NEUROPSYCHOLOGICAL, PSYCHOLOGICAL AND FUNCTIONAL OUTCOMES 12-MONTHS POST-SUBARACHNOID HAEMORRHAGE POPULATION-BASED SAMPLE COMPARED TO MATCHED CONTROLS

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ABSTRACT

Stroke is a common neurological disorder that is a leading cause of disability worldwide and may result in deterioration of functioning in neuropsychological, psychological and functional abilities. While accounting for a small proportion of all strokes, subarachnoid haemorrhage (SAH) affects a comparatively young age group who live with its burden for longer. A thorough understanding of SAH survivors' short-term (≤12-months) outcomes and trajectory is imperative as most change and rehabilitation occurs during this time. However, previous research has been limited by use of hospital- and clinic-based samples, comparison to normative data, limited outcomes focus, and use of brief measures.

The current population-based study examined SAH survivors' (n=30) outcomes throughout the first 12-months compared to control participants (n=29) matched on age, gender and ethnicity. Both groups were assessed using a neuropsychological test battery (e.g., verbal and visual memory, cognitive flexibility, processing speed, cognitive screening measure) and on psychological (anxiety, depression, overall) and functional (stroke symptoms, disability, health related quality of life [HRQoL]) outcomes; the SAH group at 28-days, 6- and 12-months post-SAH.

As compared to controls, the SAH group performed significantly worse with greater proportions falling in the impaired range on some neuropsychological outcomes (e.g., cognitive screening measure) and most psychological and HRQoL outcomes throughout the 12-months, despite good outcomes regarding stroke symptoms and disability. Some early improvement in outcomes was found but this plateaued during the 6- to 12-month period and SAH survivors' outcomes remained poor compared to controls. Psychological and HRQoL outcomes in particular were interrelated, with previous stroke and surgical clipping related to worse HRQoL outcomes.

The current findings demonstrate the importance of psychological and HRQoL outcomes in particular throughout the first 12-months post-SAH as compared to the more frequent emphasis of stroke symptoms and disability, suggesting a different direction for assessment and intervention focus. Neuropsychological outcomes are also impaired, though more research using a larger population-based sample and test battery are required to better understand domain specific impairment, trajectory and relation to other outcomes.

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LIST OF ABBREVIATIONS

Abbreviation	Full Expression
ADL	Activities of daily living
ANOVA	Analysis of variance
ARCOS IV	Auckland Regional Community Outcome Stroke Study IV
BDS	Behavioural Dyscontrol Scale
BI	Barthel Index
BMI	Body mass index
BP	Bodily pain (SF-36)
CNS-VS	CNS Vital Signs
CT	Computed tomography
EIF	Environmental Independence Factor (BDS)
FAI	Frenchay Activities Index
FIF	Fluid Intelligence Factor (BDS)
FIM	Functional Independence Measure
GCS	Glasgow Coma Scale
GH	General health perceptions (SF-36)
GHQ-28	General Health Questionnaire 28
HADS	Hospital Anxiety and Depression Scale
HRQoL	Health related quality of life
ICH	Intracerebral haemorrhage
LHS	London Handicap Scale
LIADLS	Lawton Instrumental Activities of Daily Living Scale
MCS	Mental Component Summary (SF-36)
MH	General mental health (SF-36)
MMSE	Mini-Mental State Examination
MoCA	Montreal Cognitive Assessment
MPF	Motor Programming Factor (BDS)
MRI	Magnetic resonance imaging
mRS	Modified Rankin Scale
NCI	Neurocognition Index (CNS-VS)
NIHSS	National Institute of Health Stroke Scale
NZ	New Zealand

PCS ..... Physical Component Summary (SF-36)
PH ..... Physical functioning (SF-36)
PIS ..... Participant Information Sheet
PSA ..... Post-stroke anxiety
PSAHD ..... Post-subarachnoid haemorrhage depression
PSD ..... Post-stroke depression
PTSD ..... Post-traumatic stress disorder

RE ..... Role limitations due to emotional health (SF-36)
RP ..... Role limitations due to physical health problems (SF-36)

SAH ..... Subarachnoid haemorrhage
SD ..... Standard deviation
SF ..... Social functioning (SF-36)
SF-36 ..... Short Form 36
SIP ..... Sickness Impact Profile

TICS ..... Telephone Interview for Cognitive Status

US ..... United States

VT ..... Vitality, energy and fatigue (SF-36)

WHO ..... World Health Organisation

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CHAPTER I:

INTRODUCTION

Neuropsychological, Psychological and Functional Outcomes 12-Months Post-Subarachnoid Haemorrhage: Population-Based Sample Compared to Matched Controls

Synopsis

The purpose of this study was to examine the neuropsychological, psychological and functional outcomes of survivors' 12-months post-subarachnoid haemorrhage in comparison to matched controls. As such, the following literature review provides an introduction to stroke including an overview of definitions and epidemiology, with a focus on subarachnoid haemorrhage (SAH). This is followed by a section on stroke outcomes again focussed on SAH outcomes. As this is a New Zealand (NZ) based study, data from NZ are presented where available.

Introduction to Stroke

Stroke: Definition and Subtypes

Stroke, also known as a cerebrovascular accident, is a spontaneous acute vascular injury to the brain, resulting in rapidly developing clinical symptoms and/or signs of focal, and at times global (i.e., patients in deep coma) loss of brain function, with symptoms lasting more than 24-hours or leading to death (Feigin, 2004; Warlow, 2001; World Health Organisation [WHO], 1989). The cardinal pathogenic feature of a stroke is the disruption of the supply of nutrients, primarily oxygen and glucose, to the brain as a result of disrupted blood flow (Bogousslavsky, Hommel, & Bassetti, 1998). As the brain does not store required nutrients but rather relies on a constant 24-hour supply from circulating arterial blood,

disrupted supply rapidly (within several minutes) starves brain tissue, creating area(s) of damaged or dead brain tissue (i.e., infarct; Feigin, 2004; Lezak, Howieson, & Loring, 2004).

Stroke is a pathologically heterogeneous condition. Two prominent mechanisms that account for infarction are obstruction of blood vessels, or ischemic stroke; and rupture of blood vessels, known as haemorrhagic stroke (Bogousslavsky et al., 1998). Ischemic and haemorrhagic stroke are depicted in Figure 1. An undetermined stroke is a stroke in which the causal pathology cannot be determined by radiological scanning or autopsy (Brown, Whisnant, Sicks, O'Fallon, & Wiebers, 1996b; Thrift, Dewey, Macdonell, McNeil, & Donnan, 2001).

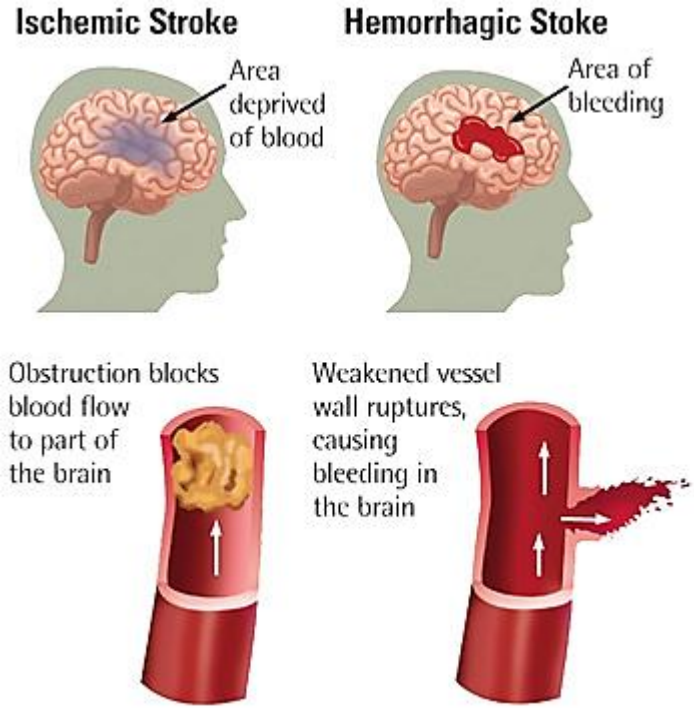

Figure 1. Types of stroke (Pahner, 2011)

Ischemic strokes, which account for 80% to 85% of all strokes (Wiebers, Feigin, & Brown, 2006), may be thrombotic, where blood particles and tissue overgrowth accumulate (a thrombus) in a build-up of fat deposits (atherosclerotic plaques) within arterial walls,

which narrows or closes the blood vessel (Bogousslavsky et al., 1998; Lezak et al., 2004); or embolic, whereby an emboli (plug of thrombic material or foreign matter) migrates from an extracranial artery and causes a blockage in an intracranial artery (Feigin, 2004; Lezak et al., 2004). Thrombotic strokes often take up to half an hour to develop fully, with up to one third of cases evolving over hours or days, whereas embolic strokes tend to be abrupt in presentation (Lezak et al., 2004; Yamamoto, Matsumoto, Hashikawa, & Hori, 2001). Ischemic stroke patients typically present with headache of a throbbing nature and symptoms consistent with neurologic dysfunction related to a single arterial territory (Rossor, 2001; Wiebers et al., 2006).

The two most common causes of haemorrhagic stroke are weakening of a vessel wall due to pathological alterations secondary to hypertension, which is present in 78% to 88% of cases; and rupture associated with a vascular abnormality such as an aneurysm or vascular malformation (Qureshi et al., 2001b). Haemorrhagic strokes can be divided into four subgroups based on the location of the primary haemorrhage. The primary haemorrhage can occur in the brain tissue (intracerebral haemorrhage, ICH), the subarachnoid space (SAH), the ventricles (intraventricular haemorrhage), or the subdural space (subdural haemorrhage; Warlow, 2001; Wiebers et al., 2006). Intraventricular haemorrhage most commonly occurs as an extension of ICH or SAH rather than being the primary haemorrhage, while subdural haemorrhage is more often traumatic than spontaneous in origin (Warlow, 2001; Wiebers et al., 2006). Thus, ICH and SAH are the most commonly considered spontaneous haemorrhagic strokes. The onset of ICH is usually rapid and continues to evolve over hours, typically presenting as severe headache and decreased level of consciousness with non-focal symptoms predominating initially over focal neurologic deficit (Wiebers et al., 2006). As the focus of this research is SAH, the following section reviews SAH and its most common causes in greater detail.

SAH: Definition and Common Mechanisms. SAH, which accounts for approximately 5% of all strokes (van Gijn, Kerr, & Rinkel, 2007), is bleeding into the subarachnoid space; the narrow space surrounding the brain between the pia matter, the fine covering that clings to the surface of the brain, and the next covering, the arachnoid matter (Feigin, 2004; Ogden, 2005). Brain tissue is not usually affected, though vasospasm (contraction of blood vessels in the region of the bleed) occurs in approximately 30% of cases, causing ischemia and infarction (Britz & Mayberg, 1997; Feigin, 2004). Diagnosis is most accurate via computed tomography (CT) brain scanning obtained within 24-hours, which detects SAH in 93% to 98% of cases, though from 24-hours up to 40-days following the haemorrhage magnetic resonance imaging (MRI) becomes increasingly superior (Al-Shahi, White, Davenport, & Lindsay, 2006; Edlow & Caplan, 2000; Wiebers et al., 2006). Patients with suspected SAH but normal CT and/or MRI require a lumbar puncture to confirm diagnosis (Al-Shahi et al., 2006; Edlow & Caplan, 2000).

SAH is commonly provoked by exertion, presenting suddenly as abrupt onset of a new, severe headache of no apparent cause, commonly compared by patients to being hit over the head (Rossor, 2001; Warlow, 2001; Wiebers et al., 2006). This headache is the most common clinical symptom and may be the only symptom in up to one third of patients (Al-Shahi et al., 2006; Kopitnik & Samson, 1993; Linn, Rinkel, Algra, & van Gijn, 1998), but is often accompanied by nausea, vomiting, neck stiffness, photophobia, and rapid alteration in level of consciousness, with brief loss of consciousness occurring in half of patients (Feigin, 2004; Rossor, 2001; Warlow, 2001; Wiebers et al., 2006). Up to one third of patients also develop focal neurological signs within the hour (Lezak et al., 2004; Wiebers et al., 2006). However, as many as 30% of all SAHs present atypically, creating the potential for misdiagnosis (Edlow & Caplan, 2000; Wiebers et al., 2006).

The most common cause of SAH is spontaneous rupture of an aneurysm (75% to 85%; Feigin, 2004; Ogden, 2005; van Gijn & Rinkel, 2001a; Wiebers et al., 2006). Non-aneurysmal SAH (up to 25% of SAHs) is most commonly caused by non-aneurysmal perimesencephalic haemorrhage (10% of SAHs), though it can also be caused by a variety of rare conditions such as arteriovenous malformation, arterial dissection, vascular lesions in the spinal cord, cocaine abuse, and trauma (5% of SAHs; Edlow & Caplan, 2000; van Gijn & Rinkel, 2001a; Warlow, 2001; Wiebers et al., 2006). In up to 15% of cases, the cause remains unknown (Wiebers et al., 2006).

Aneurysms, the most common cause of SAH, are balloon-like blood filled sacs that develop over time out of weak spots in arterial walls (Morita, Puumala, & Meyer, 1998; Ogden, 2005; van Gijn et al., 2007). Intracranial aneurysms most commonly arise at sites of large artery bifurcations involving the Circle of Willis and its major branches; specifically, the anterior communicating artery complex (29%), the internal carotid-posterior communicating artery junction (23%), the middle cerebral artery (23%), multiple lesions (15%), the vertebrobasilar circulation (5%), the internal carotid artery bifurcation (3%) and the distal portion of anterior cerebral artery (2%; Al-Shahi et al., 2006; Richardson, 1969; Wiebers et al., 2006). Approximately 2% of people have unruptured aneurysms, with prevalence increasing from the third decade (Rinkel, Djibuti, Algra, & van Gijn, 1998). The annual risk of rupture is up to 2%, and increases with age, female gender, increased aneurysm size, irregular shape, and posterior circulation location (Backes et al., 2014; Lindner, Bor, & Rinkel, 2010; Rinkel et al., 1998; Suarez, Tarr, & Selman, 2006; Wermer, van der Schaaf, Algra, & Rinkel, 2007). Approximately 20% of SAH patients have multiple aneurysms, thus identification of the ruptured aneurysm is crucial for effective management (Al-Shahi et al., 2006; Richardson, 1969).

Non-aneurysmal perimesencephalic haemorrhage, the most common cause of non-aneurysmal SAH, is defined by its characteristic distribution of blood in the subarachnoid space (Linn et al., 1998; van Gijn et al., 2007). The haemorrhaged blood is confined to the cisterns around the midbrain, with the centre of the haemorrhage located immediately anterior to the midbrain (Schwartz & Solomon, 1996; van Gijn & Rinkel, 2001a). Though the clinical presentation is very similar, patients with non-aneurysmal SAH are typically in better neurological condition and have more favourable short- and long-term outcomes compared to those with aneurysmal SAH (Cánovas, Gil, Jato, de Miquel, & Rubio, 2012; Kopitnik & Samson, 1993; Suarez et al., 2006). Headache onset is comparatively gradual (minutes rather than seconds) and loss of consciousness and focal symptoms are rare and transient (Linn et al., 1998; Schwartz & Solomon, 1996; van Gijn et al., 2007; van Gijn & Rinkel, 2001a).

Stroke Epidemiology

Having reviewed stroke definitions and subtypes along with the most common mechanisms of SAH, the following section examines the epidemiology of stroke. This includes examining incidence, prevalence, case fatality, and stroke risk factors. Specific examination of the epidemiology of SAH is presented within each subsection.

Stroke subtypes, incidence and case fatality. Stroke is the fifth most common neurological disorder in the United States (US), affecting approximately 795,000 people each year (Hill & Feasby, 2002; Lloyd-Jones et al., 2010). Globally, there are approximately 16 million new stroke victims annually, with over 80 million people suffering from the results of a stroke at any given time (Feigin, 2004; Strong, Mathers, & Bonita, 2007). It is expected that by 2030, due to population aging, the annual incidence of stroke will increase to 23 million (Strong et al., 2007).

Ischemic strokes comprise approximately 80% to 85% of all strokes (Feigin, 2004; Warlow, 2001; Wiebers et al., 2006). Of these, more than 75% are thrombotic, making this the most common stroke subtype (60% to 70% of all strokes), and up to 25% are embolic (Bogousslavsky et al., 1998; Castillo & Bogousslavsky, 1997). Haemorrhagic strokes comprise 15% to 20% of all strokes (Wiebers et al., 2006). The most common haemorrhagic stroke is ICH, causing up to 60% of brain haemorrhages and accounting for 10% of all strokes (Warlow, 2001; Wiebers et al., 2006). Approximately 5% of strokes are due to SAH (Feigin, 2004; Warlow, 2001; Wiebers et al., 2006).

A number of population-based studies support these rates. For example, in Melbourne and South London, respectively, the incidence of first-ever stroke has been reported at 73% and 73% ischemic, 15% and 14% ICH, 4% and 6% SAH, and 9% and 8% undetermined (Heuschmann, Grieve, Toschke, Rudd, & Wolfe, 2008; Thrift et al., 2001; Wolfe et al., 2002). However, stroke subtype proportional frequencies vary between countries, with high income countries (according to World Bank's country classification) averaging 82% ischemic, 11% ICH, and 3% SAH; compared to 67% ischemic, 22% ICH, and 7% SAH for low to middle income countries (Feigin, Lawes, Bennett, Barker-Collo, & Parag, 2009).

Further demonstrating the worldwide variation in stroke incidence, the highest crude subtype-specific stroke incidence rates (2000 to 2008 period) have been reported in Valley d'Aosts, Italy (ischemic 174 per 100,000 people) and Tbilisi, Georgia (ICH 44 per 100,000 people; SAH 16 per 100,000 people), while the lowest rates are in Iquique, Chile (ischemic 47 per 100,000 people) and Dijon, France (ICH 10 per 100,000 people, and SAH 2 per 100,000 people; Feigin et al., 2009). It is also significant to note that stroke incidence rates may vary considerably within countries. For example, age-adjusted stroke incidence rates in

Beijing, China were reported at 135 per 100,000 people compared to 76 per 100,000 people in Shanghai, China (Wang et al., 2007).

Over the last four decades, worldwide stroke incidence rates have declined by 42% in high income countries, but have more than doubled in low to middle income countries which now, for the first time, exceed those reported in high income countries (Feigin et al., 2009). Research suggests that the trend to decline can be attributed to increased education and intervention aimed at reducing modifiable risk factors (e.g., hypertension, smoking, alcohol consumption, obesity; de Rooij, Linn, van der Plas, Algra, & Rinkel, 2007; Wang et al., 2007; Warlow, 2001) along with more sensitive diagnostic techniques (e.g., CT scanning) to clarify diagnosis (McCarron, Smith, & McCarron, 2006; Wolf, 1997); whereas the trend to increasing stroke incidence may reflect the state of health and demographic transition in low and middle income countries, including increased exposure to modifiable risk factors (Brown et al., 1996b; Connor, Walker, Modi, & Warlow, 2007; Strong et al., 2007).

Within the general trend to decline in high income countries, regional variation exists ranging from a slight increasing trend (e.g., Rochester, Minnesota; Brown et al., 1996b), to stable trends (e.g., The Framingham Study; Wolf et al., 1992), to modest (e.g., 14% decline in Novosibirsk, Russia; Feigin, Wiebers, Whisnant, & O'Fallon, 1995; 18% decline in the US; Lloyd-Jones et al., 2010) and large (e.g., 43% decline in Perth, Australia; Islam et al., 2008) trends to decline. In NZ, stroke incident rates were stable from 1981-1982 to 1991-1992 and then declined in 2002-2003, producing an overall decline in stroke incidence of 11% during this period (Anderson et al., 2005).

In regards to case fatality, stroke is the most common life-threatening neurological disorder, ranking third amongst all causes of death in the US, accounting for 1 of every 15 deaths (Biller & Ferro, 2011; Bonita, Broad, & Beaglehole, 1997a; Brown, Baird, Shatz, &

Bornstein, 1996a; Romero, 2007). Of the approximately 16 million annual new stroke victims, case fatality at 12-months is approximately 5.7 million (Strong et al., 2007).

Early case fatality (within one-month) following ischemic stroke is approximately 20% (Wiebers et al., 2006), and ranges from 35% to 52% for haemorrhagic strokes, depending on the subtype (Carhuapoma & Hanley, 2002). In Australia, 28-day case fatality was 12% for ischemic stroke, 45% for ICH, 50% for SAH, and 38% for undetermined stroke (Thrift et al., 2001).

As with stroke incidence, regional variation in case fatality is evident (Sarti, Rastenyte, Cepaitia, & Tuomilehto, 2000). Generally, early case fatality is decreasing in both high and low to middle income countries (Feigin, Lawes, Bennett, & Anderson, 2003; Lloyd-Jones et al., 2010; McCarron et al., 2006; Warlow, 2001) though it remains 25% higher in low to middle income countries, likely due to their increasing incidence rates and less sophisticated and available interventions (Feigin et al., 2009). Over 80% of all stroke deaths occur in low to middle income countries, with stroke mortality predicted to double in these countries by 2020 (Connor et al., 2007; Murray & Lopez, 1997; Reddy, 2004; Strong et al., 2007).

As has been demonstrated above, the distribution of the burden of stroke is heterogeneous and variable over time (Sacco et al., 1997). The following subsection explores the epidemiology of SAH to place the present study within this wider context.

SAH incidence and case fatality. SAH accounts for approximately 5% of all strokes (Feigin, 2004; van Gijn et al., 2007; Wiebers et al., 2006) and affects 21,000 to 33,000 people each year in the US (Suarez et al., 2006). The international yearly incidence of SAH is reported to be approximately 10 per 100,000 people (Bonita & Thomson, 1985; Suarez et al., 2006).

However, a ten-fold variance between countries in the incidence of SAH has been reported in two multinational comparisons (de Rooij et al., 2007; Ingall, Asplund, Mähönen, & Bonita, 2000), with yearly incidence per 100,000 persons ranging from 2 in China to greater than 22 in Finland and Japan. In NZ, the age- and sex-adjusted annual incidence of SAH in the late 1990's was reported at 10 per 100,000 people, which was high compared to Australian cities (The ACROSS Group, 2000). In the 2000-2008 period NZ reported the highest crude incidence rate of SAH (10 per 100,000) and the highest proportion of SAH strokes (6%) among high income countries (Feigin et al., 2009). Rate recalculation based on ethnicity suggested that the high rates in NZ could in part be accounted for by a high rate among Maori and Pacific people (The ACROSS Group, 2000); discussed further in the broader context of stroke risk factors, which general consensus attributes overall incidence variation to (de Rooij et al., 2007). As was demonstrated with stroke in general, the incidence of SAH varies considerably on a regional basis (Kopitnik & Samson, 1993).

From 1981-1982 to 1991-1993 the incidence of SAH in NZ decreased from 15 to 11 per 100,000 people (Truelsen, Bonita, Duncan, Anderson, & Mee, 1998). Worldwide, the incidence of SAH is thought to have decreased by 0.6% per year since 1950 (de Rooij et al., 2007). Despite reports of a modest decline in the incidence of SAH, researchers generally agree that the incidence of SAH has actually remained stable for decades, with any apparent decline attributable to the increasing proportion of patients investigated with CT scanning, reducing false-positive diagnosis of SAH based on loose diagnostic criteria (Feigin et al., 2009; Islam et al., 2008; Linn, Rinkel, Algra, & van Gijn, 1996; Suarez et al., 2006; van Gijn & Rinkel, 2001a).

Since the 1960's, population-based studies have reported early case fatality rates for SAH ranging from 20% to 67% (Wiebers et al., 2006); with 50% early case fatality generally accepted as an overall estimate (Al-Shahi et al., 2006; Estol, 2001; Suarez et al., 2006; van

Gijn et al., 2007). Early case fatality for SAH in high income countries is up to 23% lower than that reported in low to middle income countries (Feigin et al., 2009), likely due to poorer identification and management of SAH in low to middle income countries (Ingall et al., 2000). Since the 1970's, early case fatality rates have decreased by 0.6% per year, resulting in a 17% decrease over the following three decades (Nieuwkamp et al., 2009). Though not reported in all populations (Islam et al., 2008), early case fatality for SAH has been decreasing in NZ, reducing from 53% in 1982, to 46% in 1992, to 32% by 1998 (Nieuwkamp et al., 2009; The ACROSS Group, 2000; Truelsen et al., 1998).

Risk factors for stroke and SAH. A risk factor is a characteristic of an individual or population associated with increased risk of a disease compared with an individual or population without that characteristic (Warlow, 2001). Risk factors for stroke have been well researched, and are commonly classified into modifiable and non-modifiable factors (Biller & Ferro, 2011; Lezak et al., 2004; Romero, 2007). Although most risk factors have an independent effect, there are likely significant interactions between factors, making stroke risk estimation increasingly complex (Bushnell et al., 2014; Feigin, 2004; Meschia et al., 2014; Nordahl et al., 2014). While review of all risk factors is beyond the scope of this study, the following section reviews some of the most important 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 are untreatable risk factors (i.e., age, sex, ethnicity, family history). They are of relevance to identifying individuals at greater risk of stroke (Biller & Ferro, 2011).

Age. Age is the most important non-modifiable stroke risk factor (Biller & Ferro, 2011; Goldstein et al., 2001; Sacco et al., 1997). From the fourth decade stroke incidence rises rapidly, with each subsequent year increasing the risk of stroke by 9% in men and 10% in women (Ariesen, Claus, Rinkel, & Algra, 2003; Asplund et al., 2009; Norris & Hachinski,

2001). In a review of population-based studies, the average age of stroke onset was 70 years for males and 75 years for females (Feigin et al., 2003). Only 4% of strokes occur in people aged 15 to 40 with no more than 25% occurring before age 65; more than half of strokes occur in people over 75 years (Feigin, 2004; Feigin et al., 2003; Warlow, 2001).

The incidence of SAH also increases with age, though less markedly (Longstreth, Koepsell, Yerby, & van Belle, 1985; van Gijn et al., 2007). Approximately 20% of SAHs occur in people aged 15 to 45, with SAH accounting for 40% to 50% of strokes in patients under 35 years of age (Biller & Ferro, 2011; Longstreth et al., 1985). SAH most frequently occurs between ages 40 and 60, with over half SAH patients being younger than 55 (Kopitnik & Samson, 1993; Ogden, 2005; van Gijn et al., 2007). A prospective community-based study in Italy demonstrated that SAH accounted for 23% of strokes in those under 45 compared to only 2% for patients over 45 (Marini et al., 2001). In NZ the mean age of SAH onset is 59 years compared to 72 and 69 for ischemic stroke and ICH, respectively (Feigin et al., 2006).

Sex. Stroke incidence rates are up to 25% greater in men, having higher age-specific incidence than women with exceptions being the 35 to 44 year age group and those over 85 years where women have greater incidence (Ariesen et al., 2003; Feigin, 2004; Goldstein et al., 2001; Norris & Hachinski, 2001; Thrift et al., 2001). However, the lifetime risk of stroke is higher in women (1 in 5) than in men (1 in 6) due to women having longer life expectancy (Carter et al., 2006; Katsiki, Ntaios, & Vemmos, 2011; Sacco et al., 1997; Seshadri et al., 2006). Stroke case fatalities are also higher in women than men (Goldstein et al., 2001; Sacco et al., 1997). The average age of stroke onset in NZ women is 76 years; approximately 5 to 10 years later than men (Anderson et al., 2005; Dyall et al., 2006). From 1981-1982 to 2002-2003, there was a significant decline (16%) in stroke incidence in males, whereas stroke incidence in women remained relatively stable during this period, with a significant decline

(14%) noted between 1991-1992 and 2002-2003 only (Anderson et al., 2005; Dyall et al., 2006).

The incidence of SAH is up to twice as high in women compared to men (Eden et al., 2008; Nilsson, Lindgren, Stahl, Brandt, & Säveland, 2000; Sandvei, Romundstad, Müller, Vatten, & Vik, 2009; Thrift et al., 2001). This sex difference appears at 55 years and increases thereafter (de Rooij et al., 2007). However, research suggests that the generally accepted sex difference in SAH incidence is not universal. In a multinational review, SAH incidence rates were essentially the same for both sexes in South London, East Germany, China, Yugoslavia, Italy, Sweden, and Lithuania; up to 40% higher in women in Poland, Northern Sweden, and Denmark; and up to 80% higher in men in Finland and Russia (Heuschmann et al., 2008; Ingall et al., 2000). In 2002-2003, SAH incidence was similar for both sexes in NZ (women 53%, men 47%; Feigin et al., 2006), though women accounted for more than 60% of SAH incidence in the previous two decades (Truelsen et al., 1998).

Ethnicity. Stroke incidence and case fatality varies with regards to ethnicity, with ethnic minorities generally being at increased risk (Agyemang et al., 2014; Feigin, 2004; Warlow, 2001). In comparison to whites, African Americans have a 2-fold increased risk of stroke (Lloyd-Jones et al., 2010; Sacco et al., 1997; Wolfe et al., 2002), while Hispanics have over a 60% increased risk (Sacco et al., 1997; White et al., 2005). The average age of stroke onset is also up to 11 years younger for ethnic minorities (White et al., 2005; Wolfe et al., 2002), with case fatality up to 50% higher in African Americans than whites (Mensah, Mokdad, Ford, Greenlund, & Croft, 2005). In NZ, Maori and Pacific people have a significantly higher relative risk of stroke (odds ratio 1.3 and 1.6, respectively) and early case fatality (odds ratio 1.4 and 1.6, respectively) compared with Europeans (Bonita et al., 1997a), with stroke onset also occurring at significantly younger ages for Maori/Pacific peoples (62 years) and Asian/other (64 years) than in Europeans (75 years; Feigin et al., 2006). Ethnic

variations are commonly attributed to higher incidence of modifiable stroke risk factors, particularly hypertension, diabetes, and obesity (Bonita et al., 1997a; Feigin et al., 2006; Feigin & Rodgers, 2004; Mensah et al., 2005), combined with limited access to and delivery of appropriate health services (Feigin, McNaughton, & Dyall, 2007). From 1981-1982 to 2002-2003, stroke incidence rates in NZ declined significantly for European (19%), but remained high for Maori (non-significant 2% increase) and increased by 4- and 8-fold for Pacific and 'other' peoples, respectively (Anderson et al., 2005; Carter et al., 2006; Feigin et al., 2007). During this same period declining trends in early case fatality for all ethnic groups were reported, ranging from non-significant for Maori to a 70% decline for Asian/other (Carter et al., 2006).

Non-white ethnicity is also a significant risk factor for SAH, increasing risk more than 3-fold (Feigin et al., 2005b; Suarez et al., 2006). Population-based research in the US reported that Mexican Americans account for 60% of SAH though they make up only 48% of the population, producing a risk ratio of 1.7 compared to white Americans (Eden et al., 2008). In NZ, the proportional stroke subtype frequency of SAH is similar across ethnic groups (6% European, 7% Maori/Pacific, 5% Asian/other; Feigin et al., 2006); however, Maori/Pacific SAH incidence rates range from 17 to 25 per 100,000 people compared to 8 to 9 per 100,000 people of non-Maori/Pacific ethnicity (The ACROSS Group, 2000).

Family history. A family history of stroke, particularly if occurring before age 65, increases the risk of stroke (Feigin, 2004). The Framingham Study demonstrated that stroke in parents under 65 years was associated with increased risk of stroke of the same type in their offspring, producing a hazard ratio of 2.8 (Seshadri et al., 2010). This increased risk could be mediated through genetic heritability of other risk factors (e.g., hypertension, diabetes, heart disease), familial sharing of environmental and lifestyle factors, or an interaction of these (Feigin, 2004; Goldstein et al., 2001).

Multivariate models have shown that family history of SAH is an independent risk factor for SAH (Biller & Ferro, 2011). A positive family history (first-degree) of SAH could account for approximately 11% of SAH cases (Ruigrok, Buskens, & Rinkel, 2001), and reduces the average age at which SAH occurs compared to patients without a family history (van Gijn et al., 2007). For individuals with one first-degree relative affected by SAH, the odds ratio of SAH is 2.5 compared to individuals with no family history (Bor et al., 2008; Woo et al., 2009). This odds ratio increases to 51.0 for individuals with two or more first-degree relatives affected by SAH (Bor et al., 2008).

Modifiable risk factors. Modifiable risk factors (e.g., hypertension, diabetes mellitus, heart disease, cigarette smoking) can be reduced via significant lifestyle changes and medical management, thus reducing the risk of stroke (Biller & Ferro, 2011; Wolf, 1997). Stroke is thought to be preventable in up to 85% of cases by controlling these modifiable risk factors (Feigin, 2004; Wiebers et al., 2006).

Hypertension. Hypertension is an important risk factor for all stroke types, though more so for haemorrhagic strokes for which it is *the* key risk factor (Feldmann et al., 2005; Lezak et al., 2004; McCarron et al., 2006; Sturgen et al., 2007; Wolf, D'Agostino, Belanger, & Kannel, 1991; World Health Organisation, 1989). Uncontrolled hypertension is associated with ischemic stroke (risk ratio 1.5) and strongly related to haemorrhagic stroke (risk ratio 3.0) and is responsible for 27% and 57% of these stroke types, respectively (Klungel et al., 2000). Patients with treated hypertension are also at increased stroke risk (odds ratio 1.4; Woo et al., 2004).

Stroke incidence and case fatality increase in proportion to increases in systolic, diastolic, and combined blood pressures, with a relative stroke risk of up to 33.0 for stage three hypertension compared to normotensive individuals (Ariesen et al., 2003; Goldstein et al., 2001; Kannel, Vasan, & Levy, 2003). Systolic blood pressure may be particularly

problematic, with every 10mm Hg increase increasing stroke risk by more than 25% (Asplund et al, 2009; Harmsen, Lappas, Rosengren, & Wilhelmsen, 2006). Normotensive patients have approximately half the lifetime risk of stroke of patients with hypertension (Lloyd-Jones et al., 2010; Seshadri et al., 2006).

Systematic reviews report that hypertension increases the risk of SAH up to 3-times (Feigin et al., 2005b; Teunissen, Rinkel, Algra, & van Gijn, 1996), with specific populations reporting odds ratios up to 4.3 (Inagawa, 2005). Approximately 17% of SAH cases can be attributed to hypertension and it is up to 30% more hazardous for women (Feigin et al., 2005b; Ruigrok et al., 2001). As with stroke in general, the risk of SAH increases in proportion with increases in hypertension (Sandvei et al., 2009), with every 10mm Hg increase in systolic blood pressure increasing the risk of SAH by 31% (Feigin et al., 2005a).

Diabetes mellitus. Diabetes mellitus is an independent risk factor for stroke and increases case fatality (Harmsen et al., 2006; Warlow, 2001). Stroke incidence among diabetic patients is 1.7 to 6.3 times higher than in non-diabetics (Adams, Putman, Kassell, & Torner, 1984; Giorda et al., 2007). However, this relationship varies with stroke type (Adams et al., 1984). Patients with aneurysmal SAH have lower or equivalent prevalence of diabetes mellitus compared to the general population, suggesting that diabetic patients are not at increased risk of aneurysmal SAH (Adams et al., 1984; Qureshi et al., 2001a). Conversely, diabetic patients may have up to a 30% risk reduction of aneurysmal SAH, possibly due to better medical treatment and altering lifestyle factors (Feigin et al., 2005b).

Heart disease. People with heart problems, such as angina, heart failure, valve disorders, artificial valves and congenital heart defects are at increased risk of stroke, particularly via cardiogenic embolism (Feigin, 2004). The most significant cardiac stroke risk factor is atrial fibrillation (irregular heartbeat where the left atrium beats rapidly and unpredictably); accounting for up to 50% of all cardiogenic embolic strokes (Goldstein et al.,

2001; Sacco et al., 1997). Atrial fibrillation independently increases the risk of stroke by up to 10-fold (Feigin, 2004; Harmsen et al., 2006; Lloyd-Jones et al., 2010; Romero, 2007). With regards to SAH, heart disease is an important risk factor for aneurysm formation though it is also associated with a decreased risk of aneurysm rupture, perhaps partially due to atherosclerosis and restriction of strenuous physical activity (Inagawa, 2010).

Cigarette smoking. Cigarette smoking is a considerable stroke risk factor and the single largest contributor to preventable stroke in some populations (Kleinig, Kimber, & Thompson, 2009b). In NZ current smokers have a 4-fold increased risk of stroke compared with people who never smoked (Bonita, Duncan, Truelsen, Jackson, & Beaglehole, 1999). A dose-response relationship exists between smoking (quantity and duration) and stroke risk (Feigin, 2004). One prospective cohort study reported that smoking up to 21 cigarettes per day had a relative stroke risk of 1.6 and 1.4 for men and women, respectively; increasing to 2.2 and 3.9, respectively, with greater quantities (Ueshima et al., 2004). Passive smoking among non-smokers has also been shown to increase the risk of stroke by 80% (Bonita et al., 1999). Each year of smoking cessation substantially decreases stroke risk, nearly matching that of a never smoker after 5 years (Wiebers et al., 2006).

