The Neuropsychological and Functional Outcomes of Primary Intracerebral Haemorrhage (ICH) at 6 and 12 Month Follow Up: A Population-Based Study

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Abstract

This exploratory population-based study examined the short-term (6-month) and long-term (12-month) mood, cognitive, and functional outcomes of Intracerebral Haemorrhage (ICH). Previous research has tended to use hospital-based samples; examine stroke outcomes generally rather than specific to stroke subtype; assess outcomes only in the acute phase post-stroke; and favour functional outcomes, while neglecting neuropsychological outcomes. The aims of the current study, therefore, were: to compare 6- and 12-month outcomes of ICH survivors to matched controls; to describe the natural course of recovery of mood, cognitive, and functional outcomes over the first year post-ICH; to examine the interrelationships between mood, cognitive, and functional outcomes of ICH; and to determine if there are characteristics of the ICH survivor at baseline that predicts good versus poor functional and cognitive outcomes at 12 months. The study used a sample of 35 ICH survivors drawn from the Auckland Regional Community Stroke (ARCOS) IV Study and 34 healthy controls matched for age, gender, and ethnicity who were recruited from the Auckland community. Both the ICH group and the control group were administered a series of measures assessing their mood, cognition, and functioning (independence in activities of daily living [ADLs] and health related quality of life [HRQoL]). The ICH group were assessed, where possible, at three timeframes: baseline (within 2 weeks of ICH), 6 months post-ICH, and 12 months post-ICH.

The results indicate that cognitive impairment following ICH is common, widespread, and persisting. Mood difficulties, particularly mild depression, were also present for ICH survivors over the study period. The ICH group had significantly less independence in ADLs, although improvement over the 12 months was observed. There were mixed findings when it came to HRQoL. Physical HRQoL post-ICH was impaired but not necessarily mental HRQoL. Younger age was found to correlate with poorer mood outcomes. Given the exploratory nature of the study, and limited power due to a small sample size, the findings are not considered conclusive. However, the results highlight the need for a greater focus on cognition and mood in rehabilitation of ICH survivors and on future research in these areas.

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List of In-Text Abbreviations

Abbreviated Form	Full Form
ACE-R	Addenbrooke's Cognitive Examination- Revised
ADLs	Activities of Daily Living
ARCOS	Auckland Region Community Stroke Study
BDS-2	Behavioural Dyscontrol Scale Second Edition
BI	Barthel Index
DSM	Diagnostic and Statistical Manual of Mental Disorders
GHQ-28	General Health Questionnaire 28
HADS	Hospital Anxiety and Depression Scale
HRQoL	Health Related Quality of Life
IADLs	Instrumental Activities of Daily Living
ICF	International Classification of Functioning, Disability, and Health
ICH	Intracerebral Haemorrhage
MMSE	Mini Mental State Examination
NZ	New Zealand
PICH	Primary Intracerebral Haemorrhage
PSA	Post Stroke Anxiety
PSD	Post Stroke Depression
PTSD	Post-Traumatic Stress Disorder
SAH	Subarachnoid Haemorrhage
SF-36	Short-Form 36
WHO	World Health Organisation

Chapter 1: Introduction

This study examined the short-term (6 month) and long-term (1 year) outcomes of intracerebral haemorrhage (ICH) survivors from a 12-month incidence sample in Auckland, New Zealand (NZ). Its overarching aims were to describe the cognitive, mood, and functional outcomes of ICH at each time frame; to determine the extent to which ICH impacts on cognitive, mood, and functional outcomes in contrast to age, gender, and ethnicity matched controls; to determine any interrelationships between outcome variables; and to determine if demographic variables are predictive of outcome.

Given these aims, the following literature review provides an introduction to ICH and stroke in general- including key definitions and epidemiological information; this is followed by an in-depth discussion of the stroke outcomes of interest to this study (mood, cognitive, and functional outcomes). Literature on ICH is referred to where possible but the shortage of this literature often made it necessary to reference stroke more generally. The review also references NZ studies and data where possible. The introduction section ends with the specific aims of this study and a discussion of how the literature reviewed informed these aims.

Section 1: Introduction to Stroke and ICH

Definition and classification of stroke.

A stroke or cerebrovascular accident has been defined by the World Health Organisation (WHO) as "rapidly developed clinical signs of focal or global disturbance of cerebral function, lasting more than 24 hours or until death, with no apparent non-vascular cause" (p. 108, Tunstall-Pedoe, Puska, & Tuomilehto, 1988). Strokes are broadly classified based on the associated pathology as being ischaemic or haemorrhagic. Ischaemic stroke occurs if a blood vessel in the brain is blocked, which obscures blood and therefore oxygen flow to areas fed by that vessel (Norrving, 2009). This leads to areas of infarction or dead tissue. By contrast, haemorrhagic strokes occur when a blood vessel ruptures; if this rupture occurs between the brain and a layer of the meninges called the subarachnoid membrane the stroke is termed a subarachnoid haemorrhage

(SAH) (Norrving, 2009). If it occurs within the tissue of the brain itself, it is classified as an ICH (Qureshi, Mendelow, & Hanley, 2009). The proportion of all strokes that these subtypes make up are approximately as follows: ischaemic 69-82%, ICH 8-15%, and subarachnoid haemorrhage 4-11%, with the remaining proportion being strokes that are undefined (Barker-Collo, Feigin, Parag, Lawes, & Senior, 2010; Cabral et al., 2009; Thrift, Dewey, Macdonell, McNeil, & Donnan, 2001; Thrift et al., 2009). Of note is that this data has mainly originated from high income countries; the relative proportion of ICH's appears to be higher in low-middle income countries, reported in one review as comprising 14-27% of all strokes (Feigin, Lawes, Bennett, Barker-Collo, & Parag, 2009).

Subtypes of ICH based on aetiology.

ICH's can be classified as being of traumatic or of spontaneous origin. A traumatic ICH resulting from the mechanical forces associated with trauma is classified as a traumatic brain injury rather than a stroke. Spontaneous ICH's can be further classified as being primary or secondary based on the cause of the bleed (Sutherland & Auer, 2006). A secondary ICH occurs when the rupture of the vessel is associated with a secondary condition; the bleed may be secondary to a structural abnormality (e.g., tumour or arteriovenous malformation), secondary to a clotting disorder known as a coagulopathy (e.g., induced by aspirin or warfarin), or secondary to severe acute hypertension (e.g., due to sympathomimetic drugs such as cocaine; Xi et al., 2006).

If the WHO definition of stroke is strictly followed, secondary ICH cannot be classified as a stroke because there is an associated cause that is not of entirely vascular origin. The outcome of a secondary ICH may therefore be representative of the ICH and additionally the secondary condition. In contrast to a secondary ICH, a primary ICH (PICH) can be defined as spontaneous non-traumatic bleeding when there is no obvious known precipitating cause other than vascular (Alway, 2009; Nilsson, Lindgren, Brandt, & Säveland, 2002; Roob et al., 2000). PICH is therefore the only type of ICH which can be considered a stroke under the WHO definition and, as it is the outcome of ICH itself (rather than any secondary conditions) which is of interest to this study, it is therefore the definition of ICH being used in this thesis. This approach is consistent with other

researchers who use the WHO definition of stroke (e.g., Bamford, Sandercock, Dennis, Burn, & Warlow, 1990; Islam et al., 2008).

Risk factors for primary ICH.

Whilst a PICH by definition has no known cause, the two most important risk factors for PICH are hypertension and age (Alway, 2009). Hypertension, defined as raised blood pressure, was found to have a population prevalence of 30% in a recent NZ survey (McLean, Williams, Mann, & Parnell, 2012). Chronic hypertension is thought to cause arteries and arterioles to become stiff, brittle, and narrow (which increases vascular resistance as less blood can flow through); leaving them more vulnerable to rupturing (Enache, Zaharia, Georgescu, & Tenovivi, 2005; Intengan & Schiffrin, 2000; Serné, de Jongh, Eringa, IJzerman, & Stehouwer, 2007). One review found that a history of hypertension increased the odds of PICH by 2.6 times (O'Donnell et al., 2010). It appears that as people age arteries and arterioles also become more fragile meaning that increased age is associated with a greater prevalence of PICH (Alway, 2009).

Mechanisms of injury in ICH.

An ICH occurs when a diseased or weakened blood vessel in the brain ruptures, causing blood to erupt into the surrounding brain tissue (de Bustos & Moulin, 2012). As the blood rushes out of the vessel, the mechanical force is enough to sever white matter tracts in surrounding brain tissue (Sutherland & Auer, 2006). These white matter tracts transmit signals within the brain; therefore, this damage may lead to a slowed speed of information processing. The mass of blood that collects in the tissue is known as a haematoma and it disrupts the brains cellular architecture; it compresses surrounding tissue and increases intracranial pressure potentially to the extent where a shift in the brain's midline may occur (Keep, Hua, & Xi, 2012). The volume of blood in the haematoma at cessation of bleeding is important for the patient's outcome on the clinical spectrum from asymptomatic to fatal (Sutherland & Auer, 2006). If this volume exceeds 150ml then the pressure of the blood flow in the brain will drop to zero and the patient will die; if it is below 150ml then the patient has a chance of survival (Xi, Keep, & Hoff, 2006). Further damage is caused by

haemoglobin (an oxygen-carrying protein in blood) released into areas surrounding the haematoma because this protein and its degradation products are harmful to brain tissue (Xi et al., 2006).

In reaction to the infiltration of blood into the brain a clot starts to form in order to stop the bleeding. This is known as the coagulation cascade and one of the key components in this clotting cascade is the protein thrombin (Xi et al., 2006). Thrombin can set off some potentially harmful processes which result in necrosis (un-programmed cell death), hence why after clotting has occurred some people will be treated with thrombin inhibitors (Xi et al., 2006). The coagulation cascade can result in a ring of swelling or oedema around the haematoma (Xi et al., 2006). Swelling peaks several days after the initial bleed (Xi et al., 2006), and leads to further pressure on other parts of the brain, potentially resulting in transtentorial herniation (the downward displacement of brain tissue through the tentorial notch) which places pressure on the brainstem and is therefore life threatening (Keep et al., 2012). Cerebral oedema, which is defined as swelling of brain tissue due to either cellular enlargement or extracellular fluid build-up, may also cause further damage because of pressure effects (Elliott & Smith, 2010).

In addition to the coagulation cascade the brain also activates an acute inflammatory response soon after the ICH. Whilst this is helpful in some respects, too much inflammation can aggravate the injury (Wang, 2010; Xi et al., 2006) and excessive inflammation is associated with poorer functional outcomes (Rodriguez-Yanez et al., 2012). The haematoma tends to gradually resolve itself within a couple of weeks; though as the blood is reabsorbed this sometimes leaves behind a cavity where brain tissue has been destroyed (Keep et al., 2012). ICH survivors may therefore be left with lasting neurological deficits and may even die from later complications (Xi et al., 2006).

It has been a controversial topic whether or not secondary ischaemia occurs in the brain after an ICH (Xi et al., 2006). Secondary ischaemia would occur if the blood flow adjacent to the haematoma decreases to the point where it can no longer provide the areas of the brain reliant on it with enough blood, and therefore oxygen, resulting in some neuronal death (Xi et al., 2006). Some

researchers believe a compromised artery can clot quick enough to prevent secondary ischaemia (Xi et al., 2006). It appears, however, that vasospasm, which is when blood vessel spasm leads to vasoconstriction, may occur in up to one fifth of ICH patients- potentially leading to secondary ischaemia (Kochanowitz et al., 2012). Despite this, recent research indicates that only a small number of ICH survivors have ischaemic lesions near the haematoma site visible on scans (Prabhakaran & Sheth, 2012).

Clinical presentation and diagnosis of ICH.

The classic presentation of ICH is sudden onset of a focal neurological deficit that worsens over minutes to hours with accompanying elevated blood pressure (90% of patients), alteration in consciousness (50% of patients), vomiting (49% of patients) and severe headache (40% of patients) (Broderick et al., 1999; Case, 2010).

