participant Ethics Committee on 20th June for a period of 3 years from June 2007 to June 2010 Reference Number 2007/206

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