Cigarette smoking is an independent risk factor for SAH and has been identified as *the* most important modifiable cause of SAH (Biller & Ferro, 2011; Anderson et al., 2004; Feigin et al., 2005a). During 2002-2003 in NZ smoking was the most common risk factor (54%) identified by SAH patients (Feigin et al., 2006) and up to 30% of cases are attributable to current smoking (Anderson et al., 2004; Ruigrok et al., 2001). Population-based research and systematic reviews report that compared to never smokers, current smoking increases the risk of SAH between 3- and 6-fold (Anderson et al., 2004; Feigin et al., 2005b; Qureshi et al., 2001a; Sandvei et al., 2009; Teunissen et al., 1996). Ex-smokers have a 2- to 4-fold increased risk (Feigin et al., 2005b; Qureshi et al., 2001a; Sandvei et al., 2009; Teunissen et

al., 1996). A dose-response relationship between current and lifetime cigarette smoking and SAH risk is evident (Kurth et al., 2003), as is an increased risk from passive smoking (Anderson et al., 2004).

Obesity. Being overweight as determined by Body Mass Index (BMI; normal range 19-25 kg/m^2) increases the risk of stroke (Feigin, 2004). For every increase of $2kg/m^2$ in baseline BMI, the relative risk of stroke increases by 6.1%; an association that has been reported in both genders and different ethnic populations (Katsiki et al., 2011). Though BMI is the most widely reported measure of obesity, central obesity (waist circumference) may relate even more closely to increased stroke risk (Biller & Ferro, 2011; Romero, 2007).

Research regarding BMI and SAH is inconsistent (Feigin et al., 2005b). A review of 26 cohort studies found no significant association between BMI and SAH (Feigin et al., 2005a), however, some research suggests that being underweight increases the risk of SAH (Anderson et al., 2004; Sandvei et al., 2009), whereas overweight people may have lower risk of SAH (Sandvei et al., 2009).

Alcohol consumption. There is ample evidence that excessive alcohol consumption increases risk of stroke. Compared to abstainers, individuals who consume over 60g of alcohol per day have an increased risk of ischemic (odds ratio 1.7) and haemorrhagic (odds ratio 2.2) stroke (Wiebers et al., 2006). However, this relationship depends on the type of stroke and amount and consistency of alcohol consumed (Wiebers et al., 2006). A linear dose-response relationship exists between haemorrhagic stroke and alcohol consumption, whereas a curvilinear relationship is apparent between ischemic stroke and alcohol consumption, suggesting low to moderate alcohol consumption has a protective effect, increasing ischemic stroke risk only with higher exposure (Ariesen et al., 2003; Mazzaglia, Britton, Altmann, & Chenet, 2001; Patra et al., 2010). For all stroke types, chronic heavy drinking and binge drinking are associated with increased stroke risk (Mazzaglia et al., 2001).

Multivariate models confirm that alcohol consumption is an independent risk factor for SAH (Biller & Ferro, 2011). Systematic reviews of SAH risk factors demonstrate a linear dose-response relationship, whereby consuming less than 150g of alcohol per week increases relative risk of SAH by 2.8, increasing to 4.7 for consumption in excess of this (Feigin et al., 2005b; Teunissen et al., 1996). Consuming 100-299g of alcohol per week may account for 11% of SAH cases, and up to 21% of cases when consumption exceeds 300g of alcohol per week (Ruigrok et al., 2001).

Carotid stenosis. Carotid stenosis, the narrowing or blockage of the inner surface of the carotid artery most often caused by atherosclerotic processes is another important stroke risk factor (Lezak et al., 2004; Murros & Toole, 1997; Rajamani & Fisher, 1997; Sacco, 2001). Research using specific populations demonstrates that up to 70% of stroke victims have over 50% carotid stenosis, with ischemic events up to 3-times more likely in people with severe (over 75%) or progressing carotid stenosis (Chambers & Norris, 1986; Schwartz et al., 1995). Furthermore, every 0.1mm increase of intima-media thickness results in a 1.5 hazard ratio, increasing stroke risk by 19-fold once intima-media thickness extends beyond 1.2mm (Silvestrini et al., 2010). An association between bilateral carotid stenosis and SAH has also been reported, with SAH more likely to occur on the side of severest stenosis (Kleinig, Kimber, & Thompson, 2009a).

Summary

While accounting for a small proportion of all strokes, SAH has the highest case fatality rates and affects a comparatively young age group; thus survivors live with its burden for longer. A number of important risk factors have been presented, with cigarette smoking, hypertension, and alcohol consumption emerging as particularly relevant to SAH (Biller & Ferro, 2011; Feigin et al., 2005b; Teunissen et al., 1996). The following section focuses on

outcomes up to 12-months post-stroke. As literature specific to SAH is sparse, literature on stroke outcomes in general is reviewed with literature on SAH presented where available.

Stroke and SAH Outcomes

Stroke is the leading cause of disability in the world, resulting in significant burden on patients, their families, health care systems, and society (Biller & Ferro, 2011; Feigin, 2004; Gloede et al., 2014; McDowell, 1997; Warlow, 2001). Furthermore, declining stroke fatality rates increase the number of survivors living with stroke outcomes; thus, the importance of furthering stroke outcomes research cannot be overstated.

Stroke outcomes are complex and may manifest in varied and multiple domains of a survivor's functioning (WHO, 2001). In consideration of the WHO International Classification of Functioning, Disability and Health framework's core sets for stroke (Geyh et al., 2004) and the terminology commonly employed within the literature, the outcome domains of neurological, neuropsychological, psychological, and functional have been used within the context of this study.

As stroke impacts functioning across multiple domains, it is important to exercise a multifocal approach to studying stroke outcomes to provide a holistic understanding of its impact (Barker-Collo & Feigin, 2006). However, to date stroke outcomes research has favoured neurological and functional outcomes at the relative cost of neuropsychological and psychological outcomes, despite their prevalence and the substantial contribution they make to patients' stroke recovery (Morris, Wilson, & Dunn, 2004; Salmond et al., 2006). There is also a lack of research exploring outcome profiles associated with various stroke subtypes (Barker-Collo & Feigin, 2006).

Researching stroke outcomes throughout the first year post-stroke is particularly important as a means of informing rehabilitation and care. Stroke outcomes tend to change

over time yet their natural course remains to be delineated, which will assist in developing appropriately targeted rehabilitation (Haug et al., 2007; Patel, Coshall, Rudd, & Wolfe, 2003). Furthermore, while recovery may continue in the years post-stroke, the most substantial changes occur in this first year (Samra et al., 2007), making informed rehabilitation during this time imperative. Finally, population-based research is limited, though necessary to provide an accurate view of outcomes given that hospital- and clinic-based samples suffer from selection bias which often results in the exclusion of stroke survivors with mild and severe outcomes (Barker-Collo & Feigin, 2006).

The following section reviews literature on the short-term (≤12-month) neurological, neuropsychological, psychological, and functional outcomes post-stroke with a focus on SAH, after which the associations between these outcomes are examined. Somewhat greater attention is given to neuropsychological, psychological, and functional outcomes and studies which are population-based as these are the primary focus of the current study.

Neurological Outcomes

The neurological impact of stroke may include recurrence and impairments such as motor, sensory, verbal and perceptual deficits, and behaviour change. Various scales are used to assess neurological outcomes; those commonly used include the National Institute of Health Stroke Scale (NIHSS; Brott et al., 1989) and the Hunt and Hess grading system (Hunt & Hess, 1968).

One of the most thorough examinations of the prevalence of acute stroke impairments was conducted by Lawrence and colleagues (2001) using population-based data from the South London Stroke Register (n=1259). Nearly all (98%) patients were found to have some impairment, with over 50% experiencing 6 to 10 impairments. The most common impairments were upper (77%) and lower (72%) limb weakness, urinary incontinence (48%), impaired consciousness (45%), and dysphagia (45%), though visual field defects, sensory

deficits, dysarthria, gaze paresis, and ataxia were also common. Similar findings have been reported in other population- (Taub, Wolfe, Richardson, & Burney, 1994) and hospital- (Hochstenbach, Mulder, van Limbeek, Donders, & Schoonderwaldt, 1998) based research. In a multi-centre European study (Di Carlo et al., 1999) comparing stroke impairments in the very old (≥80 years) with younger stroke survivors (n=4499), older patients were shown to experience more severe neurological outcomes, with urinary incontinence (57%), paralysis (49%), weakness (42%), aphasia (40%) and dysphagia (39%) occurring significantly more often in this age group. Increased stroke symptoms (NIHSS) has repeatedly been associated with increasing age as well as female gender, though these demographic characteristics do not relate to neurological improvement or deterioration post-stroke (Abdul-Rahim et al., 2015; Gattringer et al., 2014; Irie et al., 2015). At 12-months post-stroke, a significant portion of survivors still experience impairments (Cederfeldt, Gosman-Hedström, Gutiérrez Pérez, Sävborg, & Tarkowski, 2010).

In comparison to impairments following other stroke subtypes, SAH patients have the highest rates of impaired consciousness (82%), coma (67%), and gaze paresis (46%; Lawrence et al., 2001). Urinary incontinence (71%), dysphagia (68%) and limb weakness (66%) are other common impairments following SAH (Lawrence et al., 2001). At 3-months post-stroke, Powell, Kitchen, Heslin and Greenwood (2002) found that 61% of their SAH sample (n=52) still had at least one impairment of dysphasia (28%), impaired visual acuity (24%), tinnitus (16%), vertigo (12%), visual field deficit (8%), and/or diplopia (6%) though none suffered from nystagmus, ataxia, or locomotor deficits.

Neuropsychological Outcomes

Neuropsychological functioning refers to the behavioural expression of brain functioning and includes abilities such as planning, memory, language, psychomotor, and information processing speed (Barker-Collo & Feigin, 2006; Lezak et al., 2004).

Neuropsychological outcomes are an important sequel of stroke and are considerably impaired in 30% to 65% of stroke survivors (Ballard, Rowan, Stephens, Kalaria, & Kenny, 2003; Srikanth et al., 2003).

Given the diversity and complexity of neuropsychological functioning, neuropsychological test batteries are required to adequately assess these outcomes. However, in the few population-based studies that have examined neuropsychological functioning post-stroke, brief global screening measures such as the Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975) were often used (Douiri, Rudd, & Wolfe, 2013; Lisabeth et al., 2014; Patel et al., 2002; Patel et al., 2003). Such measures only assist in detecting gross and severe cognitive decline, and are biased towards attention, memory, and language, resulting in an overly simplified and insensitive measure of neuropsychological outcomes, likely under-identifying neuropsychological deficits (Hochstenbach et al., 1998; Rasquin, Lodder, & Verhey, 2005). For example, Cao and colleagues (2007) found that a portion (not reported) of their ischemic stroke survivors classified as having no cognitive impairment using the MMSE were concurrently assessed as having global cognitive impairment as measured by a neuropsychological test battery. Considerable under-identification of cognitive deficits using the MMSE was also demonstrated by Cederfeldt and colleagues (2010), whereby this measure identified cognitive impairment in 29% and 10% of stroke survivors in the acute phase and 12-month follow-up, respectively, compared to 91% and 77% identified by a neuropsychological test battery. Hence, the literature presented here focuses on research using neuropsychological test batteries to examine short-term post-stroke and -SAH neuropsychological outcomes.

Stroke. As summarised in Table 1, stroke survivors may experience a variety of domain specific cognitive impairments in such areas as orientation, attention, memory, information processing speed, language, arithmetic, visuospatial abilities, and executive

Table 1

Neuropsychological outcomes using battery of tests ≤ 12 months post-stroke

Author/Study	Sample	Time post-stroke	Measures	Outcomes
Multiple assessment points				
Cederfeldt et al., (2010) Longitudinal Hospital-based Sweden	Stroke N=45 (sample ≥65 years) $\bar{x}$=76 years Exclusion: severe medical conditions, aphasia, dementia	Discharge, 12-months	CIQ, MMSE, WMS-R (LM), Cronholm-Molander, copy (mirror image cup, cube), count (cube), EXIT, ST, TMT, RCM	% with impairment at discharge vs. 12-months: - any cognitive impairment 91% vs. 77% - visual memory 35% vs. 49% - verbal memory 34% vs. 39% - logical deductive ability 51% vs. 61% - visuospatial 49% vs. 46% - executive function 66% vs. 55% - speed and attention 76% vs. 70%
Rasquin et al., (2005) Longitudinal Hospital-based The Netherlands	First-ever hemispheric stroke N=156 $\bar{x}$=69 years Exclusion: <40 years, baseline MMSE <15, aphasia, other neurological deficits, psychiatric disorder	1-, 6-, and 12-months	AVLT, ST, CST, GIS, CAMCOG	- ↓ in memory, mental speed, executive function, orientation, attention, language, praxis, visuospatial, and calculation - memory, mental speed, and calculation steadily ↑ - most impaired throughout: mental speed (39-60%), calculation (39-51%), and executive function (26-34%) - least impaired throughout: orientation (5-7%)
Serrano et al., (2007) Longitudinal Hospital-based Spain	Stroke (excluded SAH) N=327 $\bar{x}$=70.9 years	3- and 12-months	MMSE, SPMSQ, hearing and simple/random visual reaction time, Bell Test, COWA, WAIS (PR, WL, LM, BD, SM), BAB (naming), TT	- 3-months: 19.6% cognitive impairment (no dementia); 30% dementia - 12-months: 26.8% cognitive impairment (no dementia); 22.6% dementia

Author/Study	Sample	Time post-stroke	Measures	Outcomes
Controlled comparisons				
Cao et al., (2007) Cross sectional Hospital-based Italy	First-ever ischemic stroke N=40 (sample <49 years) $\bar{x}$=38.8 years Exclusion: aphasia, mental retardation, psychiatric illness, alcohol/drug abuse, metabolic disorders, other CNS diseases Matched control group (age, education) N=40 $\bar{x}$=37.8 years	$\bar{x}$=9.2 months (range 6- to 12-months)	MMSE, AVLT, DS, BSRT, TT, COWA, BNT, CBTB, SM, RPM	- ↓ performance compared to controls on every test (mean scores and proportion failing each test; sig for AVLT delayed, TT, RPM) - 5=dementia (MMSE) - 3=global impairment - 8=partial impairment
Hochstenbach et al., (1998) Cross-sectional Hospital-based The Netherlands	Stroke N=229 $\bar{x}$=55.9 years Exclusion: >70, other major physical illness or mental disorders Control group (not matched) N=33 $\bar{x}$=52.4 years	$\bar{x}$=72.2 days (range 5- to 293-days)	RAVLT (Dutch version), WAIS (IN, DS, DSy, BD, SM), RBMT, TMT, letter cancellation task, BIT (PS, CT), Bobertag, structured clock test, clock-drawing task, MRMT, DAS (comprehension, COWA, naming), arithmetic (money handling)	- 70% slowed information processing speed - 50% left- 44% right-sided neglect (attention) - 40% visuospatial and constructive impairment - 40% arithmetic impairment - 40% language impairment - 30% memory impairment (long- worse than short-term)

Author/Study	Sample	Time post-stroke	Measures	Outcomes
Srikanth et al., (2003) Cross sectional Population-based Australia	First-ever stroke (excluded SAH) N=89 $\bar{x}$=70.5 years Exclusion: aphasia, severe auditory/visual impairment Matched control group (age, sex) N=89 $\bar{x}$=69.9 years	3-months	WAIS-R (IN, DS, SM, AR, PC, BD, DSy), K-SNAP (mental status, gestalt closure, number recall, 4-letter words), MMSE, RAVLT, RBMT, ROCF, clock drawing, COWA	- stroke 1.5 times ↑ to have any cognitive impairment - stroke ↑ impaired in attention, orientation, visuospatial, language, executive domains - memory deficits comparable between groups
Weinstein et al., (2014) Cohort Population-based United States	First-ever stroke N=132 $\bar{x}$=76.8 years Exclusion: cognitive decline pre-stroke, no educational information Matched control group (age, sex) N=132 $\bar{x}$=77.4 years	6-months	WAIS-R (LM, VR, PA, SM, DS,BD, IN), COWA, WRAT	- stroke sig ↑ impaired in short- and long-term verbal and visual memory, learning, visuospatial, language, abstract reasoning, executive - no sig differences in attention, visual perception

AR=Arithmetic; AVLT=Auditory Verbal Learning Test; BAB=Boston Aphasia Battery; BD=Block Design; BIT=Behavioural Inattention Task; BNT=Boston Naming Test; BSRT=Babcock Story Recall Test; CAMCOG=Cambridge Examination for Mental Disorders of the Elderly; CBTB=Corsi's Block-Tapping Board; CIQ=Cognitive Impairment Questionnaire; COWA=Controlled Oral Word Association; CST=Concept Shifting Test; CT=Copying Task; DAS=Dutch Aphasia Society; DS=Digit Span; DSy=Digit Symbol; EXIT=Executive Interview; GIS=Groninger Intelligence Scale; HVOT=Hooper Visual Organisation Test; IN=Information; K-SNAP=Kaufman Short Neuropsychological Assessment; LM=Logical Memory; MMSE=Mini Mental Status Examination; MRMT=Money's Road Map Test; PA=Paired Associates; PC=Picture Completion; PR=Picture Recognition; PS=Picture Scanning; RAVLT=Rey Auditory Verbal Learning Test; RBMT=Rivermead Behavioural Memory Test; RCM=Raven's Coloured Matrices; ROCF=Rey-Osterreith Complex Figures; RPM=Raven's Progressive Matrices; SM=Similarities; SPMSQ=Short Portable Mental Status Questionnaire; ST=Stroop Test; TMT=Trail Making Test; TT=Token Test; VR=Visual Reproduction ; WAIS=Wechsler Adult Intelligence Scale; WAIS-R=Wechsler Adult Intelligence Scale-Revised; WL=Word Learning; WRAT=Wide Range Achievement Test; WMS-R=Wechsler Memory Scale-Revised

functions, as well as global cognitive decline including dementia up to 12-months post-stroke (Cao et al., 2007; Cederfeldt et al., 2010; Hochstenbach et al., 1998; Rasquin et al., 2005; Serrano, Domingo, Rodríguez-Garcia, Castro, & del Ser, 2007; Srikanth et al., 2003; Weinstein et al., 2014). It has been suggested that cognitive impairment prior to stroke significantly increases the risk of global cognitive impairment and dementia 3-months post-stroke (Serrano et al., 2007), with increasing age and ethnic minority status also associated with less favourable 3-month neuropsychological outcomes (Douiri et al., 2013; Lisabeth et al., 2014; Patel et al., 2002). Though considerable variability is evident, the cognitive domains most commonly and most extensively impaired appear to be attention, executive functions, and information processing speed (Cederfeldt et al., 2010; Hochstenbach et al., 1998; Rasquin et al., 2005). Memory deficits are often reported but are not as impaired as the previous domains (Cederfeldt et al., 2010; Hochstenbach et al., 1998), with one population-based study concluding comparable memory abilities between stroke patients (excluding SAH) 3-months post-stroke and matched control participants (Srikanth et al., 2003).

Few studies have examined short-term changes in neuropsychological functioning post-stroke. Those that have report few assessment periods spanning long time periods (typically 9- to 12-months), creating substantial gaps when mapping the natural course of recovery (Cederfeldt et al., 2010; Rasquin et al., 2005; Serrano et al., 2007). Hochstenbach and colleagues (1998) reported that cognitive functioning does not change significantly during 12-months post-stroke, though they made between-subject comparisons of patients assessed only once at variable times post-stroke. Others report both improvement (Ballard et al., 2003; Cederfeldt et al., 2010) and deterioration (Serrano et al., 2007) in global cognitive functioning during this period. Though using the MMSE to assess cognitive functioning, Patel and colleagues (2003) demonstrated that in their population-based sample cognitive functioning assessed at 3- and 12-months post-stroke most commonly remained stable, but

improved (24%) and deteriorated (13%) in some cases, highlighting the variable course cognitive outcomes may take. The presence of visuospatial neglect was found to compromise cognitive recovery during this period (Patel et al., 2003).

With regards to domain specific cognitive functions, attention, visuospatial abilities, information processing speed, and executive functions have been shown to improve over the initial 12-months post-stroke, though often remain impaired compared to norms and control participants (Cederfeldt et al., 2010; Rasquin et al., 2005). Memory has been shown to both improve (Rasquin et al., 2005) and deteriorate (Cederfeldt et al., 2010) during this time. In a systematic review of the post-stroke memory literature, Snaphaan and de Leeuw (2007) conclude that memory dysfunction generally improves between 3- and 12- months post-stroke, with prevalence rates ranging from 23% to 55% and 15% to 31%, respectively.

As is apparent from the above, there is considerable variation in the research findings regarding neuropsychological outcomes post-stroke and their short-term progression. Such variation is likely due to a combination of factors, including sampling differences, stroke subtype inclusion, variable and broad follow-up points, and the use of different neuropsychological tests and cut-off criteria to quantify cognitive impairment. Additional methodological limitations such as the use of hospital- and clinic-based samples, small sample sizes, considerable loss to follow-up, and the exclusion of very old patients or those with severe neurological and cognitive outcomes or psychiatric disorders also limit the generalizability of these findings.

SAH. Table 2 summarises the literature on short-term neuropsychological outcomes of SAH survivors, focusing on research using neuropsychological test batteries for reasons previously described. As can been seen in Table 2, the literature consistently reports that SAH survivors experience a range of cognitive deficits up to 12-months post-SAH, whether they have good (Cheng, Shi, & Zhou, 2006) or poor (Haug et al., 2010) neurological grade

Table 2

Neuropsychological outcomes using battery of tests ≤ 12 months post-SAH

Author/Study	Sample	Time post-SAH	Measures	Outcomes
Cheng et al., (2006) Prospective cohort Hospital-based China	Aneurysmal SAH pre surgery N=37 $\bar{x}$=46.2 years Exclusion: >70 years, acute Fisher score >III, complications (e.g., neurological deterioration, vasospasm), Hunt and Hess grade >II, serious medical or mental conditions	$\bar{x}$=22 days (range 1-day – 5-years)	WAIS-R (AR, DS, DSy), WMS-R (PC, VR), COWA, TMT, MMSE	- 29.7% no cognitive ↓ - 37.8% 1 cognitive deficit - 32.4% ≥2 cognitive deficits - 70.3% ↓ in some aspect of cognition - attention, memory, and executive functions most impacted
Egge et al., (2005) Prospective cohort Hospital-based Scandinavia	Aneurysmal SAH N=42 $\bar{x}$=47.6 years	12-months	HRTB (SRT, TMT), WMS-R (verbal and visual PA), COWA, WCST (computerised), Grooved Pegboard	- 7% normal cognition - 93% ↓ cognition; most commonly memory, information processing speed, mental flexibility, problem solving
Haug et al., (2009) Prospective cohort Hospital-based Norway	ACoA vs MCA aneurysmal SAH N=24 compared to 22 $\bar{x}$=53.0 years Exclusion: Hunt and Hess grade >III, ≥70 years, aphasia, other diagnosis, alcohol/drug abuse	12-months	CVLT, Continuous Visual Memory Test, Grooved Pegboard, WAIS-III (DS, DSy), WAIS-R (VC, SM, BD, MR), D-KEFS	- within 1SD of mean on verbal and visual memory, attention, fine motor, psychomotor, most measures of executive and IQ

Author/Study	Sample	Time post-SAH	Measures	Outcomes
Haug et al., (2010) Retrospective cohort Hospital-based Norway	Severe grade (Hunt and Hess grade V) aneurysmal SAH N=26 $\bar{x}$=49 years	12-months	Grooved Pegboard, WAIS-III (DSy, DS), D-KEFS, CVLT, ROCF, WAIS-R (VC, SM, BD, MR)	- half=good cognitive function: mild decline in psychomotor, memory, attention; normal executive and IQ - half=poor cognitive function: severe decline in memory; moderate decline in psychomotor, executive, attention, IQ
Mayer et al., (2002) Prospective cohort Hospital-based United States	Non- and aneurysmal SAH (excluded AVM) N=113 $\bar{x}$=49.4 years Exclusion: admission >14 days post ictus	3-months	TICS, SBT, VSAT, WMS-R (VR), ROCF, CVLT, CalCAP, Grooved Pegboard, Trails A and B, WCST (modified), WAIS-R (BD), BNT, TT	% of patients >2 SD ↓ mean: - up to 43%=global deficits - 42% verbal memory - 39% psychomotor - 28% reaction time - 27% language - 23% visual memory - 21% visuospatial - 18% executive
Multiple assessment points				
Alfieri et al., (2008) Longitudinal Hospital-based Italy	Non-aneurysmal SAH N=30 $\bar{x}$=41.7 years Exclusion: >80 years, cerebral lesions, admission GCS <13	Inpatient, 1-, 3-, 6-, and 12-months	ZVNCT, D2TA, WAIS-III, WCST (modified), WASI (BD, MR), RAVLT, BVRT, RRT, TLT, ROCF	- sig ↓ in visuoconstructional and executive -↓ in attention, memory, information processing speed
Brand et al., (2014) Longitudinal Hospital-based Germany	SAH, all inclusive N=60 $\bar{x}$=48.6 years Exclusion: >65 years, GOS>2, aphasia, alexia, agraphia, alcohol/drug abuse, mental disorder	6- and 12- months	D2TA, PTS, IST, H-WIT, VLRT, BVRT, TMT	% impaired (performing under 2SD↓ mean) at 6- and 12-months: - concentration > 60% reduced to 50% - executive up to 70% reduced to 60%; several specific functions deteriorated - working memory 45% reduced to 35%; long-term memory deteriorated from 40% to 50% impaired - sig ↓ processing speed 6- to 12-months

Author/Study	Sample	Time post-SAH	Measures	Outcomes
Haug et al., (2007) Longitudinal Hospital-based Norway	Aneurysmal SAH N=32 $\bar{x}$=54 years Exclusion: >70 years, aphasia, previous cerebrovascular or neurological disease, alcohol/drug abuse	3-, 6-, and 12-months	Grooved Pegboard, WAIS-III (DS, DSy), CVLT, ROCF, D-KEFS (colour word interference), WASI (SM, BD), BNT (short version)	- sig ↓ in all cognitive domains at 3-months - fine motor improved by 6-months; remained sig ↓ - psychomotor and visual memory gradually improved, reaching normal range at 12-months - verbal memory improved 6-12months; remained ↓ - executive: inhibition gradually improved, ↑ normative mean 12-months; switching sig ↓ at each time point - attention sig ↓ at each time point - IQ was least affected
Hillis et al., (2000) Cohort study Hospital-based (surgical clipping) United States	Aneurysmal SAH N=27 N=6 at 12 months $\bar{x}$=51 years Exclusion: <21 years, previous neurosurgery, other neurological disease	3- and 12-months	WAIS-R (DS, BD, Dsy), WMS-R , BNT, WRMT (faces, words), RAVLT, ROCF, ST, COWA, Grooved Pegboard	- 3-months: performance sig ↓ mean for attention, memory, language, visuospatial, executive, psychomotor - minority with severe deficits account for the overall results (bimodal distribution) - 12-months: sig ↑ in language only
Ogden et al., (1993) Longitudinal Hospital-based NZ	SAH N=89 $\bar{x}$ women=46.4 years $\bar{x}$ men=44.7 years Exclusion: >70, not 'alert' prior to discharge, other diagnosis, head injury, alcohol/drug abuse	Inpatient, 10-weeks, 12-months	WAIS-R (DS, VC, SM, CM, PC, BD, PAr, DSy), RMT (words, faces), WMS (LM, Associate Learning), OSR (Form I), ROCF, MOQ, TT, NART, GNT, MTDDA, VNT, TMT, WCST	- IQ in average range throughout - memory and visuospatial sig ↑ over time; verbal, sustained attention, psychomotor speed and executive did not. - sig % with ↓ at 12-months = memory (esp. nonverbal), visuospatial construction, sustained attention, psychomotor speed, executive functions

Author/Study	Sample	Time post-SAH	Measures	Outcomes
Samra et al., (2007) Longitudinal Hospital-based International, multicentre	Aneurysmal SAH N=185 $\bar{x}$=51.3 years Exclusion: obese, WFNS>III, GOS>2 Matched control group (age, sex, ethnicity, education) N=45 $\bar{x}$=51.9 years	3- and 9-months	BVRT, COWA, Grooved Pegboard, ROCF, TMT, NART	- cognitive impairment 35.7% at 3-months, 25.8% at 9-months (23.3% at 15 months) - significant cognitive improvement from 3-9 months - plateau from 9-months onwards
Controlled comparisons				
Berry et al., (1997) Cross sectional Hospital-based (surgical treatment) United Kingdom	Aneurysmal SAH N=48 Mean= 44.1 years Exclusion: >60 years, visual deficits Matched control group (age, sex, estimated IQ) N=48 $\bar{x}$=41.9 years	8-months	WAIS-R (DS, VC, AR, SM, PAr, BD, OA, DSy), NART, RMT, WMS-R, CALEV recall and recognition test, subjective memory, WCST, Cognitive Estimates Test, COWA	- 37% sig ↓ in IQ compared to premorbid - memory: sig ↓ in recall and recognition - executive functions intact
Fontanella et al., (2003) Cross sectional Hospital-based Italy	Aneurysmal SAH N=37 (20 clipped, 17 coiled) $\bar{x}$=55.3 years Exclusion: WFNS > II, multiple or large aneurysms Control group (not matched) N=18	6-months	VST, BSWRT, CBS, Verbal Learning, LM, WAIS (Par), Elithorn's Test, Fluency (literal, category, associative), Verbal Judgement, Raven Coloured Matrices, Sentence Construction Test	- selective attention, memory, IQ comparable to controls - clipped: sig ↓ logical memory, language, and executive functions - coiled: sig ↓ in only one measure of executive (literal fluency)

Author/Study	Sample	Time post-SAH	Measures	Outcomes
Germanò et al., (1998) Cross sectional Hospital-based Italy	SAH of unknown cause (not aneurysm or AVM) N=20 $\bar{x}$=45.4 years Exclusion: >60 years, Hunt and Hess grade >2, Fisher score >II, vasospasm, complications (neurological deterioration, fever), GOS >1 at discharge, <5 years formal education, alcohol/drug history, other medical illness Matched control group (age, sex, education) N=20 $\bar{x}$=38 years	12-months	WAIS-R, WMS-R, RAVLT, TPT, TT, COWA, BVRT	- no significant differences between SAH, controls, and normative data re IQ, memory, attention, language, visuospatial/constructional, executive
Hadjivassiliou et al., (2001) Cross sectional Hospital-based United Kingdom	Aneurysmal SAH N=80 (40 surgical clipping; 40 endovascular coiling) Matched control group (mean age, sex, premorbid IQ) N=31	12-months	NART, WAIS-R (VC, DS, SM, AR, PAr, BD), word/face recognition, story/complex figure recall, CANTAB (CBS, ID/ED shift test, SWMT, TLT), COWA, digit ordering, BNT, TMT	- sig ↓ in IQ, memory, executive function, visual perception, and attention compared to controls - trend toward ↓ performance for surgical compared to endovascular (significant in VC, complex figure recall, semantic fluency, ID/ED shift test).

Author/Study	Sample	Time post-SAH	Measures	Outcomes
Passier et al., (2010) Cross sectional Hospital-based The Netherlands	Aneurysmal SAH N=111 $\bar{x}$=52.8 years Exclusion: institutionalised Control group (not matched) N=62 $\bar{x}$=57.8 years	3-months	WAIS-III (DS), COWA, RAVLT, ROCF (copy, delayed), BSAT, ST (colour word)	- 46.8% mild (1.5-2SD ↓ controls) and 25.2% severe (>2SD ↓ controls) impairment on ≥1 task - most commonly memory; less often visuospatial and executive; rarely attention.

ACoA=Anterior communicating artery; AR=Arithmetic; BD=Block Design; BNT=Boston Naming Test; BSAT=Brixton Spatial Anticipation Test; BSWRT=Bi-syllabic Words Repetition Test; BVRT=Benton Visual Retention Test; CalCAP=California Computerised Assessment Package; CANTAB=Cambridge Automated Neuropsychological Test Battery; CBS=Corsi block span test; CM=Comprehension; COWA=Controlled Oral Word Association; CVLT=California Verbal Learning Test; D-KEFS=Delis-Kaplan Executive Functional System; DS=Digit Span; DSy=Digit-Symbol; D2TA=d2 Test of Attention; GCS=Glasgow Coma Scale; GNT=Graded Naming Test; GOS=Glasgow Outcome Scale; H-WIT=Hamburg-Wechsler Intelligence Test; HRTB=Halstead-Reitan Test Battery; ID/ED shift test =Intradimensional/Extradimensional shift test; IQ=Intelligence Quotient; IST=Intelligence Structure Test; LM=Logical Memory; MCA=Middle cerebral artery; MMSE=Mini Mental Status Examination; MOQ=Memory Observation Questionnaire; MR=Matrix Reasoning; MTDDA=Minnesota Test for the Differential Diagnosis of Aphasia; NART=National Adult Reading Test; OA=Object Assembly; OSR=Oral Selective Reminding; PA=Paired Associates; PAr=Picture Arrangement; PC=Picture Completion; PTS=Performance Testing System; RAVLT=Rey Auditory-Verbal Learning Test; RMT=Recognition Memory Test; ROCF=Rey-Osterreith Complex Figures; RRT=Rey Recovery Test; SBT=Short Blessed Test; SM=Similarities; SRT=Seashore Rhythm Test; ST=Stroop Test; SWMT=Spatial Working Memory test; TICS=Telephone Interview of Cognitive Status; TLT=Tower of London Task; TMT=Trail Making Test; TPT=Toulouse-Pieron Test; TT=Token Test; VC=Vocabulary; VLRT=Verbal Learning and Retention Test; VNT=Visual Neglect Test; VR=Visual Reproduction; VSAT=Verbal Series Attention Test; VST=Visual Search Test; WAIS=Wechsler Adult Intelligence Scale; WAIS-R=Wechsler Adult Intelligence Scale-Revised; WAIS-III=Wechsler Adult Intelligence Scale 3rd edition; WASI=Wechsler Abbreviated Scale of Intelligence; WCST=Wisconsin Card Sorting Test; WFNS=World Federation Neurological Surgeons; WMS-R=Wechsler Memory Scale-Revised; WRMT=Warrington Recognition Memory Test; ZVNCT=Zahlen-Verbindungs Number Combination Test.

SAH. Global cognitive functioning and IQ are reportedly least affected (Haug et al., 2007; Ogden et al., 1993), though up to 43% do experience global cognitive decline (Mayer et al., 2002); perhaps more likely detected when performance is compared to pre-stroke functioning rather than standardised norms or control participants (Berry, Jones, West, & Brown, 1997). With regards to domain specific cognitive functioning, up to 93% of SAH survivors still experience deficits 12-months post-SAH (Egge et al., 2005). The only studies to report no significant decline in SAH cognitive functioning at 12-months compared to standardised norms and/or control participants (Germanò et al., 1998; Haug et al., 2009) based findings on a small sample size ($n \leq 24$) that consisted of rare and/or particular SAH mechanisms, and favourable selection bias (e.g., ≤60 years, mild severity, formal education).

Some research suggests a bimodal distribution in SAH cognitive outcomes, whereby patients have either a good cognitive outcome profile (mild impairment) or severely impaired cognitive outcome profile (Haug et al., 2010; Hillis, Anderson, Sampath, & Rigamonti, 2000). For example, on a visual recognition memory test nearly half (44%) of SAH patients (n=26) scored at the 10^{th} percentile or below and 12% performed in the 10-25 percentile range demonstrating very severe and moderate-severe deficits, respectively, while the remaining majority (44%) scored at the 60^{th} percentile or greater, suggesting average or above ability (Hillis et al., 2000). Patients more likely to have a good cognitive outcome profile were younger, had more years of education, and were less likely to have been shunted (Haug et al., 2010).

The cognitive deficits most commonly reported by SAH survivors are difficulties with attention, memory, and information processing speed (Hackett & Anderson, 2000; Passier et al., 2010). Though reported at higher rates than found by neuropsychological testing, these deficits, along with impaired executive functions are most prevalent throughout the initial 12-

months (Alfieri et al., 2008; Al-Khindi, Macdonald, & Schweizer, 2010; Brand et al., 2014; Cheng et al., 2006; Egge et al., 2005; Hadjivassiliou et al., 2001; Ogden et al., 1993).

Different domains of cognitive functioning have been shown to have different time courses. Assessment of global cognitive functioning (Telephone Interview for Cognitive Status [TICS]; Brandt, Spencer, & Folstein, 1988) suggests that up to 34% of SAH survivors improve from 3- to 12-months (Springer et al., 2009), though it appears that recovery begins to plateau at 9-months (Samra et al., 2007). Similar findings have also been reported for those with poor neurological grade SAH, but despite improvement they are likely to remain cognitively impaired at 12-months (Mocco et al., 2006). Higher premorbid intellect and younger age has been associated with better global cognitive recovery post-SAH (Ogden et al., 1993; Samra et al., 2007). Haug and colleagues (2007) provide the most thorough delineation of short-term domain specific cognitive recovery thus far. However, their findings must be interpreted with caution given the sample (n=32) was hospital-based and excluded patients over 70 years and/or with aphasia. However, these authors report that cognition may demonstrate early improvement before plateauing at 6-months (fine motor control); remain static before improving between 6- and 12-months (verbal memory); show gradual improvement throughout this period (psychomotor, visual memory, inhibition); or may not significantly improve at all (attention, mental flexibility). Of note, different aspects of the same cognitive domain may have different time courses (e.g., verbal and visual memory). Research also shows different rates of impairment of specific aspects within a cognitive domain. For example, Mayer and colleagues (2002) found that 42% and 23% of their sample (n=113) had verbal and visual memory deficits, respectively, 3-months post-SAH. Hence, the common practice of reporting complex cognitive domains as one entity (e.g., memory) may be misleading and likely contributes to variability between findings. Again, despite improvement, the most severely impaired domains (attention, memory,

information processing speed, executive function) frequently remain impaired at 12-months (Brand et al., 2014; Ogden et al., 1993).