This presentation shares similarities with other stroke subtypes although headaches are more common in the presentation of ICH compared to ischaemic stroke; severe 'thunderclap' headaches are however nearly universal in SAH (Alway, 2009). Decreased consciousness and vomiting is uncommon in early ischaemic stroke; but similar to ICH, these symptoms accompany half of SAHs (Broderick et al., 1999). The rapid worsening of neurological symptoms is typically more marked in ICH than ischaemic stroke, and is often caused by ongoing bleeding and enlargement of the haematoma (Alway, 2009; Broderick et al., 1999).

Clinical signs are generally not specific enough for differentiation into stroke subtype; therefore definite diagnosis can only be made with the aid of brain imaging (de Bustos & Moulin, 2012; Morgenstern et al., 2010). A non-contrast head computed tomography (CT) scan is a way of diagnosing ICH that is widely available, accurate, and fast and which shows blood within the substance of the brain (Aguilar & Freeman, 2011). While Magnetic Resonance Imaging (MRI) is also accurate, it is not as widely available nor as rapid as a CT (Alway, 2009).

Acute medical treatment of ICH.

ICH is a medical emergency (Elliott & Smith, 2010) that has been described by some medical professionals as the least treatable form of stroke, with limited options available for medical intervention (Ciccone, Pozzi, Motto, Tiraboschi, & Sterzi, 2008). In the acute stage post-ICH intervention focusses on initial medical stabilisation, treatment of high blood pressure and neurotoxic factors, therapies to promote hematoma stabilisation, and in some cases minimally invasive neurosurgical treatment to drain blood or reduce clot size in order to decrease swelling and the resulting brain damage (de Bustos & Moulin, 2012; Elliott & Smith, 2010; Morgenstern et al., 2010; Naval, Nyquist, & Carhuapoma, 2008). Surgery is risky in this group because patients are often in a fragile state and due to the traumatic nature of the invasive procedure there is a high rate of post-operative haemorrhaging (Sutherland & Auer, 2006). Further treatment may also be necessary for ongoing complications of ICH such as raised intracranial pressure and hydrocephalus (Alway, 2009).

Incidence and prevalence of stroke and ICH.

Incidence rate is the rate of new strokes that occur in a defined population over a certain time period, typically 1 year. The incidence rate of ICH is typically much lower than that for ischaemic stroke and higher than that for SAH. An Australian study found the following incidence rates by subtype: ischaemic stroke 197, ICH 47, and SAH 19 per 100,000 person years (Thrift et al., 2009). Put more simply the incidence rate of ICH means that if you observed 100,000 people during a one year time frame you would expect approximately 47 ICH's to occur in this group.

Research indicates that in NZ (Anderson et al., 2005) and other developed countries (Heuschmann, Grieve, Toschke, Rudd, & Wolfe, 2008; Wieberdink, Ikram, Hofman, Koudstaal, & Breteler, 2012) there has been a reduction in overall age adjusted stroke incidence over the last several decades. A study that combined the data of population-based studies around the world (mainly developed countries) estimated that the incidence of total stroke for individuals aged over 55 is 420 to 650 per 100,000 person years (Feigin et al., 2003).

Whilst the incidence rate for stroke in general may be decreasing, there is some evidence the incidence of ICH is either remaining stable or increasing. An extensive review of 36 studies publishing data on the incidence of ICH between 1980 and 2008 was undertaken by van Asch et al. (2010). The review found that the overall incidence of ICH among Western countries has stayed stable over the last 3 decades at 24.6 per 100,000 person years, in contrast to other types of stroke which were reported to decrease (van Asch et al., 2010). A NZ study found that between 1999 and 2008 the incidence rate of ICH increased from 14.4 to 21.4 per 100,000 person years, which is attributed to increasing numbers of elderly, neuroimaging technologies which make observance of ICH easier, and increased incidence of ICH associated with blood-thinning treatment via Warfarin (Irwin, Wright, & Reeve, 2011).

The reported incidence rates discussed thus far have mainly come from studies in developed countries; yet stroke incidence rates appear to differ in developing nations. A review of population-based stroke incidence studies conducted over the past four decades found that in high income countries there has been a 42% decrease in overall stroke incidence, however in low to middle income countries there has been a 100% increase in overall stroke incidence (Feigin et al., 2009). The overall incidence rate of ICH in low to middle income countries (22 per 100,000) was found to be twice as high as that reported in high income countries (10 per 100,000; Feigin et al., 2009). Increasing incidence rates in low to middle income countries may relate to increasing urbanisation alongside lifestyle and dietary changes which have resulted in higher rates of risk factors for stroke such as diabetes, smoking, poor diet, physical inactivity and obesity (Johnston et al., 2009; Kim & Johnston, 2011). Few resources also tend to be allocated to stroke prevention and treatment in low-income countries (Johnston et al., 2009). Recent research has, however, highlighted that death rates are declining over time in both developed and developing countries (Krishnamurthi et al., 2015).

Differences can also be seen in stroke incidence within countries based on income level. Heeley et al. (2011) pooled data across two Australian studies and one NZ study and found that stroke incidence is higher in more socioeconomically deprived areas, which may be explained by

more risk factors associated with people living in these areas (e.g., minority ethnicity, cigarette smoking, hypertension, and diabetes).

In regards to ethnicity, differences in stroke incidence have also been noted in the literature. In NZ, Maori and Pacific Islanders have a higher estimated relative risk of developing a stroke than NZ Europeans (Bonita, Broad, & Beaglehole, 1997; Carter et al., 2006). A Hawaiian study found that native Hawaiians and Pacific Islanders tend to be younger and have a higher burden of risk factors for stroke when they have an ICH, compared to white Hawaiians' (Nakagawa, Koenig, Seto, Asai, & Chang, 2012). A review of the incidence of ICH among different ethnicities found that the incidence rates for most ethnic groups were comparable, but was two times higher in people of East or Southeast Asian ethnicity (van Asch et al., 2010).

In contrast to incidence, prevalence is the proportion of people in the population who are affected by a disease at a particular point in time. The age-standardised prevalence of stroke for people aged 65 or more in a review that pooled data from various population-based studies was 4,610 to 7,330 per 100,000 people in the population (Feigin et al., 2003). This means that approximately 4 to 8% of people over the age of 65 are affected by a stroke at any given time. There was no significant variation in the prevalence rates according to the population studied, except in L'Aquila, Italy and Newcastle, United Kingdom, which reported higher prevalence rates than the other studies.

Case fatality rate of stroke and ICH.

Case fatality rate is defined as the percentage of people diagnosed as having a specified disease who die as a result of that disease in a given time period. The early case fatality rate (generally 28 days to 1 month post-stroke) of ICH is far greater than that for ischaemic stroke (Fu et al., 2013) with the majority of deaths occurring within the first two days post-ICH (Rønning, Sorteberg, Nakstad, Russell, & Helseth, 2008; Tatu et al., 2000). A review of population-based studies over the last decade found that the early case fatality rate for the various subtypes of stroke were as follows; ischaemic stroke 14%, ICH 41%, and SAH 30% (Feigin et al., 2009).

Improvements in fatality rates for stroke in general have been reported recently; however, whilst there is a trend towards improvement in ICH fatality rates (e.g., from 46 to 41% over the last 3 decades; Feigin et al., 2009) no significant improvement has yet been documented (Feigin et al., 2009; Manno, 2012; van Asch et al., 2010).

Studies reporting long-term survival post-ICH have found the following fatality rates: 49% at 5 years (Feigin et al., 2010), 76% at 10 years, and 92% at 21 years (Anderson et al., 2004). In Anderson et al.'s (2004) study, which was conducted in NZ, the 21 year fatality rate post-ICH was nearly twice the fatality rate of the general NZ population (Anderson et al., 2004).

In terms of risk factors for mortality post-stroke it appears that increased age is an important factor (Fiorelli et al., 1995; Henon et al., 1995); there is a higher frequency of secondary complications among older age groups (Demchuk & Buchan, 2000) and older age is known to be a factor associated with greater brain swelling and neurological deficits (Xi et al., 2006). Gender is another factor that may influence fatality with a systematic review finding that the 1 month case fatality rate is higher among women than men (Appelros et al., 2009). However, studies that take the age distribution of stroke into account (women generally have strokes at an older age than men), such as Thrift et al. (2009), have found that the poorer case fatality rate for women is no longer apparent at 28-days or 1 year post-stroke.

Certain ethnic groups may be at increased risk of fatality following stroke. In NZ the early case fatality rate of stroke has been found to be 1.3 times higher for Maori and 1.6 times higher for Pacific Islanders in NZ than for NZ Europeans (Bonita et al., 1997). This may be attributable to the greater number of risk factors associated with these groups, and the higher rate of the more fatal subtypes of stroke (ICH and SAH) occurring among Maori and Pacific Islanders compared to Europeans (Bonita et al., 1997). Socioeconomic factors may also relate to fatality. One global study found that national per capita income was the strongest predictor of stroke fatality rates, even when adjustments were made for cardiovascular risk factors; Eastern Europe, North Asia, Central Africa, and the South Pacific therefore had the highest case fatality rates (Johnston, Mendis, & Mathers,

2009). This is consistent with a review that found there is a difference in the early case fatality rate of ICH in high income countries (25-35%) compared to low to middle income countries (30-48%), which may be due to a different level of care being available in these countries (Feigin et al., 2009).

Post-stroke cognitive impairment has been found to correlate with increased fatality; with executive dysfunction and deficits in visuospatial/visuoperceptual abilities in particular, relating to poorer survival (Oksala et al., 2009). Depression also increases the risk of post-stroke fatality; Morris et al. (1993) found that those who had a diagnosis of depression were three times more likely to have died over the ten year follow-up period, independent of other major risk factors. The depressed individuals particularly at risk were those who had few social contacts; of this group over 90% had died at follow-up which is considerably more than the overall rate of 53% (Morris et al., 1993).

Stroke burden and risk factors.

In NZ approximately 9000 people have a stroke every year (Stroke Foundation of NZ, 2013). Stroke is the second leading cause of death worldwide (Mathers, Fat, & Boerma, 2008) and the third leading cause of death in NZ (Tobias, Cheung, Carter, Anderson, & Feigin, 2007). It is the biggest cause of disability both worldwide and in NZ (Mathers et al., 2008; Tobias et al., 2007).

The financial burden of stroke is high and is estimated to increase in the coming decades due to the ageing population (Feigin, Lawes, Bennett, & Anderson, 2003; Gommans et al., 2003; Payne, Huybrechts, Caro, & Klittich, 2002). Over a lifetime it is estimated that ICH on average incurs 1.7 times greater financial costs to society than ischaemic stroke (Dewey et al., 2003). Stroke survivors with cognitive impairment have also been found to incur 3 times greater financial cost to society compared to those without cognitive impairment (Claesson, Linden, Skoog, & Blomstrand, 2005).

Stroke risk factors can be broken down into those that are modifiable and those that are non-modifiable. Non-modifiable risk factors that are well established include age (older age equals greater risk), gender (which has varying affects over different age ranges and subtypes), ethnicity (with ethnic minorities at greater risk), and hereditary factors (at greater risk if family history of

stroke; Abbott et al., 2003; Alberts, McCarron, Hoffman, & Graffagnino, 2002; Ionita et al., 2005; Sacco, 2001; Tegos, Kalodiki, Daskalopoulou, & Nicolaides, 2000). To expand on age as a risk factor, the average age of stroke onset in NZ is 70 years for males and 75 years for females (Feigin et al., 2003); when ethnic groups are examined specifically the average age is 75 years for NZ Europeans, 61 years for Māori, and 64 years for Pacific Islanders living in NZ (Feigin et al., 2006). In terms of gender the majority of studies have found that men have a greater incidence rate of ICH compared to women (Appelros, Stegmayr, & Terént, 2009; Thrift et al., 2009; Tveiten et al., 2012). However, other research has found no significant gender difference in incidence rate of ICH (van Asch et al., 2010); and other studies report that while the incidence rate of ICH in under 85 year olds is higher in men, in over 85 year olds the reverse is true leading to women having an overall greater lifetime risk of stroke (Petrea et al., 2009). Other potential non-modifiable stroke risk factors are mild dementia (2.6 times greater risk of stroke) and cognitive impairment (2 times greater risk of stroke; Zhu, Fratiglioni, Guo, Winblad, & Viitanen, 2000).