The factors likely contributing to the variability in findings and the methodological limitations which were discussed in relation to stroke cognitive outcomes research (page 28) applies to the SAH literature, hence the need for further research.

Psychological Outcomes

Emotional problems are a common though often overlooked consequence of stroke (Anderson et al., 1995). A considerable range of assessment measures are used in the literature, including self-report or observer-rated questionnaires and clinical interview schedules (Hackett, Yapa, Parag, & Anderson, 2005; Poynter et al., 2009). The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) and the General Health Questionnaire 28 (GHQ-28; Goldberg, 1978) are two of the most commonly employed (Hackett & Pickles, 2014). Post-stroke depression (PSD) and anxiety (PSA) have received the most research attention as they are considered to be the most common post-stroke psychiatric conditions (Burvill et al., 1995). Although PSA will be reviewed in this section, the literature on PSA remains comparatively scarce, therefore the main focus will be on PSD and post-SAH depression (PSAHD). Stroke survivors living with PSD are willing to hypothetically trade more years of life compared with matched (cognitively and functionally) non-depressed survivors, demonstrating the reduced value they place on life and the subsequent importance of these outcomes (Bosworth, Horner, Edwards, & Matchar, 2000).

Prevalence rates of PSD vary considerably between studies, with a recent meta-analysis concluding that PSD is present in 31% of stroke survivors at any time (Hackett & Pickles, 2014; Hackett et al., 2005). During the acute phase, PSD rates range from 5% to 56% (Cao et al., 2007; Farner et al., 2010; Townend et al., 2007; West, Hill, Hewison, Knapp, & House, 2010); 21% to 53% at 3- to 4-months (Barker-Collo, 2007; Herrmann,

Black, Lawrence, Szekely, & Szalai, 1998; Kauhanen et al., 1999; Kim & Choi-Kwon, 2000; Kwok et al., 2006; Townend et al., 2007); 20% to 42% at 6-months (Dennis et al., 2000; Rasquin et al., 2005); and 11% to 48% at 12-months (Anderson et al. 1995; Farner et al., 2010; Herrmann et al., 1998; Kauhanen et al., 1999; West et al., 2010). Unfortunately, only one study was population-based (Hackett & Anderson, 2006), reporting PSD (GHQ-28) prevalence of 27% at 6-months. However, these results were based on only 60% of their sample due to incomplete data collection. The majority of PSD cases (up to 79%) develop within the first months post-stroke, most of which (up to 55%) continue to experience PSD at 12-months, though PSD may also develop after 3-months post-stroke (Anderson et al., 1995; Farner et al., 2010; Herrmann et al., 1998; Townend et al., 2007).

Prevalence of PSA ranges from 5% to 27% in the acute phase (Castillo, Schultz, & Robinson, 1995; Castillo, Starkstein, Fedoroff, Price, & Robinson, 1993; Townend et al., 2007); 12% to 37% at 3- to 4-months (Barker-Collo, 2007; Burvill et al., 1995; Castillo et al., 1995; Townend et al., 2007); 22% to 36% at 6-months (Castillo et al., 1995; Dennis et al., 2000); and 17% to 34% at 12-months (Castillo et al., 1995; Rasquin et al., 2005). High rates of comorbidity have also been reported, with up to 75% of people with PSA also having PSD at 3-months post-stroke (Castillo et al., 1995; Barker-Collo, 2007).

Regarding SAH, studies generally show increased rates of PSAHD and anxiety compared to controls (Powell et al., 2002). Those that report comparable rates were based on rare SAH cases (i.e., undetermined SAH) and/or samples with characteristics considered predictive of favourable outcome (e.g., single and small aneurysm, good neurological grade SAH, no complications; Fontanella et al., 2003; Germanò et al., 1998). PSAHD reportedly affects between 5% and 50% of survivors, while anxiety is experienced by 27% to 54% of survivors (Al-Khindi et al., 2010; Kreiter et al., 2013). Prevalence rates vary over time, ranging from 14% to 64% (PSAHD) and 16% to 52% (anxiety) at 3-months (Hillis et al.,

2000; Passier et al., 2010; Powell et al., 2002); 9% (PSAHD) and 17% (anxiety) at 9-months (Powell et al., 2002); and up to 33% (PSAHD) at 12-months (Kreiter et al., 2013), with up to 25% experiencing PSAHD throughout the first year post-SAH (Kreiter et al., 2013). In a population-based study, 39% reported ongoing difficulties with mood 12-months post-SAH (Hackett & Anderson, 2000). However, the assessment measure used (telephone interview asking a series of simple yes/no questions) limits its findings. Depressed patients also experience a pessimistic perception of their recovery at 12-months compared to matched (cognitively and functionally) non-depressed survivors (Hedlund, Zetterling, Ronne-Engstrom, Ekselius, & Carlsson, 2010), highlighting the need to attend to depression post-SAH.

The sudden, unexpected, and potentially life threatening nature of SAH has been shown to precipitate anxiety indicative of post-traumatic stress disorder (PTSD; Sheldrick, Tarrier, Berry, & Kincey, 2006). Such symptomology includes intrusive thoughts and avoidance of reminders, and is experienced at clinically significant levels in 18% to 60% of survivors during the first 3-months (Powell et al., 2002; Sheldrick et al., 2006), and 30% to 37% at 9- to 12-months (Noble et al., 2008; Powell et al., 2002).

As with most clinical research, discrepancies in prevalence rates may be attributed to differences in sample characteristics, testing time periods, definitions and assessment measures of psychiatric conditions, cut-off criteria, and whether or not participants are receiving treatment (Hackett et al., 2005; Poynter et al., 2009). Additional methodological limitations such as selection bias, broad assessment periods, and the lack of population-based research further limit the literature.

Controversy exists as to the relationship between various demographic variables and psychological outcomes (Kutlubaev & Hackett, 2014). Most studies report no association between age and PSD (Anderson et al., 1995; Brodaty et al., 2007; Herrmann et al., 1998;

Kim & Choi-Kwon 2000), though higher prevalence rates have been reported in both younger (Bosworth et al., 2000; Paradiso & Robinson, 1998) and older (Kauhanen et al., 1999) age groups. Gender has also been found to be irrelevant (Kauhanen et al., 1999; Kim & Choi-Kwon, 2000) but others report higher rates among women, particularly as the severity of PSD increases (Anderson et al., 1995; Herrmann et al., 1998; Paradiso & Robinson, 1998; Poynter et al., 2009; Reeves et al., 2008; West et al., 2010). Women may also be more likely to experience chronic PSD (Chemerinski, Robinson, & Kosier, 2001). Psychiatric history is occasionally reported as unrelated to PSD and PSA (Brodaty et al., 2007) but most research reports that about half of those with PSD or PSA have pre-stroke psychiatric histories (Burvill et al., 1995; Hackett & Anderson, 2006; Herrmann et al., 1998; Naess, Lunde, Brogger, & Waje-Andreassen, 2010). Psychiatric history may be particularly relevant to those who develop emotional difficulties acutely and to those with more severe symptomology (Castillo et al., 1995; West et al., 2010).

Functional Outcomes

Functional outcomes include disability (limitations in ability to perform normal functional activities), handicap (limitations in functional roles resulting from impairment or disability), and arguably the most important patient-centred outcome (Almborg, Ulander, Thulin, & Berg, 2010), health related quality of life (HRQoL; person's general wellbeing resulting from physical, psychological, and social aspects of life possibly affected by changes in health states; Barker-Collo & Feigin, 2006; Sturm et al., 2002; WHO, 2001). Commonly used measures to assess functional outcomes post-stroke include the Barthel Index (BI; Granger, Dewis, Peters, Sherwood, & Barrett, 1979), Frenchay Activities Index (FAI; Holbrook & Skilbeck, 1983), and the Lawton Instrumental Activities of Daily Living Scale (LIADLS; Lawton & Brody, 1969) (disability); the modified Rankin Scale (mRS; Bamford, Sandercock, Warlow, & Slattery, 1989) and the London Handicap Scale (LHS; Harwood,

Rogers, Dickinson, & Ebrahim, 1994) (handicap); and the Short Form 36 (SF-36; Ware & Sherbourne, 1992) and Sickness Impact Profile (SIP; Damiano, 1996) (HRQoL).

A substantial body of research demonstrates that stroke survivors experience considerable disability and handicap compared to norms and control participants (Barker-Collo & Feigin, 2006; Bonita, Solomon, & Broad, 1997b; Cao et al., 2007; Clarke, Marshall, Black, & Colantonio, 2002), with up to 20% requiring institutionalised care post-stroke (Barker-Collo & Feigin, 2006; Bonita et al., 1997b; Cederfeldt et al., 2010). Disability prevalence ranges from 71% during the acute phase (Cederfeldt et al., 2010); 47% to 54% at 3-months (Kwok et al., 2006; Taub et al., 1994); 62% at 6-months (Cederfeldt et al., 2010); and 34% to 58% at 12-months (Cederfeldt et al., 2010; Taub et al., 1994). Research findings are inconsistent regarding the recovery of functional abilities, with some demonstrating significant improvement from the acute phase and 3- to 12-months post-stroke (Cederfeldt et al., 2010), whereas others report no evidence of change (Kwok et al., 2006; Taub et al., 1994). Conversely, in a population-based study utilising the Swedish Stroke Register (Ullberg, Zia, Petersson, & Norrving, 2015), activities of daily living (ADL) dependency rates increased from 3- (16%) to 12-months (28%) post-stroke, with females more likely to deteriorate (relative risk of 1.6). However, this finding is limited by the simplistic measure employed (i.e., three questions, e.g., “Do you need help to visit the toilet? yes/no”).

What is clear is that stroke survivors continue to experience significantly reduced HRQoL up to 12-months post-stroke (Bays, 2001; Clarke et al., 2002; Hochstenbach et al., 2001). In a longitudinal multi-rehabilitation centre study (n=528), Franceschini and colleagues (2010) found that half to three quarters of their stroke participants still experienced problems in each of the HRQoL domains assessed (usual activities=77%, mobility=74%, self-care=62%, mood=60%, pain/discomfort=58%) at 12-months. Kwok and colleagues (2006) found that HRQoL significantly decreased in up to 31% of their stroke

sample (n=268) during the initial 12-months, demonstrating that HRQoL is at risk of further deterioration in the short-term for many patients.

Regarding SAH, deficits in basic and instrumental ADL may be observed in up to 12% and 93% of survivors, respectively, with up to 38% requiring constant supervision at the time of discharge (Al-Khindi et al., 2010; Saciri & Kos, 2002). However, improvement in such abilities is noted in the majority of SAH patients from discharge to 3-, 6- and 12-months (Mocco et al., 2006; Navi, Kamel, Hemphill, & Smith, 2012). High rates of handicap, particularly related to occupational roles, are also reported (Powell et al., 2002). Of those fully employed at the time of SAH, 41% to 77% return to full-time employment by 12-months, though reduced hours and demotion are common (Alfieri et al., 2008; Ogden, Mee, & Henning, 1993).

The HRQoL of SAH patients is considerably reduced. Up to 92% and 81% experience moderate or severe problems in at least one domain of HRQoL acutely and at 12-months, respectively, with most HRQoL domains remaining significantly impaired compared to norms at 12-months (SF-36; Haug et al., 2009; Meyer et al., 2010). At 4-months post-SAH, Katati and colleagues (2007) concluded that 43% of their sample (n=70) had impaired HRQoL overall (SF-36); most commonly in the domains of role limitations due to physical functioning (60%), mental health (47%), vitality (43%) and role limitations due to emotional health (40%). The least affected domain was bodily pain, though over 20% were still impaired. Physical, emotional, and social functioning domains have been shown to improve during the first 12-months, whereas other domains (e.g., household management, recreational activities, energy, general health) remain stable or deteriorate over this time (Al-Khindi et al., 2010; Noble et al., 2008).

Hackett and Anderson (2000) summarised the 12-month functional outcomes of SAH survivors in their population-based study (Australia and NZ). At 12-months, 4% required

institutional care and 46% reported they had still not completely recovered from the SAH. Approximately 10% were completely dependent in ADL, but two thirds had successfully returned to pre-stroke occupational status. With regards to HRQoL (SF-36), SAH survivors experienced significantly more role limitations resulting from physical problems compared with Australian population norms. Though summarising a range of functional outcomes, the results of this study are limited by the simple and brief assessment tools used (i.e., ADL were assessed by asking "do you require help from anyone for everyday activities?"; the acute SF-36 was used, assessing HRQoL over the prior week only), which likely underestimate the prevalence of poor 12-month functional outcomes post-SAH.

As will be seen, neurological, neuropsychological, and psychological outcomes are shown to relate to functional outcomes post-stroke and -SAH, however, relationships also exist between disability, handicap, and HRQoL. For example, The North East Melbourne Stroke Incidence Study (Sturm et al., 2002) assessed disability (BI) and handicap (LHS) at 3- and 12-months post-stroke. Not only did disability and handicap scores significantly correlate at both assessment points, but the mean handicap score significantly increased alongside level of disability. This study also assessed the relationship between perceived recovery ("Have you made a complete recovery from your stroke?") and disability. Of those reporting incomplete recovery, 37% were free from disability, demonstrating that multiple factors contribute to overall recovery and wellbeing post-stroke. Though not sufficient to predict HRQoL by themselves, disability and handicap do relate to HRQoL (Passier, Visser-Meily, Rinkel, Lindeman, & Post, 2013). For example, SAH survivors who are independent with only mild functional symptomology often have considerable reductions in HRQoL (SIP and SF-36); but HRQoL does significantly reduce as the level of dependence increases (Hop, Rinkel, Algra, & van Gijn, 1998). Other factors associated with poor HRQoL include inability to return to work, and limited social activities and social support (Bays, 2001). The

complex phenomenon of post-stroke fatigue experienced by 39% to 72% of stroke survivors and up to 89% of SAH survivors is also significantly related to each aspect of functional outcome (Barker-Collo, Feigin, & Dudley, 2007; Ogden et al., 1994).

Inconsistent results have been reported with respect to the demographic variables of age and gender (Bays, 2001; Noble & Schenk, 2010; Taub et al., 1994), though older age (Castonguay et al., 2014; Di Carlo et al., 1999; Franceschini et al., 2010; Passier et al., 2013; Vibo et al., 2007) and female gender (Franceschini et al., 2010; Gattringer et al., 2014; Irie et al., 2015; Meyer et al., 2010; Passier et al., 2013; Reeves et al., 2008; Vibo et al., 2007) have frequently been linked with the range of less favourable functional outcomes.

After examining the four major domains of short-term stroke outcomes, the interactions between each pair of outcomes are now examined.

Neurological and Neuropsychological Outcomes

Stroke neuropathology and neurological outcomes have been associated with neuropsychological, psychological, and functional outcomes post-stroke and -SAH. With regards to neuropsychological outcomes, global cognitive impairment 3-months post-stroke is associated with acute urinary incontinence (odds ratio 4.8), visual field defect (odds ratio 2.0), left hemispheric lesion (odds ratio 1.6), dysphasia (odds ratio 1.5), and loss of consciousness (odds ratio 1.4; Patel, Coshall, Rudd, & Wolfe, 2002). Other stroke severity markers such as increased stroke volume (infarction or haemorrhage) and regions of impaired cerebral perfusion are also determinants of post-stroke and -SAH cognitive deficits up to 12-months (Egge et al., 2005; Ogden, Levin, & Mee, 1990; Snaphaan & de Leeuw, 2007). In a longitudinal study of 32 SAH survivors, Hunt and Hess grades were significantly associated with IQ, memory, and aphasia test scores while loss of consciousness related to psychomotor and visual memory functioning at 3-, 6-, and 12-months post-SAH (Haug et al., 2007). Complications such as hydrocephalus and fever have been associated with early cognitive

deficits in SAH patients, though the significance of these relationships diminishes during the first year (Ogden, Mee, & Henning, 1993; Springer et al., 2009). Surgical clipping of aneurysmal SAH may relate to poorer global cognitive functioning, memory, and information processing speed up to 12-months post-SAH in comparison to endovascular coiling (Fontanella, Perozzo, Ursone, Garbossa, & Bergui, 2003; Hadjivassiliou et al., 2001; Scott et al., 2010), though not all research supports this association (Haug et al., 2007).

Neurological and Psychological Outcomes

Depression and anxiety post-stroke and -SAH are frequently associated with left hemisphere lesions (Barker-Collo, 2007; Robinson, 2003). However, a number of studies refute these findings (Anderson, Vestergaard, Ingemaan-Nielsen, & Lauritzen, 1995; Brodaty, Withall, & Sachdev, 2007; Carota et al., 2005) with some suggesting that lesion location, specifically anterior location, is more significant (Kim & Choi-Kwon, 2000). Early deterioration in neurological impairment score (NIHSS) is an independent determinant of PSD (Townend et al., 2007), as is impaired upper and lower limb motor function (Kim & Choi-Kwon, 2000). Other neurological impairments associated with PSD include urinary incontinence and visual field defects (Dennis, O'Rourke, Lewis, Sharpe, & Warlow, 2000). Depression is also an independent risk factor for mortality, with acutely depressed patients having 50% higher mortality at 12-months compared to non-depressed patients (Robinson, 2003).

Neurological and Functional Outcomes

With regards to functional outcomes, increased stroke symptoms (NIHSS) is associated with worse disability and handicap at 3-months (Abdul-Rahim et al., 2015), with specific impairments of paralysis, urinary incontinence, dysphagia, speech difficulties, aphasia, and gaze paresis independently predicting severe disability and handicap at this time (Di Carlo et al., 1999; Lawrence et al., 2001). Post-SAH, patients having undergone coiling

as compared to clipping have significantly better disability outcomes (mRS) at 3-months (Mortimer et al., 2014). Urinary incontinence emerged as the single best neurological predictor of disability and handicap up to 12-months (Taub et al., 1994), while increased stroke symptoms and incomplete motor recovery are the strongest neurological predictors of poor HRQoL 12-months post-stroke and -SAH (Franceschini, Porta, Agosti, & Massucci, 2010; Meyer et al., 2010; Passier et al., 2013; Vibo, Kõrv, & Roose, 2007). Though related to other functional outcomes, clipping versus coiling has not been related to HRQoL post-SAH (12-months; Al-Khindi et al., 2010; Meyer et al., 2010; Proust et al., 2009).

Conversely, it is important to note that many stroke and SAH survivors classified as having *good* neurological outcomes have been shown to have considerable neuropsychological (Cao, Ferrari, Patella, Marra, & Rasura, 2007; Mayer et al., 2002), psychological (Alfieri et al., 2008), and functional (Abdul-Rahim et al., 2015; Cao et al., 2007; Ogden et al., 1990) deficits, demonstrating the importance of examining the full spectrum of outcome domains.

Neuropsychological and Psychological Outcomes

Numerous studies have reported a significant association between cognitive impairment and PSD and PSA. Research using multivariate regression analysis demonstrates that cognitive impairment at 1-month can account for 42% of the variance in self-reported depression 12-months post-stroke (Anderson et al., 1995). At 3-months post-stroke, Barker-Collo (2007) found that 51% and 39% of the variance in depression and anxiety, respectively, could be explained by poor cognitive performance, which accounted for the greatest proportion of overall variance explained. Both global and domain specific cognitive impairments have been linked with PSD, with deficits in attention, memory, psychomotor speed, language, and executive functions most commonly cited (Brodaty et al., 2007; Kauhanen et al., 1999; Pohjasvaara et al., 2002; Robinson, 2003). Patients with psychiatric

symptoms are also at greater risk of experiencing cognitive decline rather than recovery during the first 12-months post-stroke (Rasquin et al., 2005). Contradictory evidence exists regarding the impact of successful treatment of psychological difficulties on cognition, but overall, treatment appears to be associated with improved cognition (Robinson, 2003).

Research exploring the relationship between cognitive functioning and psychological outcomes post-SAH is limited and the findings much more variable. Mayer and colleagues (2002) explored the relationship between global cognitive functioning (TICS) and domain specific cognitive functioning (neuropsychological test battery) with concurrent measures of depression and anxiety 3-months post-SAH. They concluded that global cognitive functioning is significantly associated with depression and anxiety, but domain specific cognitive functioning (e.g., memory, information processing speed, language) is not. Similar findings were reported by Ogden and colleagues (1993) at 10-weeks and 12-months post-SAH. Conversely, research suggests that attention, memory, and language deficits are closely related to PSAHD, as are memory impairments and anxiety up to 12-months post-SAH (Alfieri et al., 2008; Berry et al., 1997; Powell et al., 2002). A strong relationship between self-reported cognitive complaints (attention, memory, information processing speed) and depression (r=.59) and anxiety (r=.48; Passier et al., 2010) has also been found. These contradictory findings, based on a small body of research, all use hospital-based samples, necessitating the need for further population-based research.

Neuropsychological and Functional Outcomes

In a review of the literature, Barker-Collo and Feigin (2006) report that neuropsychological functioning (e.g., attention, memory, aphasia, neglect, anosognosia) predicts post-stroke functional outcomes and that neuropsychological rehabilitation may benefit functional outcomes. For example, Patel and colleagues (2002, 2003) assessed global cognitive functioning (MMSE) 3-months post-stroke and disability (BI, FAI) 12-months

post-stroke in a population-based sample. Subjects cognitively impaired at 3-months were more likely to be severely disabled and institutionalised at 12-months.

The impact of cognitive functioning on recovery of functional abilities has recently been highlighted by Cederfeldt and colleagues (2010). In their sample (n=45) of elderly (≥65 years) stroke survivors, subjects with intact cognition (MMSE) acutely demonstrated significant improvement in their ability to perform personal cares (BI) up to 12-months, whereas those with impaired cognition deteriorated in their personal cares from 6- to 12-months post-stroke. Research also suggests that the relationship between cognition and functional outcomes is incremental; as cognitive deficits (MMSE and neuropsychological test battery) increase in severity, so too does functional disability (BI; Cao et al., 2007).

Associations between domain specific cognitive functioning and functional outcomes have also been identified. Acute visual neglect is a key predictor of poor functional recovery (FAI) and lower HRQoL 12-months post-stroke (Franceschini et al., 2010; Jehkonen et al., 2000), while aphasia and deficits in processing and motor speed, attention, and mental flexibility have also been associated with poorer HRQoL post-stroke (Bays, 2001; Hochstenbach, Anderson, van Limbeek, & Mulder, 2001).

Research examining the relationship between these outcomes in SAH survivors is minimal and limited by the frequent use of brief global cognitive measures. Sacri and Kos (2002) looked at global cognitive (MMSE) and functional (Functional Independence Measure; FIM) outcomes at discharge from early rehabilitative treatment following aneurysmal SAH (n=59). A stepwise relationship was observed, whereby 0%, 36%, and 100% of subjects with severe, mild, and no cognitive impairment, respectively, were independent in ADL. During rehabilitation, these authors also noted that severely cognitively impaired subjects made much slower progress in the recovery of their ADL compared to mildly cognitively impaired subjects. At 3- and 12-months post-SAH, cognitive impairment

(TICS) has been associated with worse concurrent disability (BI, LIADLS), handicap (mRS), and HRQoL (Mayer et al., 2002; Springer et al., 2009).

Research that thoroughly assesses cognitive functioning (neuropsychological test battery) in relation to functional outcomes post-SAH gives inconsistent results. Mayer and colleagues (2002) report that visual memory, visuospatial, psychomotor, and reaction time domains are only related to disability (BI, LIADLS) 3-months post-SAH (not handicap [mRS] or HRQoL), while other cognitive domains (verbal memory, language, executive) are not related to any functional outcome. In contrast, Haug and colleagues (2010) conclude that poor cognitive functioning is associated with poor HRQoL (SF-36, GHQ-28) 12-months post severe neurological grade (Hunt and Hess grade V) SAH. These findings likely differ in part due to differences in sample selection, different follow-up periods, and the use of different measures to assess both neuropsychological and functional outcomes.

Psychological and Functional Outcomes

Numerous studies have confirmed an association between psychological and functional outcomes during the first 12-months post-stroke and -SAH, with depressed patients demonstrating significantly higher levels of disability, handicap, institutionalisation, and poorer HRQoL as compared to non-depressed patients (Bays, 2001; Brodaty et al., 2007; Dennis et al., 2000; Farner et al., 2010; Goodwin & Devanand, 2008; Herrmann et al., 1998; Kauhanen et al., 1999; Kreiter et al., 2013; Kutlubaev & Hackett, 2014; Kwok et al., 2006; Passier et al., 2013; Townend et al., 2007). Depressed patients are also less likely to return to their pre-stroke employment status 12-months later (Ogden, Mee, & Henning, 1994), and have poorer social cognition and few or decreased levels of social activities (Anderson et al., 1995; Herrmann et al., 1998). Research also demonstrates that the severity of PSD is positively associated with severity of functional disability (Robinson, 2003; West et al., 2010) and that it may have a more generalised adverse effect on HRQoL than disability and

handicap (Kreiter et al., 2013; Kwok et al., 2006). Furthermore, remission of PSD, whether spontaneous or due to treatment, significanlty improves performance of ADL and HRQoL as compared to those with stable PSD (Chemerinski et al., 2001; Kreiter et al., 2013; Robinson, 2003).

Summary

The stroke outcomes literature demonstrates that neurological, neuropsychological, psychological and functional deficits are common short-term outcomes post-stroke and -SAH. Furthermore, complex relationships exist between these outcome domains whereby each may help and hinder recovery in other domains. However, a comprehensive understanding of short-term stroke and particularly SAH outcomes is restricted as the current body of literature is limited in a number of ways. Few population-based studies have examined short-term neuropsychological and psychological outcomes post-SAH, let alone the interaction between these outcomes, and those that have are limited by the use of brief outcome measures. The natural course of outcomes has been investigated by few, requiring delineation of the natural course of outcomes to be pooled from multiple studies at this stage. Studies that have delineated the natural course typically assess at few time points and are limited by the sample and exclusion criteria. Research employing matched control participants is also limited. Combining these limitations, there is not currently any population-based studies that thoroughly assess the neuropsychological (test battery), psychological and functional outcomes, and the relationship between these outcomes, throughout the first 12-months post-SAH compared to control participants matched on age, gender, and ethnicity.

Purpose

Following from the above, the purpose of this study was to examine the short-term neuropsychological, psychological and functional outcomes of a population-based sample of SAH survivors at 28-days, 6- and 12-months post-SAH compared to healthy matched (age, gender, ethnicity) control participants. Four aims were examined in the current study. First, SAH survivors' performance on measures of neuropsychological, psychological and functional outcomes at each time point were examined and compared to matched control participants at 6- and 12-months. Second, the natural course of recovery during the first 12-months post-SAH on measures of neuropsychological, psychological and functional outcomes was delineated. Third, the extent to which demographic factors, injury characteristics, and 28-day and 6-month outcome measures related to SAH survivors' HRQoL at 12-months was explored. Finally, the relationships between 12-month neuropsychological, psychological and functional outcomes in SAH survivors were explored.

CHAPTER II:

METHODOLOGY

Context of Study

This is a prospective population-based study of short-term (≤12-months) neuropsychological, psychological and functional outcomes of SAH survivors, with matched controlled comparison. This study sourced SAH participants and used existing baseline, 28-day, 6- and 12-month data from a stroke incidence and outcomes study (Auckland Regional Community Outcome Stroke [ARCOS] Study IV) carried out in Auckland, NZ. The ARCOS IV used a prospective population-based register with multiple overlapping sources to ascertain all new and recurrent (occurring ≥28-days post index stroke) stroke cases that occurred among adults (≥16 years) in the "usually resident" population of Auckland during a 12-month period from 1 March 2011 to 29 February 2012. While methods for ARCOS IV are described thoroughly elsewhere (Krishnamurthi et al., 2014), those relevant to this study are described below.

Participants

SAH Sample

Potential SAH participants included all hospitalised and non-hospitalised adult patients of SAH in the Auckland region during the 12-month period specified above. Of the 86 SAH cases that occurred during this period, 14 (16%) died prior to notification. Of the remaining SAH cases (n=72), 31 met inclusion criteria (i.e., ≥16 years, "usually resident" in the Auckland region; provided written informed consent). Despite repeated and various attempts to make contact (e.g., phone calls, letter, contact with general practitioner), one participant was immediately lost to follow-up. Thus, the SAH sample consisted of 30

participants; 42% of the SAH survivors alive during the study period. Of this sample, 30% (n=9) had experienced a previous stroke though no participants experienced recurrent stroke during the study period. Most SAH participants had undergone surgical clipping (57%, n=17) as compared to endovascular coiling (27%, n=8); neither procedure was recorded for several participants (17%, n=5).

Sensitivity analyses were conducted to contrast the SAH sample with those SAH survivors who chose not to participate in the study (n=42) to determine whether the demographics and stroke symptoms (Glasgow Coma Scale, GCS; Teasdale & Jennett, 1974) of the present sample differed from the wider group. Independent t-tests were used for continuous variables (i.e., age, GCS) and chi-square analyses were used for discrete variables (i.e., gender, ethnicity). The analyses indicated that while the groups did not differ significantly in terms of age, gender, and stroke symptoms ($p>.05$), they did differ significantly regarding ethnicity ($\chi 2(1)=6.58$, $p=.01$), with greater proportions of SAH survivors self-identifying as non-European choosing not to participate in the present study.

Table 3 summarises the demographic characteristics of the SAH and control samples. As can be seen in Table 3, the mean age of SAH group participants was just over 50 years, with age at time of SAH ranging from 21 to 82 years. Over two thirds of the sample was female and most self-identified as NZ European ethnicity. For the purpose of data analysis, participants were grouped together as European (n=22) or Other (n=8) due to the small sample size. In accordance with the NZ Census (Census, 2013), the European group included those self-identifying as NZ European and English, the Other group consisting of all other ethnicities including NZ European/Maori.

Control Sample

Control participants were recruited from the Auckland region and matched to the SAH sample on age (within 2 years), gender and ethnicity, as research shows these variables

Table 3

Demographic characteristics of SAH and control groups

Variable	SAH group (n=30)	Control group (n=29)	Differences between groups
Age mean years(SD)	52.40(13.10)	51.83(13.04)	F(1,57)=.03, p=.87
Gender			
Female(%)	22(73.3%)	22(75.9%)	$\chi 2(1)=.05$, p=.82
Male(%)	8(26.7%)	7(24.1%)	
Ethnicity			
NZ European(%)	20(66.7%)	20(69.0%)	$\chi 2(1)=.04$, p=.85
NZ European/Maori(%)	1(3.3%)	1(3.4%)	
Maori(%)	3(10.0%)	3(10.3%)	
Samoan(%)	1(3.3%)	1(3.4%)	
Cook Island Maori(%)	1(3.3%)	1(3.4%)	
Tongan(%)	1(3.3%)	- -	
Niuean(%)	1(3.3%)	1(3.4%)	
English(%)	2(6.7%)	2(6.9%)	
Marital status			
Married, civil union, defacto(%)	26(86.7%)	23(79.3%)	$\chi 2(2)=.83$, p=.66
Separated, divorced, widowed(%)	2(6.7%)	4(13.8%)	
Single (never married)(%)	2(6.7%)	2(6.9%)	
Living arrangements			
Living with partner/family(%)	28(93.3%)	25(86.2%)	$\chi 2(2)=1.35$, p=.51
Living with others(%)	- -	1(3.4%)	
Living alone(%)	2(6.7%)	3(10.3%)	
Dwelling place			
Own home(%)	20(66.7%)	21(72.4%)	$\chi 2(2)=1.07$, p=.59
Rented(%)	7(23.3%)	8(27.6%)	
Living with family/friends(%)	1(3.3%)	- -	
Missing(%)	2(6.7%)	- -	
Complete secondary school?			
Yes(%)	20(66.7%)	18(62.1%)	$\chi 2(1)=.31$, p=.58
No(%)	9(30.0%)	11(37.9%)	
Missing(%)	1(3.3%)	- -	
Highest tertiary qualification			
Degree(%)	4(13.3%)	8(27.6%)	$\chi 2(2)=2.03$, p=.36
Diploma or Certificate(%)	7(23.3%)	7(24.1%)	
Trade or Technical(%)	1(3.3%)	- -	
Socioeconomic status			
Professional(%)	- -	6(20.7%)	$\chi 2(6)=14.22$, **p=.03**
Managerial/Technical(%)	12(40.0%)	8(27.6%)	
Skilled non-manual(%)	3(10.0%)	9(31.0%)	
Skilled manual(%)	2(6.7%)	1(3.4%)	
Partly skilled(%)	2(6.7%)	1(3.4%)	
Unskilled(%)	1(3.3%)	1(3.4%)	
Unemployed/Retired(%)	10(33.3%)	3(10.3%)	

Note: Bold typeface highlights significant difference.

impact the outcomes of interest (Lezak et al., 2004; Strauss, Sherman & Spreen, 2006). Although consideration was given to matching controls on education as well, matching on four variables would have meant significant extension to recruitment time lines. As education is known to impact neuropsychological outcomes (Strauss et al., 2006), information regarding completion of secondary school and tertiary education was collected. Potential control participants were excluded if they were extremely unwell; had a neurological condition or cognitive disability (i.e., stroke, traumatic brain injury, other neurological or current mental health diagnosis); or a history of substance abuse as these conditions would potentially affect their performance. Unfortunately one control participant who matched on age, gender and ethnicity could not be found, thus the final control group consisted of 29 participants.

As can be seen from Table 3, the control sample appeared similarly matched to the SAH sample across each demographic characteristic. To check for equivalence, between-group comparisons were made utilising analysis of variance (ANOVA) for the continuous variable (i.e., age, independent variable=group [SAH and control], dependent variable=age) and Pearson's chi-squared tests for the discrete variables (e.g., gender, ethnicity). The analyses indicated that the groups did not differ significantly on any of the demographic characteristics, importantly on age, gender, ethnicity and education; with the exception of socioeconomic status, whereby the highest proportions of SAH participants were managerial/technical and unemployed/retired compared to the highest proportions of control participants being professional, managerial/technical and skilled non-manual.

Measures

A list of the measures used and time points of assessment of the SAH group is outlined in Table 4. As seen in Table 4, baseline assessments, conducted within 2-weeks of

SAH, included demographic information (e.g., age, gender, ethnicity, relationship status, education), neurological outcomes (GCS, NIHSS), and an assessment of disability (BI). At 28-days, with follow-up assessments at 6- and 12-months, SAH participants were assessed on neurological outcomes (NIHSS), and completed all neuropsychological, psychological and functional measures listed. To allow for direct between-group comparison, control participants were assessed once using the same battery of tests, excluding neurological measures, as those completed by SAH participants at follow-up assessments. Control participants' demographic information was also obtained at this time. The section below describes and reviews each outcome measure used. For reference, the demographic information, neuropsychological, psychological, and functional measures used are attached in Appendix A.

Table 4

Measures used and time points of administration for SAH sample

	Time point			
Measures	Baseline	28-day	6-month	12-month
Demographic Information (e.g., age, gender)	X			
Neurological				
Glasgow Coma Scale	X			
National Institute of Health Stroke Scale	X	X	X	X
Neuropsychological				
Montreal Cognitive Assessment		X	X	X
CNS Vital Signs		X	X	X
Behavioural Dyscontrol Scale		X	X	X
Psychological				
Hospital Anxiety and Depression Scale		X	X	X
General Health Questionnaire 28		X	X	X
Functional				
modified Rankin Scale		X	X	X
Barthel Index	X	X	X	X
Short Form 36		X	X	X

Neurological Measures

To assess the neurological impact of SAH on the SAH sample, two commonly used scales, the GCS and the NIHSS were used.

Glasgow Coma Scale (GCS; Teasdale & Jennett, 1974). The GCS is a neurological scale used by medical professionals to objectively assess the depth and duration of impaired consciousness and coma. The GCS operates on a scale from 3 to 15 where 3 indicates deep coma or death and 15 indicates a fully conscious person. Three aspects of behaviour are measured and scored and sum together to comprise the total score: eye opening, verbal performance, and motor responsiveness. Scores for these categories range from 1 (does not open eyes) to 4 (opens eyes spontaneously), 1 (makes no sounds) to 5 (oriented, converses normally), and 1 (makes no movements) to 6 (obeys commands), respectively. A score of <8 is thought to represent the recognised definition of coma (Jennett & Teasdale, 1977).

The GCS is the most universally used scale for grading level of consciousness and is widely used post-stroke (Starke et al., 2009; Sternbach, 2000; Weir, Bradford, & Lees, 2003). The GCS has high test-retest reliability (r=.85; Prasad, 1996) and high inter-rater reliability among experienced users (96% agreement; Prasad, 1996; Rowley, & Fielding, 1991). In SAH patients, its inter-rater reliability is superior to that of other neurological scales (e.g., Hunt and Hess grading system; Lindsay, Teasdale, & Knill-Jones, 1983). The GCS correlates with measures that reflect the extent of brain damage, which depth of coma is considered to reflect, thus demonstrating construct validity (Prasad, 1996). The prognostic value of the GCS has also been confirmed as it relates to mortality and functional outcomes up to 12-months post-stroke and -SAH (Broderick, Brott, Duldner, Tomsick, & Huster, 1993; Prasad, 1996; Starke et al., 2009; Weir et al., 2003).

National Institute of Health Stroke Scale (NIHSS; Brott et al., 1989). The NIHSS is used by healthcare providers to objectively quantify stroke-related neurological deficit. Originally designed for use in acute stroke clinical trials, it is now widely used as a clinical assessment tool to evaluate and document neurological status in stroke patients, serving as a measure of stroke severity. The NIHSS consists of 15 items which address level of consciousness, extra-ocular movements, visual fields, facial muscle function, extremity strength, coordination, sensory function, language, speech, and hemiattention. Each item is scored between 0 and 2 to 4. A score of 0 indicates normal functioning, while a higher score indicates some level of impairment. Item scores are summed to calculate a total NIHSS score, ranging from a minimum score of 0 (no stroke symptoms) to a maximum score of 42 (severe stroke symptoms). Commonly used cut-off scores to categorize mild, moderate, and severe stroke are NIHSS scores ≤5, from 6 to 13, and ≥14, respectively (Cederfeldt et al., 2010; Schlegel et al., 2003).

The NIHSS is one of the major stroke impairment scales used today (Appelros & Terént, 2004). Its content validity has been demonstrated by exploratory factor analysis, which reveals two underlying factors representing left and right brain function, with each factor further dividing into two sub-factors representing cortical and motor function for each hemisphere (Lyden et al., 1999). The underlying structure of the NIHSS remains consistent over time, demonstrating its validity when used serially (Lyden et al., 1999). Inter-rater reliability for most items is high to moderate (κ=.41 to .79; Goldstein, Bertels, & Davis, 1989), with its reliability established for neurologists and other physicians, and for experienced and inexperienced raters (Goldstein & Samsa, 1997; Hinkle, 2014; Lyden et al., 1999). Baseline NIHSS scores repeatedly demonstrate predictive validity with regards to functional outcomes up to 12-months post-stroke, with good sensitivity (r=.71), specificity

(r=.90), and overall accuracy (r=.83; Adams et al., 1999; Appelros & Terént, 2004; Lyden et al., 1999; Muir, Weir, Murray, Povey, & Lees, 1996).

Neuropsychological Measures

The literature demonstrates that SAH survivors experience a range of neuropsychological deficits within 12-months post-SAH, particularly in relation to attention, memory, executive functioning, and information processing speed. The test battery described below was selected to assess a range of neuropsychological outcomes inclusive of the domains most commonly impaired.

CNS Vital Signs (CNS-VS; Gualtieri & Johnson, 2005). The CNS-VS is a brief (30 minute) self-administered computerised neuropsychological test battery comprised of nine common neuropsychological tests: verbal memory, visual memory, finger tapping, symbol digit coding, Stroop Test, shifting attention, continuous performance, perception of emotions, and four part continuous performance. A description of each neuropsychological test is outlined in Table 5 (CNS Vital Signs, 2014).

Each test generates a range of raw scores which are used to generate 12 domain scores: Composite Memory, Verbal Memory, Visual Memory, Processing Speed, Executive Function, Psychomotor Speed, Reaction Time, Complex Attention, Cognitive Flexibility, Working Memory, Sustained Attention, and Social Acuity. Based on five domain scores, a summary score, the Neurocognition Index (NCI), represents a global score of neuropsychological cognition. An outline of each domain and its contributing neuropsychological tests are in Table 6 (CNS Vital Signs, 2014). Domain scores are standardized using age-appropriate normative data with a mean of 100 and standard deviation (SD) of 15 and assigned qualitative categories of very low (<70), low (70 to 79), low average (80 to 89), average (90 to 109), or above average (>109).

Table 5

Brief description of CNS Vital Signs neuropsychological tests (CNS Vital Signs, 2014)

Test Name	Description
Verbal memory	Participant presented with 15 words, one by one, to remember and recognise in a field of 15 distractors. Repeated at the end of the test battery
Visual memory	Participant presented with 15 geometric figures, one by one, to remember and recognise in a field of 15 distractors. Repeated at the end of the test battery
Finger tapping	Three 10 second rounds of tapping with index finger with each hand
Symbol digit coding	Participant types the appropriate number below the paired symbol for 120 seconds
Stroop Test	Three part test. Participant responds when a word is displayed; when the word matches the colour; and when the word does not match the colour
Shifting attention	Participant adjusts response to randomly changing rules of 'match colour' or 'match shape'
Continuous performance	Participant responds to 'B' in a series of letters presented for 5 minutes
Perception of emotions	Participant identifies matches between photographs depicting facial expressions and descriptions of 'happy', 'calm', 'sad', and 'angry'
Four part continuous performance	Four part test. Participant responds when any shape is displayed; when the identified shape and colour is displayed; when the shape and colour immediately preceding was the same; and when the shape and colour two displays prior was the same

The CNS-VS was chosen over a conventional battery of tests as it ensures consistency in administration and scoring; times responses with millisecond accuracy; and generates numerous alternative forms suitable for repeat testing (Gualtieri & Johnson, 2005; Gualtieri & Johnson, 2006). Its use also minimized the potential impact assessment for this study could have on clinically driven assessment of the same constructs for SAH survivors. A minimum number of response keys are used, so keyboard skills have minimal influence on performance (Gualtieri & Johnson, 2006). The CNS-VS has been translated into 52 languages and found to be as reliable and valid as the conventional neuropsychological tests upon which it is based (Gualtieri & Johnson, 2005; Gualtieri & Johnson, 2006). Test-retest

Table 6

Brief description of CNS Vital Signs domains with contributing neuropsychological tests in parenthesis (CNS Vital Signs, 2014)

Domain (test contribution)	Description
Neurocognition Index (CM, PmS, RT, CA, CF)	General assessment of the overall neuropsychological status of the participant
Composite Memory (verbal and visual memory)	Overall ability to recognize, remember, and retrieve words and figures
Verbal Memory (verbal memory)	Ability to recognize, remember, and retrieve words
Visual Memory (visual memory)	Ability to recognize, remember, and retrieve figures
Processing Speed (symbol digit coding)	Ability to recognize and process information, including perceiving, attending/responding to incoming information, motor speed, fine motor coordination, visual-perceptual ability
Executive Function (shifting attention)	Ability to recognize rules, categories, and manage/navigate rapid decision-making
Psychomotor Speed (finger tapping, symbol digit coding)	Ability to perceive, attend, and respond to visual-perceptual information, and motor speed and fine motor coordination
Reaction Time (Stroop Test)	Ability to quickly react (milliseconds) to simple and increasingly complex set of directions
Complex Attention (Stroop Test, shifting attention, continuous performance)	Ability to track and respond to information over lengthy periods of time and perform mental tasks requiring vigilance quickly and accurately
Cognitive Flexibility (shifting attention, Stroop Test)	Ability to adapt to rapidly changing and increasingly complex set of directions and to manipulate information
Working Memory (four part continuous performance)	Ability to perceive and attend to symbols using short-term memory
Sustained Attention (four part continuous performance)	Ability to direct and focus cognitive activity on specific stimuli
Social Acuity (perception of emotions)	Ability to perceive, process, and respond to emotional cues

CA=Complex Attention; CF=Cognitive Flexibility; CM=Composite Memory; PmS=Psychomotor Speed; RT=Reaction Time
Note: Neurocognition Index is generated from domain scores, not test scores.

reliability for individual tests range from r=.45 to .87 (with the exception of one aspect of the Stroop Test r=.31), and from r=.65 to .87 for domain scores (Gualtieri & Johnson, 2005; Gualtieri & Johnson, 2006). Concurrent validity has been established by comparison of the CNS-VS to conventional and other computerised neurocognitive tests, where moderate

correlations were found, which is as well correlated as conventional tests are with each other (Gualtieri & Johnson, 2005; Gualtieri & Johnson, 2006). The CNS-VS has been used in various clinical populations (e.g., mild cognitive impairment, dementia, traumatic brain injury, depression) and found to be sensitive to cognitive impairment caused by different conditions, demonstrating its discriminant validity (Gualtieri & Johnson, 2006; Gualtieri & Johnson, 2008). The computerised test battery has good sensitivity (90% for domain scores) and specificity (64% to 82% across domain scores) with regards to mild cognitive impairment (Gualtieri & Johnson, 2005).

Behavioural Dyscontrol Scale (BDS; Grigsby, Kaye, & Robbins, 1992). The BDS is a measure of an individual's capacity for behavioural self-control, defined as one's ability to initiate purposeful, goal-directed behaviour and inhibit inappropriate activity, as an indicator of executive functioning (Grigsby & Kaye, 1996). Though the CNS-VS assesses executive functioning, executive functioning refers to a broad range of abilities and is often impaired post-SAH, hence the inclusion of another measure. The BDS consists of 9 items, taking approximately 10 minutes to administer. Seven of the 9 items assess control of motor functioning (e.g., squeeze examiners hand if examiner says "red" and do nothing if they say "green"). One item assesses working memory and the capacity for cognitive flexibility and shifting of attention (i.e., count, alternating it with the alphabet). The final item assesses the individual's level of insight into their performance. The BDS has three underlying factors: the Motor Programming Factor (MPF), which taps ability to volitionally generate and sustain motor responses (4 items); the Environmental Independence Factor (EIF), which taps impulsivity (2 items); and the Fluid Intelligence Factor (FIF), which taps ability to use feedback (3 items; Ecklund-Johnson, Miller, & Sweet, 2004; Grigsby & Kaye, 1996; Suchy, Blint, & Osmon, 1997). Each item is scored on a 4 point scale with scores assigned to represent performance that is high (3 points), adequate (2 points), mildly to moderately

deficient (1 point), and very impaired (0 points). Factor scores are sums of the relevant items and the total score ranges from 0 to 27. Higher scores indicate better behavioural control/executive functioning.

The BDS has been used in a range of populations, including clinic- and community-based samples (Grigsby & Kaye, 1996). It has high internal consistency (Cronbach's α=.87) and test-retest reliability ($r \geq .86$; Grigsby & Kaye, 1996). Inter-rater reliability is also high for individual items (Spearman's rho ranged .84 to .98) and total BDS score (Spearman's rho=.98; Grigsby & Kaye, 1996). The construct validity of the BDS is demonstrated by its relationships with other measures of executive functioning such as the Stroop Test, Controlled Oral Word Association, Card Sorting Test, and Trail Making Test (Suchy, Blint, & Osmon, 1997; Suchy, Leahy, Sweet, & Lam, 2003), and its ability to differentiate patients with frontal as compared to nonfrontal brain injuries (Leahy, Suchy, Sweet, & Lam, 2003). The BDS also correlates with measures of non-executive functions (e.g., the MMSE), though it nevertheless is dissociable from them (Grigsby & Kaye, 1996; Suchy, Blint, & Osmon, 1997). The BDS also demonstrates ecological validity in its relationship to everyday functional competence (Grigsby, Kaye, Kowalsky, & Kramer, 2002; Suchy et al., 1997).

Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005). The MoCA is a rapid (10 minute) screening tool used to assist health professionals screen for cognitive impairment. It briefly assesses a range of cognitive domains (i.e., attention, concentration, working and short-term memory, executive functions, language, visuo-constructional skills, orientation) and is made up of seven subtests: visuo-executive, naming, attention, language, abstraction, delayed recall, and orientation. Examples of items include two learning trials of five nouns followed by 5 minute delayed recall (delayed recall subtest assessing short-term memory), naming low-familiarity animals (language subtest), and clock-drawing (visuo-executive subtest assessing visuo-constructional skills). The number of items and associated

points for each subtest varies. The total possible score is 30, with scores ≥26 considered normal and scores below this indicative of cognitive impairment, requiring further assessment.

Though the literature demonstrates that brief cognitive screening measures are inadequate for assessing neuropsychological outcomes, the MoCA was included in this test battery as patients too unwell to complete the full neuropsychological test battery were often able to complete this brief cognitive screen. It also allowed for comparison to the literature, which commonly employs such tools. The MoCA was selected over other brief cognitive screening measures (e.g., the MMSE) given its superiority in detecting post-stroke and -SAH cognitive impairment (Chiti & Pantoni, 2014; Dong et al., 2010; Pasi et al., 2015; Schweizer, Al-Khindi, & Macdonald, 2012; Webb et al., 2014; Wong, Lam, Ngai, Wong, Mok, & Poon, 2012). The MoCA has high test-retest reliability (r=.92) and good internal consistency (Cronbach α=.83; Nasreddine et al., 2005). Content validity has been established via the close correlation between MoCA scores and those of other cognitive screening tools (i.e., the MMSE, r=.87; Nasreddine et al., 2005). The MoCA also demonstrates sensitivity (90%) and specificity (87%) for mild cognitive impairment determined by clinical diagnosis (Nasreddine et al., 2005). The MoCA has been translated into multiple languages and has been validated in a number of populations (Fujiwara et al., 2010; Lee et al., 2008; Rahman & Gaafary, 2009; Thissen, van Bergen, de Jonghe, Kessels, & Dautzenberg, 2010).

Psychological Measures

Depression and anxiety are considered the most common psychiatric conditions post-stroke and -SAH. The two measures used here to assess these outcomes are those most commonly employed in the stroke literature.

Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983). The HADS is a 14 item self-administered scale that provides a brief state measure of current

levels of anxiety and depression. Seven items contribute to the anxiety (e.g., "I feel tense or wound up") and 7 to the depression (e.g., "I still enjoy the things I used to) subscales. The HADS items do not include somatic symptomology (e.g., dizziness, headaches) as these could be endorsed due to a physical rather than psychological state (Zigmond & Snaith, 1983). Each item has four possible responses, for example, ranging across "Not at all", "From time to time, occasionally", "A lot of the time", and "Most of the time". Individuals are instructed to respond to each item in order to reflect how they have felt during the past week. Items are scored from 0 to 3, with total scores for each subscale ranging from 0 to 21. Subscale scores are classified as indicating "normal" (0 to 7), "mild" (8 to 10), "moderate" (11 to 14), and "severe" (15 to 21) levels of anxiety or depression. The overall HADS score (out of 42) may give a general indication of emotional distress (Johnston, Pollard, & Hennessey, 2000). The scale takes about 5 minutes to complete.

The validity of the HADS has repeatedly been demonstrated in both general and clinic populations, including acute and chronic stroke patients (Aben, Verhey, Lousberg, Lodder, & Honig, 2002; Bjelland, Dahl, Haug, & Neckelmann, 2002; Johnston et al., 2000; Kang et al., 2013). Average internal consistency ratings are high (Cronbach α=.83 and .82 for the anxiety and depression subscales, respectively), with Cronbach α of up to .93 and .90, respectively, in some populations (Bjelland et al., 2002; Moorey et al., 1991). Concurrent validity has been demonstrated by comparison with psychiatric evaluation, whereby HADS anxiety and depression subscale scores correlate significantly with their respective diagnoses (r=.54 and .79, respectively), but not with the contrary disorder (r=.08 and .19, respectively; Zigmond & Snaith, 1983). Sensitivity and specificity for both HADS subscales are approximately 80% (Bjelland et al., 2002). Correlations between the HADS and other commonly used questionnaires of anxiety and/or depression range from r=.49 to .83, demonstrating the construct validity of the measure (Bjelland et al., 2002). The HADS is also sensitive to

changes in anxiety and depression and can be repeated at intervals to gauge such changes (Zigmond & Snaith, 1983). The psychometric properties of the HADS are maintained when administered via telephone (Gallagher, McKinley, & Dracup, 2003).

General Health Questionnaire 28 (GHQ-28; Goldberg, 1978). The GHQ-28 is a 28 item self-administered scale used to assess overall psychological health and wellness. It focuses on two major classes of phenomena; inability to continue to carry out one's normal functions, and the appearance of symptoms of a distressing nature (Goldberg & Hillier, 1979). It is designed to detect a range of psychological disorders across four specific subscales: somatic symptoms (e.g., "Have you recently been getting any pains in your head?"), anxiety and insomnia (e.g., "Have you recently lost much sleep over worry?"), social dysfunction (e.g., "Have you recently been managing to keep yourself busy and occupied), and severe depression (e.g., "Have you recently been thinking of yourself as a worthless person?"). However, the subscales are not independent of each other, thus the total score which indicates general psychological disorder has better utility (Goldberg & Hillier, 1979). Each item has four possible responses, typically ranging from "Not at all", to "No more than usual", "Rather more than usual", and "Much more than usual", scoring from 0 to 3, respectively. Participants respond in regards to the extent their psychological health and wellness has differed, in general, from their usual state over the past few weeks, thereby assessing change in characteristics not lifelong personality traits. The total score ranges from 0 to 84 with higher scores indicating poorer psychological health and wellbeing. A cut-off score of 24 has been determined as the best threshold for psychological disorder (Goldberg et al., 1997; Noorbala, Bagheri Yazdi, & Mohammad, 2009).

The GHQ-28 has been translated into 38 different languages for use in diverse cultural groups, which gives testament to the reliability and validity of the measure (Jackson, 2007). Its internal consistency is high (α=.92), as is its Spearman-Brown split-half reliability

(r=.84; Kiliç et al., 1997). The GHQ-28 correlates with self-rated overall health (r=.43) and general practitioner ratings of psychological illness (r=.30; Kiliç et al., 1997). It is also a valid measure in relation to concurrent psychiatric assessment, with a sensitivity of 87% and specificity of 77% (Lykouras et al., 1996). The GHQ-28 has been used in NZ populations to assess psychological health and wellbeing post-stroke (Hackett & Anderson, 2006; Hackett et al., 2010).

Functional Measures

Functional outcomes include disability, handicap, and HRQoL. Three measures commonly used to assess functional outcomes in stroke research were selected for use in this study.

Modified Rankin Scale (mRS; Bamford, Sandercock, Warlow, & Slattery, 1989). The mRS is a clinician-reported outcome classification scale that is widely used to evaluate disability post-stroke (Banks & Marotta, 2007). 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 enabling finer discrimination of mild disability, ranging from grade 0 (no symptoms at all) to grade 5 (severe disability), with grade 6 denoting "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 (de Haan, Limburg, Bossuyt, van der Meulen & Aaronson, 1995; Wolfe, Taub, Woodrow & Burney, 1991). Based on studies with stroke survivors, good outcome is defined as a mRS grade of ≤2, and poor outcome as grade ≥3 (Sulter, Steen & De Keyser, 1999).

The mRS has strong test-retest reliability (weighted k=.94 to .99) and moderate inter-rater reliability (weighted κ=.69 to .75) for both experienced and inexperienced raters (Banks

& Marotta, 2007; Wilson et al., 2005; Wolfe, Taub, Woodrow, & Burney, 1991; Zhao, Collier, Quah, Purvis, & Bernhardt, 2010). Some research suggests structured interviews may increase inter-rater reliability, though most finds no significant difference in reliability between structured and standard mRS administration (Quinn, Dawson, Walters, & Lees, 2009; Wilson et al., 2005). Numerous studies demonstrate the construct validity of the mRS by its relationships to physiological indicators such as stroke type, location, lesion size, perfusion and neurological impairment (Banks & Marotta, 2007; Lai & Duncan, 1999; Nedeltchev et al., 2005). High construct validity has also been reported when comparing the mRS to other measures of disability and impairment such as the BI, motor component of the FIM, LIADLS, and NIHSS (Banks & Marotta, 2007; Kwon, Hartzema, Duncan, & Lai, 2004; Lai & Duncan, 1999; New & Buchbinder, 2006; Tilley et al., 1996).

Barthel Index (BI; Granger, Dewis, Peters, Sherwood, & Barrett, 1979). The BI is a measure of disability originally developed by Mahoney and Barthel (1965) and later modified by Granger and colleagues (1979). The BI is used to assess patient ability to perform ten ADL associated with personal care (i.e., feeding, bathing, grooming, dressing, bowel and bladder care, toilet use) and mobility (i.e., mobility on level surfaces, chair/bed transfers, stair climbing). The BI can be self-administered or completed by a clinician, taking approximately 5 to 10 minutes. With 5 point increments used for BI scoring, the maximum score of 100 indicates that the patient is fully independent. The lowest score is 0 indicating a bed ridden state. The definition of a good outcome on the BI varies considerably, with cut-off scores of ≥50 to ≥95 employed (Sulter, Steen & De Keyser, 1999). Increasingly research suggests that the BI cut-off score be determined in relation to the mRS cut-off score used (e.g., if good outcome on mRS ≤2, BI cut-off ≥90 is appropriate; Uyttenboogaart, Stewart, Vroomen, De Keyser, & Luijckx, 2005) and was employed in the current study.

The BI is considered a sensitive/responsive measure, able to detect minimal yet clinically significant changes in physical functioning (Dromerick, Edwards, & Diringer, 2003; Hsueh, Lin, Jeng, & Hsieh, 2002). Its test-retest and inter-rater reliability is high (weighted κ=.98 and $\geq$.88, respectively; Wolfe, Taub, Woodrow, & Burney, 1991). Comparison of the BI with other measures of disability such as the motor component of the FIM demonstrates high construct validity ($r \geq .92$; Hsueh, Lin, Jeng, & Hsieh, 2002). Acute BI scores are strongly related to post-stroke recovery, demonstrating its validity as a prognostic tool (Huybrechts & Caro, 2007).

Short Form 36 (SF-36; Ware & Sherbourne, 1992). The SF-36 is the most widely used generic measure of HRQoL (de Haan, 2002). The survey is suitable for individuals over 13 years and can be self-administered or completed by face-to-face or telephone interview, taking 5 to 10 minutes (Anderson, Laubscher, & Burns, 1996; Watson, Firman, Baade, & Ring, 1996). It comprises 36 multiple choice items which assess eight areas of HRQoL: physical functioning (PH); role limitations due to physical health problems (RP); social functioning (SF); bodily pain (BP); general mental health (MH); role limitations due to emotional health (RE); vitality, energy and fatigue (VT); and general health perceptions (GH). The number of items contributing to each scale varies. Most items are related to the person's functioning in the past month, and there are a variety of response formats ranging from yes/no, through three, four, five, and six category responses. For example, the six category response item "How much of the time during the past 4 weeks have you felt calm and peaceful?" has choices from 1=None of the time, to 6=All of the time.

The SF-36 was scored using RAND guidelines and involves a two-step process (Hays, Sherbourne, & Mazel, 1993). Items are assigned scores ranging from 0 to 100, where higher scores indicate better HRQoL. These are used to calculate a mean score for each of the eight scales. Dimensions such as PH, RP, BP, and RE measure health status as the

absence of disability (e.g., a score of 100 on BP scale=no pain related limitations). Other scales such as MH, VT, and GH measure both positive and negative states of health (e.g., 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 (MCS) score, derived from the SF, VT, RE and MH scales, and the Physical Component Summary (PCS) score, derived from the PH, RP, BP and GH scales. The summary scores have been standardized to have a mean of 50 and SD of 10. As employed in previous research (Katati et al., 2007), scale and summary scores more than 1 SD below the mean are considered to indicate poor HRQoL. New Zealand normative data has been developed for each scale (Scott, Tobias, Sarfati, & Haslett, 1999) and was used in this study.

The SF-36 has been tested for validity and reliability across general and patient populations including stroke (de Haan, 2002; Jenkinson, Wright, & Coulter, 1994). Importantly, it has been found to be a valid and reliable measure of HRQoL for NZ European, Maori, and Pacific ethnic groups (Scott, Sarfati, Tobias, & Haslett, 2000; Scott et al., 1999). The internal consistency of the SF-36 is high for each of the eight scales (Cronbach's $\alpha \geq .85$), though lower for social functioning (Cronbach's $\alpha = .73$) which partly reflects the low number of items (2 items) in that dimension (Brazier et al., 1992). Test-retest reliability is also high, with mean differences in scores for each scale less than 1 point on the 100 point scale (Brazier et al., 1992). The construct validity of the SF-36 has been demonstrated by the relationships the physical and mental health domains have with the BI and the GHQ-28, respectively (Anderson, Laubscher, & Burns, 1996). The SF-36 is considered a valid prognostic tool when considering the outcomes of SAH patients (Scharbrodt, Stein, Schreiber, Böker, & Oertel, 2009).

Procedure

Ethical approval for this study was obtained from the Northern X Regional Ethics Committee (SAH group) and the University of Auckland Human Participants Ethics Committee (control group). The procedure for the SAH group and that for the control group are described below.

SAH Group

The SAH group consisted of participants of ARCOS IV (i.e., SAH between 1 March 2011 and 29 February 2012, ≥16 years, "usually resident" in the Auckland region, provided written informed consent). Potential participants were ascertained using a prospective population-based register with multiple overlapping sources (i.e., daily, weekly, monthly and quarterly searches of hospital and community records). As soon as possible after notification/identification, potential participants were given a participant information sheet (PIS; Appendix B) detailing information about ARCOS IV and their role in the study. The opportunity to ask questions was given, along with notification that they may withdraw from the study at any time and that this would not affect their future medical care in any way. Written informed consent (Appendix B) was obtained from those who agreed to participate, or their representative, as per regulatory and legal requirements.

Following provision of written informed consent, trained research nurses completed baseline assessments with the participant, or their representative if the participant was too unwell. Neuropsychological, psychological and functional outcome measures were administered by trained community researchers with the participant, or their representative, in their usual place of residence at 28-day, 6- and 12-month follow-up assessments. As seen in Table 7, varied methods of data collection were employed to reduce patient burden and fatigue. At each assessment, the telephone assessment, which took approximately 15 to 20 minutes, was completed prior to the face-to-face assessment, which took approximately 90

Table 7

Data collection methods for SAH and control groups

Measures	Telephone	Face-to-face
Demographic Information	X (C)	X (S)
Neuropsychological		
Montreal Cognitive Assessment		X (C + S)
CNS Vital Signs		X (C + S)
Behavioural Dyscontrol Scale		X (C + S)
Psychological		
Hospital Anxiety and Depression Scale	X (C + S)	
General Health Questionnaire 28		X (C + S)
Functional		
modified Rankin Scale		X (C + S)
Barthel Index		X (C + S)
Short Form 36	X (C + S)	

C=Control group; S=SAH group.

minutes. Prior to beginning assessment, time was taken by the researcher to establish rapport and reduce anxiety, which is important given that increased anxiety has been shown to negatively impact test performance (Lezak et al., 2004). As telephone and face-to-face assessments were carried out with the participant in their usual place of residence, the researcher also took steps to ensure optimal testing conditions (i.e., adequate lighting, reducing distractions such as noise, presence of other people in the room, turning television and cell phones off) prior to assessment. Participants were reminded to use any aids they may need (e.g., glasses). They were monitored for fatigue and offered breaks during assessment and/or multiple sessions, which were provided as the participant required. If a participant's responses on either of the psychological measures indicated depression, anxiety, or general psychological distress the researcher sought permission to contact the participant's general practitioner and/or advised the participant to do so as soon as possible about this.

Control Group

The control group included healthy participants from the general community residing in the Auckland region who were matched to the SAH group on age, gender and ethnicity.

Potential participants were recruited in a number of ways, including postings of an advertisement (Appendix C) and PIS (Appendix D) in churches, libraries, community centres, retirement homes, and medical services throughout the Auckland region; and the snowballing technique, whereby potential control participants recruit additional potential control participants from among their own networks. Where requested and where practicable, the researcher also conducted brief presentations about the research to community groups and their patrons. Where presentations were made, PISs and consent forms (Appendix E) were made available with freepost return envelopes in which to return completed consent forms, should any attendees be interested in participating.

Following contact from interested potential participants, the researcher answered further questions about the study and, if not already available, sent a PIS, consent form and freepost return envelope to return the signed consent form should they decide to participate. Once the researcher received a signed consent form, each potential control participant was contacted via telephone or email using the contact details they provided on the consent form. Potential control participants were matched to SAH participants and checked for exclusion criteria previously described. Potential control participants who could not be matched or who met exclusion criteria were excluded from the study, but were thanked for their interest and offered a brief written summary of the findings upon study completion. Potential control participants who matched and did not meet any exclusion criteria were recruited and assessment sessions were scheduled at times agreeable to both parties.

In line with the assessment procedure for SAH participants, control participants first completed telephone assessments (approximately 15 to 20 minutes) followed by face-to-face assessments (approximately 90 minutes) at their usual place of residence. As was done with SAH participants, time was taken prior to beginning assessment to establish rapport and reduce anxiety and to ensure optimal testing conditions. Participants were also reminded to

use any aids they may need (e.g., glasses), and were asked to advise the researcher if they needed breaks during testing, which were then provided. If a participant's responses on either of the psychological measures indicated depression, anxiety, or general psychological distress the researcher, consistent with ethical approval obtained, sought permission to contact the participant's general practitioner and/or advised the participant to do so as soon as possible about this.

During recruitment participants were informed via the PIS and verbally by the researcher that participation may benefit them in two ways. First, as data would be stored for 10 years in accordance with Health Act regulations (Privacy Commissioner, 2008), their neuropsychological test data may serve as personal baseline data should they be referred for neuropsychological assessment during this time. Second, an offer of compensation for their participation time was available (i.e., $10 petrol voucher). Participants indicated on their consent form whether they would like to accept both/either of these potential benefits. A letter to their general practitioner (Appendix F) to facilitate future contact and access to the participant's neuropsychological test data if required and/or compensation were provided at the face-to-face assessment as directed by the participant.

Data Management

After administration and scoring of all measures in accordance with standard procedures, all data were entered into SPSS20.0 file for analysis. Each participant was given a code number and all identifying information was removed from the file. Prior to data analysis, preliminary analyses of the data set were conducted to address accuracy of data input and missing data.

Accuracy of data input. To ensure accuracy of the data file, descriptive statistics for each variable and all individual items that make up the variables were generated and examined. This involved checking that values entered for each variable fell within the range

of possible values for that variable and, for continuous variables, mean scores were plausible. (Pallant, 2011; Tabachnick & Fidell, 2013).

Missing data. A moderate to large amount of data was missing in the data set that needed to be dealt with.

At 28-day assessment, 11 SAH participants (36.7%) did not complete any assessments due to being too unwell (n=5), speech impairment (n=2), cognitive impairment (n=1), and unspecified reasons (n=3). Most mRS (n=7) and BI scores (n=6) were able to be provided by these SAH participants' nominated proxy. In addition, another 11 SAH participants (36.7%) did not complete CNS-VS at this time due to being too unwell (n=2), fatigue (n=6), or unwilling to participate (n=3). One SAH participant (3.3%) did not complete the BDS due to time limitations and another participant had two outcomes missing on this measure (EIF and total score). Two SAH participants (6.7%) did not complete the MoCA due to time limitations (n=1) and fatigue (n=1). Single values were also missing on the HADS (depression subscale and total score) and the SF-36 (VT, SF, MH, MCS).

At 6-month assessment, 7 SAH participants (23.3%) did not complete any assessments due to being too unwell (n=1), speech impairment (n=2), and unspecified reasons (n=4). Nominated proxy provided mRS and BI scores for 3 of these SAH participants. In addition, another 13 SAH participants (43.3%) did not complete CNS-VS at this time due to being too unwell (n=5), fatigue (n=3), or unwilling to participate (n=5). Values were also missing for a number of CNS-VS domains; NCI (x2), composite, verbal and visual memory (x1), complex attention (x1), working memory (x2), and sustained attention (x2). Two SAH participants (6.7%) did not complete the BDS or MoCA due to being too unwell, and another (3.3%) did not complete the BDS due to swollen hands. Single values were also missing on the HADS (depression subscale and total score) and the GHQ-28 (social dysfunction subscale and total score).

At 12-month assessment, 5 SAH participants (16.7%) did not complete any assessments due to being too unwell (n=1), speech impairment (n=2), and unspecified reasons (n=2). Nominated proxy provided mRS and BI scores for 3 of these SAH participants. In addition, another 15 SAH participants (50%) did not complete CNS-VS at this time due to being too unwell (n=4), fatigue (n=5), or unwilling to participate (n=6). Single values were also missing for working memory and sustained attention CNS-VS domains. One SAH participant (3.3%) did not complete the BDS or MoCA due to being too unwell, and single values on the BDS (FIF and total scores) were missing for another participant.

All neuropsychological, psychological and functional outcomes data was complete for the control group.

There are various ways of handling missing data, including deleting cases or variables and estimating missing data (e.g., substituting mean values; Pallant, 2011; Tabachnick & Fidell, 2013). As is generally recommended, pairwise exclusion of missing data was employed, where the participant was excluded only if they were missing the data required for the specific analysis, but was included in all analyses for which they had the necessary information (Pallant, 2011). This was chosen over listwise exclusion, which totally excludes a participant from all analyses if any of their data is missing, which can result in substantial and unnecessary loss of subjects (Pallant, 2011; Tabachnick & Fidell, 2013). Estimating missing data is generally ill advised as it can severely distort the results of the analysis, thus was not employed (Pallant, 2011).

CHAPTER III:

RESULTS

Overview

The analyses for this study are presented in a number of sections. The first section presents overall performance of SAH and control groups and group comparisons. Means and SDs are presented to describe overall performance of the SAH group at each time point and the control group across measures. One-way ANOVAs were used to determine which neuropsychological, psychological and functional outcomes SAH survivors differed on average from matched controls at 6- and 12-months. As reliance on means may not provide a full picture, further group comparison involved examination of the proportion of SAH group scores at 6- and 12-months and control group scores that fell within qualitative categories (CNS-VS) or fell below or exceeded cut-off scores suggestive of poor functioning (other measures). Chi square analyses were used to determine whether group proportions differed significantly on the outcomes of interest.

Section two examines SAH group performance over time on measures of neuropsychological, psychological and functional outcomes. Repeated measures within-subjects ANOVAs were used to examine change over time across measures. Change over time was also plotted to provide a better picture of the data for the CNS-VS.

In section three, one-way ANOVAs (discrete variables) and Pearson's bivariate correlations (continuous variables) were conducted to determine what demographic factors (i.e., age, gender, ethnicity, education), injury characteristics (i.e., SAH severity, surgical clipping or coiling, previous stroke), and 28-day and 6-month neuropsychological, psychological and functional outcomes were most related to 12-month HRQoL (SF-36) for the SAH group.

Finally, section four presents interrelationships amongst 12-month outcomes for SAH survivors. A series of Pearson's bivariate correlation matrices explored the degree and direction of relationships between areas of neuropsychological, psychological and functional outcomes at 12-months.

Section 1: Overall Performance and Group Comparison

This section examines overall performance by the SAH group at 28-days, 6- and 12-months and the control group across measures, and compares SAH group performance at 6- and 12-months to control group performance. SAH group performance at 28-days was not compared to control group performance as fewer SAH participants were able to be assessed acutely and acute outcomes post-SAH have been more extensively researched in the literature (Alfieri et al., 2008; Ogden et al., 1993; Meyer et al., 2010; Navi et al., 2012). As outlined above, change in performance over time will be examined in the following section.

Tables 8, 10 and 12 present the means and SDs for the SAH group at each time point and the control group on neuropsychological, psychological and functional outcomes, respectively. To compare SAH group mean performances at 6- and 12-months to the control group one-way ANOVAs were run with group (i.e., SAH and control) as the independent variable and the neuropsychological, psychological and functional variables listed in Tables 8, 10 and 12 entered as dependent variables. As reliance on means may not provide a full picture, the proportion of SAH group scores at 6- and 12-months and control group scores falling within qualitative categories (CNS-VS; Table 9) or falling below or exceeding cut-off scores suggestive of poor functioning (other measures; Table 11 and 13) were also compared. Chi square analyses were conducted to determine whether group proportions differed significantly on the outcomes of interest. This analysis was not conducted for the BDS as qualitative categories or cut-off scores are not specified for this measure. These results are

first presented for neuropsychological outcomes, followed by psychological then functional outcomes.

Neuropsychological Outcomes

Table 8 presents overall performance of the SAH and control groups on neuropsychological outcomes and comparisons of 6- and 12-month SAH group and control group means. As can be seen in Table 8, while SAH group means tended to be slightly lower, both SAH and control group mean performances were within the average range (90 to 109) across domain scores of the CNS-VS at each assessment. The only exception was an above average mean performance by the SAH group on the processing speed domain at 28-days. At 6-months SAH and control group mean performances were comparable for most domain scores excluding working memory and sustained attention, for which the SAH group were on average significantly more impaired compared to matched controls. The effect sizes for these differences approached what is defined as a medium effect (Cohen, 1992). At 12-months mean SAH and control group performance no longer differed significantly on the sustained attention domain, but the SAH group remained on average significantly more impaired on the working memory domain, but with a small effect size (Cohen, 1992).

In contrast with the executive functioning results on the CNS-VS, the SAH group was on average significantly more impaired on executive functioning compared to matched controls at both 6- and 12-months as assessed by the BDS (total score). The effect sizes for these differences approached medium (Cohen, 1992). Contributing to this, the SAH group was on average significantly more impaired on the FIF (ability to use feedback) compared to matched controls at both time points with effect sizes being large (Cohen, 1992). Mean performances did not differ significantly on the MPF (ability to volitionally generate and sustain motor responses) or EIF (impulsivity).