Whilst it is important to note these non-modifiable risk factors, it is more productive to focus on modifiable risk factors because being aware of these can inform interventions to reduce the risk of stroke. Efforts to reduce modifiable risk factors for stroke can go a long way to reducing the sizeable burden that stroke places on society (Feigin, Barker-Collo, Krishnamurthi, Theadom, & Starkey, 2010). A large case-control study that examined 3000 cases of stroke from 22 countries worldwide found that significant risk factors for all types of strokes were: history of hypertension, current smoking, high waist-to-hip ratio, diet risk score, reduced regular physical activity, diabetes, alcohol intake (>30 drinks per month or binge drinking), and psychosocial stress (O'Donnell et al., 2010). These risk factors accounted for 90% of the variance in stroke. Other research has established that someone experiencing depressive symptoms is also at increased risk of suffering a stroke; therefore emotional well-being may also potentially be considered a modifiable risk factor for stroke (Ostir, Markides, Peek, & Goodwin, 2001). A NZ study has noted some favourable downwards trends in modifiable stroke risk factors such as cigarette smoking in recent years;

however, the decreases noted have been offset partially by increasing body mass indexes, diabetes levels, and increasing age of the population (Anderson et al., 2005).

When ICH is looked at specifically, hypertension, smoking, high waist-to-hip ratio, diet and alcohol intake are the most significant risk factors (Casolla et al., 2012; O'Donnell et al., 2010; Tatu et al., 2000). In terms of smoking, there is evidence of a dose-response relationship, that is, the more you smoke the more likely you are to have an ICH (Shah & Cole, 2010). Past stroke is also an important risk factor for recurrent ICH (Easton et al., 2009; Ionita et al., 2005). It is of note also that regular exercise before an ICH not only reduces the chances of an ICH occurring, but is associated with a better 5 year outcome post-ICH (Hankey, Jamrozik, Broadhurst, Forbes, & Anderson, 2002). Other modifiable risk factors specific to ICH that likely increase the overall risk of having an ICH to a smaller degree include: no high school education (Martini et al., 2012), anger proneness in people under 65 years old (Arthur, 2002), lower socioeconomic status (Addo et al., 2012), cocaine use (which results in acute periods of very high blood pressure; Martin-Schild et al., 2010), and low consumption of green tea or coffee (Kokubo et al., 2013).

Section 2: Outcomes of Stroke and ICH

Stroke can have a devastating range of outcomes across various domains. Survivors of stroke commonly experience physical impairments such as loss of arm and leg functioning (e.g., Desrosiers et al., 2003), motor speech disorders (e.g., Mackenzie, 2011), vision disorders (e.g., Zhang, Kedar, Lynn, Newman, & Biousse, 2006), urinary incontinence (e.g., Divani, Majidi, Barrett, Noorbaloochi, & Luft, 2011), sleep difficulties (e.g., Sterr, Herron, Dijk, & Ellis, 2008), fatigue (e.g., Feigin et al., 2012), impaired cognitive functioning (e.g., Brady, Kelly, Godwin, & Enderby, 2012), personality changes (e.g., Stone et al., 2004), emotional difficulties, difficulties with family relationships, deterioration in sexual life, financial difficulties, deterioration in leisure activities, and reduced quality of life (e.g., Daniel, Wolfe, Busch, & McKevitt, 2009). The neurological symptom(s) a patient presents with are related to the area of the brain damaged. Table 1 highlights common neurological syndromes arising from ICH in specific locations in the brain.

Table 1

Neurological Syndromes Associated with ICH

Location	Possible Neurological Outcomes
Lobar	Coma, muscle weakness, delirium, sensory loss, hemianopia.
Supratentorial	Coma, hemiparesis, sensory loss, hemianopia, respiration problems, dilated and fixed pupils, decerebrate posturing, gaze palsy.
Thalamic	Coma, oculomotor abnormalities, contralateral eye deviation, unilateral hemiplegia, hemiparesis, unusual sensory syndromes, (E.g. spontaneous pain).
Putaminal	Coma, deviation of gaze, hemiplegia, hemiparesis, global aphasia, hemineglect.
Caudal	Disorientation/confusion, gaze paresis, hemiparesis.
Basal ganglion	Muscle weakness, sensory changes, motor inertia.
Pons	Coma, quadriplegia, decerebrate rigidity, locked-in syndrome, hypernea, hyperhydrosis, hyperthermia.
Midbrain and medulla	Coma, quadriplegia, hemiplegia, ocular-motor paralysis.
Cerebellar	Coma, vertigo, inability to walk or stand, limb ataxia, facial palsy, nystagmus, decreased corneal reflex, gaze paresis, abducens palsy.

Note. Adapted from Wiebers, Feigin, & Brown (2006).

Whilst all the areas across which stroke outcomes span deserve research, the body of literature available, mainly coming from a medical perspective, has emphasised physical functioning (Donovan et al., 2008) with neuropsychological outcomes (including both cognition and mood) being largely neglected (Dafer, Rao, Shareef, & Sharma, 2008)**.** In fact one review of over 8000 stroke outcome studies revealed that only 6% of studies included a cognitive or mood measure (Lees, Fearon, Harrison, Broomfield, & Quinn, 2012). This has led to a lack of understanding of the neuropsychological outcomes of stroke despite nearly 50% of stroke survivors having neuropsychological sequelae (Dennis, O'Rourke, Lewis, Sharpe, & Warlow, 2000).

This thesis therefore focussed on cognitive and mood outcomes; as well as the relationship of these outcomes to functional outcomes because it is important to look at a range of stroke outcomes that cover various areas of functioning, giving a more holistic view of the impact of the stroke on the patient as a whole (Patel, Coshall, Rudd, & Wolfe, 2002). Functional outcomes refers to both general functional outcomes, measured by independence in activities of daily living (ADLs),

and wider functional outcomes such as health related quality of life (HRQoL). Whilst physical and sensory limitations are also important predictors of wider functional outcomes (Patel et al., 2006) they were not examined in this thesis because, as noted at the outset, one of the aims of this thesis was to examine the independent impact of mood and cognitive difficulties on wider functional outcomes over and above any physical limitations. To approach these outcomes using a common language for researchers that crosses disciplines the World Health Organisation (WHO) framework is used in order that the content of this thesis can be more accessible to a range of people (Üstün, Chatterji, Bickenbach, Kostanjsek, & Schneider, 2003).

World Health Organisation (WHO) model of health outcomes.

In 2001 the WHO released a model for classifying the consequences of health conditions, known as the International Classification of Functioning, Disability, and Health (ICF; Üstün et al., 2003). This model aims to provide an international scientific tool for understanding peoples' functioning and disability in the context of clinical research, policy development, and other aspects of public health (Üstün et al., 2003). The ICF will therefore be the framework for reporting the outcomes of stroke used in this study.

According to the ICF model, functioning and disability can be understood as a dynamic interaction between health conditions and contextual factors, both relating to the individual person and their environment (WHO, 2001). Disability is the negative aspects of the interaction between a person with a health condition and their context and is therefore not an attribute of a person (WHO, 2011). Disability is an umbrella term which includes impairments ("problems in body function or structure such as significant deviation or loss"), activity limitations ("difficulties an individual may have in executing activities"), and participation restrictions ("problems an individual may experience in involvement in life situations"; WHO, 2001, p. 10). The interactional nature of disability outlined in the ICF model highlights the importance of studying multiple aspects of post-stroke outcomes; this will provide information about how factors are interacting to impact on the

individual's overall outcome. Figure 1 shows the broad domains of outcomes included in the ICF model.

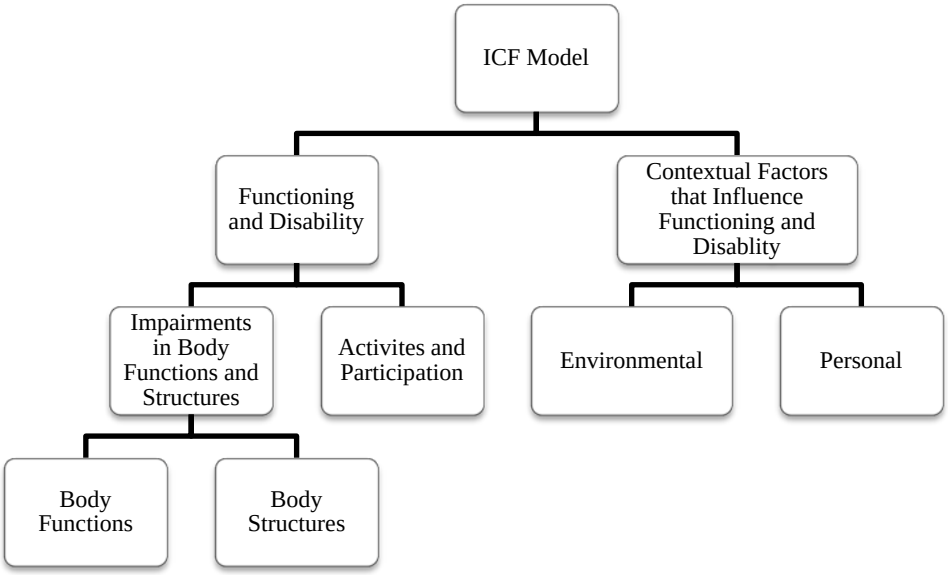

Figure 1. The ICF Model

A core set of ICF outcomes that relate specifically to stroke have been designated by consensus of experts in the field and are shown in Appendix A (Geyh et al., 2004). The outcomes of interest in this study can be placed within this core set of ICF stroke outcomes; mood outcomes are classed under 'Body Functions' and are listed simply as emotional functions (b152) in the core set; cognitive outcomes also are classed as 'Body Functions' and are made up of attention (b140), memory (b144), perceptual functioning (b156), higher level cognitive functions (b164), mental functions of language (b164), and calculation functions (b172). Functional outcome comes under the heading 'Activities and Participation' and includes carrying out daily routine (d230), washing oneself (d510), toileting (d530), dressing (d540), eating (d550), preparing meals (d630), doing housework (d640) and moving around (d455). This set of behaviours or skills which allow individuals to achieve independence in their ADLs is captured by general functional outcome. Wider functional outcome, which was also of interest in this thesis, refers to HRQoL which can be defined as an individual's happiness or satisfaction with domains of their life (including the

behaviours or skills listed above) insofar as they affect or are affected by health (Wilson & Cleary, 1995).

Each of these outcomes (mood, cognitive, general functional outcome [independence in ADLs], and wider functional outcome [HRQoL]) will now be discussed in turn, with reference to both acute and long-term outcomes. In line with other research in this area acute outcomes are considered to be within the first few days post-stroke up until 6 months post-stroke, whereas long-term outcomes will refer to 1 year or more post stroke (Barker-Collo & Feigin, 2006).

Mood outcomes of stroke and ICH.

Emotional sequelae, including depression and anxiety, are common post-stroke (Hackett, Kohler, O'Brien, and Mead, 2014), yet only 3% of stroke outcome studies include a measure of mood (Lees et al., 2012). The available literature on these outcomes is therefore limited, but will be reviewed in the following section.

Post-stroke depression (PSD).

Definition and causation of PSD.

Post-stroke depression (PSD) can be defined as clinically significant and persistent sadness and loss of interest/pleasure in activities following a stroke (Berg, Palomäki, Lehtihalmes, Lönnqvist, & Kaste, 2003). Stroke survivors with PSD commonly endorse feeling as if they are a burden on their family (97%), experiencing sadness (85%), and having crying episodes (85%) (Angelelli et al., 2004). The onset of PSD appears to be either within a few hours to days of the stroke (early onset PSD) or months after the stroke (delayed onset PSD; Whyte & Mulsant, 2002). Theories about the causal pathway of PSD tend to view either post-stroke structural changes within the brain as playing a central role or the psychosocial changes associated with experiencing a stroke as being key (Whyte & Mulsant, 2002).