Table 8

Performance of SAH and control groups and comparison of SAH group at 6- and 12-months to control group across neuropsychological outcomes

	SAH group															
	28-days			6-months			12-months			Control group (N=29)		Differences between groups 6-months			Differences between groups 12-months		
Measures	N	Mean	SD	N	Mean	SD	N	Mean	SD	Mean	SD	F	p	eta2	F	p	eta2
CNS Vital Signs Domains																	
NCI	8	100.13	10.40	8	98.88	10.70	10	101.20	14.70	102.59	6.98	1.40	0.25	0.04	0.16	0.69	0.00
Composite memory	8	90.75	17.20	9	101.56	19.53	10	93.80	21.04	100.90	11.70	0.02	0.90	0.00	1.77	0.19	0.05
Verbal memory	8	95.00	17.11	9	98.56	19.59	10	95.10	22.26	99.28	11.74	0.02	0.89	0.00	0.58	0.45	0.02
Visual memory	8	90.75	14.92	9	103.78	16.16	10	94.30	16.53	102.34	14.71	0.06	0.80	0.00	2.09	0.16	0.05
Processing speed	8	119.50	22.68	10	108.20	28.25	10	102.30	64.34	103.38	12.90	0.54	0.47	0.01	0.01	0.93	0.00
Executive function	8	103.50	18.97	10	97.60	15.21	10	106.10	20.87	105.00	8.81	3.54	0.07	0.09	0.06	0.82	0.00
Psychomotor speed	8	107.38	19.64	10	100.30	17.34	10	109.00	29.94	101.38	13.51	0.04	0.84	0.00	1.21	0.28	0.03
Reaction time	8	99.38	11.01	10	99.80	14.37	10	102.40	12.32	102.48	12.79	0.31	0.58	0.01	0.00	0.99	0.00
Complex attention	8	100.75	19.79	9	99.89	14.99	10	97.80	18.60	103.72	8.33	0.97	0.33	0.03	1.91	0.18	0.05
Cognitive flexibility	8	102.88	19.22	10	97.10	15.24	10	102.90	19.70	104.24	9.46	3.05	0.09	0.08	0.08	0.78	0.00
Working memory	8	97.88	9.94	8	95.75	11.59	9	95.78	22.30	108.48	9.58	10.15	**<.01**	0.23	6.10	**0.02**	0.15
Sustained attention	8	96.88	10.67	8	90.75	20.89	9	100.67	12.16	105.66	8.27	9.81	**<.01**	0.22	1.99	0.17	0.05
Social acuity	8	104.25	14.68	10	95.40	11.06	10	96.60	14.22	102.00	17.60	1.23	0.28	0.03	0.77	0.39	0.02
Behavioural Dyscontrol Scale																	
MPF	18	10.83	1.51	20	9.90	2.15	24	10.17	1.95	10.76	0.95	3.63	0.06	0.07	2.08	0.16	0.04
EIF	17	5.71	0.59	20	5.50	0.76	24	5.33	0.82	5.52	0.57	0.01	0.93	0.00	0.92	0.34	0.02
FIF	18	7.28	1.41	20	6.65	1.35	23	6.83	1.50	8.41	0.63	37.97	**<.01**	0.45	26.79	**<.01**	0.35
Total score	17	23.94	2.97	20	22.05	3.68	23	22.30	3.82	24.69	1.11	13.31	**<.01**	0.22	10.28	**<.01**	0.17
Montreal Cognitive Assessment																	
Total score	17	25.65	3.41	21	26.67	2.42	24	26.13	2.79	28.52	0.91	14.31	**<.01**	0.23	18.99	**<.01**	0.27

EIF=Environmental Independence Factor; FIF=Fluid Intelligence Factor; MPF=Motor Programming Factor; NCI=Neurocognition Index.
Note: Bold typeface highlights significant difference.

Regarding performance on the MoCA, at 28-days the SAH group mean fell below the cut-off score of 26, which is indicative of cognitive impairment. At 6- and 12-months, the SAH group mean fell on this cut-off score, suggesting performance in the normal range. The control group mean was above the cut-off score, indicating performance in the normal range. Though group means suggested performance in the normal range, the SAH group were on average significantly more impaired on the MoCA compared to the matched control group at 6- and 12-months with moderate effect sizes (Cohen, 1992).

To further compare group performances, Table 9 presents the proportion of SAH group scores at 6- and 12-months and control group scores falling within each qualitative category of the CNS-VS domains. As can be seen in Table 9, the majority of the SAH group at 6- and 12-months and the control group performed in the average and above average ranges on each domain. However, greater proportions of the SAH group performed in the low average, low and very low ranges at 6- and 12-months in comparison to the control group and in excess of what would be expected under a normal distribution.

With regards to the very low range, at 6-months 10% to 12.5% of the SAH group performed in the very low range on memory (composite, verbal), speed (processing speed, reaction time) and sustained attention domains. At 12-months, 10% to 20% of the SAH group performed in the very low range on memory (composite, verbal, visual, working), processing speed, higher functioning (executive function, complex attention, cognitive flexibility) and social acuity domains. This is in comparison to the control group, whereby only 3.5% performed in the very low range on the social acuity domain only. Thus, though mean performances of the SAH group at 6- and 12-months and the control group on the CNS-VS domains were in the average range and largely comparable in the previous analyses, this analysis demonstrates qualitative differences in group performance.

Table 9

Performance on CNS Vital Signs domains of SAH group at 6- and 12-months and control group as proportion falling within qualitative categories

CNS Vital Signs Domains		Qualitative performance ranges and associated standard scores				
		Very low (N)	Low (N)	Low average (N)	Average (N)	Above average (N)
	N	<70	70-79	80-89	90-109	>109
Normal distribution %		2.2%	6.7%	16.1%	50%	25%
SAH group 6-months						
NCI	8	- -	- -	12.5% (1)	**75% (6)**	12.5% (1)
Composite memory	9	11.11% (1)	- -	11.11% (1)	**44.44% (4)**	**33.33% (3)**
Verbal memory	9	11.11% (1)	11.11% (1)	- -	**55.55% (5)**	**22.22% (2)**
Visual memory	9	- -	- -	**22.22% (2)**	**44.44% (4)**	**33.33% (3)**
Processing speed	10	10% (1)	- -	- -	**40% (4)**	**50% (5)**
Executive function	10	- -	**20% (2)**	10% (1)	**40% (4)**	**30% (3)**
Psychomotor speed	10	- -	10% (1)	**20% (2)**	**60% (6)**	10% (1)
Reaction time	10	10% (1)	- -	- -	**70% (7)**	**20% (2)**
Complex attention	9	- -	11.11% (1)	11.11% (1)	**44.44% (4)**	**33.33% (3)**
Cognitive flexibility	10	- -	**20% (2)**	10% (1)	**50% (5)**	**20% (2)**
Working memory	8	- -	12.5% (1)	12.5% (1)	**75% (6)**	- -
Sustained attention	8	12.5% (1)	- -	**25% (2)**	**62.5% (5)**	- -
Social acuity	10	- -	10% (1)	10% (1)	**70% (7)**	10% (1)
SAH group 12-months						
NCI	10	- -	- -	**20% (2)**	**60% (6)**	**20% (2)**
Composite memory	10	10% (1)	10% (1)	10% (1)	**40% (4)**	**30% (3)**
Verbal memory	10	**20% (2)**	- -	**20% (2)**	**20% (2)**	**40% (4)**
Visual memory	10	10% (1)	- -	**30% (3)**	**50% (5)**	10% (1)
Processing speed	10	**20% (2)**	- -	- -	**50% (5)**	**30% (3)**
Executive function	10	10% (1)	10% (1)	- -	**20% (2)**	**60% (6)**
Psychomotor speed	10	- -	10% (1)	10% (1)	**60% (6)**	**20% (2)**
Reaction time	10	- -	- -	10% (1)	**60% (6)**	**30% (3)**
Complex attention	10	10% (1)	10% (1)	- -	**50% (5)**	**30% (3)**
Cognitive flexibility	10	10% (1)	10% (1)	- -	**30% (3)**	**50% (5)**
Working memory	9	11.11% (1)	- -	**22.22% (2)**	**44.44% (4)**	**22.22% (2)**
Sustained attention	9	- -	11.11% (1)	- -	**55.55% (5)**	**33.33% (3)**
Social acuity	10	10% (1)	- -	**20% (2)**	**50% (5)**	**20% (2)**
Control group						
NCI	29	- -	- -	3.45% (1)	**86.21% (25)**	**10.34% (3)**
Composite memory	29	- -	3.45% (1)	10.34% (3)	**62.07% (18)**	**24.14% (7)**
Verbal memory	29	- -	3.45% (1)	17.24% (5)	**58.62% (17)**	**20.69% (6)**
Visual memory	29	- -	6.90% (2)	13.79% (4)	**37.93% (11)**	**41.38% (12)**
Processing speed	29	- -	3.45% (1)	10.34% (3)	**62.07% (18)**	**24.14% (7)**
Executive function	29	- -	- -	3.45% (1)	**62.07% (18)**	**34.48% (10)**
Psychomotor speed	29	- -	6.90% (2)	13.79% (4)	**58.62% (17)**	**20.69% (6)**
Reaction time	29	- -	6.90% (2)	10.34% (3)	**44.83% (13)**	**37.93% (11)**
Complex attention	29	- -	- -	6.90% (2)	**62.07% (18)**	**31.03% (9)**
Cognitive flexibility	29	- -	- -	6.90% (2)	**68.97% (20)**	**24.14% (7)**
Working memory	29	- -	- -	- -	**48.28% (14)**	**51.72% (15)**
Sustained attention	29	- -	- -	3.45% (1)	**68.97% (20)**	**27.59% (8)**
Social acuity	29	3.45% (1)	3.45% (1)	6.90% (2)	**44.83% (13)**	**41.38% (12)**

NCI=Neurocognition Index

Note: Bold typeface highlights proportions ≥ 20

In relation to the MoCA, 28.57% (n=6) and 37.50% (n=9) of SAH participant scores at 6- and 12-months fell below the cut-off score of 26, respectively, indicating cognitive impairment. However, no control participant scores fell below the cut-off score. Chi square analyses indicated that significantly greater proportions of SAH participants' scores fell below the cut-off as compared to control participants at 6- ($\chi 2(1)=9.42$, $p=.00$) and 12-months ($\chi 2(1)=13.10$, $p<.01$).

Psychological Outcomes

Table 10 presents overall performance of the SAH and control groups on psychological outcomes and comparisons of 6- and 12-month SAH group and control group means. As can be seen in Table 10, SAH and control groups scored on average within the "normal" range (0 to 7) on the anxiety and depression subscales of the HADS at each assessment. Conversely, the SAH group mean exceeded the total score cut-off of 24 on the GHQ-28 at 28-days, suggesting poor psychological health and wellbeing. SAH group mean total scores at 6- and 12-months fell below the GHQ-28 cut-off, as did the control group mean total score.

At 6-months the SAH group mean total scores on the HADS and the GHQ-28 were significantly higher compared to matched controls, indicating greater emotional distress and poorer psychological health and wellbeing. At this time SAH group means were also significantly higher (i.e., more symptomology) on the HADS depression subscale and the GHQ-28 somatic symptoms, anxiety and insomnia and social dysfunction subscales, with the severe depression subscale approaching significance. At 12-months the SAH group continued to produce significantly higher mean total scores on both measures, and the HADS depression subscale and the GHQ-28 somatic symptoms subscale. At 12-months the GHQ-28 anxiety and insomnia and social dysfunction mean subscale scores no longer differed

Table 10

Performance of SAH and control groups and comparison of SAH group at 6- and 12-months to control group across psychological outcomes

	SAH group									Control group (N=29)		Differences between groups 6-months			Differences between groups 12-months		
	28-days			6-months			12-months										
Measures	N	Mean	SD	N	Mean	SD	N	Mean	SD	Mean	SD	F	p	eta2	F	p	eta2
Hospital Anxiety and Depression Scale																	
Anxiety	19	6.26	4.15	23	6.00	4.78	25	5.48	4.41	4.10	2.04	3.73	0.06	0.07	2.27	0.14	0.04
Depression	18	5.56	3.91	22	3.73	3.68	25	4.12	3.38	1.83	1.49	6.38	**0.02**	0.12	10.89	**<.01**	0.17
Total score	18	11.94	7.53	22	10.00	7.53	25	9.60	7.07	5.93	2.73	7.26	**0.01**	0.13	6.69	**0.01**	0.11
General Health Questionnaire 28																	
Somatic symptoms	19	10.63	3.64	23	7.13	4.21	25	7.00	3.81	3.10	1.88	21.26	**<.01**	0.30	23.73	**<.01**	0.31
Anxiety and insomnia	19	7.42	5.45	23	6.78	5.05	25	4.96	4.36	3.52	2.52	9.28	**<.01**	0.16	2.29	0.14	0.04
Social dysfunction	19	9.32	3.87	22	7.86	2.51	25	7.64	2.60	6.72	0.92	5.09	**0.03**	0.09	3.16	0.08	0.06
Severe depression	19	1.68	3.53	23	1.52	3.23	25	1.04	2.73	0.28	0.84	3.99	0.05	0.07	2.05	0.16	0.04
Total score	19	29.05	12.12	22	22.00	9.53	25	20.64	9.94	13.62	4.96	16.57	**<.01**	0.25	11.25	**<.01**	0.18

Note: Bold typeface highlights significant difference.

significantly between groups. Effect sizes at 6- and 12-months ranged from small to medium (Cohen, 1992).

Table 11 presents the proportion of SAH group scores at 6- and 12-months and control group scores that exceeded cut-offs on the HADS and the GHQ-28, thus suggesting poor psychological health and wellbeing. As can be seen in Table 11, significantly greater proportions of SAH participants produced scores that exceeded cut-offs on the HADS subscales (>7) and the GHQ-28 total (>24) at 6- and 12-months as compared to control group participants. Interestingly, the proportion of SAH group participants whose scores exceeded cut-offs on each outcome was similar at 6- and 12-months. Thus, though all mean scores fell below cut-offs at these time points in the previous analysis, the current analysis demonstrates that significantly higher proportions of the SAH group would be classified as experiencing anxiety, depression, and generally poor psychological health and wellbeing than controls. Particularly noteworthy is the HADS anxiety subscale, where approximately one third of the SAH group exceeded the cut-off score at 6- and 12-months compared to less than 5% of the control group, despite SAH and control group means not differing significantly.

Table 11

Proportion of SAH group scores at 6- and 12-months and control group scores exceeding cut-off (i.e., poor functioning) on psychological measures

	SAH group				Control group		Significance between groups			
	6-months		12-months		(N=29)		6-months		12-months	
Measures	N(total)	%	N(total)	%	N	%	$\chi2$	p	$\chi2$	p
Hospital Anxiety and Depression Scale										
Anxiety	9(23)	39.13%	8(25)	32%	1	3.45%	10.51	**<.01**	7.88	**0.01**
Depression	4(22)	18.18%	4(25)	16%	- -	- -	5.72	**0.02**	5.01	**0.03**
General Health Questionnaire 28										
Total score	8(22)	36.36%	9(25)	36%	1	3.45%	9.33	**<.01**	9.43	**<.01**

Note: Bold typeface highlights significant difference.

Functional Outcomes

Table 12 presents overall performance of the SAH and control groups on functional outcomes and comparisons of 6- and 12-month SAH group and control group means. As can be seen in Table 12, the SAH group were assessed on average to have minor stroke symptoms (NIHSS≤5) and good outcomes regarding disability (mRS≤2; BI≥90) at each assessment. As would be expected, the control group was assessed to have no disability. At 6- and 12-months the SAH group were on average significantly more disabled regarding instrumental and basic ADL than matched controls as assessed by the mRS. Both comparisons yielded large effect sizes (Cohen, 1992). However, the group means did not differ significantly on disability related to personal care and mobility as assessed by the BI at 6- and 12-months.

Regarding HRQoL, the SAH group scored on average significantly lower than the matched control group, suggesting poorer HRQoL, on the MCS and PCS and across all scales of the SF-36 at 6-months. This was with the exception of the GH scale, though this scale approached significance. Summary score effect sizes approached large and scale effect sizes ranged from small to medium (Cohen, 1992). At 12-months, the SAH group continued to score on average significantly lower than the matched control group on both summary scores and all scales of the SF-36, including the GH scale, with effect sizes ranging from small to medium (Cohen, 1992).

Table 13 presents the proportion of SAH group scores at 6- and 12-months and control group scores that fell below (BI, SF-36) or exceeded (mRS) cut-offs, thus suggesting poor functional outcomes and HRQoL. As can be seen in Table 13, reasonably small proportions of SAH group scores were indicative of poor outcomes regarding disability, and these proportions were not significantly greater compared to matched controls at either time point. However, greater proportions of SAH group scores at 6- and 12-months were more

Table 12

Performance of SAH and control groups and comparison of SAH group at 6- and 12-months to control group across functional outcomes

	SAH group									Control group (N=29)		Differences between groups 6-months			Differences between groups 12-months		
	28-days			6-months			12-months										
Measures	N	Mean	SD	N	Mean	SD	N	Mean	SD	Mean	SD	F	p	eta2	F	p	eta2
NIHSS	19	0.84	1.34	23	0.57	1.34	25	1.08	1.32	- -	- -	- -	- -	- -	- -	- -	- -
mRS	26	1.85	0.97	26	1.46	0.91	28	1.50	0.96	0.00	.00	75.85	**<.01**	0.59	70.52	**<.01**	0.56
Barthel Index	25	90.60	20.58	26	96.73	11.83	28	96.96	11.49	100.00	.00	2.22	0.14	0.04	2.02	0.16	0.04
Short Form 36																	
Physical function	19	62.89	26.53	23	79.78	19.51	25	74.60	22.54	94.83	7.73	14.45	**<.01**	0.22	20.60	**<.01**	0.28
Role physical	19	14.47	28.03	23	50.00	47.07	25	54.00	44.88	94.83	20.46	21.31	**<.01**	0.30	19.37	**<.01**	0.27
Bodily pain	19	67.37	26.76	23	70.43	29.25	25	72.30	29.72	85.17	11.06	6.26	**0.02**	0.11	4.70	**0.04**	0.08
General health	19	60.13	23.10	23	69.78	21.98	25	65.60	24.47	79.31	11.86	3.99	0.05	0.07	7.17	**0.01**	0.12
Vitality	18	41.94	20.66	23	52.83	19.76	25	48.20	27.38	73.97	13.32	21.14	**<.01**	0.30	20.18	**<.01**	0.28
Social function	18	51.39	34.80	23	72.28	31.07	25	70.50	35.70	93.97	10.89	12.28	**<.01**	0.20	11.34	**<.01**	0.18
Role emotional	19	71.93	40.47	23	66.67	44.95	25	77.33	39.35	97.70	12.38	12.67	**<.01**	0.20	6.99	**0.01**	0.12
Mental health	18	68.22	20.05	23	75.13	15.72	25	75.84	21.12	85.79	6.89	10.77	**<.01**	0.18	5.75	**0.02**	0.10
PCS	19	51.22	18.73	23	67.50	19.50	25	66.63	25.69	88.53	9.53	26.02	**<.01**	0.34	18.24	**<.01**	0.26
MCS	18	58.91	21.26	23	66.73	21.37	25	67.97	25.08	87.86	6.97	25.10	**<.01**	0.33	16.78	**<.01**	0.24

mRS=modified Rankin Scale; MCS=Mental Component Summary; NIHSS=National Institutes of Health Stroke Scale; PCS=Physical Component Summary.

Note: Bold typeface highlights significant difference.

than 1 SD below the mean on SF-36 summary scores and scales, indicating poor HRQoL. In comparison to the control group, where fewer than 5% of participant scores were more than 1 SD below the mean on RP, VT, SF and RE scales only, the SAH group proportions were significantly greater on the MCS and all scales at 6- and 12-months. At 12-months, a significantly higher proportion of SAH group scores were more than 1 SD below the mean on the PCS also. From 6- to 12-months, the proportion of the SAH group performing poorly appeared to remain stable on some outcomes (PH, RP, GH, SF) but increase on others (BP, VT, MH, PCS, MCS). The only scale to decrease in proportion performing poorly was RE. The proportion of SAH group scores indicating poor HRQoL at 12-months was ≥20% on all SF-36 outcomes.

Table 13

Proportion of SAH group scores at 6- and 12-months and control group scores falling below or exceeding cut-off (i.e., poor functioning) on functional measures

	SAH group				Control group (N=29)		Significance between groups			
	6-months		12-months				6-months		12-months	
Measures	N(total)	%	N(total)	%	N	%	χ^2	p	χ^2	p
mRS	2(26)	7.69%	3(28)	10.71%	- -	- -	2.32	0.13	3.28	0.07
Barthel Index	1(26)	3.85%	1(28)	3.57%	- -	- -	1.14	0.29	1.05	0.31
Short Form 36										
Physical function	6(23)	26.09%	7(25)	28%	- -	- -	8.55	**<.01**	9.33	**<.01**
Role physical	11(23)	47.83%	11(25)	44%	1	3.45%	14.23	**<.01**	12.77	**<.01**
Bodily pain	6(23)	26.09%	8(25)	32%	- -	- -	8.55	**<.01**	10.89	**<.01**
General health	6(23)	26.09%	6(25)	24%	- -	- -	8.55	**<.01**	7.83	**0.01**
Vitality	8(23)	34.78%	11(25)	44%	1	3.45%	8.80	**<.01**	12.77	**<.01**
Social function	9(23)	39.13%	9(25)	36%	1	3.45%	10.51	**<.01**	9.43	**<.01**
Role emotional	8(23)	34.78%	6(25)	25%	1	3.45%	8.80	**<.01**	5.03	**0.03**
Mental health	4(23)	17.39%	8(25)	32%	- -	- -	5.46	**0.02**	10.89	**<.01**
PCS	1(23)	4.35%	6(25)	24%	- -	- -	1.29	0.26	7.83	**0.01**
MCS	3(23)	13.04%	5(25)	20%	- -	- -	4.01	**0.04**	6.39	**0.01**

MCS=Mental Component Summary; mRS=modified Rankin Scale; PCS=Physical Component Summary.
Note: Bold typeface highlights significant difference.

Section 2: Change Over Time

This section examines change in SAH group performance over time on measures of neuropsychological, psychological and functional outcomes. These changes are presented for the SAH group only as the control group was only assessed once. While means and SDs are presented in the previous section, they are replicated here for ease of reading.

Tables 14, 15 and 16 present means and SDs at each assessment and examination of change over time on neuropsychological, psychological and functional outcome measures, respectively. Analyses of change over time were conducted using repeated measures within-subjects ANOVAs with the neuropsychological, psychological and functional variables listed in Tables 14, 15 and 16 entered as dependent variables. Change over time was also plotted (Figures 2 to 14) to further explore the data for the CNS-VS. These results are first presented for neuropsychological outcomes, followed by psychological then functional outcomes.

Neuropsychological Outcomes

As can be seen in Table 14, performance on the MoCA improved significantly from 28-days (mean performance indicated cognitive impairment) to 6-months (mean performance in normal range). None of the other comparisons were significant ($p>.05$). Mean performance appeared to remain stable on most CNS-VS domains (NCI, verbal and working memory, reaction time, complex attention, cognitive flexibility) and all BDS outcomes. However, though not significant, an overall trend towards initial deterioration then stable (processing speed, social acuity) or improved performance (executive function, psychomotor speed, sustained attention) was observed on several CNS-VS domains. Interestingly, the visual memory domain demonstrated the reverse trajectory, where initial improvement preceded later decline and this contributed to a similar pattern for the composite memory domain.

Table 14

Within-subject change over time for SAH participants on measures of neuropsychological outcomes

	Time of measure									Significance of change					
	28-days			6-months			12-months			28-days to 6-months			6- to 12-months		
Measures	N	Mean	SD	N	Mean	SD	N	Mean	SD	N	F	p	N	F	p
CNS Vital Signs Domains															
NCI	8	100.13	10.40	8	98.88	10.70	10	101.20	14.70	4	0.44	0.55	7	0.01	0.95
Composite memory	8	90.75	17.20	9	101.56	19.53	10	93.80	21.04	5	2.18	0.21	8	0.98	0.36
Verbal memory	8	95.00	17.11	9	98.56	19.59	10	95.10	22.26	5	0.02	0.91	8	0.18	0.68
Visual memory	8	90.75	14.92	9	103.78	16.16	10	94.30	16.53	5	3.41	0.14	8	2.50	0.16
Processing speed	8	119.50	22.68	10	108.20	28.25	10	102.30	64.34	6	0.58	0.48	9	0.03	0.87
Executive function	8	103.50	18.97	10	97.60	15.21	10	106.10	20.87	6	0.03	0.87	9	0.49	0.51
Psychomotor speed	8	107.38	19.64	10	100.30	17.34	10	109.00	29.94	6	0.84	0.40	9	1.39	0.27
Reaction time	8	99.38	11.01	10	99.80	14.37	10	102.40	12.32	6	3.56	0.12	9	0.18	0.69
Complex attention	8	100.75	19.79	9	99.89	14.99	10	97.80	18.60	5	0.19	0.69	8	0.31	0.60
Cognitive flexibility	8	102.88	19.22	10	97.10	15.24	10	102.90	19.70	6	0.02	0.89	9	0.13	0.73
Working memory	8	97.88	9.94	8	95.75	11.59	9	95.78	22.30	4	1.00	0.39	7	0.97	0.36
Sustained attention	8	96.88	10.67	8	90.75	20.89	9	100.67	12.16	4	0.97	0.40	7	2.10	0.20
Social acuity	8	104.25	14.68	10	95.40	11.06	10	96.60	14.22	6	0.86	0.39	9	2.65	0.14
Behavioural Dyscontrol Scale															
MPF	18	10.83	1.51	20	9.90	2.15	24	10.17	1.95	15	2.15	0.16	18	1.76	0.20
EIF	17	5.71	0.59	20	5.50	0.76	24	5.33	0.82	14	0.00	1.00	18	0.68	0.42
FIF	18	7.28	1.41	20	6.65	1.35	23	6.83	1.50	15	1.28	0.28	17	4.23	0.06
Total score	17	23.94	2.97	20	22.05	3.68	23	22.30	3.82	14	2.83	0.12	17	1.48	0.24
Montreal Cognitive Assessment															
Total score	17	25.65	3.41	21	26.67	2.42	24	26.13	2.79	15	9.55	**0.01**	19	0.00	1.00

EIF=Environmental Independence Factor; FIF=Fluid Intelligence Factor; MPF=Motor Programming Factor; NCI=Neurocognition Index.
Note: Bold typeface highlights significant change.

Given the non-significant results based on the small number of participants available for change over time analysis on CNS-VS domains (n=4 to 9), it was hypothesised that different individual trajectories could have cancelled out an overall effect. Thus, performance over time on each CNS-VS domain was also plotted, as seen in Figures 2 to 14, to further understand the findings.

As can be seen in Figures 2 to 14, there was much variability in the trajectory between 28-day to 6-month and 6- to 12-month time points in each CNS-VS domain, with scores improving, declining, and remaining stable (within 10 standard score points) for different participants. Overall, it appeared that the trajectory between 28-days and 6-months on most domains was for performance to remain stable or decline, but to remain stable or improve between 6- and 12-months. In contrast, the overall trajectory on memory domains (composite, verbal and visual memory, Figures 3 to 5), showed the reverse pattern with greater proportions of improved performance between 28-days and 6-months and decline between 6- and 12-months.

These figures also demonstrate that each participant's trajectory varied for different domains, though when domains were considered in broader cognitive functioning domains (e.g., memory) participant trajectories within these were similar. For example, overall participant 10's performance demonstrated various levels of stability or improvement over time on most domains, but deteriorated by up to 60 standard score points on memory domains (composite, verbal, visual) between 6- and 12-months. This was particularly noteworthy in respect to the trajectories depicted for higher cognitive functioning domains (i.e., executive function, complex attention, cognitive flexibility, Figures 11 to 13). These three domains not only generated similar overall trajectories, which were consistent with the pattern described for most domains, but individual participants' performance within a time point and their trajectory was markedly similar across these domains. For example, on all three of these

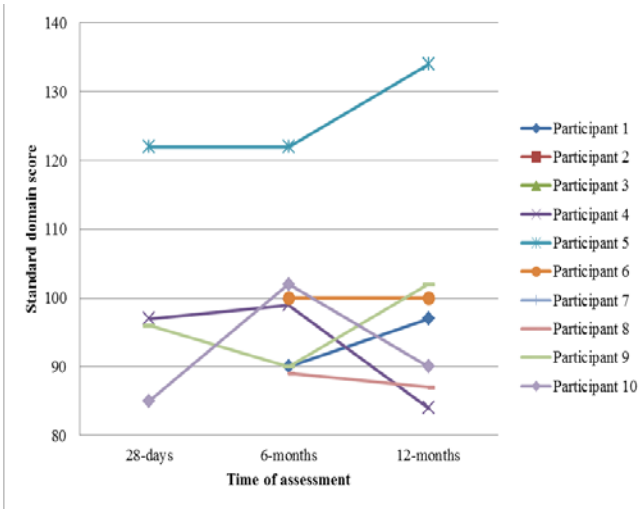

Figure 2. Change over time on Neurocognition Index for SAH participants

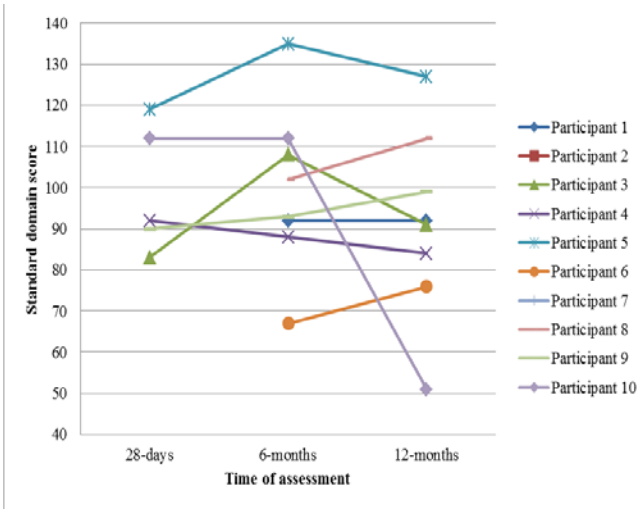

Figure 3. Change over time on composite memory domain for SAH participants

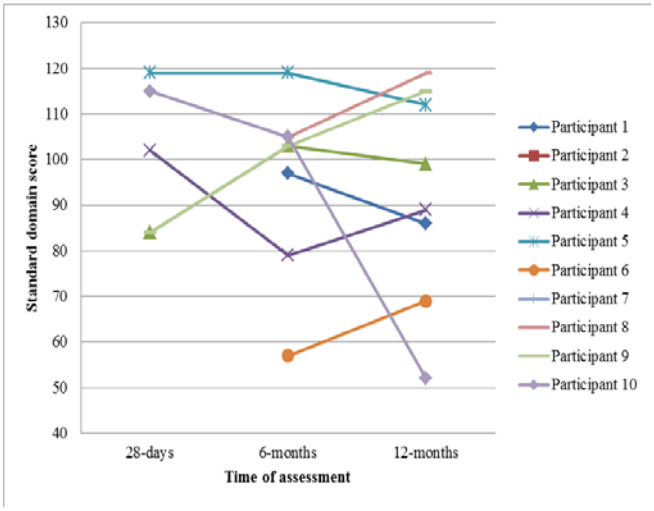

Figure 4. Change over time on verbal memory domain for SAH participants

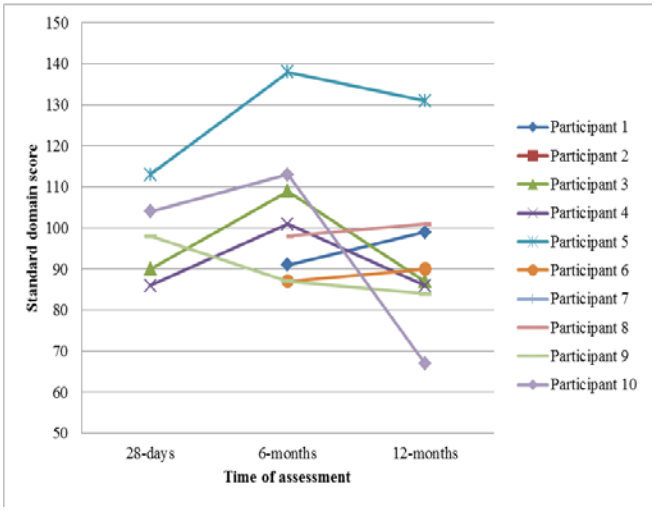

Figure 5. Change over time on visual memory domain for SAH participants

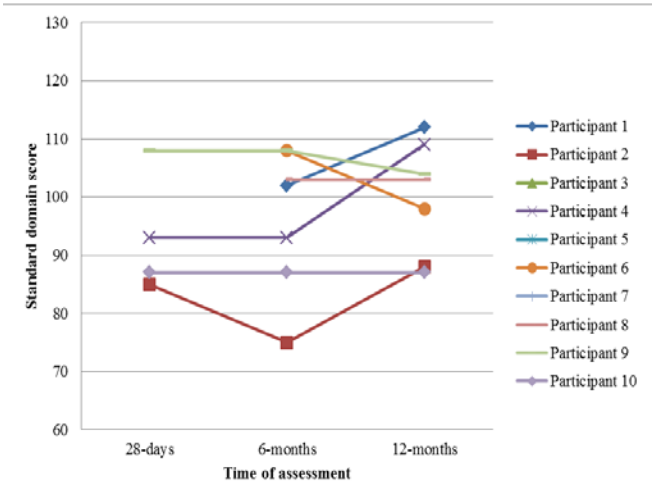

Figure 6. Change over time on working memory domain for SAH participants

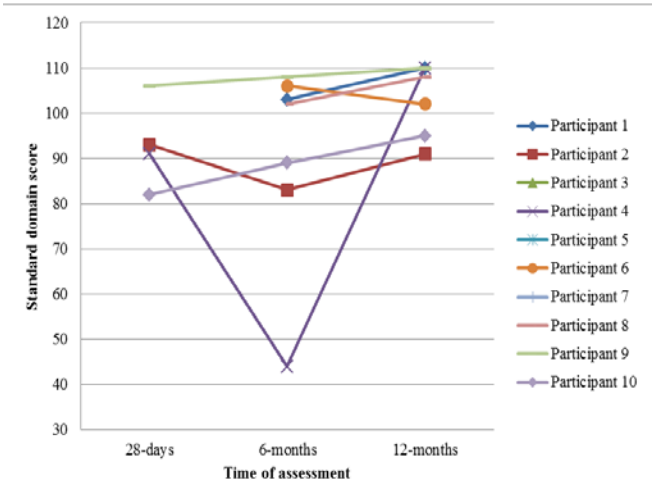

Figure 7. Change over time on sustained attention domain for SAH participants

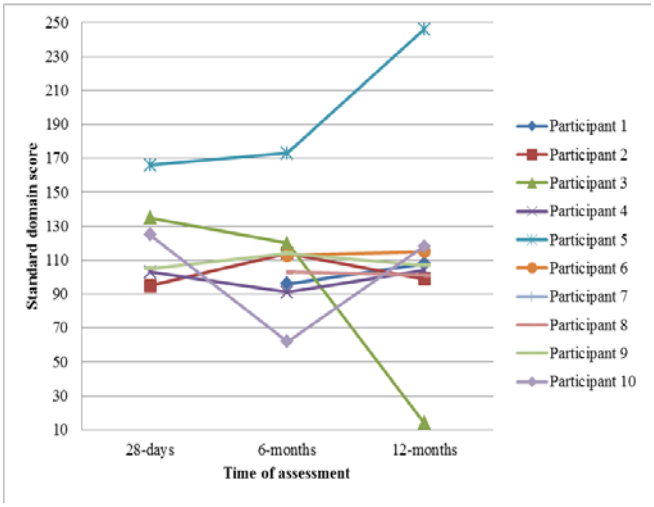

Figure 8. Change over time on processing speed domain for SAH participants

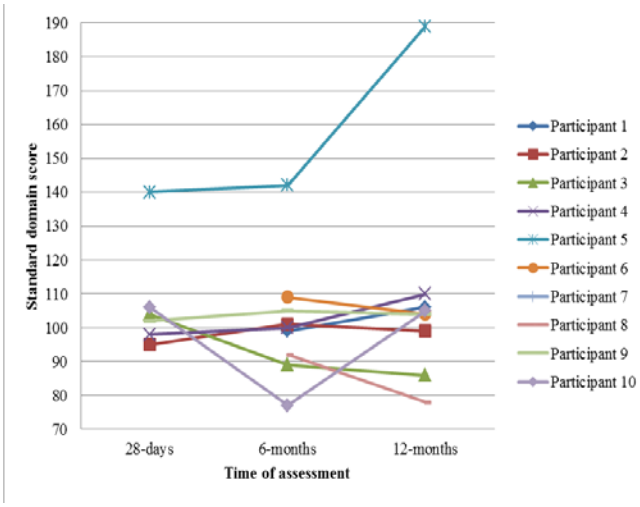

Figure 9. Change over time on psychomotor speed domain for SAH participants

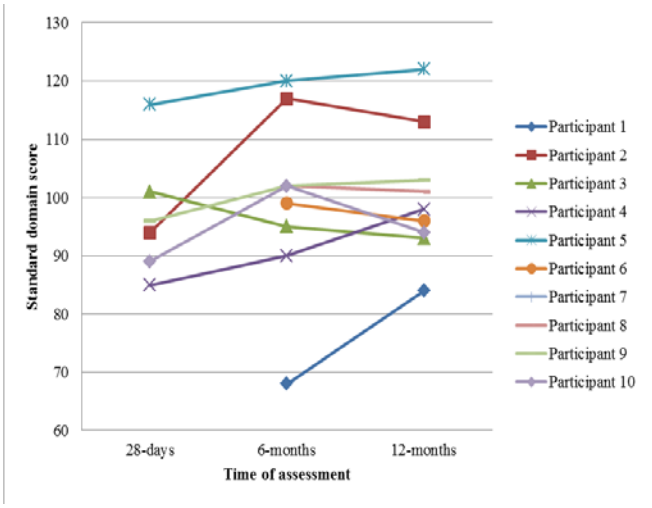

Figure 10. Change over time on reaction time domain for SAH participants

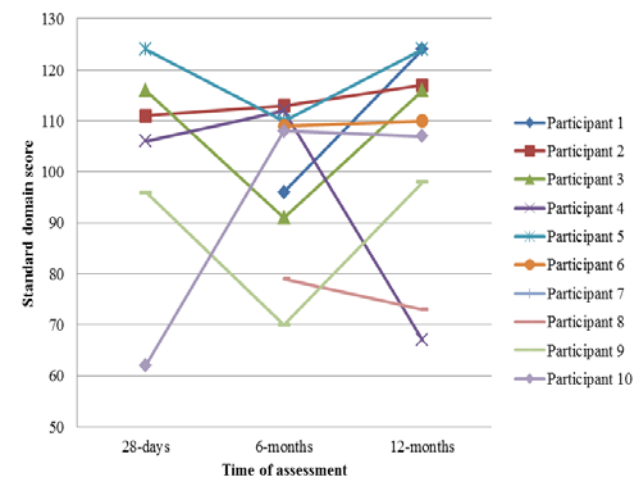

Figure 11. Change over time for executive function domain for SAH participants

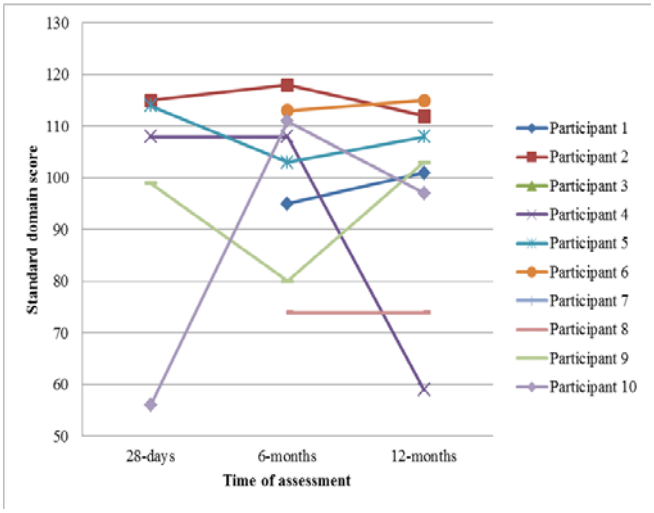

Figure 12. Change over time on complex attention domain for SAH participants

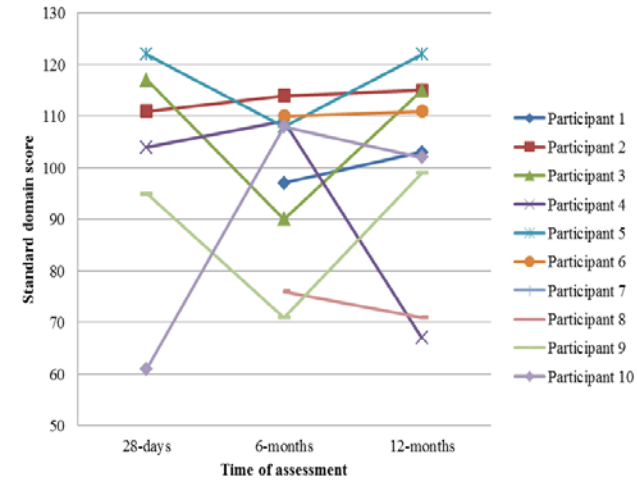

Figure 13. Change over time for cognitive flexibility domain for SAH participants

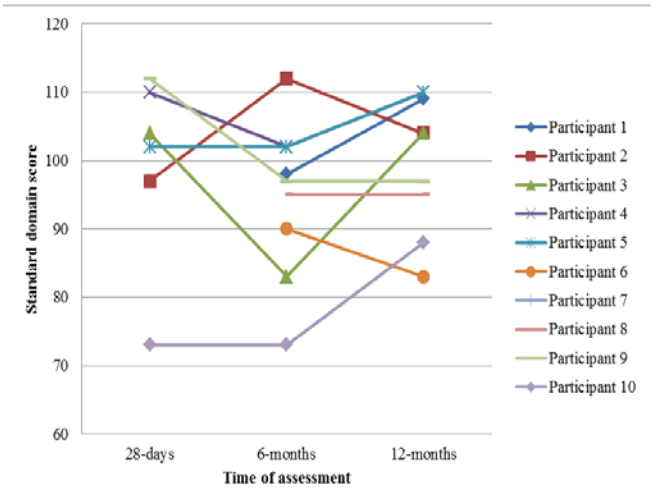

Figure 14. Change over time on social acuity domain for SAH participants

domains participant 9 scored a standard score of 90 to 100 at 28-days, deteriorating to 70 to 80 at 6-months, before improving to 95 to 105 at 12-months.