According to the psychosocial theory of causation, if a stroke survivor does not have adequate coping skills or resources to deal with the dramatic changes and often loss of independence associated with a stroke then they may become overwhelmed and depressed (Whyte

& Mulsant, 2002). This view is consistent with the fact that stroke patients report that they have a loss of social contact and the valued roles which were embedded in everyday functions they had been able to perform pre-stroke (Pound, Gompertz, & Ebrahim, 1998; Sienkiewicz-Jarosz et al., 2010). To provide a specific example, stroke survivors have reported feeling a loss of control within their role as a spouse and a change in their sense of self (Thompson & Ryan, 2009).

Further evidence supportive of a psychosocial causation of PSD includes that risk factors for PSD have been identified that are not specific to stroke. Studies have found that the severity of disability is a risk factor for both PSD and idiopathic depression (depression without a known medical cause; Burvill et al., 1997; Murphy, 1982), although other studies, focusing on the acute phase post-stroke, have failed to support this association (Andersen et al., 1995; Berg et al., 2001). Research has also found that factors such as low education, low income (Sienkiewicz-Jarosz et al., 2010), living alone, having experienced social loss in the 6 months preceding stroke (Andersen et al., 1995; Bush, 1999), and having a psychiatric history increase the risk of PSD (Eastwood, Rifat, Nobbs, & Ruderman, 1989; Morris, Robinson, Raphael, Samuels, & Molloy, 1992; Quaranta, Marra, & Gainotti, 2012).

Some research has found that stroke survivors' cognitions likely play a role in the development of PSD, specifically, stroke survivor's satisfaction with advice they had received from medical professionals and their confidence in their recovery at 1 month post-stroke jointly explained 32% of the variance in PSD at 6 months post-stroke (Morrison, Johnston, & MacWalter, 2000). Other research has found that significant predictors of greater levels of depression in stroke survivors include lower levels of social support and family functioning, a tendency to use avoidance coping, and difficulty finding meaning in the experience of the stroke (King et al., 2002). Further support for psychosocial causation of PSD is that the typical symptom profile of PSD does not differ from idiopathic depression (Alexopoulos et al., 1997; Hopkins, 1986). However, because two disorders have the same symptoms does not necessarily mean they share the same causation.

In contrast to the psychosocial view, the biological model of PSD causation hypothesises that brain lesions caused by the stroke may directly affect neural circuits involved in mood regulation (Whyte & Mulsant, 2002). In support of this view depression has been reported in some studies as being far more common following stroke than other physical illnesses which result in a similar level of disability (Folstein, Maiberger, & McHugh, 1977; Nilsson, Kessing, Sorrensen, Andersen, & Bolwig, 2003); however, other studies have found no difference (Lieberman et al., 1999).

Some studies report that certain lesion locations, such as left anterior lesions, left basal ganglia lesions, and lesions closer to the frontal pole, are associated with a higher rate of PSD, with the strongest associations being found in the acute phase post-stroke (Whyte & Mulsant, 2002). It is important to keep in mind that these studies do not prove a direct causal link between brain damage and emotional problems; it may be that there is a mediating factor involved, such as executive dysfunction. Further, the effect sizes reported in lesion studies tend to be small (Whyte & Mulsant, 2002) and a systematic review by Carson et al. (2000) of research examining this issue concluded that lesion location within the brain does not have an impact on PSD outcome.

More recent research, however, indicates that stroke location in the left hemisphere is associated with increased risk of developing PSD (Barker-Collo, 2007). This relationship only holds true for early onset PSD, raising the possibility that that there may be different mechanisms for the development of acute and delayed-onset PSD (Bhogal, Teasell, Foley, & Speechley, 2004). Although there is a lack of relevant research available at the current time to draw any conclusions on whether different mechanisms are involved, one research finding that supports the hypothesis of biological mechanisms having a greater role in acute onset PSD is that the association between lesion location and PSD tends to be stronger in the first 6 months post-stroke (Whyte & Mulsant, 2002). Further, research determining that social isolation is a risk factor for depression at 1 year post-stroke but not immediately after stroke (Whyte & Mulsant, 2002) is also suggestive of a

possible psychosocial aetiology of delayed onset PSD. Whilst further research is needed to clarify the causation of PSD it seems likely that both psychosocial and biological factors may play a role.

Prevalence and issues in the assessment of PSD.

Regardless of its causation, PSD is an important outcome to assess because it has been found to be a common experience for stroke survivors and may impact on their recovery. Prevalence rates of PSD range from 20 to 50% within the first year, with a trend for this rate to be at its highest within the first 6 months following a stroke (Dafer et al., 2008). However, a systematic review found that the pooled estimate rate of PSD of 51 studies was 33% in the acute phase post-stroke and 34% in the long-term (Hackett, Yapa, Parag, & Anderson, 2005). A further systematic review of PSD literature found an average prevalence rate of 38% within a month of stroke, 31% at 1-6 months, 33% at 6 months to 1 year, and 25% at more than 1 year (Ayerbe, Ayis, Wolfe, & Rudd, 2013).

Depression rates will vary depending on the severity of symptoms being measured and method of assessment. Nys et al., (2005) found that within the first 3 weeks post-stroke 40% of patients exhibited mild depressive symptoms, while 12% experienced moderate to severe depressive symptoms. When the Diagnostic and Statistical Manual of Mental Disorders (DSM; American Psychiatric Association, 1980, 1987, 1994, 2000) criteria are used to diagnose depression, rates tend to be in the lower range of prevalence estimates (12% at 3 to 6 months post-stroke; Brodaty, Withall, Altendorf, & Sachdev, 2007). By contrast, when the Hospital Anxiety and Depression Scale (HADS) was used to assess stroke survivors at 2, 4, and 6 months post-stroke, rates of probable depression ranged between 24 and 30% (De Wit et al., 2008). When SAH survivors were asked to self-report mood problems at 1 year post-stroke the prevalence rate was 39% (Hackett & Anderson, 2000). By contrast Vykopal (2010) and also Gill (2011) found very low rates of depression using the General Health Questionnaire-28 (GHQ-28) to assess PSD in ICH and SAH survivors, but this is because the GHQ-28 measures depression in its most severe form. The pooled estimate of acute PSD has also been found to be slightly higher in hospital-based studies compared

to population-based studies (36% versus 33%; Hackett et al., 2005). It appears then that the disparity of prevalence rates of PSD reported in the literature is in part due to differences in the way depression is assessed and what sample is used (Dafer et al., 2008). It is also of interest to find out if the PSD rate varies according to stroke subtype; the few studies reporting on this have not found a significant difference in the rate (Carod-Artal, Egido, González, & De Seijas, 2000; Pohjasvaara, Vataja, Leppävuori, Kaste, & Erkinjuntti, 2001; Townend et al., 2007).

Course of PSD.

Multiple studies have shown that the prevalence of PSD varies over time, with depression in some cases having a chronic course (Berg et al., 2003; Brodaty et al., 2007; Burvill et al., 1995b). In a study by Berg et al. (2003) 46% of those people who experienced depressive symptomology at 2 months post-stroke were still experiencing these symptoms at either 12 or 18 month follow-up. Longer term studies have found that stroke and ICH survivors continue to have a higher rate of depression than controls at 5 years (prevalence of 30%; Bergersen, Frøslie, Stibrant Sunnerhagen, & Schanke, 2010; Gill, 2011; Lincoln et al., 2013; Vykopal, 2010) and 7 years post-stroke (prevalence of 20%; Dam, 2001). A systematic review of PSD literature found that the prevalence of depression remains stable up to 10 years post-stroke (Ayerbe et al., 2013). They further found that the rate of recovery from PSD ranged from 15 to 57% at 1 year post-stroke (Ayerbe et al., 2013).

Risk factors for PSD.

With regards to gender differences in the rates of PSD, many studies have found that women are significantly more likely to become depressed than men (Carod-Artal et al., 2000; Kotila, Numminen, Waltimo, & Kaste, 2003; Nys et al., 2006), with some studies reporting as much as a two-fold higher rate of both subjectively reported and objectively rated depressive symptomology in women compared to men (Cassidy, O'Connor, & O'Keane, 2004). A large review of relevant studies, however, concluded that women tend to be only slightly more likely to become depressed post-stroke; with the prevalence rate for women being higher in only 35 out of 47 studies (Poynter

et al., 2009). A study looking at ICH specifically did not find any significant gender difference in the rate of depressive symptomology (Vykopal, 2010).

Other baseline factors that have been found to be risk factors for developing PSD are younger age (Barker-Collo, 2007), low level of education and income (Altieri et al., 2012; Sienkiewicz-Jarosz et al., 2010), more severe physical disability (Demchuk & Buchan, 2000; Dennis et al., 2000; Hackett et al., 2005), and greater stroke severity (Hackett et al., 2005). A factor found to correlate with better emotional functioning post-stroke is having strong religious beliefs (Giaquinto, Spiridigliozzi, & Caracciolo, 2007). Risk of PSD has also been suggested to vary by level of development of a country. Consistent with this, a study in rural Tanzania found a high prevalence of PSD (53%) compared to developed countries (Jones et al., 2012). However, no such difference was found in a study in Uganda, with a prevalence rate of 31.5% reported for PSD (Gyagenda et al., 2015).

Post-stroke anxiety (PSA).

Definition and causation of PSA.

PSA, an umbrella term for a range of anxiety disorders in which severe anxiety is the central feature has, to some extent, been neglected by researchers (Campbell Burton et al., 2012; De Wit et al., 2008). To give an idea of the type of symptoms experienced by someone with PSA, Angelelli et al. (2004) found that PSA is primarily manifested by excessive preoccupation about upcoming events (71%), inability to relax and subjective tension (58%), and by separation anxiety (33%). Another study that used the Beck Anxiety Inventory to assess anxiety found that the most common PSA symptoms that participants endorsed were ‘fearing the worst’ (63%), feeling ‘scared’ (60%), and ‘fear of dying’ (59%; Barker-Collo, 2007). The symptoms endorsed by stroke survivors indicate that the high rate of anxiety recorded among this population is not merely a reflection of the physical and cognitive consequences of a stroke resulting in higher scores (e.g., it’s not just that they have trouble with attention which could be either a symptom of anxiety on a rating scale or directly due to a lesion rather than anxiety itself).

Studies that have reported the type of anxiety disorder according to the DSM classification system suggest that generalised anxiety disorder, agoraphobia, and specific phobias are common diagnoses post-stroke. Post-traumatic stress disorder (PTSD) has not appeared much in the PSA literature; however a recent pilot study found that 25% of stroke survivors had symptoms of PTSD with 10% of people overall meeting the DSM-IV-TR (American Psychiatric Association, 2000) criteria, suggesting this may also be a common disorder encountered in stroke survivors (Favrole et al., 2013).

Causal factors associated with PSA are not well established but, similarly to PSD, there is evidence that both biological and psychosocial variables may play a role (Vataja & Caste, 2013). Support for biological causation comes from studies which indicate that PSA is more common following lesions in certain regions of the brain. Several studies have found that survivors of stroke affecting the anterior circulation have a greater likelihood of experiencing PSA than those with strokes impacting the posterior circulation (Dennis et al., 2000; Leppavuori, Pohjasvaara, Vataja, Kaste, & Erkinjunth, 2003). PSA alone has also been found to correlate significantly with lesions in the right hemisphere (Tang et al., 2012), whereas PSA comorbid with PSD is associated with left hemisphere cortical lesions (Astrom, 1996; Castillo et al., 1993; Shimoda & Robinson, 1998). In terms of support for the role of psychosocial factors in PSA, some research indicates that cognitions may play a role in both initiating and exacerbating anxiety. Specifically, satisfaction with treatment and confidence in recovery at 1 month post-stroke explained 31% of the variance in PSA at 6 months (Morrison et al., 2000). Previous psychiatric history has also been found to correlate with early onset PSA, but not delayed onset PSA (Castillo et al., 1995), indicating that, similarly to PSD, there may be different causal mechanisms involved in early and late onset PSA. No further literature of interest was able to be found during the current literature review, thus no conclusions can safely be drawn at the current time on the causal mechanisms of PSA.

Prevalence and course of PSA.

Whilst the causation of PSA is not yet clear, research has established that PSA is common, having similar prevalence as PSD (Campbell Burton et al., 2012; De Wit et al., 2008). A systematic review of studies that assessed PSA found that the overall pooled estimate of anxiety disorders was 18% if anxiety was assessed by clinical interview and 25% if a rating scale was used (Campbell Burton et al., 2012). This latter estimate is consistent with a NZ study which found a self-rated prevalence rate of 21% for anxiety at 3 months post-stroke (Barker-Collo, 2007).

In terms of the timeframe post-stroke that people experience anxiety the combined prevalence rates of anxiety from Campbell Burton et al.'s (2012) review were 20% within 1 month of stroke, 23% between 1 to 5 months, and 24% at 6 months post-stroke or after (Campbell Burton et al., 2012). Studies which tracked anxiety to 3 (Åström, 1996) and 5 (Bergersen et al., 2010; Lincoln et al., 2013) years post-stroke found that there was no significant decrease in prevalence from the 6 month mark onwards. Burvill et al. (1995a) looked at the chronicity of PSA by following up a group of stroke survivors 1 year after they were diagnosed with anxiety at 4 months post-stroke; half no longer met criteria for an anxiety disorder, a quarter still had anxiety, and the remaining quarter had died. Castillo et al. (1995) looked at the prevalence of early onset PSA (beginning during hospital stay) and also delayed onset PSA (beginning after discharge) and found the rates to be 27% and 23% respectively.

Clearly then PSA is an ongoing issue for stroke survivors despite interventions being available to effectively reduce anxiety (Campbell-Burton et al., 2011). One review concluded that anxiolytic drugs combined with psychotherapy decreases the mean level of anxiety in stroke survivors with PSA by 71% compared to standard care (Campbell-Burton et al., 2011). The fact that high rates of PSA tend to persist for years post-stroke, despite effective interventions being available to reduce anxiety, may indicate that a lack of attention is being given to emotional functioning in routine post-stroke rehabilitation.

Risk factors for PSA.

Gender differences in PSA prevalence have been examined in the literature, with mixed results. PSA has been found to be more prevalent among female, compared to male, stroke survivors at 4 months (Burvill et al., 1995a), 1 year (Broomfield et al., 2014), 2 years (Schultz et al., 1997), and 3 years post-stroke (Morrison, Pollard, Johnston, & MacWalter, 2005), following the trend in the general population of greater prevalence of anxiety among women (Morrison, et al., 2005). In contrast, other research has failed to find a gender difference in PSA prevalence at 6 weeks (Schottke & Giabbiconi, 2015), 6 months (Dennis et al., 2000), and 3 years post-stroke (Astrom, 1996) and at 5 years post-ICH (Vykopal, 2010).

When considering factors that can account for the discrepancy in the findings listed above, it is possible that in the case of Vykopal's (2010) study the small sample size offered reduced power to detect a significant difference. Upon examination of Schottke and Giabbiconi's (2015) and Dennis et al.'s (2000) study it is noted that the sample reported on presented with less disability than found in the samples of those studies detecting a difference. This is because Schottke and Giabbiconi (2015) used a sample from a rehabilitation service which did not accept patients needing intensive medical treatment and Dennis et al. (2000) report that only 67% of their sample completed the Hospital Anxiety and Depression Scale (HADS) which was given to participants to mail back after an interview; those with greater disability were less likely to return the measure. Astrom's (1996) study was population-based on the other hand so was more representative of the range of disability experienced by stroke survivors. However, Astrom (1996) assessed only Generalised Anxiety Disorder, which is more restrictive than assessing anxiety more widely (as was reported in the studies of Broomfield et al., [2014], Shultz [1997], and Morrison et al [2005] study) or in accordance with all DSM anxiety disorders (as was the case with Burvill et al., [1995a] study). Burvill et al. (1995a) found that, in fact, Agoraphobia was the most common anxiety disorder present in their sample; thus, it is possible that Astrom's study offers limited scope for examination of PSA. Therefore, it appears that the impact of gender on PSA is not a straightforward relationship;

rather it is possible that factors such as level of disability and type of anxiety disorder assessed may play a role. Further research is needed to clarify the relationship between gender and PSA.

Age has also been examined as a risk factor for PSA in several studies. Findings were mixed with some research suggesting younger stroke survivors are at greater risk of PSA than older stroke survivors (Broomfield et al., 2014; Schultz et al., 1997;) and other research finding no correlation between age and PSA (Morrison et al., 2000). The sample used in Morrison's study, however, was not typical of stroke survivors because the women were significantly younger than the men in this hospital-based study. Further research is needed to determine whether younger age is in fact a risk factor for stroke. A recent systematic review of risk factors for PSA determined that PSD, stroke severity, and dementia or cognitive impairment were the main predictors of PSA (Menlove et al., 2015).

Interaction of PSA with PSD.

Comorbidity of depression and anxiety is high in stroke survivors (Åström, 1996; Barker-Collo, 2007; Castillo et al., 1995; Morrison et al., 2005; Schottke & Giabbiconi, 2015; Vykopal, 2010). Of those stroke survivors with PSA 75 to 85% will have comorbid major or minor depression (Åström, 1996; Castillo et al., 1995). NZ research has found a 12% prevalence of comorbid anxiety and depression at 3 months post-stroke (Barker-Collo, 2007) and determined that the positive correlation between symptoms of anxiety and depression remains even at 5 years post-ICH (Vykopal, 2010). Anxiety in the acute phase post-stroke had been found to predict depression at 6 months (Borst, 2009) and 3 years post-stroke (Morrison et al., 2005). The converse has not been found to be true, with acute depression post-stroke not having predictive utility in regards to later anxiety (Morrison et al., 2005). The predictive utility of anxiety in regards to depression but not vice versa has also been found in research looking at the general population and it has been suggested this may reflect anxiety as more determined by an underlying personality trait than depression (Wetherel et al., 200l). It is thought that PSA and PSD interact to result in greater severity and longer duration of emotional disturbance (Shimoda & Robinson, 1998). Specifically,

anxiety is thought to interact with depression to influence the level of impairment in ADLs and the course of recovery of social functioning (Shimoda & Robinson, 1998).

Cognitive outcomes of stroke and ICH.

Prevalence and issues in the assessment of post-stroke cognitive difficulties.

Only 5% of stroke outcome studies have included a measure of cognitive functioning (Lees et al., 2012) yet cognitive impairment is a common outcome of stroke with reported prevalence rates varying from 20 to 70% in the acute phase post-stroke and 21 to 56% in the long-term post-stroke (del ser et al., 2005; Douriri et al., 2013; Hochstenbach, Mulder, van Limbeek, Donders, & Schoonderwaldt, 1998; Patel et al., 2002, 2003; Pendlebury et al., 2012; Rasquin et al., 2004; Sachdev et al., 2009; Saxena et al., 2006; Serrano et al., 2007; Srikanth et al., 2003). These estimates likely vary as a result of a number of methodological differences between studies, including: 1) the criteria used to determine when 'cognitive impairment' is present, 2) the method of testing cognitive impairment, with full neuropsychological batteries included in some studies and only brief screening measures or self-reported accounts of cognitive impairment included in others, 3) the timeframe post-stroke that testing is carried out- with greater cognitive impairment in the acute stage (Gottesman & Hillis, 2010; Leśniak, Bak, Czepiel, Seniów, & Członkowska, 2008) compared to the long-term (Patel, Coshall, Rudd, & Wolfe, 2003; Rasquin et al., 2004), 4) the type of stroke being sampled (e.g., ischaemic versus haemorrhagic), 5) the location of the stroke lesion (e.g., left hemisphere lesions involving the cortex are associated with the greatest prevalence of cognitive problems; Nys et al., 2007), or similarly what circulatory system(s) the stroke affects- with more cognitive difficulties noted in strokes affecting the anterior compared to posterior circulation (Barker-Collo et al., 2012), 6) the sampling method used (e.g., population-based versus clinical study), and 7) the criteria used to exclude participants. In many studies people with aphasia are excluded due to difficulties in their completing the required assessments, which may lead to an underestimation of the prevalence of cognitive difficulties (Andersen et al., 1995).

To expand on the point that prevalence varies according to stroke type, research indicates that there is a higher rate of cognitive impairment following ICH compared to ischaemic stroke (Fuh & Wang, 1995; Nys et al., 2007; Su, Chen, Kwan, Lin, & Guo, 2007). In fact, in Nys et al.'s (2007) study it was found that despite there being no significant differences in lesion sizes and demographic characteristics between patients with ischaemic stroke and ICH, those with ICH were six times more likely to have cognitive impairments (Nys et al., 2007). Whilst no studies could be found that contradict the findings of those above, given the infancy of the research in this area and the limitations of some of the studies above (specifically the small sample size [n=6] used in Fuh and Wang's [1995] study and the fact Su et al.,'s [2007] study looked exclusively at a subtype of ICH-basal ganglia haemorrhage) further studies replicating those above would give more certainty around the level of cognitive impairment post-ICH. Nevertheless, it can be hypothesised that the apparent higher prevalence rate of cognitive deficits post-ICH may be due to the widespread effects of increased intracranial pressure associated with ICH which could result in more widespread cortical damage (Arboix et al., 2002).

Despite variations in the prevalence of cognitive difficulties it seems fair to assume that at least half of stroke patients on average will experience some form of cognitive difficulties within the first few weeks post-stroke (Nys et al., 2007; Saxena, 2006). A large scale population-based study supports this conclusion and notes improvement in prevalence of cognitive impairment at 3 months post-stroke (ischaemic stroke 32%, PICH 40%, and SAH 21%); however, there was little variation in these rates over the 15 year study follow-up period in which the same group of stroke survivors were assessed annually (Douiri, Rudd, & Wolfe, 2013). Being population-based this study provides a good sampling of the stroke community, and it is to its credit that it reports findings based on subtypes. However, a limitation of these findings is that cognitive evaluation was conducted using the Mini Mental State Exam (MMSE) or a similar measure called the Abbreviated Mental Test. These brief measures are, by definition, only screening tools for significant cognitive impairment, and cannot yield the same detailed picture of cognitive functioning that a battery of

neuropsychological tests can. As such the MMSE lacks sensitivity to mild cognitive impairment; one recent study found that the MMSE only picked up 70% of stroke survivors determined to have mild cognitive impairment by a full neuropsychological examination (Pendlebury, Mariz, Bull, Mehta, & Rothwell, 2012). Other screening tools such as the Montreal Cognitive Assessment (MoCA) and Addenbrooke's Cognitive Examination- Revised (ACE-R) have been found to be superior to the MMSE in terms of sensitivity in recent studies (Dong et al., 2010; Pendlebury et al. 2012).

While neuropsychological assessment can give a more detailed account of cognitive functioning, some researchers believe it is not feasible to conduct neuropsychological examination in the acute stage post-stroke because of factors like impaired arousal and fatigue. However, a study that conducted neuropsychological assessment 4 to 20 days post-stroke found that these were actually only a minor issue (Van Zandvoort, Kessels, Nys, De Haan, & Kappelle, 2005). In the same study three quarters of the sample completed the majority of the administered tasks within an acceptable time of 1.5 hours, which was interpreted as evidence substantiating the feasibility of neuropsychological examination in the acute stages post-stroke (Van Zandvoort et al., 2005). Despite the foregoing issues, which have impacted the reporting of neuropsychological deficits post stroke, it cannot be denied that cognitive deficits result from stroke (Barker-Collo & Feigin, 2006). The sections which follow review the available literature on the nature of these cognitive deficits, and attempts to identify patterns of findings within that literature.

Post-stroke dementia.