Thus, change over time on neuropsychological outcomes as assessed by the CNS-VS varied between domains, between participants, and for a participant on different domains, though somewhat similar levels of performance and trajectories for participants were seen within broader cognitive functioning domains (e.g., memory, higher cognitive functioning).

Psychological Outcomes

Table 15 shows means and significance of change over time for psychological outcomes. As seen in this table, several scores decreased significantly between 28-days and 6-months, suggesting reduced symptomology. The HADS total and depression subscale scores reduced significantly (mean scores in "normal" range at both time points), while the anxiety subscale did not change significantly. The GHQ-28 total score did not change significantly. However, the somatic symptoms and social dysfunction subscales did reduce significantly during this time. These results indicate that anxiety and severe depression seemed to change very little during the first 6-months post-SAH as compared to depression, somatic symptoms and social dysfunction which improved during this time. No significant

Table 15

Within-subject change over time for SAH participants on measures of psychological outcomes

	Time of measure									Significance of change					
	28-days			6-months			12-months			28-days to 6-months			6- to 12-months		
Measures	N	Mean	SD	N	Mean	SD	N	Mean	SD	N	F	p	N	F	p
Hospital Anxiety and Depression Scale															
Anxiety	19	6.26	4.15	23	6.00	4.78	25	5.48	4.41	16	0.48	0.50	21	0.08	0.77
Depression	18	5.56	3.91	22	3.73	3.68	25	4.12	3.38	15	9.24	**0.01**	20	0.98	0.33
Total score	18	11.94	7.53	22	10.00	7.53	25	9.60	7.07	15	6.81	**0.02**	20	0.07	0.79
General Health Questionnaire 28															
Somatic symptoms	19	10.63	3.64	23	7.13	4.21	25	7.00	3.81	16	5.97	**0.03**	21	0.07	0.79
Anxiety and insomnia	19	7.42	5.45	23	6.78	5.05	25	4.96	4.36	16	0.05	0.82	21	2.07	0.17
Social dysfunction	19	9.32	3.87	22	7.86	2.51	25	7.64	2.60	16	6.12	**0.03**	20	1.19	0.29
Severe depression	19	1.68	3.53	23	1.52	3.23	25	1.04	2.73	16	0.41	0.53	21	0.01	0.93
Total score	19	29.05	12.12	22	22.00	9.53	25	20.64	9.94	16	3.12	0.10	20	0.62	0.44

Note: Bold typeface highlights significant change.

Table 16

Within-subject change over time for SAH participants on measures of functional outcomes

	Time of measure									Significance of change					
	28-days			6-months			12-months			28-days to 6-months			6- to 12-months		
Measures	N	Mean	SD	N	Mean	SD	N	Mean	SD	N	F	p	N	F	p
NIHSS	19	0.84	1.34	23	0.57	1.34	25	1.08	1.32	16	1.77	0.20	21	7.01	**0.02**
modified Rankin Scale	26	1.85	0.97	26	1.46	0.91	28	1.50	0.96	23	8.21	**0.01**	25	0.06	0.80
Barthel Index	25	90.60	20.58	26	96.73	11.83	28	96.96	11.49	22	6.02	**0.02**	25	0.14	0.71
Short Form 36															
Physical function	19	62.89	26.53	23	79.78	19.51	25	74.60	22.54	16	10.59	**0.01**	21	0.54	0.47
Role physical	19	14.47	28.03	23	50.00	47.07	25	54.00	44.88	16	8.37	**0.01**	21	0.03	0.87
Bodily pain	19	67.37	26.76	23	70.43	29.25	25	72.30	29.72	16	0.34	0.57	21	0.13	0.72
General health	19	60.13	23.10	23	69.78	21.98	25	65.60	24.47	16	3.96	0.07	21	1.52	0.23
Vitality	18	41.94	20.66	23	52.83	19.76	25	48.20	27.38	15	1.91	0.19	21	1.54	0.23
Social function	18	51.39	34.80	23	72.28	31.07	25	70.50	35.70	15	4.47	0.05	21	0.04	0.85
Role emotional	19	71.93	40.47	23	66.67	44.95	25	77.33	39.34	16	0.15	0.70	21	0.00	1.00
Mental health	18	68.22	20.05	23	75.13	15.72	25	75.84	21.12	15	0.89	0.36	21	0.02	0.88
PCS	19	51.22	18.73	23	67.50	19.50	25	66.63	25.69	16	13.62	**<.01**	21	0.02	0.91
MCS	18	58.91	21.26	23	66.73	21.37	25	67.97	25.08	15	3.20	0.10	21	0.06	0.80

MCS=Mental Component Summary; NIHSS=National Institutes of Health Stroke Scale; PCS=Physical Component Summary.

Note: Bold typeface highlights significant change.

changes regarding psychological outcomes were found from 6- to 12-months post-SAH ($p>.05$), though mean scores suggest continued, small reductions in symptoms.

Functional Outcomes

Means and significance of change over time for functional outcomes are presented in Table 16 (previous page). As seen in this table, stroke symptoms as assessed by the NIHSS did not change significantly from 28-days to 6-months, but increased significantly from 6- to 12-months. However, this significant result reflects a mean increase of less than 1 point on a 42 point scale and is still reflective of minor stroke symptoms. Conversely, disability as assessed by the mRS and BI reduced significantly between 28-days and 6-months (mean scores indicate good outcomes at both time points), but did not change significantly from 6- to 12-months. Regarding HRQoL, the PCS improved significantly from 28-days to 6-months, as did the PH and RP scales, indicating better physical functioning and fewer role limitations due to physical functioning. Thus, it can be seen that physical abilities improve over the first 6-months post-SAH, with this being reflected in improved HRQoL in physical domains. The MCS and other scales of the SF-36 did not change significantly between 28-days and 6-months. Between 6- and 12-months, no significant changes in HRQoL were found ($p>.05$).

Section 3: Factors Related to 12-Month HRQoL

In this section, analyses were conducted to examine which demographic factors (i.e., age, gender, ethnicity, education), injury characteristics (i.e., SAH severity, surgical clipping or coiling, previous stroke), and 28-day and 6-month neuropsychological, psychological and functional outcomes were most related to 12-month HRQoL (SF-36). These analyses were conducted for the SAH group only. Twelve-month HRQoL was selected as the outcome of interest as it is arguably the most important patient-centred outcome (Almborg et al., 2010)

and was found to have the poorest outcomes for SAH survivors when compared to matched controls in the previous sections.

To analyse these relationships, one-way ANOVAs were conducted between discrete variables (i.e., gender, ethnicity, education, clipping, coiling, previous stroke) and 12-month HRQoL (SF-36 scale and summary scores), while Pearson's bivariate correlations were used to examine the degree and direction of relationships between continuous variables (i.e., age, SAH severity, 28-day and 6-month neuropsychological, psychological and functional outcomes) and 12-month HRQoL (SF-36 scale and summary scores). Due to the number of correlations being generated increasing the possibility of Type 1 error and the small sample size a conservative level of significance was used ($p<.01$) for correlation analyses. Those significant at the $p<.05$ level are identified, but interpreted with caution. These results are presented in several sections, beginning with demographic factors, followed by injury characteristics, then neuropsychological, psychological and functional outcomes.

Demographic Factors

Age at time of SAH, ethnicity (i.e., European or Other) and education (i.e., completion of secondary school, further education) were not significantly related to 12-month HRQoL ($p>.05$). Regarding gender, females had significantly lower scores on average as compared to males on the BP domain ($F[1,23]=5.22$, $p=.03$), thus they reported worse bodily pain than males at 12-months. Female and male mean scores were comparative for all other HRQoL domains and summary scores at 12-months ($p>.05$).

Injury Characteristics

With regards to injury characteristics, SAH severity as assessed by the GCS at time of SAH was not significantly related to 12-month HRQoL ($p>.01$). SAH survivors who underwent clipping scored on average significantly lower PCS scores ($F[1,23]=9.54$, $p=.01$) and on HRQoL domains contributing to this summary score, that is PH ($F[1,23]=6.12$,

p=.02), RP (F[1,23]=5.41, p=.03), BP (F[1,23]=6.22, p=.02), and GH (F[1,23]=7.29, p=.01), indicating that they had worse physical HRQoL outcomes at 12-months as compared to SAH survivors who did not undergo clipping. They also produced significantly lower MCS scores (F[1,23]=4.67, p=.04) and VT domain scores (F[1,23]=6.92, p=.02), indicating worse overall emotional HRQoL and less vitality at 12-months. Coiling was not significantly related to most HRQoL domains (p>.05), with the exception of VT domain scores (F[1,23]=4.57, p=.04) whereby SAH survivors who underwent coiling scored on average significantly higher, indicating more vitality at 12-months than those who did not undergo coiling.

SAH survivors who had had a previous stroke scored on average significantly lower MCS scores (F[1,23]=5.30, p=.03) and on HRQoL domains contributing to this summary score; that is VT (F[1,23]=5.32, p=.03), SF (F[1,23]=5.30, p=.03), and MH (F[1,23]=4.62, p=.04), indicating that they had worse vitality, social functioning, mental health, and overall emotional HRQoL outcomes at 12-months as compared to those who had not previously had a stroke. Previous stroke was also significantly related to lower PH domain scores (F[1,23]=11.07, p=.00), suggesting worse physical functioning outcomes at 12-months, but was not significantly related to any other physical HRQoL domains (p>.05).

Neuropsychological Outcomes

Table 17 presents bivariate correlations between 28-day and 6-month neuropsychological outcomes and 12-month HRQoL. For ease of presentation and interpretation, only variables that generated significant results were included in the table. As seen in Table 17, better performance (higher scores) at 28-days on several higher cognitive functioning domains of the CNS-VS (i.e., executive function, complex attention, cognitive flexibility), was significantly related to better BP domain scores at 12-months (better HRQoL regarding bodily pain; p<.01). There was also a trend towards better performance (higher scores) at 28-days on the sustained attention domain of the CNS-VS being associated with

Table 17

Correlations between 28-day and 6-month neuropsychological outcomes and 12-month HRQoL

	Short Form 36					
	Role physical	Bodily pain	General health	Vitality	Social functioning	PCS
Measures(N)	28-days					
CNS Vital Signs Domains						
Executive function(7)	.367	.893**	-.457	.134	-.268	.299
Complex attention(7)	.522	.887**	-.372	.216	-.143	.427
Cognitive flexibility(7)	.339	.915**	-.485	.127	-.295	.278
Sustained attention(7)	.694	.460	.336	.702	.405	.765*
Social acuity(7)	.531	.640	-.310	.189	-.088	.391
Montreal Cognitive Assessment						
Total score(16)	-.069	-.085	-.051	.111	.045	-.072
	6-months					
CNS Vital Signs Domains						
Executive function(10)	.274	.096	.266	.258	.511	.298
Complex attention(9)	.233	.257	.411	.350	.671*	.364
Cognitive flexibility(10)	.299	.142	.319	.308	.558	.353
Sustained attention(8)	-.223	.159	.259	.494	-.165	.056
Social acuity(10)	.707*	.491	.113	.362	.197	.580
Montreal Cognitive Assessment						
Total score(20)	-.533*	-.415	-.527*	-.556*	-.209	-.561*

PCS=Physical Component Summary
* =p<.05
**=p<.01

higher 12-month PCS scores (better overall physical HRQoL; p<.05). No other 28-day neuropsychological outcomes (remaining CNS-VS domains, BDS factor and total scores, MoCA total scores) were associated with 12-month HRQoL (p>.05).

No 6-month neuropsychological outcomes were significantly related to 12-month HRQoL (p>.01). However, several trends towards significance were found (p<.05). Better performance (higher scores) on the complex attention domain of the CNS-VS trended towards higher SF scores (better social functioning) at 12-months, while higher social acuity domain scores trended towards increased 12-month RP scores (fewer physical health related role limitations). Conversely, MoCA total scores trended towards negative associations with

several 12-month HRQoL outcomes (RP, GH, VT, PCS), suggesting that better performance (higher scores) on the MoCA at 6-months related to more physical health related role limitations and worse general health, vitality, and overall physical HRQoL (lower scores) at 12-months.

Psychological Outcomes

Table 18 presents bivariate correlations between 28-day and 6-month psychological outcomes and 12-month HRQoL. As can be seen in Table 18, 28-day HADS anxiety and total scores significantly negatively correlated with most 12-month HRQoL domains and summary scores ($p<.01$), indicating that better psychological outcomes at 28-days (lower scores) were associated with better HRQoL at 12-months (higher scores). However, 28-day HADS depression scores only significantly negatively related to 12-month PH and SF domains ($p<.01$), though trends to significance were found in relation to 12-month VT and several physical HRQoL outcomes (PCS, RP, GH; $p<.05$). No 28-day HADS scores significantly related to 12-month BP or RE outcomes ($p>.01$), though the anxiety subscale trended towards negative associations with BP domain ($p<.05$). Subscale and total scores on the GHQ-28 at 28-days were not related to any 12-month HRQoL outcomes ($p>.05$).

Table 18 also demonstrates that 6-month HADS total scores continue to significantly negatively relate to most 12-month HRQoL domains and summary scores ($p<.01$). However, though 6-month HADS anxiety scores trend towards significance in relation to HRQoL ($p<.05$), these were only significant for the SF domain and summary scores ($p<.01$). Conversely, 6-month HADS depression scores were significantly negatively related to 12-month physical (i.e., PH, GH) and emotional (i.e., VT, SF) HRQoL scales ($p<.01$), and trended towards significance in relation to both 12-month HRQoL summary scores ($p<.05$). No 6-month HADS scores were related to 12-month BP or RE outcomes ($p>.05$).

Table 18

Correlations between 28-day and 6-month psychological outcomes and 12-month HRQoL

	HADS			GHQ-28				
Short Form 36	Anxiety	Depression	Total score	Somatic symptoms	Anxiety and insomnia	Social dysfunction	Severe depression	Total score
	28-days							
	N=17	N=16	N=16	N=17	N=17	N=17	N=17	N=17
Physical function	-.852**	-.639**	-.828**	.056	-.023	.084	.029	.048
Role physical	-.765**	-.521*	-.726**	.042	-.176	.178	.158	.048
Bodily pain	-.557*	-.094	-.382	.040	-.432	.250	-.107	-.140
General health	-.628**	-.538*	-.641**	.279	.067	-.106	-.084	.049
Vitality	-.786**	-.557*	-.744**	.241	-.173	.192	-.136	.016
Social function	-.815**	-.725**	-.847**	.116	.228	-.125	.045	.114
Role emotional	-.200	.025	-.093	.402	-.256	.161	.039	.102
Mental health	-.700**	-.370	-.602*	.335	-.072	.178	.077	.161
PCS	-.828**	-.528*	-.761**	.110	-.180	.137	.026	.006
MCS	-.715**	-.468	-.653**	.364	-.079	.105	.010	.116
	6-months							
	N=21	N=20	N=20	N=21	N=21	N=20	N=21	N=20
Physical function	-.545*	-.601**	-.635**	-.537*	-.490*	-.305	-.301	-.516*
Role physical	-.535*	-.558*	-.609**	-.526*	-.496*	-.227	-.232	-.518*
Bodily pain	-.337	-.109	-.247	-.457*	-.496*	-.141	.133	-.279
General health	-.505*	-.652**	-.625**	-.411	-.435*	-.175	-.456*	-.450*
Vitality	-.452*	-.568**	-.523*	-.364	-.451*	-.171	-.321	-.498*
Social function	-.624**	-.708**	-.730**	-.602**	-.514*	-.485*	-.632**	-.574**
Role emotional	-.316	-.127	-.243	-.349	-.211	.221	-.141	-.111
Mental health	-.510*	-.332	-.467*	-.435*	-.392	-.072	-.316	-.401
PCS	-.563**	-.560*	-.620**	-.567**	-.562**	-.245	-.240	-.525*
MCS	-.569**	-.512*	-.587**	-.537*	-.462*	-.122	-.425	-.450*

GHQ-28=General Health Questionnaire-28; HADS=Hospital Anxiety and Depression Scale; HRQoL=Health related quality of life; MCS=Mental Component Summary; PCS=Physical Component Summary.
* =p<.05
**=p<.01

Regarding the GHQ-28, all 6-month subscale and total scores were negatively associated with 12-month SF domain ($p<.05$), though these were only significant for somatic symptoms and severe depression subscales and total scores ($p<.01$). This suggests that better psychological functioning at 6-months (lower scores) is associated with better social functioning at 12-months (higher scores). Better 6-months outcomes on the somatic symptoms and anxiety and insomnia subscales (lower scores) were significantly related to better 12-month overall physical HRQoL (higher scores; $p<.01$). No other 6-month GHQ-28 outcomes significantly related to 12-month HRQoL ($p>.01$), though somatic symptoms and anxiety and insomnia subscales and total scores trended towards significant relationships with most HRQoL outcomes ($p<.05$).

Functional Outcomes

Table 19 presents bivariate correlations between 28-day functional outcomes and 12-month HRQoL. No 6-month functional outcomes were associated with 12-month HRQoL at either the $p<.01$ or $p<.05$ significance level, hence they were not included in the table. For further ease of presentation and interpretation, only variables that generated significant results were included in the table. As seen in Table 19, no 28-day functional outcomes were significantly related to 12-month HRQoL ($p>.01$). However, trends towards significance

Table 19

Correlations between 28-day functional outcomes and 12-month HRQoL

	Short Form 36			
Measures(N)	Physical function	Bodily pain	General health	Social function
NIHSS(17)	-.490*	.176	-.519*	-.533*
modified Rankin Scale(21)	.241	.453*	.007	-.029
Barthel Index(21)	.209	-.139	.241	.522*

NIHSS=National Institutes of Health Stroke Scale
* =p<.05
**=p<.01

were found, particularly in relation to stroke symptoms (NIHSS) and several HRQoL domains, where less 28-day stroke symptoms (lower scores) were associated with better 12-month HRQoL (higher scores; $p<.05$).

Section 4: Relationships between 12-Month Neuropsychological, Psychological and Functional Outcomes

In this section, a series of bivariate correlation matrices were generated to examine the degree and direction of relationships between areas of neuropsychological, psychological and functional outcomes 12-months post-SAH. These analyses were conducted for the SAH group only. Due to the number of correlations being generated increasing the possibility of Type 1 error and the small sample size a conservative level of significance was used ($p<.01$). Those significant at the $p<.05$ level are identified, but interpreted with caution.

Neuropsychological and Psychological Outcomes

Table 20 presents Pearson's bivariate correlations generated between 12-month neuropsychological and psychological outcomes. For ease of presentation and interpretation, only variables that generated significant results were included in the table. These correlations indicate that aspects of higher cognitive functioning (i.e., executive function, complex attention and cognitive flexibility CNS-VS domains) significantly negatively correlated with HADS anxiety subscale scores ($p<.01$), and trended towards significance in relation to HADS total scores and GHQ-28 somatic symptoms and anxiety and insomnia subscale scores ($p<.05$), such that better higher cognitive functioning (higher scores) was associated with lower levels of anxiety/insomnia, somatic symptoms and overall emotional difficulties (lower scores). In contrast, better BDS performance (higher total scores) and higher levels of depression (higher HADS depression scores) trended towards significance ($p<.05$). No other

aspects of neuropsychological functioning (other CNS-VS domains, BDS factor scores, MoCA total score) were related to psychological functioning (p>.05).

Table 20

Correlations between neuropsychological and psychological outcomes at 12-months

	HADS			GHQ-28	
Measures(N)	Anxiety	Depression	Total score	Somatic symptoms	Anxiety and insomnia
CNS Vital Signs Domains					
Executive function(10)	-.793**	-.518	-.713*	-.711*	-.745*
Complex attention(10)	-.732*	-.583	-.711*	-.665*	-.695*
Cognitive flexibility(10)	-.775**	-.539	-.714*	-.648*	-.724*
Behavioural Dyscontrol Scale					
Total score(24)	-.067	.416*	.150	.238	.078

GHQ-28=General Health Questionnaire-28; HADS=Hospital Anxiety and Depression Scale.
* =p<.05
**=p<.01

Neuropsychological and Functional Outcomes

Table 21 presents Pearson's bivariate correlations generated between 12-month neuropsychological and functional outcomes. For ease of presentation and interpretation, only variables that generated significant results were included in the table. Better performance on the composite memory domain of the CNS-VS (higher scores) was significantly associated with less stroke symptoms (lower scores on NIHSS; p<.01). Verbal memory trended towards significance in relation to stroke symptoms (p<.05) but no other neuropsychological outcomes related to stroke symptoms (p>.05). No neuropsychological outcomes were associated with level of disability or handicap (mRS, BI; p>.05).

Regarding HRQoL (SF-36), no neuropsychological outcomes were significantly related to scale or summary scores (p>.01). However, several trends towards significance were found (p<.05). Better overall level of neurocognitive functioning (NCI) trended towards association with better VT, but no other HRQoL domains. Aspects of higher

cognitive functioning trended towards associations with better HRQoL in the domains of BP (executive function and cognitive flexibility) and VT (complex attention). Conversely, better performance (higher scores) on the EIF of the BDS (impulsivity) trended towards relations to worse GH outcomes (lower scores). No other aspects of neuropsychological functioning (other CNS-VS domains, BDS outcomes, MoCA total score) were related to functional outcomes (p>.05).

Table 21

Correlations between neuropsychological and functional outcomes at 12-months

			Short Form 36	
Measures(N)	NIHSS	Bodily pain	General health	Vitality
CNS Vital Signs Domains				
NCI(10)	-.256	.567	.317	.639*
Composite memory(10)	-.772**	.407	-.167	.192
Verbal memory(10)	-.735*	.264	-.263	.001
Executive function(10)	.187	.690*	.207	.421
Complex attention(10)	.098	.581	.454	.743*
Cognitive flexibility(10)	.231	.682*	.189	.501
Behavioural Dyscontrol Scale				
EIF(24)	.000	-.133	-.417*	-.194

EIF=Environmental Independence Factor; NIHSS=National Institutes of Health Stroke Scale; NCI=Neurocognition Index.
* =p<.05
**=p<.01

Psychological and Functional Outcomes

Table 22 presents Pearson's bivariate correlations generated between 12-month psychological and functional outcomes. Stroke symptoms (NIHSS) were not significantly related to psychological outcomes (p>.01), though worse stroke symptoms in relation to increased anxiety and insomnia and worse overall psychological wellbeing (GHQ-28) trended towards significance (p<.05). Greater disability (mRS) was significantly associated with higher levels of anxiety, somatic symptoms, and worse overall psychological functioning (HADS and GHQ-28; p<.01).

Table 22

Correlations between psychological and functional outcomes at 12-months

	HADS			GHQ-28				
Measures(N)	Anxiety	Depression	Total score	Somatic symptoms	Anxiety and insomnia	Social dysfunction	Severe depression	Total score
NIHSS(25)	.337	.091	.254	.265	.435*	.106	.311	.406*
mRS(25)	.547**	.370	.518**	.520**	.392	.360	.325	.555**
BI(25)	-.077	.038	-.030	-.100	-.046	-.295	.108	-.106
Short Form 36								
Physical functioning(25)	-.663**	-.671**	-.735**	-.461*	-.445*	-.227	-.413*	-.545**
Role physical(25)	-.547**	-.689**	-.671**	-.530**	-.574**	-.380	-.375	-.658**
Bodily pain(25)	-.685**	-.431*	-.633**	-.721**	-.725**	-.145	.049	-.619**
General health(25)	-.378	-.756**	-.598**	-.297	-.351	-.426*	-.555**	-.532**
Vitality(25)	-.507**	-.589**	-.598**	-.420*	-.480*	-.188	-.397*	-.530**
Social functioning(25)	-.542**	-.724**	-.685**	-.349	-.279	-.367	-.484*	-.485*
Role emotional(25)	-.471*	-.354	-.464*	-.482*	-.653**	-.301	-.560**	-.704**
Mental health(25)	-.660**	-.520**	-.661**	-.518**	-.615**	-.326	-.462*	-.681**
PCS(25)	-.672**	-.753**	-.780**	-.612**	-.642**	-.360	-.373	-.713**
MCS(25)	-.655**	-.667**	-.728**	-.537**	-.616**	-.369	-.598**	-.737**

BI=Barthel Index; GHQ-28=General Health Questionnaire-28; HADS=Hospital Anxiety and Depression Scale; MCS=Mental Component Summary; mRS=modified Rankin Scale; NIHSS=National Institutes of Health Stroke Scale; PCS=Physical Component Summary.

* =p<.05

**=p<.01

Regarding HRQoL (SF-36), better overall psychological wellbeing (lower total scores on HADS and GHQ-28) and less anxiety and depression (lower HADS subscale scores) were significantly associated with better HRQoL (higher scores) overall (PCS and MCS) and in relation to most domains ($p<.01$). Within the subscales of the GHQ-28, less somatic symptoms, and anxiety and insomnia (lower scores) were significantly related to better HRQoL (higher scores) overall (PCS and MCS) and in relation to RP, BP and MH domains ($p<.01$); and trended towards significance in relation to PH, VT and RE domains ($p<.05$). Higher levels of severe depression (higher scores) trended towards significance in relation to worse HRQoL (lower scores) in all emotional domains (VT, SF, MH; $p<.05$) and was significantly related to worse overall emotional HRQoL (MCS), more role limitations due to emotional health (RE), and poorer general health perceptions (GH; $p<.01$). Social dysfunction was not significantly associated with any HRQoL outcomes ($p>.01$).

Relationships amongst Neuropsychological Outcomes

Pearson's bivariate correlations generated to examine the relationships amongst neuropsychological outcomes are presented in Table 23. For ease of presentation and interpretation, only variables that generated significant results were included in the table. Within the CNS-VS, overall neuropsychological functioning (NCI) trended towards ($p<.05$) or was significantly related ($p<.01$) to half of the domain scores, however, was not related to composite nor verbal memory ($p>.05$), though composite memory (based on verbal and visual memory) contributes to NCI score. As expected, composite memory was significantly related to verbal and visual memory ($p<.01$). Processing and psychomotor speed were significantly related ($p<.01$), though neither related to the other speed domain, reaction time ($p>.05$). Interestingly, slower processing and psychomotor speed (lower scores) was significantly associated with better working memory (higher scores; $p<.01$). Several higher cognitive functioning domains (i.e., executive function, complex attention, cognitive

Table 23

Correlations amongst neuropsychological outcomes at 12-months

		CNS Vital Signs Domains									Behavioural Dyscontrol Scale			
	NCI	Composite memory	Visual memory	Processing speed	Executive function	Psychomotor speed	Reaction time	Complex attention	Working memory	Sustained attention	MPF	EIF	FIF	Total score
CNS Vital Signs Domains														
	N=10	N=10	N=10	N=10	N=10	N=10	N=10	N=10	N=9	N=9				
Verbal memory	.343	.910**												
Visual memory	.765**	.858**												
Executive function	.684*	.097	.312	.003										
Psychomotor speed	.795**	.392	.680*	.775**	.364									
Reaction time	.771**	.606	.614	.236	.241	.553								
Complex attention	.685*	.092	.237	.036	.867**	.277	.382							
Cognitive flexibility	.758*	.131	.308	-.021	.950**	.389	.419	.902**						
Working memory	-.661(9)	-.372(9)	-.534(9)	-.895**(9)	-.293(9)	-.810**(9)	-.458(9)	-.219(9)						
Social acuity	-.026	.337	.226	.465	-.186	.249	-.249	-.443	-.389	.748*				
Behavioural Dyscontrol Scale														
	N=10	N=10	N=10	N=10	N=10	N=10	N=10	N=10	N=9	N=9	N=24	N=24	N=23	
MPF	.449	.571	.837**	.517	.178	.509	.198	.079	-.445	.257				
EIF	.186	.119	.412	-.082	.256	.318	.057	-.147	-.074	-.175	.811**			
FIF	-.827**	-.664	-.692*	-.656	-.490	-.748*	-.670*	-.440	.779*	.100	.589**	.598**		
Total score	-.101	-.002	.286	-.124	.019	.062	-.268	-.263	.137	.134	.930**	.881**	.829**	
Montreal Cognitive Assessment														
	N=10	N=10	N=10	N=10	N=10	N=10	N=10	N=10	N=9	N=9	N=24	N=24	N=23	N=23
Total score	.570	.238	.258	.307	.509	.558	.414	.278	-.561	-.215	.508*	.401	.371	.502*

EIF=Environmental Independence Factor; FIF=Fluid Intelligence Factor; MPF=Motor Programming Factor.

* =p<.05

**=p<.01

Note: Parenthese within the table reflect a different N than otherwise indicated.

flexibility) significantly correlated with one another ($p<.01$). Sustained attention and social acuity were not related to any other domains, but trended towards significance in relation to each other ($p<.05$).

Within the BDS, all factor and total scores significantly related to each other ($p<.01$). In relation to the CNS-VS, the MPF was significantly associated with visual memory ($p<.01$). Surprisingly, better FIF performance (higher scores) was significantly associated with worse overall neuropsychological functioning (lower scores on NCI; $p<.01$) and trended towards relations with worse visual memory, psychomotor speed and reaction time performance (lower scores), but better working memory (higher scores; $p<.05$). The EIF and total score were not related to any CNS-VS domains ($p>.05$). No BDS outcomes were related to any of the CNS-VS higher cognitive functioning domains ($p>.05$).

Regarding the MoCA, performance was not significantly related to any CNS-VS domains nor BDS outcomes ($p>.01$), though a trend towards significance in relation to BDS MPF and total scores was found ($p<.05$).

Relationships amongst Psychological Outcomes

Pearson's bivariate correlations generated to examine the relationships amongst psychological outcomes are presented in Table 24. HADS subscale and total scores significantly correlated with each other and with the GHQ-28 total score ($p<.01$). HADS anxiety and total scores significantly related to GHQ-28 somatic symptoms and anxiety and insomnia subscales ($p<.01$), but HADS depression scores only trended towards significance in relation to these subscales ($p<.05$). No HADS outcomes significantly related to GHQ-28 social dysfunction nor severe depression subscales ($p>.01$). Within the GHQ-28, all subscales significantly related to the total score to which they contribute ($p<.01$). The somatic symptoms and anxiety and insomnia subscales were significantly related ($p<.01$), as

Table 24

Correlations amongst psychological outcomes at 12-months

	HADS			GHQ-28			
Measures(N)	Anxiety	Depression	Total score	Somatic symptoms	Anxiety and insomnia	Social dysfunction	Severe depression
HADS							
Depression(25)	.639**						
Total score(25)	.930**	.877**					
GHQ-28							
Somatic symptoms(25)	.710**	.501*	.683**				
Anxiety and insomnia(25)	.779**	.486*	.718**	.767**			
Social dysfunction(25)	.023	.427*	.219	.308	.216		
Severe depression(25)	.095	.360	.232	.124	.161	.572**	
Total score(25)	.646**	.616**	.698**	.835**	.834**	.631**	.543**

GHQ-28=General Health Questionnaire-28; HADS=Hospital Anxiety and Depression Scale.
* =p<.05
**=p<.01

Table 25

Correlations amongst functional outcomes at 12-months

Measures(N)	NIHSS	modified Rankin Scale
modified Rankin Scale(25)	.490*	
Barthel Index(25)	-.055	-.578**(28)
Short Form 36		
Physical function(25)	-.496*	-.620**
Role physical(25)	-.515**	-.382
Bodily pain(25)	-.480*	-.530**
General health(25)	-.176	-.333
Vitality(25)	-.474*	-.408*
Social function(25)	-.158	-.524**
Role emotional(25)	-.418*	-.395
Mental health(25)	-.555**	-.575**
PCS(25)	-.515**	-.535**
MCS(25)	-.467*	-.574**

MCS=Mental Component Summary; NIHSS=National Institutes of Health Stroke Scale; PCS=Physical Component Summary.
* =p<.05
**=p<.01
Note: Parenthese within the table reflect a different N than otherwise indicated.

were the social dysfunction and severe depression subscales ($p<.01$), but no other relationships between subscales were found ($p>.05$).

Relationships amongst Functional Outcomes

Pearson's bivariate correlations generated to examine the relationships amongst functional outcomes are presented in Table 25 (previous page). Correlations amongst SF-36 scale and summary scores were not generated as doing so would have resulted in too many correlations, reducing power to detect real effects. Furthermore, the relationships between scale and summary scores are well established (Brazier et al., 1992; Wade & Sherbourne, 1992).

As seen in Table 25, less stroke symptoms (lower NIHSS scores) trended towards significance in relation to less disability (lower mRS scores) and better HRQoL (higher SF-36 scores; $p<.05$), and was significantly related to better overall physical HRQoL (PCS) and RP and MH domains ($p<.01$). Less disability (lower mRS scores) was significantly related to most HRQoL outcomes ($p<.01$), though was not associated with role limitations due to physical or emotional health (RP, RE) nor general health perceptions (GH; $p>.05$). While the disability measures significantly related with each other (mRS and BI; $p<.01$), the BI did not relate to any other functional outcomes at either level of significance ($p>.01$ and $>.05$).

CHAPTER IV:

DISCUSSION

This study examined short-term (≤12-months) neuropsychological, psychological and functional outcomes of SAH survivors' in comparison to healthy age, gender, and ethnicity matched control participants. Specific aims were to describe SAH survivors' performance on measures of neuropsychological, psychological and functional outcomes at 28-days, 6- and 12-months post-SAH and to identify outcomes on which they differed from matched controls at 6- and 12-months; delineate the natural course of recovery during the first 12-months post-SAH; identify factors related to SAH survivors' HRQoL at 12-months; and examine relationships between 12-month neuropsychological, psychological and functional outcomes for SAH survivors. The findings in relation to each of these are discussed in turn below.

Overall Performance and Group Comparison

Neuropsychological Outcomes

The findings of the current study demonstrate that on average SAH survivors generally performed in the average (CNS-VS) or non-impaired (MoCA) range on neuropsychological outcomes at each time point, as did matched controls. Though average group performance was comparable on many outcomes at each time point, SAH survivors on average performed significantly worse on sustained attention at 6-months and on working memory, aspects of executive functioning (BDS only), and on a cognitive screening measure (MoCA) at 6- and 12-months compared to matched controls. Furthermore, a significantly higher proportion of SAH survivors performed in the impaired range on the cognitive screening measure (MoCA); and in the low average, low and very low ranges on attention,

memory, and higher cognitive functioning domains (CNS-VS) at 6- and 12-months compared to matched controls.

That the SAH group on average performed in the average range and did not significantly differ in comparison to control participants at 6- and 12-months on most domain specific neuropsychological outcomes (CNS-VS) was surprising, particularly given that the domains assessed (i.e., attention, memory, speed, and executive functions) were those most commonly impaired during the first 12-months post-SAH in the literature (Alfieri et al., 2008; Al-Khindi et al., 2010; Cheng et al., 2006; Egge et al., 2005; Hadjivassiliou et al., 2001; Ogden et al., 1993). Those areas of cognition that were found to be significantly worse than controls (i.e., working memory, sustained attention, aspects of executive function) are consistent with the literature (Alfieri et al., 2008; Al-Khindi et al., 2010; Cheng et al., 2006; Egge et al., 2005; Hadjivassiliou et al., 2001; Ogden et al., 1993). Few studies have found little significant differences between SAH cognitive functioning at 6- and/or12-months and standardised norms and/or control participants; those that have based findings on small ($n \leq 24$) hospital-based samples comprised of rare and/or specific SAH mechanisms, and favourable selection bias (e.g., ≤60 years, mild severity, formal education; Germanò et al., 1998; Haug et al., 2009).

A number of factors may have contributed to the current study's results. First, while approximately two thirds of the SAH group completed the BDS and MoCA at each time point, a maximum of one third of the SAH group completed the CNS-VS, the primary neuropsychological measure, at each time point. The main reasons for non-completion were fatigue and unwillingness to participate. This suggests that the test battery required too much time and energy of participants and/or that the method of administration (computer-based) may have been off-putting. The resulting small sample size (n=8 to 10) likely reduced ability to detect real effects on this measure (CNS-VS). It is also possible that those participants

who did not participate experienced more severe outcomes, possibly resulting in an underestimation of the severity of impairment. These hypotheses may be supported by the fact that on both measures completed by most of the SAH group (BDS and MoCA), the SAH group's performance on average was significantly worse compared to matched controls. Previous research comparing the use of cognitive screening measures such as the MoCA to neuropsychological test batteries has consistently found that screening tools under-identify neuropsychological deficits (Cao et al., 2007; Cederfeldt et al., 2010), further supporting the hypothesis that more significant differences may have been found had more SAH survivors completed the CNS-VS.

That the SAH group performed on average significantly worse than controls on executive functioning as assessed by the BDS but not on any higher cognitive functioning domains of the CNS-VS could be due to more impaired SAH survivors not completing the CNS-VS, as described above. However, it could also demonstrate that the two measures were assessing different aspects of higher cognitive functioning. Thus, it could be concluded that SAH survivors on average were impaired compared to controls regarding ability to modify behaviour based on feedback (FIF on BDS), but not on reasoning, decision-making, strategy formation, and ability to sequence, manage and switch tasks (executive function and cognitive flexibility domains on CNS-VS). Different findings based on different measures are reflected throughout the literature (Alfieri et al., 2008; Al-Khindi et al., 2010; Cheng et al., 2006; Egge et al., 2005; Hadjivassiliou et al., 2001; Mayer et al., 2002; Ogden et al., 1993) and support the need to employ comprehensive neuropsychological test batteries, particularly when assessing broad and complex cognitive functions.

Whether performing significantly worse in comparison to controls or not, the current findings still show that, on average, the SAH group's performance was generally in the average range, which is much more optimistic than previous findings would suggest (Alfieri

et al., 2008; Al-Khindi et al., 2010; Cheng et al., 2006; Egge et al., 2005; Hadjivassiliou et al., 2001; Ogden et al., 1993). Previous research that thoroughly assesses neuropsychological outcomes via test battery have employed hospital- and clinic-based samples, which often result in exclusion of SAH survivors with both good and severe outcomes (Cheng et al., 2006; Fontanella et al., 2003; Ogden et al., 1993; Samra et al., 2007). Thus, it is possible that the population-based sample used in the present study provides a more inclusive, and therefore balanced, account of SAH survivors' outcomes, where on average their neuropsychological outcomes may be less impaired than previously demonstrated.

Another possibility could be that the SAH group participants were very high functioning prior to their SAH, thus significant deterioration in neuropsychological functioning may have occurred but would not be evident by their normative data description range (e.g., average) or compared to controls. Though the control group was matched on key demographic characteristics and did not differ significantly from the SAH group on education, factors known to confound neuropsychological assessment performance; between-group differences in pre-SAH cognitive functioning cannot be excluded. Perhaps, as some research suggests, greater levels of neuropsychological impairment post-SAH may have been found had performance been compared to estimates of premorbid functioning instead (Berry et al., 1997).

In saying that, examination of the proportion of SAH survivors performing in the impaired and below average ranges compared to both controls and the normal curve (CNV-VS) demonstrated that while average between-group differences were few, there were significant between-group differences in a qualitative, clinical sense.

The cognitive domains of attention, memory, and higher cognitive functioning (CNS-VS) in which high proportions of SAH survivors performed poorly is consistent with the literature, though was found at much lower proportions in this study (Alfieri et al., 2008; Al-

Khindi et al., 2010; Cheng et al., 2006; Egge et al., 2005; Hadjivassiliou et al., 2001; Ogden et al., 1993). For example, Egge and colleagues (2005) reported that 93% of their SAH sample experienced significant decline in some domain of cognitive functioning at 12-months. The differences in results compared to the current study could reflect different sample selection criteria (hospital- versus population-based) and the use of different measures. Unfortunately, no other study employed the CNS-VS making direct comparison of findings difficult.

The proportion of SAH survivors performing in the impaired range on the cognitive screening measure (MoCA) in this study is comparable to other population-based studies, which range from 22% to 39% between 3- and 12-months, though these used stroke samples and employed different measures (i.e., MMSE; Dourir et al., 2013; Lisabeth et al., 2014; Patel et al., 2003). Studies on SAH samples using the MoCA report higher proportions of impairment than that found here, ranging from 42% to 73% (Schweizer et al., 2012; Wong et al., 2012); which may reflect their use of hospital- rather than population-based samples, effectively over-estimating their findings.

Finally, some authors have suggested a bimodal distribution of SAH cognitive outcomes (Haug et al., 2010; Hillis et al., 2000). Though the sample size and analysis conducted in this study is not adequate to conclude the presence of a bimodal distribution, it provides some support for this hypothesis. That is, most of the SAH group performed in the average or above average (CNS-VS) and non-impaired ranges (MoCA), indicating good neuropsychological outcomes, but up to one-third of them were doing poorly at 6- and 12-months.

Psychological Outcomes

The current study found that on average both groups generally scored in the “normal” range on psychological outcomes at each time point, though the SAH group had significantly

more depressed symptoms (HADS), somatic symptoms (GHQ-28), and reported significantly greater overall emotional distress and poorer psychological health and wellbeing (HADS and GHQ-28) compared to controls at 6- and 12-months post-SAH. This finding is consistent with but adds to the literature (Powell et al., 2002), through supporting these outcomes in a population-based sample. In the current study, compared to controls, a significantly higher proportion of the SAH group also reported anxiety, depression, and overall emotional distress and poor psychological wellbeing in the clinical range at 6- and 12-months post-SAH.

Hackett and Anderson (2000) indicated that 39% of their population-based sample reported overall mood difficulties 12-months post-SAH. The current study is consistent with this finding, in that 36% of SAH survivors reported poor overall psychological wellbeing in the clinical range at 6- and 12-months. The current study supports Hackett and Anderson's findings through the use of commonly used, validated and reliable measures (HADS and GHQ-28), as compared to their use of a brief and informal measure (telephone interview asking a series of simple yes/no questions).

The proportion of the SAH group reporting depression in the clinical range in the current study (18% and 16% at 6- and 12-months, respectively) falls towards the lower end of the range reported in the literature (Al-Khindi et al., 2010; Hillis et al., 2000; Passier et al., 2010). This discrepancy is likely due to differences in samples (hospital- and clinic-based versus population-based) and measures used. In particular, those measures that are observer-based tend to report higher proportions of depression as compared to self-report measures, such as those used in the current study (Herrmann et al., 1998; Kauhanen et al., 1999).

That the SAH group reported significantly more depressive symptoms compared to controls on the depression subscale of the HADS but not the severe depression subscale of the GHQ-28 likely reflects the different content and level of depression assessed by these measures. While HADS depression items tap depressive symptomology (e.g., "I feel

cheerful"), GHQ-28 severe depression items target hopelessness, self-harm, and suicidal ideation explicitly (e.g., "Have you recently felt that life isn't worth living?"). Thus, it seems reasonable to interpret this finding as SAH survivors being significantly more depressed than controls (HADS), but not significantly more likely to be experiencing the severe end of hopelessness, self-harming, or suicidal ideation (GHQ-28). Similar findings have been reported in other studies using different measures of depression as compared to the GHQ-28 in SAH samples (Gill, 2010).

That the SAH group on average did not differ significantly in comparison to controls on the primary anxiety measure (HADS anxiety subscale) was surprising as previous research demonstrates that anxiety post-stroke and -SAH is common (Al-Khindi et al., 2010; Barker-Collo, 2007; Passier et al., 2010). However, the SAH group were significantly more likely to report anxiety in the clinical range at 6- and 12-months compared to controls. As was the case with neuropsychological outcomes, this demonstrates that while on average quantitative between-group differences were not found, there were marked between-group differences in a qualitative, clinical sense. In fact, twice as many SAH survivors in this study would be classified as experiencing anxiety in the clinical range as would be classified as experiencing depression in the clinical range at 6- and 12-months (HADS), despite average group comparisons suggesting the reverse. The proportion of SAH survivors experiencing anxiety in the clinical range in this study (39% and 32% at 6- and 12-months, respectively) falls within the upper end of the range reported in the literature (Al-Khindi et al., 2010; Passier et al., 2010; Powell et al., 2002), likely due to reasons cited in regards to depression (i.e., different samples and measures).

Functional Outcomes

The SAH group on average had minor stroke symptoms and good outcomes regarding disability at each time point, but were significantly more disabled regarding basic and

instrumental ADLs (mRS) compared to matched controls at 6- and 12-months. Significantly reduced HRQoL on average and significantly greater proportions of impaired HRQoL outcomes in comparison to controls were evident at 6- and 12-months post-SAH.

It was unexpected that the SAH group had very minor stroke symptoms throughout the first 12-months post-SAH. In contrast, Lawrence and colleagues (2001) reported that nearly their entire population-based sample had some impairment during the acute phase post-stroke, with over 50% experiencing 6 to 10 impairments. Research demonstrates that older age, particularly >70 years, is associated with greater neurological impairment post-stroke (Di Carlo et al., 1999; Irie et al., 2015); thus, one possible interpretation of the current finding could be that few stroke symptoms were found due to the younger age of SAH survivors in general, and in this study in particular ($\bar{x}$=52 years), compared to other stroke subtypes. However, research looking at the SAH subtype also suggests that higher levels of neurological impairment would be expected, though this research was not population-based (Powell et al., 2002). Another explanation could be that SAH survivors with more severe neurological impairments were less likely to participate in this study. The SAH sample did not differ in stroke severity from the original SAH population as assessed by the GCS, though this measure does not provide a comprehensive assessment of neurological impairment (Middleton, 2012).

While the literature is reasonably conclusive that stroke and SAH survivors experience considerable disability and handicap compared to norms and control samples (Al-Khindi et al., 2010; Barker-Collo & Feigin, 2006; Cao et al., 2007; Cederfeldt et al., 2010), the findings in the current study generally suggest otherwise, with average disability outcomes falling in the good outcome range and SAH survivors being no more likely to be classified as impaired compared to controls at 6- and 12-months. However, the current findings are consistent with those reported in a comprehensive review of the literature by Al-

Khindi and colleagues (2010), where only 4% to 12% of SAH survivors experienced deficits in basic ADL as assessed by various measures, including the BI which was employed in the current study. Higher rates of disability post-SAH reported in the literature likely reflect the measure used, particularly if it assesses instrumental ADL which are impaired in up to 93% of SAH survivors (Al-Khindi et al., 2010). That the mRS includes some instrumental ADL while the BI is limited to basic ADL likely accounts for the slightly higher proportions of SAH survivors classified as having poor outcomes on the mRS as compared to the BI in the current study, as well as the significant between-group difference on average on the mRS at 6- and 12-months, but not the BI. The current study adds to the literature in that these outcomes were supported in a population-based sample at 6- and 12-months post-SAH.

Reduced HRQoL during the first 12-months post-SAH is suggested in the literature, though this is sometimes limited to specific areas of reduced HRQoL (Al-Khindi et al., 2010; Meyer et al., 2010; Noble et al., 2008). Particularly noteworthy, Hackett and Anderson (2000) found that SAH survivors reported significantly reduced HRQoL at 12-months in the domain of role limitations due to physical functioning only, compared to Australian population norms. Conversely, the current study's findings were much broader in that SAH survivors reported on average significantly reduced HRQoL and were significantly more likely to report HRQoL in the impaired range across all domains at 6- and 12-months compared to controls. These results are more comparable to those of Katati and colleagues (2007), who concluded that on each HRQoL domain at least 20% of their hospital-based SAH sample were impaired, most commonly on domains of role limitations due to physical functioning and vitality, as was the case at both time points in the current study. While Hackett and Anderson's sample was population-based, their sample was limited to first-ever stroke while the current sample included previous stroke victims. This could contribute to the broader findings in the current study, as previous stroke was found to relate to worse

outcomes on several HRQoL domains in the current sample. Comparison to population norms rather than matched control participants also likely contributed to the differences in results; thus this study adds to the literature in this respect. Furthermore, Hackett and Anderson employed the acute SF-36 (assesses HRQoL over the prior week only), whereas the standard version employed in this study (and by Katati and colleagues) refers to a 4-week frame of reference, enabling a broader assessment.

Interestingly, while the SAH group on average reported significantly worse outcomes on the social functioning domain on the SF-36 at 6- and 12-months compared to controls, they were only significantly worse compared to controls on the social dysfunction subscale of the GHQ-28 at 6-months. This likely reflects that the two measures include different items aimed at assessing slightly different things. While the SF-36 measures the extent and amount of time to which physical and emotional health has interfered with one's social activities, GHQ-28 items relate to the manner in which social tasks are undertaken (e.g., being occupied, doing things well, task satisfaction, indecisiveness).

Summary

While the SAH group on average performed in the average or normal range on most outcomes throughout the first 12-months post-SAH, their outcomes were generally significantly worse compared to matched controls at 6- and 12-months. Furthermore, not only were they doing poorly compared to controls on average, but significantly greater proportions actually performed in the impaired range, highlighting clinical concern. This is particularly the case for psychological and HRQoL outcomes, despite the SAH group having minor stroke symptoms and good disability outcomes. Some evidence was found in regards to poor neuropsychological outcomes, particularly on a cognitive screening measure and in reference to proportions performing in the lower qualitative ranges and it may be reasonable

to hypothesise that this would have been further supported had a larger sample size completed the primary measure.

Change Over Time

Neuropsychological Outcomes

The findings of the current study demonstrate that the SAH group's neuropsychological functioning did not significantly change during the first 12-months post-SAH on most outcome measures, though performance significantly improved on a cognitive screening measure (MoCA) from 28-days to 6-months. This improvement is somewhat consistent with the stroke and SAH literature, though other authors report improvement on screening measures throughout the first 12-months post-stroke and -SAH (Mocco et al., 2006; Patel et al., 2003; Springer et al., 2007). However, Samra and colleagues (2007) found that improved cognitive functioning plateaued at 9-months post-SAH. Thus, perhaps had previous research used more regular follow-up assessments, a plateauing of cognitive outcomes similar to that found in the current study may have been identified at an earlier time point.

That no other significant changes in any other neuropsychological outcomes were found was surprising, particularly during the 28-day to 6-month period given that the least sensitive measure (MoCA) yielded a significant improvement. As discussed previously, the small sample size (n=4 to 9) available for within-subject comparisons on CNS-VS domains likely reduced ability to detect real effects. However, this explanation is less likely in respect of the BDS as sample sizes comparable to the MoCA were available for within-subject comparisons.

Alternatively, given that, to the researchers knowledge, this is the first population-based study delineating the trajectory of neuropsychological outcomes (test battery) during

the first 12-months post-SAH, it is possible that there was actually little change in domain specific cognitive functioning during this period, contrary to the literature (Alfieri et al., 2008; Haug et al., 2007; Ogden et al., 1993; Samra et al., 2007).

However, further exploration of the findings (CNS-VS Figures) in the current study revealed much variability within the 28-day to 6-month and 6- to 12-month trajectories for different participants on neuropsychological domains, further suggesting that the small sample size limited ability to detect an overall effect. On most domains, an overall trend towards stable or declining performance was observed from 28-days to 6-months, whereas stable or improved performance was more likely from 6- to 12-months. Exceptions were memory domains, some of which (composite, visual) showed the reverse overall trend (initial improvement followed by later decline), others a much more variable trajectory (verbal). In contrast, the working memory domain demonstrated a trajectory trend similar to those described for most domains, demonstrating that different aspects of the same broad cognitive domain have different trajectories. Haug and colleagues (2007) also found that cognitive domains have different short-term trajectories, as do different aspects of the same broad cognitive domain, consistent with the trends observed in this study; though the trajectory of particular domains differed to those found here (e.g., visual memory improved steadily compared to improvement then decline). Differences in findings may be due to hospital- versus population-based samples, greater exclusion criteria, and different assessment points and measures used. Nonetheless, the current study's observed trajectory trends must be interpreted with much caution given that these were not statistically significant results.

One final point of interest was how similar each SAH survivor's performance and trajectory was across higher cognitive functioning domains in particular (executive function, complex attention, cognitive flexibility on CNS-VS). Most research reports executive functioning as one domain (Alfieri et al., 2008; Cheng et al., 2006; Mayer et al., 2002; Passier

et al., 2010), but those that report on specific aspects of this complex cognitive domain find different trajectories. For example, Haug and colleagues (2007) found that inhibition showed gradual improvement during the first 12-months post-SAH, while cognitive flexibility did not improve at all. The present observation could reflect these three domains being generated by performance on some of the same CNS-VS tests, though the domains also draw on different tests and raw score equations. However, were this the case, it may suggest that the CNS-VS is not a sensitive measure in respect to different aspects of higher cognitive functioning, though research suggests otherwise (Gualtieri & Johnson, 2005). Alternatively, it could reflect the interconnectedness of these cognitive domains, where performance in one domain is associated with performance in the others. This possibility is supported in the current study, as performance on each of these three higher cognitive functioning domains was significantly related at 12-months post-SAH in this sample.

Psychological Outcomes

Though the SAH group continued to have poorer psychological outcomes throughout the 12-months post-SAH compared to controls as previously discussed, significant reductions in depressive symptoms (HADS), somatic symptoms and social dysfunction (GHQ-28), and overall emotional distress (HADS) from 28-days to 6-months were evident. Longitudinal research assessing the trajectory of psychological outcomes post-stroke and -SAH is limited, however, generally improved psychological functioning throughout the first 12-months is reported (Herrmann et al., 1998; Kauhanen et al., 1999; Kreiter et al., 2013). That the current study found initial improvement followed by a plateau in psychological outcomes while previous research reports improvement throughout this period may be accounted for by their use of fewer assessment points (e.g., 3- to 12-months), which may have simplified the trajectory of their outcomes (Herrmann et al., 1998; Kauhanen et al., 1999; Kreiter et al., 2013). Alternatively, the findings could differ due to the use of hospital-based and generally

stroke (rather than SAH) samples and different assessment measures. The current findings are somewhat supported by Powell and colleagues (2002), who found that depressive symptomology as assessed by the HADS decreased from 3- to 9-months in their SAH sample. Unfortunately, these authors did not conduct a later assessment point which would have allowed comparison of the continued trajectory of these outcomes, and was limited by its sample (hospital-based, good grade neurological outcomes). Thus, the current study adds to the literature in that as far as the researcher is aware it is the first population-based study to delineate the trajectory of psychological outcomes post-SAH using several assessment points across the first 12-months.

The SAH group's anxiety outcomes did not change significantly during the first 12-months post-SAH, which is consistent with the stroke and SAH literature (Powell et al., 2002; Rasquin et al., 2005). This is concerning as, though the SAH group did not differ significantly on average compared to controls, they were significantly more likely to report anxiety in the clinical range, as previously discussed.

Finally, it is interesting to note that the SAH group improved on the HADS depression subscale and total score from 28-days to 6-months, but not on the GHQ-28 severe depression subscale or total score. As previously discussed, this likely demonstrates that the two measures include different content and assess different aspects and severity levels of psychological functioning. Regarding the depression outcomes, this may suggest that SAH survivors are less depressed at 6-months as compared to 28-days (HADS), though severe levels of depression (hopelessness, self-harming, suicidal ideation) are more persistent (GHQ-28). Regarding the overall (total score) outcomes, HADS items assess anxiety and depression only whereas GHQ-28 items include somatic symptoms and social dysfunction, perhaps suggesting that broader, more general psychological wellbeing (GHQ-28) is less

likely to improve as compared to clinically defined emotional distress such as depression (HADS).

Functional Outcomes

While disability significantly improved from 28-days to 6-months post-SAH, stroke severity significantly worsened from 6- to 12-months. Outcomes on most HRQoL domains did not change significantly during the first 12-months post-SAH though, consistent with improvement in disability outcomes, overall physical HRQoL (PCS) and several related physical domains improved significantly from 28-days to 6-months.

Though remaining in the minor stroke symptoms range, the worsening of SAH survivors' stroke symptoms from 6- to 12-months was puzzling. The maximum deterioration was by 3 points on the 42 point scale (n=2), though most of those whose stroke symptoms deteriorated did so by only 1 to 2 points (n=6). The most common areas to deteriorate in this sample were sensory abilities, visual fields and facial paralysis. Somewhat consistent with the current study's findings, moderate and severe stroke symptoms have been shown to decrease from discharge to 12-months post-stroke, while minor stroke symptoms have increased during this time (Cederfeldt et al., 2010).

Though some research reports that disability post-stroke remains stable or deteriorates during the first 12-months (Kwok et al., 2006; Ullberg et al., 2015), most previous research reports significant improvement in disability during this period, consistent with the current study. Though based on an elderly (≥65 years), hospital-based stroke sample, Cederfeldt and colleagues (2010) reported findings similar to the current study, where disability (BI) improved significantly from discharge to 6-months, though not from 6- to 12-months, suggesting a plateau in disability outcomes. Mocco and colleagues (2006) reported improved disability (BI) from discharge to 3-months and 3- to 12-months in their hospital-based SAH

sample, though it is possible that an earlier plateau may have been identified had their second follow-up assessment not spanned 9-months.

Significantly improved HRQoL over the first 12-months post-SAH has been documented, particularly in physical, emotional and social functioning domains (Al-Khindi et al., 2010). For example, Mocco and colleagues (2006) found significantly improved HRQoL overall and on physical and psychosocial subscales (SIP) from 3- to 12-months post-SAH, though using a hospital-based, severe stroke symptom sample. However, stable or even deteriorating HRQoL in relation to specific domains such as recreational activities, energy, and general health has also been reported during the first 12-months post-SAH (Al-Khindi et al., 2010; Noble et al., 2008). This is somewhat more consistent with the current findings, where most HRQoL outcomes did not significantly change during the first 12-months post-SAH. Though they completed assessments at different time points (4- and 18-months) and used a hospital-based SAH sample, Hop and colleagues (2001) reported strikingly similar findings, where the only HRQoL domains to improve significantly were physical functioning and role limitations due to physical functioning (they did not report summary scores such as the PCS), though these outcomes remained significantly impaired compared to normative data, as the current SAH sample's HRQoL outcomes did compared to controls.

Summary

Overall, improvement in some psychological and functional outcomes occurred between 28-days and 6-months post-SAH but plateaued from 6- to 12-months, and SAH survivors outcomes typically remained significantly impaired compared to controls. While there was no significant change in domain specific neuropsychological outcomes during the first 12-months post-SAH, initial significant improvement on a cognitive screening measure (MoCA) and the variability and trends observed on the primary neuropsychological outcome measure (CNS-VS Figures) suggest that more change may have been identified had a larger

sample completed this measure. That there was no significant change in anxiety outcomes and on most HRQoL outcomes is concerning, particularly as these outcomes are those which SAH survivors were found to have the poorest outcomes in comparison to controls both on average and in regards to proportions falling in the clinical/impaired range.

Factors Related to 12-Month HRQoL

Demographic Factors

Age, gender, ethnicity and education were not significantly related to 12-month HRQoL post-SAH in the current study. Though the literature relating demographic characteristics to HRQoL reports variable findings, the general consensus appears to be that increasing age and female gender in particular are related to worse HRQoL outcomes post-SAH, contrary to the current findings (Katati et al., 2007; Meyer et al., 2010; Passier et al., 2013). However, in their meta-analysis Noble and Schenk (2010) concluded that age and gender had "negligible" (p. 779) effects on HRQoL post-SAH, accounting for a minimal degree of variance explained. Perhaps this study being a meta-analysis, which potentially draws together a more representative overall sample than any individual hospital-based study, explains the similarity of its findings to those of the current population-based study. Regardless, the current findings are somewhat hopeful as, though relevant demographic factors can help direct who receives intervention resources, they are static in nature thus provide no guidance as to what factors interventions should target to improve outcomes.

Injury Characteristics

Though clipping versus coiling post-SAH has not previously been related to HRQoL outcomes (Al-Khindi et al., 2010; Meyer et al., 2010; Proust et al., 2009), clipping was significantly associated with worse physical HRQoL outcomes (PCS and all contributing domains) 12-months post-SAH while coiling was associated with significantly better

outcomes regarding vitality in the current study. Research does demonstrate, however, an association between clipping and poor disability outcomes (Mortimer et al., 2014), perhaps offering some support of its relationship with 12-month physical HRQoL domains in particular in the current study. Previous findings have also used limited sample selection; for example, Proust and colleagues (2009) used a hospital-based sample including aneurysmal SAH on anterior communicating artery only, thus the current study's results likely also reflect the use of a population-based sample inclusive of all SAH mechanisms and locations.

Another injury characteristic significantly associated with worse 12-month HRQoL outcomes post-SAH in the current study was previous stroke, particularly in respect of emotional HRQoL (MCS and several contributing domains). While this finding seems to make logical sense, comparison to the literature is difficult as previous outcome studies post-SAH use first-ever stroke samples (Hackett & Anderson, 2000; Haug et al., 2009; Sturm et al., 2004) or have not examined or reported this potential relationship (Katati et al., 2007; Kreiter at al., 2013; Meyer et al., 2010). Thus, the impact of previous stroke on 12-month HRQoL using a population-based SAH sample is another finding adding to the literature.

Neuropsychological Outcomes

In the current study few neuropsychological outcomes at 28-days and/or 6-months post-SAH were significantly associated with 12-month HRQoL, with better performance on several higher cognitive functioning domains (executive function, complex attention, cognitive flexibility on CNS-VS) at 28-days significantly related to better HRQoL in the domain of bodily pain only. In contrast, the literature reports relationships between both cognitive screening measures and specific cognitive domains with HRQoL post-stroke and -SAH (Bays, 2001; Haug et al., 2010; Hochstenbach et al., 2001; Mayer et al., 2002; Springer et al., 2009). Specific cognitive domains have included executive functioning (Bays, 2001; Hochstenbach et al., 2001), thus the current findings are consistent with the literature in this

respect. However, somewhat similar to the current findings, Mayer and colleagues (2002) reported that while several cognitive domains (e.g., visual memory, psychomotor) significantly related to other functional outcomes (e.g., disability) 3-months post-SAH, no specific cognitive domains, including higher cognitive functioning, related to HRQoL outcomes.

As discussed previously, the lack of significant findings could reflect the small sample size that completed the primary neuropsychological measure (CNS-VS), though this would not account for the two other measures (BDS, MoCA). Differences in findings are perhaps more likely due to the use of hospital-based samples and associations based on concurrent outcomes in previous studies (Haug et al., 2010; Springer et al., 2009), compared to the population-based sample and exploration of relationships between outcomes at different time points in the current study.

Psychological Outcomes

Consistent with the literature which generally supports an association between psychological outcomes and HRQoL post-stroke and -SAH (Bays, 2001; Kreiter et al., 2013; Kutlubaev & Hackett, 2014; Kwok et al., 2006; Passier et al., 2013), psychological outcomes at 28-days and 6-months were significantly related to 12-month HRQoL overall (both summary scores) and on most domains in the current study. This was particularly the case with psychological outcomes as assessed by the HADS at both time points, whereas outcomes on the GHQ-28 were not significantly associated with any 12-month HRQoL outcomes as assessed at 28-days and only associated with a couple of domains as assessed at 6-months. This was unexpected, as the GHQ-28 provides a more general measure of psychological health and wellness which may capture outcomes similar to those reflected in aspects of HRQoL, and is in fact itself occasionally used as a measure of HRQoL (Haug et al., 2010). Perhaps the current finding can be interpreted as demonstrating that early clinical

psychological outcomes such as those assessed by the HADS (e.g., anxiety, depression) are particularly related to 12-month HRQoL post-SAH, more so than early indications of more general psychological ill health and wellbeing.

In particular, the HADS anxiety as opposed to the depression subscale was more often significantly related to 12-month HRQoL outcomes as assessed at 28-days and 6-months in the current study. As has been discussed throughout, previous research has focused on depression post-stroke and -SAH in general and particularly as it relates to HRQoL (Kreiter et al., 2013; Kutlubaev & Hackett, 2014; Kwok et al., 2006). The current study again demonstrates the importance of and need for an increased focus on anxiety outcomes post-SAH.

Functional Outcomes

Stroke symptoms and disability 28-days and 6-months post-SAH were not significantly related to 12-month HRQoL outcomes in this study, contrary to previous research (Katati et al., 2007; Meyer et al., 2010; Passier et al., 2013). The SAH sample in the current study had on average good outcomes regarding stroke symptoms and disability at each assessment, thus it is possible that significant associations between these outcomes and HRQoL would have been found had these functional outcomes been more problematic. In saying that, the literature does also report survivors with good stroke symptoms and disability outcomes having considerable reductions in HRQoL (Noble & Schenk, 2010). This is consistent with the current findings and contributes to an understanding of the lack of significant relationships between these functional outcomes, which is clearly complicated and mediated by various other factors.

Summary

Surgical clipping, previous stroke, and worse psychological outcomes at 28-days and 6-months were significantly associated with worse HRQoL outcomes 12-months post-SAH,

though demographic characteristics, most neuropsychological outcomes, and functional outcomes were not in the current study. Consistent with the literature (Kreiter et al., 2013; Kwok et al., 2006), these findings demonstrate the relevance of psychological outcomes in relation to HRQoL post-SAH, perhaps more so than other outcome areas, though a causal relationship cannot be claimed.

Relationships between 12-Month Neuropsychological, Psychological and Functional Outcomes

Few neuropsychological outcomes were related to psychological or functional outcomes assessed concurrently 12-month post-SAH in the current study. Those significant findings did suggest that better performance on several higher cognitive functioning domains (executive function, cognitive flexibility on CNS-VS) was associated with less anxiety (HADS), while better memory abilities (composite memory domain on CNS-VS) was associated with less severe stroke symptoms (NIHSS). Conversely, the post-stroke literature reports quite consistent and strong relationships between both neuropsychological and psychological (Anderson et al., 1995; Barker-Collo, 2007; Brodaty et al., 2007; Kauhanen et al., 1999; Pohjasvaara et al., 2002; Robinson, 2003), and neuropsychological and functional outcomes (Barker-Collo & Feigin, 2006; Franceschini et al., 2010; Hochstenbach et al., 2001; Patel et al., 2002; Patel et al., 2003), inclusive of the significant findings reported here. However, the SAH literature is comparatively limited and reports quite varied results, all of which use hospital-based samples and employ various exclusions (e.g., >60 years, poor neurological grade, non-aneurysmal SAH only; Alfieri et al., 2008; Berry et al., 1997; Mayer et al., 2002; Powell et al., 2002; Springer et al., 2009). The general lack of significant associations of neuropsychological with psychological and functional outcomes 12-months post-SAH in this study should be considered with caution due to the small sample size (CNS-

VS), but nonetheless, suggests that neuropsychological outcomes post-SAH may not associate with psychological and functional outcomes to the extent demonstrated in the general stroke literature.

Regarding relationships between psychological and functional outcomes at 12-months post-SAH, psychological outcomes were not significantly related to stroke symptoms (NIHSS). However, worse psychological outcomes (HADS and GHQ-28 total score) were significantly associated with worse disability outcomes (mRS) in the current study. The literature generally reports significant relationships between psychological outcomes and both of these functional outcomes, though has usually been based on hospital-based stroke samples and use of different measures and cut-off points, likely limiting its applicability to SAH survivors (Brodaty et al., 2007; Farner et al., 2010; Herrmann et al., 1998; Kutlubaev & Hackett, 2014; Townend et al., 2007; West et al., 2010). For example, using a hospital-based stroke sample Dennis and colleagues (2000) concluded that higher levels of depression were associated with higher levels of disability, though less of a relationship was reported between anxiety and disability. Conversely, the current study found the opposite where higher levels of anxiety (HADS) were significantly related with higher levels of disability (mRS), while depression (HADS) was not. This again highlights the relevance of anxiety post-SAH, and demonstrates the importance of population-based, stroke subtype-specific samples.

However, congruent with the findings and discussion in the previous section, psychological outcomes (HADS and GHQ-28) were significantly related to HRQoL overall (both summary scores) and on most domains as assessed at 12-months post-SAH in the current study. Of particular note, the GHQ-28 (total score) now significantly related to most HRQoL outcomes, demonstrating its utility when assessed concurrently with HRQoL, which is more consistent with assessment procedure and findings in the literature (Haug et al., 2009; West et al., 2010).

Summary

Few significant relationships between 12-month outcomes were found in the current study. Though these results should be considered with some caution due to the small sample size, particularly regarding neuropsychological outcomes, this demonstrates the importance of population-based and stroke subtype-specific studies to dis/confirm findings on the particular population of interest. In saying that, psychological outcomes continued to significantly relate to HRQoL outcomes as assessed at 12-months post-SAH, as well as concurrent disability outcomes. This continues to demonstrate the relevance and interconnectedness of these outcomes post-SAH, though a causal relationship cannot be concluded.

Clinical Implications

The clinical utility of this study primarily concerns the findings in relation to the psychological and HRQoL outcomes experienced by SAH survivors throughout the first 12-months post-SAH. On average, SAH survivors experienced worse outcomes compared to controls and were more likely to be reporting psychological outcomes in the clinical range and HRQoL outcomes in the impaired range. Though some improvement was noted during the first 6-months post-SAH, these outcomes plateaued during the second 6-month period, with SAH survivors remaining worse off than controls. This was despite seemingly good outcomes regarding more traditional and obvious markers of stroke outcomes such as stroke symptoms and disability.

These findings suggest that psychological and HRQoL outcomes should be repeatedly assessed throughout the first 12-months post-SAH, with psychological and psychosocial interventions available and offered as appropriate on an individual basis. This is particularly the case 6-months onwards post-SAH to encourage and support further improvement in these

outcomes which were shown to plateau thereafter. This is an especially important consideration given that over time, the availability of both formal and informal resources and supports typically reduce or are withdrawn all together, contrary to the evident need.