Whilst the following section will look at the profile of post-stroke cognitive deficits in general, this section will focus on those people who have the most severe cognitive difficulties- qualifying for a diagnosis of post-stroke dementia. Dementia is a clinical syndrome caused by neurodegeneration and characterised by a progressive deterioration in cognitive functioning and capacity to live independently (Prince et al., 2013). Post-stroke dementia can be defined as any dementia occurring after a stroke, irrespective of if the leading cause is vascular, degenerative, or

mixed (Pasi et al., 2012). The combination of white matter changes, cerebrovascular lesions, and pre-existent asymptomatic Alzheimer lesions may induce post-stroke dementia, even though each lesion on its own may not have been severe enough to induce dementia (Mackowiak & Pasquier, 2013). According to a large review, the pooled estimate for the prevalence rate of dementia for survivors of a first ever stroke is 7 to 12% and 41% for recurrent stroke survivors (Pendlebury & Rothwell, 2009). Estimates from other studies have noted prevalence to be in the range of 4 to 23% in the acute phase post-stroke (del Ser et al., 2005; Rasquin et al., 2004; Sachdev et al., 2004; Tham et al., 2002) and 8 to 23% in the long-term (Feigin et al., 2010b; Rasquin et al., 2004). The majority of the studies reported here used the DSM-IV (American Psychiatric Association, 1994) criteria to diagnose dementia, which requires evidence of impairment in memory and at least one other cognitive domain, and took into account a range of information including performance on a neuropsychological test battery. A diagnosis of dementia was only made if at least two trained professionals independently agreed. This likely resulted in greater validity of the results compared to if a screening tool was used, given the limitations of screening tools noted in the previous section.

In regard to the profile of cognitive difficulties associated with dementia, a similar typical pattern of cognitive deficits is noted in both people with post-stroke cognitive difficulties and with post-stroke dementia; however, the severity of deficits and prevalence of memory problems is greater in those with dementia (Barker-Collo & Feigin, 2006; Nyenhuis et al., 2004; Sachdev et al., 2004; Stephens et al., 2004). This indicates that cognitive difficulties post-stroke may lie on a continuum, with dementia being one of the extremes. However, it should be noted that post-stroke mild cognitive impairment does not always progress to dementia and in fact improvement in cognitive functioning can occur (Moorhouse & Rockwood, 2008). Research has found that at 2 years post-stroke 14% (del Ser et al., 2005) to one third (Rasquin et al., 2004) of survivors originally diagnosed as having dementia are reclassified as having mild cognitive difficulties. This is not surprising considering post-stroke memory difficulties, one of the criteria for a dementia

diagnosis, can show improvement (Snaphaan & de Leeuw, 2007). Given that for many people the term dementia is associated with permanence and worsening of cognitive deficits, its use in the post-stroke setting may be misleading for stroke survivors and their families (de Haan et al., 2006).

Just as there is improvement in cognitive functioning over time for some stroke survivors, for others cognitive functioning can deteriorate. In a study by Tham et al. (2002) at 1 year post-stroke 11% of survivors who were considered to only have mild cognitive difficulties at 3 months had worsened to fit the criteria for dementia. Stroke survivors most at risk of progressing to dementia appear to be those with poor memory and functional impairment in the acute stage post-stroke (Ingles, Wentzel, Fisk, & Rockwood, 2002).

Pattern and course of post-stroke cognitive difficulties.

Whilst post-stroke dementia may be the outcome for some stroke survivors, a greater proportion will experience milder cognitive difficulty which is the focus of this section. A summary of literature which has examined cognitive functioning in stroke survivors and ICH survivors specifically is presented in Tables 2 and 3 respectively. As can be seen in Table 2 research looking at cognitive functioning in both the acute and long-term stages post-stroke has found that impairment across a range of cognitive domains is common including impairments in executive functioning (prevalence of 19-39% [acute] and 14-33% [long-term]; Barker-Collo & Feigin, 2006; Nys, Van Zandvoort, De Kort, Jansen et al., 2005; Nys et al., 2005b; Nys et al., 2007; Rasquin, Verhey, Lousberg, & Lodder, 2005; Vataja et al., 2003), processing speed (prevalence of 46-70% [acute] and 33-49% [long-term]; Barker-Collo & Feigin, 2006; Hochstenbach et al., 1998; Nys et al., 2004b; Nys et al., 2005; Rasquin et al., 2004; Rasquin et al., 2005), attention (prevalence of 35-49% [acute] and 31% [long-term]; Duits et al., 2008; Lesniak et al., 2008; Nys et al., 2004b), memory (prevalence of 17-40% [acute] and 15-50% [long-term]; Duits et al., 2008; Hackett & Anderson, 2000; Hochstenbach et al., 1998), visuospatial/visuoperceptual abilities such as hemispatial neglect (prevalence of 23-40% [acute] and 3-30% [long-term]; Barker-Collo et al., 2010; Gottesman & Hillis, 2010; Hochstenbach et al., 1998; Nys et al., 2004b; Nys et al., 2005; Nys

Table 2

Cognitive Outcome of Stroke

Author/ Study	Sample	Timing	Assessment	Outcomes
Studies using only brief/screening measures				
Douiri et al. (2013)	1,618 stroke survivors, population-based	3 months & 1, 5, & 14 years post-stroke	MMSE or AMT	Prevalence of cognitive impairment: 3 months- 24%, 1 year- 22%, 5 years- 22%, & 14 years-21%.
Hackett & Anderson (2000)	230 SAH survivors, population-based	1 year post-SAH	Asked series of yes/no questions about memory & speech	50% reported memory problems & 14% reported problems with speech.
Leonard et al. (1998)	68 stroke survivors, consecutive discharge	1, 3, & 5 years post-discharge	Cognitive component of the FIM	Significant ↑ in cognitive functioning in the 1st year after discharge. Significant & steady ↓ from 3-5 years.
Patel et al. (2002)	645 stroke survivors, population-based	3 months post-stroke	MMSE	38% of sample cognitively impaired
Patel et al. (2003)	163 stroke survivors, population-based	3 months and 1, 3, & 4 years post-stroke	MMSE	At 3 months, 1, 2, & 3 years post-stroke, the prevalence of cognitive impairment was 39%, 35%, 30% & 32% respectively.
Rasquin et al. (2004)	196 stroke survivors, consecutive admission	1, 6, & 12 months post-stroke	CAMCOG	At 1, 6, & 12 months prevalence of MCI was 71%, 61%, & 56% and of dementia was 11%, 8%, & 8% respectively. Mental speed & calculation most frequently affected.
Saxena (2006)	200 stroke survivors, consecutive admission	On admission	AMT	55% cognitively impaired.
Wolfe et al. (2011)	3,773 stroke survivors, population-based	3 months, & annually thereafter until 10 years post-stroke	MMSE	Prevalence of cognitive impairment declined up to 1 year; thereafter the rate fluctuated till year 8, & then increased.
Studies using a comprehensive range of measures				
Ballard et al. (2003)	115 stroke survivors, consecutive admission	3 & 15 months post-stroke	MMSE, CAMCOG, CDRCB, BNT, & VF	Over time 50% of sample ↑ in cognitive functioning, 30% ↓, & 9% developed dementia.
Barker-Collo et al. (2010)	307 stroke survivors, population-based	5 years post-stroke	CVLT-2, WMS subtests LM & VPA, ROCFT, BNT, COWA, WAIS subtests BD & MR, Bells	Greatest % of sample in average range for neuropsychological functioning, 30-50% performed < average on most tests, few performed > average. Deficits most common in information

			Test, Trails A/B, Stroop, IVA CPT	processing speed & executive functioning.
Barker-Collo et al. (2012)	315 IS survivors, population-based	5 years post-IS	CVLT-2, WMS subtests LM & VPA, ROCFT, BNT, COWA, WAIS subtests BD & MR, Trails A/B, Stroop, IVA CPT	A range of impairments observed; tasks reliant on executive functioning and processing speed most impaired. Sorting strokes based on vascular distribution affected lead to significantly different cognitive profiles.
Brodaty et al. (2005)	167 IS survivors, consecutive admission	3-6 months post-IS	WAIS-R, WMS-R, MMSE, BNT, CFST, VF, Trails A/B, SDMT	IS survivors scored significantly worse than controls in all domains of cognitive functioning measured.
del ser et al. (2005)	193 stroke survivors, consecutive admission	3, 6, 12, & 24 months post-stroke	SS-IQCODE, SPMSQ, MMSE, visual and hearing reaction time, bell test, VF, picture recognition, word learning, WMS subtest LM, WAIS subtest BD & SM, BNT, & Token Test	3 months post-stroke 72% cognitively normal, 9% had cognitive decline without dementia & 19% demented. At 24 months post-stroke cognitive status was stable in 78%, had ↓ in 14% & had ↑ in 8%.
Desmond, Moroney, Sano, & Stern (1996)	151 IS survivors, consecutive admission	3 months & then annually post-IS	BVRT, SRT, MMSE, BNT, VF, Rosen Drawing Test, WAIS-R subtest SM, MDRS, & cancellation task	13% showed ↑ in cognitive functioning; increase evident only at the 1st annual examination for the majority.
Duits et al. (2008)	61 stroke survivors, consecutive discharge	2 & 7 weeks post-stroke	CLCE-24, RCPM, RBMT, Trails A/B, TLT, & category fluency	Subjective complaints; attention (38%), mental speed (46%), & memory (38%). If subjective complaints did worse than those who reported no cognitive difficulties on all tests.
Engstad, Almkvist, Viitanen, & Arnesen (2003)	199 stroke survivors, population-based	9 years post-stroke	MMSE, ROCFT, COWA, digit span, Trails A/B, finger tapping, & naming task.	Significantly poorer motor speed, episodic memory, visuospatial functioning, & verbal fluency.
Feigin et al. (2010b)	418 stroke survivors, population based	5 years post-stroke	CVLT-2, WMS subtests LM & VPA, ROCFT, BNT, COWA, WAIS subtests BD & MR, Bells Test, Trails A/B, Stroop, IVA CPT, & AMT	22.5% had cognitive impairment indicative of dementia. 1 year ↑ in age associated with a 5% ↑ risk of cognitive problems. Being non-European (Maori, Pacific, Asian, other) was associated with 62-67% ↑ risk of cognitive problems, language, visuoperceptual, or information processing speed impairment.

Gill (2011)	27 SAH survivors, population based	5 years post-SAH	HAMT, CVLT-2, WMS subtests LM & VPA, ROCFT, BNT, COWA, WAIS subtests BD & MR, Trails A/B, Stroop, IVA CPT	Performed significantly worse than controls in all domains of cognitive functioning assessed; executive functioning, information processing, verbal memory, visual memory, visuoperceptual ability, & language.
Hochstenbach et al. (1998)	229 stroke survivors, consecutive admission	On average 72 days post-stroke	RAVLT, WAIS subtests IN, DS, BD, & SM, RBMT, Trails A/B, Letter Cancellation Task, BIT, CDT, MRM, the Bobertag, VF, & naming task	> 70% showed a marked slowness of information processing. At least 40% of the sample had difficulty with memory, visuospatial and constructive tasks, language skills, and arithmetic.
Hochstenbach, Otter, & Mulder (2003)	65 stroke survivors, consecutive admission	2 & 27 months post-stroke	RAVLT, RBMT, Trails A/B, WAIS-III subtests DS, CN, SM, & BD, CDT, BIT, MRM, comprehension, & VF	Over time significant ↑ in all cognitive domains; largest improvement in language & attention and least improvement in memory.
Ingles et al. (2002)	125 people with vascular cognitive impairment-no dementia, randomly selected in survey	At recruitment & 5 years post-recruitment into study	BVRT, Buschke's Cued Recall Test, digit span, RAVLT, WMS subtest IN, WAIS-R subtests SM, CO, BD, & DS, Token Test, COWA, & naming	After 5 years 45% of the sample (all who had MCI) had dementia. Low baseline scores on tests of memory & category fluency best predicted progression to dementia.
Jonkman et al. (2009)	35 IS (in MCA) survivors, consecutive admission	3, 6, & 12 months post-IS	WAIS-R, WMS, & Brus Reading Test	Cognition significantly impaired compared to controls & estimated premorbid ability at all 3 time periods in study.
Kauhanen et al. (1999)	106 IS survivors, consecutive admission	3 & 12 months post- IS	WAIS-R subtests BD, PC, CO, & SM, WMS subtests LM, & VR, serial learning & interference task, visual recognition memory task, Trails A, VF, copy tasks & CDT	Association between PSD & cognitive impairment. PSD= significantly ↑ impairment in memory, nonverbal problem solving, attention & psychomotor speed.
Lesniak et al. (2008)	200 stroke survivors,	2 weeks & 1 year post-	List learning, CDT, Line	2 weeks; deficits in attention (48.5%), language (27%), short-