Ideally, psychological and HRQoL assessment should be established as a routine component of follow-up care for all SAH survivors, regardless of their stroke symptoms and disability outcomes, perhaps provided by primary health care providers over the long-term. The utility of the HADS and the SF-36 in particular were demonstrated in the current study through their ability to differentiate between participants with poor and good outcomes, and would be recommended for future use with this population. If using other psychological measures, it is recommended that one including assessment of anxiety, as well as depression, be employed given the incidence and relevance of this particular psychological outcome in this study. Intervention funding within the health system can be difficult to procure; proposals may be strengthened by highlighting the possibility of interventions resulting in broader outcome improvements than what they may target directly given the relationships identified between psychological and HRQoL outcomes throughout the first 12-months post-SAH. However, a causal relationship was not established, thus this rational should be used with caution.

In particular, the current findings indicated that SAH survivors who have had a previous stroke and/or underwent surgical clipping during acute SAH intervention had worse emotional and physical HRQoL outcomes, respectively, compared to those survivors who had not. It is therefore recommended that SAH survivors for whom these factors are relevant be prioritised for assessment and intervention and/or support if resource limitations necessitate prioritisation.

While limited conclusions and therefore recommendations regarding neuropsychological outcomes can be made from the current findings, the utility of the MoCA

as a cognitive screening tool was demonstrated and is recommended for use in this population. Approximately one third of the current sample were identified as impaired on this measure throughout the first 12-months post-SAH, thus further neuropsychological testing using a comprehensive test battery is still recommended to identify domain specific cognitive impairments to direct individualised intervention and/or support. In particular, higher cognitive functioning emerged as impaired and was related to several psychological and HRQoL outcomes, thus assessment and intervention should include particular focus on higher cognitive functioning domains. Furthermore, the high degree of variability in individual participants' trajectories suggests that a one off neuropsychological assessment is not likely to be adequate, rather multiple assessments are required. Use of the CNS-VS cannot be recommended as a test battery of choice given that most participants in the current study did not complete this measure due to fatigue or being unwilling to participate. Unfortunately, no further information was available, though this suggests that the measure was perceived as too onerous, regardless of breaks/multiple sessions being offered, and/or unappealing in some way. Perhaps use of a more conventional neuropsychological test battery (e.g., non-computerised version of tests comprising CNS-VS) is preferable, or at least an option that should be offered on an individual basis to minimise participation and engagement barriers.

Limitations and Future Directions

The following limitations of the current study should be considered in the interpretation and generalizability of the results. While this study used a population-based SAH sample, the sample size (n=30) is considered small and consisted of only 42% of the wider SAH population, thus limiting the generalizability of the current findings. Though the SAH sample did not differ from the wider SAH population on age, gender, or stroke

symptoms, they did differ regarding ethnicity, with SAH survivors self-identifying as non-European less likely to participate in the present study. Under-representation of ethnic minority participants is a fairly common research limitation (Douiri et al., 2013; Hackett & Anderson, 2006; Springer et al., 2009) but nonetheless limits the generalizability of the current findings to ethnic minority SAH survivors. This is particularly problematic given that ethnic minority status is associated with increased risk of SAH and poor outcomes post-stroke and -SAH (Douiri et al., 2013; Feigin et al., 2005b; Feigin et al., 2006; Lisabeth et al., 2014; Mayer at al., 2002; Springer et al., 2009; Suarez et al., 2006; The ACROSS Group, 2000). Under-representation could therefore also have resulted in an underestimation of the severity of SAH survivors' outcomes reported in the current study.

The small sample size also restricted the power of the statistical analyses and the type of statistical analyses able to be conducted. Had a larger sample size been available findings may have varied and the predictive value of variables, not just their relationships, could have been explored. Unfortunately, the SAH sample size was often further reduced due to moderate to large amounts of missing data at each time point. While the data management strategy (i.e., pairwise exclusion) chosen minimised this limitation by excluding participants only from analyses for which they were missing data, this further limited the power of the statistical analyses. Common reasons for missing data were the participant being too unwell or having speech or cognitive impairment, suggesting that the current findings likely underestimate the severity of SAH survivor's outcomes. This was especially the case for neuropsychological outcomes (CNS-VS). As such, neuropsychological findings should be interpreted with particular caution, as should use of this particular test battery in future.

The current study assessed outcomes at several time points throughout the first 12-months post-SAH, however, little change over time was found overall. It is possible that greater changes would have been identified had assessments been even more frequent (e.g.,

3-monthly). However, consideration would need to be given to the increased participant burden, which may result in larger amounts of missing data and/or withdrawal, and the test-retest validity of measures when employed frequently in close proximity.

Of fairly recent and developing interest in the literature is the incidence and impact of PTSD post-SAH (Noble et al., 2008; Powell et al., 2002; Sheldrick et al., 2006). Though not assessed specifically in the current study, the importance and relevance of anxiety outcomes post-SAH was highlighted. The current findings could have contributed more to the literature by including assessment and exploration of PTSD in this population-based sample.

Finally, the premorbid abilities of participants were not assessed in the current study. As such, no conclusions can be drawn regarding changes in post-SAH outcomes as compared to prior functioning. Particularly with respect to neuropsychological functioning, it is possible that these abilities did significantly deteriorate post-SAH in comparison to premorbid functioning, though are masked in normative data description (e.g., average) and compared to controls as some research suggests (Berry et al., 1997).

As far as the researcher is aware, there are no other population-based studies that comprehensively examine the neuropsychological (test battery), psychological and functional short-term outcomes of SAH survivors, hence the need for more research of this focus. Given the above limitations, future research would benefit from a larger population-based sample size to improve generalizability of findings, allow for more powerful and varied statistical analyses (e.g., predictive validity), and minimise the impact of inevitable missing data and/or participant withdrawal. Research should continue using stroke subtype-specific samples, as general stroke outcomes findings do not always apply to the stroke subtype in question. Recruitment of ethnic minority participants should also be prioritised to increase generalizability to this high risk group and may be more successful in consultation with or active involvement of culturally appropriate consultants.

It would also be useful to increase the number of assessment time points during the initial 12-months post-SAH. This would further develop the delineation of the trajectory of SAH outcomes and make findings more easily comparable to the existing literature which employs different time points (e.g., 3- to 12-months).

Regarding specific outcomes, there continues to be a particular need to explore and delineate the neuropsychological outcomes (test battery) within 12-months post-SAH in a population-based sample, and the inclusion of more thorough psychological outcomes assessment (e.g., PTSD). Alongside comparison to matched controls, assessment of and comparison to premorbid functioning would also develop understanding of SAH impact on survivors' functioning.

Strengths

Though a number of limitations have been identified, the current study has various strengths. As previously stated and as far as the researcher is aware, this is the first population-based study to comprehensively examine the neuropsychological (test battery), psychological and functional short-term outcomes of SAH survivors in comparison to matched control participants. Though the generalizability of findings was limited to an extent as described above, use of a population-based sample allows for the inclusion of all SAH survivors, regardless of SAH mechanism and outcome status, providing a more representative account of outcomes than those reported by hospital- and clinic-based studies. Examination of the range of outcome domains also provides a more thorough account of SAH survivors experience, whereas previous research often details one or few outcomes domains, leaving clinicians to collaborate multiple studies to ascertain a comprehensive picture of the range of outcome expectancies. Furthermore, the current study compared both average group performance and proportions falling in the poor/impaired outcome ranges, demonstrating two

key points. First, that average between-group comparisons can hide clinically significant results (e.g., HADS anxiety subscale). Second, that not only were SAH survivors performing poorly compared to controls, but many were actually performing in the poor/impaired range, highlighting clinical concern; a conclusion easily missed based on normative data description (e.g., average) and between-group mean comparisons.

Conclusion

This study demonstrates that throughout the first 12-months SAH survivors experience worse psychological and HRQoL outcomes compared to matched controls; a significant proportion of which report outcomes in the clinical/impaired range. This was despite seemingly good outcomes regarding more traditional and obvious markers of stroke outcomes such as stroke symptoms and disability. Psychological and HRQoL outcomes were interrelated and worse for those having had a previous stroke and/or undergone surgical clipping. Some improvement on psychological and HRQoL outcomes was noted during the first 6-months post-SAH. However, improvement plateaued during the second 6-month period with SAH survivors remaining worse off than controls; thus assessment and intervention and/or support post-SAH should have a particular focus on psychological and HRQoL outcomes. Conclusions regarding neuropsychological outcomes are limited, though worse outcomes on a cognitive screening measure in particular with only early improvement demonstrate impaired cognitive functioning throughout this period and the need for comprehensive neuropsychological assessment post-SAH to ascertain domain specific impairments. Some evidence suggests higher cognitive functioning in particular may be impaired and related to other outcomes.

APPENDIX A:

MEASURES USED

DEMOGRAPHIC INFORMATION

General Information
Date of assessment: _____
Title: ____ First name(s):_____ Last name: _____
Date of Birth: _____
Sex: □ Male □ Female

Ethnicity
What ethnic group do you belong to? (tick all appropriate)
□ New Zealand European
□ Maori
□ Samoan
□ Cook Island Maori
□ Tongan
□ Niuean
□ Chinese
□ Indian
□ Other (if other, specify) _____

Relationship Status
What is your current marital status? (tick one only)
□ Married, civil union, or living with partner
□ Separated, divorced, or widowed
□ Never married (single)

Living
Who are you living with? (tick one only)
□ Living with partner/family
□ Living with others
□ Living alone

Which of the following refers to your usual dwelling place? (tick one only)
□ Own home
□ Rented
□ Living with friends or family
□ Retirement village or similar
□ Rest home/Private hospital/Boarding house
□ Other

Education/Socioeconomic Status
Did you complete high school?
□ Yes
□ No

If yes, after leaving school, did you get any further education?
□ Yes
□ No

If yes, what was the highest qualification you received? (tick one only)
□ Degree e.g.: MA, PhD, BA, BSc, Medicine
□ Diploma or Certificate e.g.: Teaching, Nursing, Business Management
□ Trade or Technical qualification, e.g.: apprenticeship, typing
□ Other

Which of the following best describes your socioeconomic status? (tick one only)
□ Professional (Doctors, accountants, engineers)
□ Managerial/technical (Marketing, sales managers, teachers, journalists)
□ Skilled non-manual (Clerks, cashiers, retail staff)
□ Skilled manual (Carpenters, van/lorry drivers, joiner)
□ Partly skilled (Warehousemen, security guards, machine/tool operators)
□ Unskilled (Building/civil engineering labourers, other labourers, cleaners)
□ Armed forces
□ Unemployed/Retired
□ Other

Other Factors
Please indicate if you have ever received any of the following diagnoses or classes of diagnoses:
□ Stroke
□ Concussion
□ Depression
□ Personality disorder
□ Substance abuse
□ Traumatic brain injury
□ Learning disability
□ Anxiety disorder
□ Psychotic disorder

TELEPHONE ASSESSMENTS

HOSPITAL ANXIETY AND DEPRESSION SCALE

Please indicate which of the following options best describes how you have been feeling during the last week **(tick one on each line)**.

		3 Most of the time	**2 A lot of the time**	**1 From time to time, occasionally**	**0 Not at all**
1.	I feel tense or wound up				

		0 Definitely as much	**1 Not quite as much**	**2 Only a little**	**3 Hardly at all**
2.	I still enjoy the things I used to enjoy				

		3 Very definitely and quite badly	**2 Yes, but not too badly**	**1 A little, but it doesn't worry me**	**0 Not at all**
3.	I get a sort of frightened feeling as if something awful is about to happen				

		0 As much as I always could	**1 Not quite so much now**	**2 Definitely not as much now**	**3 Not at all**
4.	I can laugh and see the funny side of things				

		3 A great deal of the time	**2 A lot of the time**	**1 From time to time, but not too often**	**0 Only occasion-ally**
5.	Worrying thoughts go through my mind				

		3 Not at all	**2 Not often**	**1 Sometimes**	**0 Most of the time**
6.	I feel cheerful				

		0 Definitely	**1 Usually**	**2 Not often**	**3 Not at all**
7.	I can sit at ease and feel relaxed				

		3 Nearly all the time	2 Very often	1 Sometimes	0 Not at all
8.	I feel as if I am slowed down				

		0 Not at all	1 Occasion-ally	2 Quite often	3 Very often
9.	I get a sort of frightened feeling like 'butterflies' in the stomach				

		3 Definitely	2 I don't take as much care as I should	1 I may not take quite as much care	0 I take just as much care as ever
10.	I have lost interest in my appearance				

		3 Very much indeed	2 Quite a lot	1 Not very much	0 Not at all
11.	I feel restless as if I have to be on the move				

		0 As much as I ever did	1 Rather less than I used to	2 Definitely less than I used to	3 Hardly at all
12.	I look forward with enjoyment to things				

		3 Very often indeed	2 Quite often	1 Not very often	0 Not at all
13.	I get sudden feelings of panic				

		0 Often	1 Sometimes	2 Not often	3 Very seldom
14.	I can enjoy a good book or TV programme				

SHORT FORM 36

This questionnaire asks for your views about your health, how you feel and how well you are able to do your usual activities. Answer every question. If you are unsure about how to answer a question, please give the best answer you can.

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? (tick one on each line)

	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 a 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 your 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 spend on work or other activities
ii			**Accomplished less** than you would like
iii			Were limited in 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)

Extremely	Quite a bit	Moderately	Slightly	Not at all

7. How much **bodily pain** have you had during the **past 4 weeks**? (tick one only)

Very severe	Severe	Moderate	Mild	Very mild	No bodily pain

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)

Extremely	Quite a bit	Moderately	A little bit	Not at all

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)

	ACTIVITIES	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? (tick one on each line)

	ACTIVITIES	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					

FACE-TO-FACE ASSESSMENTS

MODIFIED RANKIN SCALE

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

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

BARTHEL INDEX

The next series of questions relate to your level of activity:

1. **Feeding** (tick one only)

	Independent: Able to use any necessary device; feeds in a reasonable time; able to cut food, use containers, spread butter etc. on his/her own. Food may be placed within reach.
	Needs Help: e.g. with cutting or spreading butter.
	Dependent: Needs to be fed.

2. **Bathing** (tick one only)

	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.

3. **Grooming** (tick one only)

	Independent: Doing all personal activities, e.g. washing hands and face, combing hair. Includes shaving and teeth. Not to need any help.
	Dependent: Needs some help.

4. **Dressing** (tick one only)

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

5. **Bowels** (tick one only)

	Continent: If needs enema, suppository, must manage him/herself
	Occasional accident: Rare (under once a week); needs help with enema.
	Incontinent

6. **Bladder** (tick one only)

	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** (tick one only)

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

8. **Chair/Bed Transfers** (tick one only)

	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: Unable to manage without major assistance.

9. **Mobility on level surfaces** (tick one only)

	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** (tick one only)

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

BEHAVIOURAL DYSCONTROL SCALE

1. Tap twice with right hand and once with the left in series (10 repetitions after allowing practice)

	SCORE	DESCRIPTION
	3	No errors. Task learned quickly and performed rapidly, smoothly, automatically and with little effort
	2	Generally smooth performance, but with 1 or 2 errors
	1	Three or 4 perseverative errors Or poor timing and slow, effortful performance with few errors
	0	Poor performance. 5 or more errors, or unable to perform task despite recalling the instructions

2. Tap twice with the left hand and once with the right in series (10 repetitions after allowing practice)

	SCORE	DESCRIPTION
	3	No errors. Task learned quickly and performed rapidly, smoothly, automatically and with little effort
	2	Generally smooth performance, but with 1 or 2 errors
	1	Three or 4 perseverative errors Or poor timing and slow, effortful performance with few errors
	0	Poor performance. 5 or more errors, or unable to perform task despite recalling the instructions

3. If I say "red" squeeze my hand. If I say "green" do nothing (15 repetitions)

	SCORE	DESCRIPTION
	3	No errors, rapid response to verbal stimuli
	2	Rapid response to stimuli and no more than 1 error or slow response (1 to 1.5 seconds) and no errors
	1	Two to 4 errors, including where patient catches him/herself, or response time > 2 seconds
	0	More than 4 errors either of inhibition or initiation

4. If I tap twice, you tap once. If I tap once, you tap twice (10 repetitions)

	SCORE	DESCRIPTION
	3	No errors, rapid response to stimuli
	2	Rapid response to stimuli and no more than 1 error or slow response with no errors
	1	Two or 3 errors, or fewer errors but response time > 2 seconds
	0	More than 3 errors

5. Alternate touching thumb and fingers (5 full reps after practice)

	SCORE	DESCRIPTION
	3	No errors. Task learned quickly and performed rapidly, smoothly, automatically and with little effort
	2	Learns with at most only a few errors. Movements become relatively automatic with practice
	1	Difficulty learning the task. Makes many errors, or best performance remains deliberate and effortful. Performance improves, but never becomes really automatic even with practice
	0	Failure to learn task, or no improvement with practice unless examiner models task constantly

6. Fist-Edge-Palm

	SCORE	DESCRIPTION
	3	No errors. Task learned quickly and performed rapidly, smoothly, relatively automatically and with little effort
	2	Learns task with at most a few errors. Movements become relatively automatic with practice
	1	Difficulty learning the task. Makes may errors, or best performance remains deliberate and effortful. Performance improves, but never becomes really automatic even with practice
	0	Failure to learn task, or no improvement with practice unless examiner models task constantly

7. Head's Test

Correct the first mirroring error, but count it as an error.
Examiner and subject should return hands to their laps and pause 2-3 seconds after copying each hand position to avoid mimicry.

a. Left fist beside head
b. Right index finger points to right eye
c. Right hand with bent fingers under chin
d. Left hand vertical right hand horizontal forming "T"
e. Left hand to left ear

	SCORE	DESCRIPTION
	3	No errors
	2	One error
	1	Two or 3 errors
	0	More than 3 errors

8. (i)Alphanumeric Sequencing Time

1-A-2-B-3-C-4-D-5-E-6-F-7-G-8-H-9-I-10-J-11-K-12-L
Time (seconds) _____

(ii)Alphanumeric score

	SCORE	DESCRIPTION
	3	Completes task with no errors in 20 seconds or less
	2	Completes task with no errors > 20 seconds
	1	One to 3 errors
	0	More than 3 errors or fails to finish the task

9. Insight Rating

To consider:
Was patient aware of making errors? Was this awareness consistent?
Did he/she spontaneously express awareness of errors (verbally or nonverbally)?
Did he/she express awareness of errors in response to examiner questions/comments?
Was this awareness consistent across time?
Was awareness of severity and/or significance of errors demonstrated?
Did he/she become understandably emotionally upset over his/her errors?

	SCORE	DESCRIPTION
	3	Awareness of (in)accuracy of performance and of its severity and significance if performance is deficient
	2	Awareness of errors but limited understanding of their severity or significance
	1	Partial and/or inconsistent awareness of deficient aspects of performance
	0	Completely lacking ability to assess performance accurately and critically

GENERAL HEALTH QUESTIONNAIRE 28

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

Have you recently …

		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 once you are off?				
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 strung-up all the time?				

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

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

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

		More than usual	About the same as usual	Less than satisfied	Much less satisfied
18.	Been satisfied with the way you've carried out your task?				

		More so than usual	Same as usual	Less useful than usual	Much less useful
19.	Felt that you are playing a useful part in things?				

		More so than usual	Same as usual	Less so than usual	Much less capable
20.	Felt capable of making decisions about things?				

		More so than usual	Same as usual	Less so than usual	Much less than usual
21.	Been able to enjoy your normal day-to-day activities?				

		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?				

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

		Not at all	No more than usual	Rather more than usual	Much more than 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?				

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

CNS-VITAL SIGNS

Deliver the computer assessment and record the scores from the report given at the end of the assessment (to be recorded in the same order as on the CNS VS reports).
All data are Standard Scores (not raw scores)

	CORE CLINICAL DOMAIN SCORES	DESCRIPTION
1		Neurocognition Index Standard Score
2		Composite Memory
3		Verbal Memory
4		Visual Memory
5		Processing Speed
6		Executive Function
7		Psychomotor Speed
8		Reaction Time
9		Complex Attention
10		Cognitive Flexibility
11(i)		Verbal Memory Hits – Immediate
11(ii)		Verbal Memory Passes – Immediate
11(iii)		Verbal Memory Hits – Delayed
11(iv)		Verbal Memory Passes – Delayed
12(i)		Visual Memory Hits – Immediate
12(ii)		Visual Memory Passes – Immediate
12(iii)		Visual Memory Hits – Delayed
12(iv)		Visual Memory Passes – Delayed
13(i)		Finger Tapping – Right Taps Average
13(ii)		Finger Tapping – Left Taps Average
14(i)		Symbol Digit Coding – Correct
14(ii)		Symbol Digit Coding – Errors
15(i)		Stroop – Simple Reaction Time
15(ii)		Stroop – Complex Reaction Time
15(iii)		Stroop – Reaction Time Correct
15(iv)		Stroop – Commission Errors
16(i)		Shifting Attention – Correct Responses
16(ii)		Shifting Attention – Errors
16(iii)		Shifting Attention – Correct Reaction Time
17(i)		Continuous Performance test – Correct Responses
17(ii)		Continuous Performance test – Omission Errors
17(iii)		Continuous Performance test – Commission Errors
17(iv)		Continuous Performance test – Choice Reaction Time Correct
18		Total Test Time
19		Working Memory
20		Sustained Attention
21		Social Acuity

MONTREAL COGNITIVE ASSESSMENT

Ask the subject to answer all questions (max score: 30)

Please perform the test in the following order:

1. Naming (animals)
2. Visuospatial/executive
   - Clock
   - Cube
   - Trail test

   Then follow the order shown on the sheet

	SCORE	DESCRIPTION
1		Visuospatial/executive
2		Naming
3		Memory
4		Attention
5		Language
6		Abstraction
7		Delayed recall
8		Orientation
9		Verbal fluency: Number of words beginning with f generated in the first 15 seconds
10		Delayed recall (category cue): Number of words recalled with category cue (if patient does not spontaneously recall all the words)
11		Delayed recall (multi-choice cue): Number of words recalled with a multi-choice cue (if patients fails to recall the words with a category cue)

APPENDIX B:

PARTICIPANT INFORMATION SHEET AND CONSENT FORM - SAH GROUP

ARCOS IV PART 1: INCIDENCE AND OUTCOMES

Participant Information Sheet

An invitation

You are invited to take part in a research study because you have recently had a stroke or a mini stroke (transient ischemic attack). This study is coordinated by the National Research Centre for Stroke, Applied Neurosciences and Neurorehabilitation, AUT University in 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 may be arranged.

What are the aims of this study?

The main aim of the study is to find out what impact stroke has in New Zealand. We will be looking how many people are affected by stroke, what are the characteristics of their stroke (for example what type of stroke and how severe) and the effects of stroke on their health and wellbeing. The study will include all people who suffer a first stroke in Auckland over a 12 month period between 1st March 2011 and 28th February 2012.

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

- How the brain works in areas such as memory and behavior
- Work, income and whanau/family systems. The study aims to identify barriers to recovery after stroke and also identify ways in which stroke patients cope and adapt to changes in their lives after their stroke

In addition, the study aims to identify possible differences in how stroke affects men and women and between people of different ethnic backgrounds. The study will also aim to look for gaps in stroke services, so that these may be improved in future.

What types of people can be in the study?

All people who live in Auckland who have had a first stroke between 1 March 2011 and 28 February 2012 are able to take part in the study.

We would also like to include a family member or carer of people who have had a stroke, so that we can ask them some questions about how your stroke may have affected them. We will ask you if you would like to nominate someone to answer these questions

How many people will be in the study?

We estimate about 2200 people will be involved in this study.

What happens if I do decide to take part?

If you decide to take part, the researchers will check your medical records to find out information about your stroke. The researchers will then contact you to arrange 4 assessments over 12 months. These assessments will take place at the start of the study and then at 1 month and 6 months and 12 months after your stroke.

Each assessment will include answering some questions about your stroke. This will take about 60 minutes and can be conducted over the telephone or in person. All researchers who will be asking you some questions will have been specifically trained for this project. You will be asked questions about your recovery, mood, treatments, care and services that you received after your stroke.

The researcher will then arrange a suitable time to visit you at home. You will also be asked to complete some activities on a computer (the computer will be provided for you at the time). These activities will look at your attention span, memory and the way you process information. This will help us to see if your stroke may have affected your skills and to monitor your recovery. The computer activities will last for 60 minutes and there will be opportunities for you to take a break. These activities can also be done over several sessions if you prefer. In previous studies people have often said that they find these activities enjoyable.

In total the four study interviews should take about 8 hours of your time over twelve months.

What is the time-span for the study?

The study is expected to start on 1 March 2011 and will continue until 1st February 2013.
Your involvement will be just for 1 year during this time.

How will the study affect me?

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 deciding not to take part in the study or withdrawing from the study at any stage. Your participation in this study will be stopped if 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 may help others with this condition in future. There is no guarantee that you will benefit directly from being involved in this study. However, you will be given an opportunity to discuss your injury with someone who is an expert in stroke.

Compensation

A $20 food/fuel voucher will provided to you after completion of the 6 and 12 month interviews ($40 in total).

Confidentiality

The study files 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 16 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 about your rights as a participant in this study, you may wish to contact a Health and Disability Advocate at the Health Advocates Trust,

Telephone **0800 555 050**, or email: advocacy@hdc.org.nz

Finally

This study has received Ethical Approval from the Northern Region X Ethics Committee dated 22 November 2010.

If you would like some more information about the study please feel free to contact the ARCOS Programme Manager:

Programme Manager on 09-921-9166 or email mgirling@aut.ac.nz
National Research Centre for Stroke, Applied Neurosciences and Neurorehabilitation (NRC-SANN), AUT University

Alternatively, you can contact;
Professor Valery Feigin, Director, NRC-SANN, AUT University on 09-921-9166 or e-mail valery.feigin@aut.ac.nz

or Dr Rita Krishnamurthi, Senior Research Fellow, NRC-SANN, AUT University on 09-921-9999 ext. 7809 or email rita.krishnamurthi@aut.ac.nz

Study Investigators

The principal investigator for this study is **Professor Valery Feigin** Tel 09-921-9166
National Research Centre for Stroke, Applied Neurosciences and Neurorehabilitation (NRC-SANN), AUT University, Private Bag 92006, Auckland 1142

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

CONSENT FORM

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 (Version 6, dated 25/08/2009) for participants taking part in the ARCOS study. I have had the opportunity to discuss this study. 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 have had the opportunity to use family/whānau support or a friend to help me ask questions and understand the study.
4. I agree to an approved auditor appointed by either the ethics committee, or the regulatory authority or their approved representative, and approved by the Northern Region X Ethics Committee reviewing my relevant medical records for the sole purpose of checking the accuracy of the information recorded for the study.
5. I give my approval for information regarding my present illness to be obtained from medical records.
6. 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.
7. I understand the compensation provisions for this study.
8. I have had time to consider whether to take part.
9. I know whom to contact if I have any questions about the study.
10. I understand that my GP will be contacted about my participation in this 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

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 Case Record File

Approved by the Northern Region X Ethics Committee

APPENDIX C:

ADVERTISEMENT - CONTROL GROUP

Interested in Clinical Psychology?

Keen to give neuropsychological and psychological testing a go?

Fancy providing valuable data for stroke research?

If you are, you may be interested in participating in research being conducted by Rebecca Nicholson, a postgraduate student enrolled in the Doctor of Clinical Psychology at The University of Auckland.

This research is examining the neuropsychological, psychological, and functional outcomes of healthy Auckland adults. By participating in this research you'll be providing important information that will allow us to examine the strengths and difficulties stroke survivors experience compared to healthy adults. Eligible participants are healthy adults residing in Auckland who speak English.

Participation involves one telephone assessment of up to 30 minutes and one face-to-face assessment session of up to 90 minutes at The University of Auckland or in your home. The assessment involves administration of tests commonly used by psychologists in New Zealand. There are no risks associated with participation, though people can find some of the tasks slightly boring and it is expected that you may find some test items difficult.

Participation is **voluntary**. All information collected will remain **confidential** and **anonymous**. Your name/identifying information will not be associated with any published results. A $10 petrol voucher will be given to participants to compensate for time spent, regardless of whether they decide to withdraw.

If you wish to participate or would like further information contact me using the information below to obtain further information and a consent form.

APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN PARTICIPANTS ETHICS COMMITTEE on 22 June 2012 for a period of 3 years, Reference Number 8289.

Clinical Psychology Study Rebecca Nicholson 373 7599 ext 85224 rgol012@aucklanduni.ac.nz	Clinical Psychology Study Rebecca Nicholson 373 7599 ext 85224 rgol012@aucklanduni.ac.nz	Clinical Psychology Study Rebecca Nicholson 373 7599 ext 85224 rgol012@aucklanduni.ac.nz	Clinical Psychology Study Rebecca Nicholson 373 7599 ext 85224 rgol012@aucklanduni.ac.nz	Clinical Psychology Study Rebecca Nicholson 373 7599 ext 85224 rgol012@aucklanduni.ac.nz	Clinical Psychology Study Rebecca Nicholson 373 7599 ext 85224 rgol012@aucklanduni.ac.nz	Clinical Psychology Study Rebecca Nicholson 373 7599 ext 85224 rgol012@aucklanduni.ac.nz	Clinical Psychology Study Rebecca Nicholson 373 7599 ext 85224 rgol012@aucklanduni.ac.nz

APPENDIX D:

PARTICIPANT INFORMATION SHEET - CONTROL GROUP

DEPARTMENT OF PSYCHOLOGY
Building 721, Tamaki Campus
261 Morrin Road, Glen Innes
Telephone 64 9 373 7599
Facsimile 64 9 373 7000
The University of Auckland
Private Bag 92019
Auckland, New Zealand

THE UNIVERSITY OF AUCKLAND
NEW ZEALAND
Te Whare Wānanga o Tāmaki Makaurau

PARTICIPANT INFORMATION SHEET

Title: Neuropsychological, Psychological, and Functional Outcomes of Auckland Adults
Name of Researcher: Rebecca Nicholson

Dear potential participant,

My name is Rebecca Nicholson. I am a postgraduate student enrolled in the Doctor of Clinical Psychology (Department of Psychology) at The University of Auckland conducting research to examine the neuropsychological, psychological, and functional outcomes of healthy Auckland dwelling adults. This research is partially funded by Research Expenses Funding for Doctoral Students provided by The University of Auckland.

You are invited to participate in this research. In doing so you will be providing important information that will allow me to compare the impact stroke has on its survivors. Neuropsychological abilities include performance on tests of memory, attention and concentration, mental flexibility, language, and visual problem solving. Psychological outcomes include depression, anxiety, and general wellbeing. Functional outcomes refer to one's ability to complete activities of daily living and overall quality of life. I am asking healthy adults who speak English and live in Auckland to participate in individual assessment sessions that include tests of these abilities. Participants will participate in one telephone assessment of up to 30 minutes and one face-to-face assessment of up to 90 minutes. Face-to-face assessments will take place in accessible facilities within The University of Auckland or in your home if preferred. The assessments will be conducted by me.

You are under no obligation to participate. Your participation is **voluntary** and you may withdraw participation at any time. All information collected will remain **confidential** and **anonymous.** This study will likely result in the publication of findings in an international journal to further local and international knowledge and practice, however, your name/identifying information will not be associated with any published results. At the conclusion of this research, a summary of the findings will be mailed to you.

The tests to be administered are commonly used by clinicians in the field. Therefore, participation in this study may benefit you in the future. All hard copy and electronic test data will be held in secure storage on University premises for a period of 10 years. If you are referred for neuropsychological assessment in the next 10 years (e.g., you experience a traumatic brain injury or neurological condition), a summary of your test results from this study can be made available following your consent and written request from your G.P. This will assist clinicians to more accurately assess the impact of the injury/condition leading to more appropriate treatment. A letter to file with your G.P. advising the above can be made available to facilitate future access should it be required. After the 10 years has elapsed all data will be shredded and/or deleted.

Participants can withdraw their information from the study by contacting the researcher at any time before August 1st 2013. There are no risks associated with the study though people sometimes find the tasks involved slightly boring and it is expected that you may find some of the items difficult. Participants will be offered a $10 petrol voucher as a token of appreciation for the time taken to participate in this research on participation, regardless of whether they decide to withdraw.

If you wish to participate please complete the attached consent form and return it in the freepost envelope provided OR contact me using the information below to obtain further information and a consent form.

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

Contact persons:

Rebecca Nicholson
Department of Psychology
The University of Auckland
Private Bag 92019
Auckland, New Zealand
Tel: (09) 373 7599 extn. 85224
Email: rgol012@aucklanduni.ac.nz

Primary Investigator:
Dr Suzanne Barker-Collo
Department of Psychology
The University of Auckland
Private Bag 92019
Auckland, New Zealand
Tel: (09) 373 7599 extn. 88517
Email: s.barker-collo@auckland.ac.nz

Head of Department 2012:
Associate Professor Douglas Elliffe
The University of Auckland
Private Bag 92019
Auckland, New Zealand
Tel: (09) 373 7599 extn. 85262
Email: d.elliffe@auckland.ac.nz

If you have any concerns of an ethical nature you can contact the Chair, The University of Auckland Human Participants Ethics Committee, The University of Auckland, Research Office, Private Bag 92019, Auckland 1142. Telephone (09) 373 7599 extn. 87830/83761. Email: humanethics@auckland.ac.nz

APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN PARTICIPANTS ETHICS COMMITTEE on 22 June 2012 for a period of 3 years, Reference Number 8289.

APPENDIX E:

CONSENT FORM - CONTROL GROUP

DEPARTMENT OF PSYCHOLOGY
Building 721, Tamaki Campus
261 Morrin Road, Glen Innes
Telephone 64 9 373 7599
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The University of Auckland
Private Bag 92019
Auckland, New Zealand

THE UNIVERSITY OF AUCKLAND
NEW ZEALAND
Te Whare Wānanga o Tāmaki Makaurau

CONSENT FORM

THIS FORM WILL BE HELD FOR A PERIOD OF 10 YEARS

Title: Neuropsychological, Psychological, and Functional Outcomes of Auckland Adults
Name of Researcher: Rebecca Nicholson

I have read the Participant Information Sheet and have understood the nature of the research, why I have been selected, and that participation is **voluntary**. I have had the opportunity to ask questions and have them answered to my satisfaction.

- I agree to take part in this research.
- I understand that participation will involve one telephone assessment and one face-to-face assessment lasting up to a total of two hours.
- I understand that all data collected will remain **confidential** and **anonymous** and that my name/identifying information will not be associated with any published results.
- I understand that I am free to withdraw participation at any time, and to withdraw any data traceable to me up to a specified date (August 1st 2013).
- I wish / do not wish to receive the summary of findings (circle one). Contact the researcher at rgol012@aucklanduni.ac.nz
- I understand that data will be kept in secure storage on University premises for 10 years, after which they will be destroyed.
- I understand that, with my written permission, a summary of my information collected can be made available to my G.P. within this 10-year period. I would / would not like a letter to file with my G.P. to facilitate future access.

Name _____ (please print)

Signature _____ Date _____

Contact Phone(s) _____

I wish to receive a $10 petrol voucher for participation in this research
□ YES □ NO

APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN PARTICIPANTS ETHICS COMMITTEE on 22 June 2012 for a period of 3 years, Reference Number 8289.

APPENDIX F:

GENERAL PRACTITIONER LETTER - CONTROL GROUP

DEPARTMENT OF PSYCHOLOGY
Building 721, Tamaki Campus
261 Morrin Road, Glen Innes
Telephone 64 9 373 7599
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The University of Auckland
Private Bag 92019
Auckland, New Zealand

THE UNIVERSITY OF AUCKLAND
NEW ZEALAND
Te Whare Wānanga o Tāmaki Makaurau

NOTIFICATION TO PARTICIPANT'S GENERAL PRACTITIONER

Dear Dr _____,

Your patient _____ has consented to participate in a research project conducted by Rebecca Nicholson, a postgraduate student enrolled in the Doctor of Clinical Psychology (Department of Psychology) at The University of Auckland, who is examining the neuropsychological, psychological, and functional outcomes of healthy Auckland dwelling adults to allow comparison to a group of stroke survivors.

This study involves neuropsychological assessment. This test data may benefit your patient in the future. All test data will be held in secure storage on University premises for a period of 10 years (until 2023), after which all data will be shredded and/or deleted. If your patient is referred for neuropsychological assessment in the next 10 years a summary of their test results can be made available to assist the assessor in more accurately determining the impact of the injury/condition.

Should this become applicable, a summary of their test results will be made available following written request with patient written consent from you to Dr Suzanne Barker-Collo, the primary investigator for this research (contact details below). This summary will be released to the neuropsychologist whom you identify as the assessor to ensure appropriate interpretation and application of the results.

We suggest that this letter be kept on file to facilitate future contact and access to your patients neuropsychological test results as required.

Kind regards,

Rebecca Nicholson

Primary Investigator and Contact Person:
Dr Suzanne Barker-Collo
Department of Psychology
The University of Auckland
Private Bag 92019
Auckland, New Zealand
Tel: (09) 373 7599 extn. 88517
Email: s.barker-collo@auckland.ac.nz

APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN PARTICIPANTS ETHICS COMMITTEE on 22 June 2012 for a period of 3 years, Reference Number 8289.

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