	consecutive admission	stroke	Cancellation, arithmetic task, naming & repetition task, Trails A/B, VF, & the 'go, no-go test'	term memory (24.5%), & executive functions (18.5%). 1 year; attention deficits most frequent. Frequency of executive dysfunction, aphasia, and long-term memory disorder ↓.
Nyenhuis et al. (2004)	103 IS survivors, consecutive admission	Within 6 months of IS	BDAE, Controlled Learning & Enhanced Recall, Self-Ordered Pointing Task, WMS-III subtests LM & DS, BDS, MMSE, Figural Recognition Test, BNT, VF, RPM, & Grooved Pegboard Test	MMSE score correlated with cognitive impairment determined by test battery. Worst impairment in immediate recall & psychomotor speed.
Nys et al. (2007)	190 stroke survivors, consecutive admission	Within 3 weeks post-stroke	RAPM, WAIS-III subtests SM & DS, RAVLT, BSAT, visual elevator, VF, LO, Test of Facial recognition, ROCFT, Token Test, BNT, & Star Cancellation	MCI: IS-53% & ICH-80%. Deficits in executive functioning (39%), visual perception/construction (38%), neglect (31%), abstract reasoning (26%), verbal memory (26%), language (36%) and visual memory (22%). More pronounced deficits in executive functioning, language, verbal memory & abstract reasoning following left cortical stroke. ICH independent determinant of acute MCI.
Nys et al. (2005b)	111 stroke survivors, consecutive admission	At admission & 7 months post-stroke	RAPM, WAIS-III subtests SM, DS, & BD, BNT, Token Test, CRT, RAVLT, WMS-R subtests LM &VR, Corsi Block Span, modified Location Learning task, BSAT, Visual Elevator, VF, semantic fluency, Stroop, Zoo Test, BLO, & face recognition	Recovery in visual perception/construction (83%) & visual memory (78%) common. An acute cognitive disorder (except for visual perception/construction) predicted a long-term disorder in the same domain.
Nys et al. (2005c)	168 stroke survivors, consecutive admission	Within 3 weeks & 7 months post-stroke	RAPM, WAIS-III subtests SM, DS, & BD, BNT, Token Test, CRT, RAVLT, WMS-R subtests LM & VR, Corsi Block Span, BSAT, Visual Elevator, VF,	Abstract reasoning & executive functioning impairments independent predictors of long-term cognitive impairment. Inattention & perceptual disorders were more important in predicting long-term functional impairment.

			semantic fluency, Stroop, Zoo Test, face recognition, star cancellation, & line bisection	
Pendlebury et al. (2012)	200 stroke survivors, consecutive admission	Between 1 & 5 years post-stroke	ACE-R, MOCA, MMSE, Trails A/B, SDMT, BNT, ROCFT, HVLT-R, & COWA	MCI- 39%. MOCA & ACE-R more sensitive to impairment than MMSE.
Rasquin et al. (2002)	139 stroke survivors, consecutive admission	1 & 6 months post-stroke	RAVLT, Stroop, CST, GIS, CAMCOG	↑ in: overall cognitive functioning-13%, memory-52%, cognitive flexibility-42%, & simple speed-37%.
Rasquin et al. (2005a)	118 stroke survivors, consecutive admissions	1, 6, 12, & 24 months	RAVLT, Stroop, CST, GIS, CAMCOG	20% of those with MCI at 1 month had normal functioning at later time. Most recovery occurred between 1-6 months post-stroke.
Rasquin et al. (2005b)	101 stroke survivors, consecutive admissions	1 & 24 months	RAVLT, Stroop, CST, GIS, CAMCOG	23% had progressive vascular brain damage- associated with worse performance in all domains. Improvement over time- particularly in executive functioning & abstract reasoning.
Sachdev et al. (2004)	170 stroke or TIA survivors, consecutive admission	3-6 months post-stroke or TIA	WMS-R subtests LM, BD, SM, PC, & VR, WAIS-R subtests DS & AR, BNT, Trails A/B, Western Aphasia Battery, CFST, VF, & NART-R	% with no impairment, MCI, & dementia was 41%, 36%, & 23% respectively. Dementia- disturbance in all cognitive domains (verbal memory most affected). MCI- similar but less severe disturbance.
Serrano et al. (2007)	327 stroke survivors, consecutive admission	3, 12, & 24 months post-stroke	MMSE, Short Portable Mental Status Questionnaire, visual reaction time, Bell Test, VF, WAIS subtests PC, SM, & BD, WMS subtest LM, naming, Token Test, & word learning	% with MCI at 3, 12, & 24 months was 20%, 27%, & 21% respectively.
Srikanth et al. (2003)	99 stroke survivors, population based	3 months post-stroke	WAIS-R subtests IN, DS, SM, AR, PC, BD, & DS, MMSE, RAVLT, RBMT, ROCFT, COWA, & CDT	1.5 times more likely than controls to have MCI. Most likely impairments: attention, spatial ability, executive function, & language.

Stephens et al. (2004)	381 stroke patients, consecutive admission	3 months post-stroke	CAMCOG, CDRCB	MCI: deficits in executive functioning, memory, & language. Dementia: similar profile but deficits more pronounced.
Tatemichi et al. (1994)	227 IS survivors, consecutive admission	3 months post-IS	SRT, BVRT, BNT, MMSE, VF, comprehension task, Rosen Drawing Test, WAIS-R subtest SM, target detection task	Cognitive impairment greater than controls (35% versus 4%). Memory, orientation, language, & attention most impaired.
Tham et al. (2002)	252 IS or TIA survivors, consecutive admission	Admission & 1 year post-IS or TIA	WAIS-R subtests DS & BD, Auditory Detection Test, BNT, VF, word list recall, story recall, WMS-R subtest VR, CDT, Visual Cancellation Task, & maze task	Admission: 40% MCI & 4% dementia. 1 year: 31% with MCI no longer impaired, 11% deteriorated from MCI to dementia.
Van Zandvoort et al. (2005)	57 IS survivors, consecutive admission	4-20 days & 12-24 months post-IS	WAIS subtests DS & VO, RAPM, BNT, VF, CBTT, RAVLT, Doors, ROCFT, LO, Test of Facial Perception, & Trails A/B	Acute stage: 77% could complete 82% of the tasks. Domain scores on intellectual functioning, language, memory, perception &visuospatial construction, attention & psychomotor-functioning were predictive of functional outcome.
Vataja et al. (2003)	214 IS survivors, consecutive admission	3 months post-IS	MMSE, Trails A/B, Stroop, WCST, & VF	Executive dysfunction- 24%. MMSE score independently correlated with executive dysfunction.

ACE-R= Addenbrooke's Cognitive Examination Revised; AMT= Abbreviated Mental Test; AR= Arithmetic; BD= Block Design; BDAE: Boston Diagnostic Aphasia Exam; BIT= Behavioural Inattention Test; BLO= Benton Line Orientation; BNT= Boston Naming Test; BSAT= Brixton Spatial Anticipation Test; BVRT= Benton Visual Retention Test; CBTT= Corsi Block Tapping Test; CDRCB= Cognitive Drug Research Computerised Battery; CDT= Clock Drawing Test; CFST= Colour Form Sorting Test; CLCE-24 Checklist for Cognitive and Emotional Consequences following stroke; CN= Cancellation; CO= Comprehension; COWA= Controlled Oral Word Association Test; CRT= Chapman Reading Task; CST= Concept Shifting Test; CVLT-2= California Verbal Learning Test (2nd edition); DS= Digit Span; FIM= Functional Independence Measure; GIS= Groninger Intelligence Scale; HAMT= Hodkinson Abbreviated Mental Test; HVLT= Hopkins Verbal Learning Test; IN= Information; IS= Ischaemic Stroke; IVA CPT= Integrated Visual Auditory Continuous Performance Test; LM= Logical Memory; LO= Line orientation; MCA= Middle Cerebral Artery; MCI= Mild Cognitive Impairment; MDRS= Mattis Dementia Rating Scale; MMSE= Mini Mental State Examination; MR= Matrix Reasoning; MRM= Money's Road Map; PC= Picture Completion; RAPM= Raven Advanced Progressive Matrices; RCPM= Raven Coloured Progressive Matrices; RAVLT= Rey Auditory Verbal Learning Test; RBMT= Rivermead Behavioural Memory Test; ROCFT= Rey Osterrieth Complex Figure Test; SAH= Subarachnoid Haemorrhage; SDMT= Symbol Digit Modalities Test; SM= Similarities; SPMSQ= Short Portable Mental Status Questionnaire; SRT= Selective Reminding Test; SS-IQCODE= Shortened Spanish version of Informant Questionnaire of Cognitive Decline in the Elderly; TIA= Transient Ischaemic Attack; TLT= Tower of London Test; VF= Verbal Fluency; VPA= Visual Paired Associates; VO= Vocabulary; VR= Visual Reproduction; WAIS= Wechsler Adult Intelligence Scale; WCST= Wisconsin Card Sorting test; WMS= Wechsler Memory Scale.

Table 3

Cognitive Outcome of ICH

Author/ Study	Sample	Timing	Assessment	Outcomes
Studies using only brief/screening measures				
Liang et al. (2001)	140 survivors of left basal ganglia haemorrhage, consecutive admission	3 months post-haemorrhage	Aphasia scale of the Scandinavian Stroke Scale	Proportion that had severe, moderate, mild, or no aphasia was 21%, 17%, 31%, & 31% respectively.
Studies using a comprehensive range of measures				
Fuh & Wang (1995)	6 survivors of caudate nucleus haemorrhage, consecutive admission	2 weeks post-haemorrhage	MMSE, DS & CO, Proteus Maze-like Maze Test, Delayed Picture Recognition Span Test, Paired Word Learning Test, Remote Memory Test, Word & Sentence Reading Test, Number Reading & Writing Test, Simple Calculation Test, Pattern Matching Test, Simple Figure Copying Test & VF	Significant impairment in short- & long-term memory & VF compared to controls. MMSE scores no different to controls.
Ibayashi, Tanaka, Joanette, & Lecours (1992)	42 survivors of thalamic or putaminal haemorrhage, consecutive admission	At admission & 3 months post-haemorrhage	SLTA, WAIS, RCPM, Miyake's Memory Test, WAIS, & BVRT	Aphasia at admission- 73%; this persisted past 1 month for 35%.
Maeshima et al. (1997)	22 survivors of thalamic haemorrhage, consecutive admission	Within 30 days of haemorrhage onset	SLTA, line cancellation, line bisection, figure copying, MMSE, VF, & digit span	56% of patients with left side lesion had aphasia. 77% of patients with right side lesion had neglect.
Su et al. (2007)	30 survivors of basal ganglia haemorrhage,	6 months post-haemorrhage	WAIS-R subtests DS &BD, WMS-R subtest VMS, MMSE, Hooper Visual	96%- defective performance on at least 3 tests (controls-0%), 60%- defective performance on 7 or more

	consecutive admission		Organisation Test, Test of Visual Perceptual Skills, BVRT, WCST, Luria-Nebraska Neuropsychological Battery	tests. Deficits in visuospatial function & memory most pronounced but also impaired executive functioning, language, & attention.
Vypokal (2010)	19 ICH survivors, population based	5 years post-ICH	Bells, Trails A/B, Stroop, WAIS-II subtests MR & BD, ROCFT, COWA, CVLT-II, WMS-III subtests VPA & LM, BNT, IVA CPT	Significantly poorer functioning than controls. Proportion impaired in: Executive functioning-30%, information processing-36%, verbal memory-13%, visual memory-0%, visuoperception-13%, & language-0%.

BD= Block Design; BNT= Boston Naming Test; BVRT= Benton Visual Retention Test; CO= Comprehension; COWA= Controlled Oral Word Association Test; CVLT-II= California Verbal Learning Test-II; DS= Digit Span; IVA CPT= Integrated /visual Auditory Continuous Performance Test; LM; Logical Memory; MMSE= Mini Mental State Examination; MR= Matrix Reasoning; RCPM= Raven Coloured Progressive Matrices; ROCFT= Rey Osterrieth Complex Figure Test; SLTA= Standard Language Test of Aphasia; VF= Verbal Fluency; VMS= Visual Memory Span; VPA= Verbal Paired Associated; WAIS= Wechsler Adult Intelligence Scale; WCST= Wisconsin Card Sorting Test; WMS=Wechsler Memory Scale.

et al., 2007; Rasquin et al., 2005), speech/language (prevalence of 21-40% [acute] and 14-26% [long-term]; Barker-Collo & Feigin, 2006; Gottesman & Hillis, 2010; Hochstenbach et al., 1998; Nys et al., 2004b; Nys et al., 2005b) and calculation skills (prevalence of 40-52% [acute] and 37% [long-term]; Hochstenbach et al., 1998; Rasquin et al., 2004a).

Whilst it is clear that after stroke in general there is a range of cognitive deficits which survivors may experience, less is known about the pattern of cognitive deficits associated with the various subtypes of stroke; there is currently inadequate data available to establish a typical neuropsychological profile following ICH (Barker-Collo & Feigin, 2006). What is known about cognitive functioning after ICH is that the proportion of survivors who experience deficits is high; reported to be in the range of 80 to 96% in the acute phase post-ICH (Nys et al., 2007 & Su et al., 2007). The proportion of ICH survivors who experience aphasia in the acute phase post-ICH was found to be 69 and 73% in two studies examining this (Ibayashi et al., 1992; Liang et al., 2001). Fuh and Wang (1995) found that at 2 weeks post-ICH the greatest cognitive impairment seen among survivors related to the domains of short- and long-term memory and verbal fluency. Another study has found that at 6 months post-ICH survivors have impairments in the domains of executive functioning, language, attention, visuospatial functioning and, memory; with the latter two domains showing the greatest difference compared to controls (Su et al., 2007).

In terms of the course of cognitive deficits, as can be seen in Table 2, there appears to be some spontaneous recovery of cognitive functioning within the first year post-stroke (Gottesman & Hillis, 2010; Rasquin et al., 2004; Rasquin, Lodder, & Verhey, 2005), commonly in the domains of memory and visuospatial/visuoperceptual functioning (Nys et al., 2005). When cognitive functioning was tracked from 3 months to 2 years post-stroke it was found that 78% of stroke survivors remained stable, 14% got worse, and 8% improved (del Ser et al., 2005). Therefore, the prevalence rate of cognitive functioning from 3 months

onwards does not appear to fluctuate greatly, which is congruent with Patel et al.'s (2003) finding that the prevalence of cognitive impairment was 39% at 3 months and 32% at 3 years post-stroke and Serrano et al.'s (2007) finding that cognitive impairment had a 20% prevalence at 3 months and a 21% prevalence at 2 years post-stroke (Serrano, Domingo, Rodríguez-Garcia, Castro, & del Ser, 2007). This may explain why cognitive deficits in the first few months post-stroke, such as impairments in abstract reasoning and executive functioning, have been found to be independent predictors of seven month cognitive outcome (Nys, Van Zandvoort, De Kort, Van der Worp et al., 2005).

Given the persistence of cognitive deficits post-stroke it is not surprising that at 3months (Jokinen et al., 2015), 1 year (Hackett & Anderson, 2000; Leśniak et al., 2008), 5 years (Barker-Collo et al., 2012; Gill, 2011; Vykopal, 2010), 10 years (Wolfe et al., 2011), and even 15 years (Douiri et al., 2013) post-stroke impairments across the major domains of cognitive functioning can still be found, with impairments in processing speed and executive dysfunction being the most common (Barker-Collo et al., 2010; Barker-Collo et al., 2012; Jokinen et al., 2015). At 3 months post-stroke neuropsychological testing showed that even those stroke survivors who had excellent clinical recovery (i.e., no functional impairment) had a 71% likelihood of cognitive impairment in at least one domain (Jokinen et al., 2015). This demonstrates that even in the cases where there is no obvious disability experienced by a stroke survivor the impact of the stroke on cognition may be ongoing. When neuropsychological assessment was conducted at 5 years post-stroke the majority of stroke survivors performed in the average range overall, with 30 to 50% performing at lower levels on most tests and few people performing above the average range (Barker-Collo et al., 2010). This suggests that stroke may in some way impose a ceiling on level of cognitive functioning, although additional research is needed on the subject.

In terms of ICH, whilst there is limited evidence about the course of cognitive deficits, the only study known to assess long-term cognitive outcome of ICH found that at 5 years post-ICH survivors had significantly poorer cognitive functioning compared to controls (Vypokal, 2010). The proportion of the ICH survivors that had deficits in the cognitive domains Vypokal (2010) examined were as follows; information processing speed-36%, executive functioning-30%, verbal memory-13%, visuoperception-13%, visual memory-0%, and language-0%. This study indicates that cognitive impairments persist in ICH survivors, although more research is needed to determine the exact course of cognitive deficits.

Factors associated with cognitive impairment.

Cognitive impairment at 3 months post-stroke was found to be independently associated with older age, ethnic minority groups, low socioeconomic class, left hemisphere stroke, and urinary incontinence in a large population-based study (Patel et al., 2002). An example of how ethnicity may relate to risk of cognitive deficits can be seen in a NZ study in which non-Europeans had a 62 to 67% increased risk of having serious cognitive problems, language, visuoperceptual, and information processing speed deficits compared to Europeans at 5 years post-stroke (Feigin et al., 2010).

Stroke severity (Burton et al., 2003; Nys et al., 2007; Sachdev et al., 2007; Stebbins et al., 2008), education level (Rasquin, Verhey, Van Oostenbrugge, Lousberg, & Lodder, 2004), and epileptic seizures soon after stroke (Cordonnier, Hénon, Derambure, Pasquier, & Leys, 2007) have also been found to be associated with cognitive functioning post-stroke.

Research has found that women have a higher rate of cognitive deficits post-stroke than men (Jacquin et al., 2014). A factor that may contribute to this observed difference is that women tend to have strokes at an older age (Appeleros et al., 2009; Gottesman & Hillis, 2010; Petrea et al., 2009; Roquer, Campello, & Gomis, 2003), with risk appearing to increase post-menopause (Haast, Gustafson, & Killian, 2012). Further, the types of strokes that

women are more prone to may be more associated with increased risk for cognitive deficits (Gottesman & Hillis, 2010). Specifically, women have more cardioemoblic strokes compared to men, who are more likely to suffer atherothrombotic and lacunar strokes (Roquer et al., 2003) and women are also at higher risk than men of stroke from arrhythmias or coronary revascularisation (Paulus et al., 2016). Males are also noted as having a higher percentage of cognitive recovery over the first year post-stroke compared to females (Patel et al., 2003)

A study that specifically looked at variables related to cognitive deterioration over the first 2 years post-stroke found that age, mental decline before stroke, and polypharmacy were significant risk factors for progression of cognitive deficits (del Ser et al., 2005). The findings of Nys et al. (2005) also supported age as an important factor for cognitive recovery and additionally pre-existent verbal ability and lesion volume.

When ICH is looked at specifically, neurological and physical functioning (Vypokal, 2010) as well as previous stroke (Srikanth et al., 2003) significantly correlate with cognitive functioning (Vykopal, 2010). No gender difference was noted in Vykopal's (2010) study of 5 year ICH survivors, contrary to the findings in the general stroke literature which indicates the type of stroke may be more important than gender in predicting cognitive outcome.

Causal factors associated with post-stroke cognitive dysfunction.

Cognitive functioning following stroke is, according to de Haan, Nys, and Van Zandvoort (2006), dependent on three factors. These factors are 1) focal damage which can lead to selective impairments related to the area of the lesion; 2) diffuse neuronal dysfunction which leads to a more uniform pattern of impairment, characterised by the breaking down of processes that rely on networks including speed of processing information, memory, and executive functioning; and 3) patient variables such as age, sex, premorbid level of functioning and comorbidity (de Haan et al., 2006). The second variable, which is effectively a disruption to white matter connectivity, has also been stated as being a key problem by

other researchers (Gottesman & Hillis, 2010), with cerebral white matter intensities being found to correlate with cognitive functioning post-stroke (Jokinen et al., 2005).

This model implies cognitive functioning post-stroke is not simply determined by a straightforward equation, such that damage to a given location in the brain has a known impact on cognitive functioning. Lesion location does, however, in some cases seem to increase the likelihood of certain cognitive functions being disrupted. Specifically, right temporoparietal cortex lesions are associated with the development of severe and persistent neglect (Ringman, Saver, Woolson, Clarke, & Adams, 2004). This makes sense because spatial attention is known to be mediated more by the right hemisphere; however, whilst right hemisphere lesions more commonly result in neglect, neglect can also occur from lesions in a variety of other cortical regions in both hemispheres (Ringman et al., 2004; Stone, Halligan, & Greenwood, 1993) indicating a network of supporting functions may be important for understanding the causation of neglect (Ganis, Thompson, & Kosslyn, 2004).

A further example of how the causation of cognitive deficits is not straightforward is post-stroke executive dysfunction. Executive functioning is commonly associated with the frontal lobes; however, evidence from both lesion studies (Vataja et al., 2003) and functional imaging studies (Fassbender et al., 2004) indicate it likely engages networks spread throughout the cortex. This is consistent with the fact that executive functioning deficits are seen when strokes cause lesions in many different areas (Nys et al., 2007). That said, impairments in executive functioning have been found to be more common following left than right hemisphere stroke (Nys et al., 2007; Vataja et al., 2003). Nys et al. (2007) indicate that this may not mean that executive functions are more likely to be localised in the left hemisphere, in fact research has demonstrated both the left (Johnson-Frey, Newman-Norlund, & Grafton, 2005) and right (Stuss & Levine, 2002) hemispheres are important, but instead

that language comprehension (usually localised in the left hemisphere) is a factor mediating performance on executive functioning tests for those with left hemisphere damage.

Functional outcomes of stroke and ICH.

Having addressed neuropsychological outcomes, the discussion will now turn towards functional outcomes, which have been more widely researched in the stroke literature. For the purposes of this research, and in accordance with the ICF framework, an adverse functional outcome refers to activity limitations ("difficulties an individual may have in executing activities") and/or participation restrictions ("problems an individual may experience in involvement in life situations"; WHO, 2001, p. 10). General functional outcome post-stroke is characterised by a stroke survivor's ability to continue going about their daily lives in the way they would have ordinarily done before the stroke; it has often been measured and reported in the literature by independence in activities of daily living (ADLs; Pound et al., 1998). ADLs can be broken down into basic ADLs which include feeding, grooming, dressing, bathing, personal hygiene, toileting, and mobility and instrumental ADLs (IADLs) which include telephoning, reading, leisure, medication management, money management, shopping, meal preparation, house-keeping, and home management (Hoffman et al., 2003).

Losing valued roles such as being an employee or being a member of the local sports team, as well as changes in key roles such as being a husband, wife, mother or father, is another reality for many stroke survivors, and is an example of some of the participation restrictions they may face. Health Related Quality of Life (HRQoL) is a measure that captures the impact of these restrictions and other limitations a stroke survivor is experiencing on the quality of their life; as a wider functional outcome (Gall et al., 2012; Kloeck, 2013). The literature on both general and wider functional outcomes of stroke and ICH will now be reviewed.

General functional outcomes post-stoke: Impairments in ADLs.

Prevalence and course of post-stroke general functional impairment.

The majority of research on general functional outcomes has focused on measuring basic ADLs using the Barthel Index (BI; Mahoney & Barthel, 1965) or modified Barthel Index (mBI) which are self-report measures of independence in carrying out various basic daily activities (Shah, Ranclay, & Cooper, 1989). Within the first week post-stroke functional ability is impaired in the majority of stroke survivors, with reports of only 12% being independent in basic ADLs (Wade & Hewer, 1987). The prevalence of independence in ADLs at 3- to 6-months (47 to 53%; Taub, Wolfe, Richardson, & Burney, 1994; Wade & Hewer, 1987) and 1-year (66 to 74%; Samanci et al., 2004; Taub et al., 1994) have been reported in other studies. As these findings indicate, there is a trend for recovery of function for most stroke survivors in the acute phase post-stroke (Bonita, Solomon, & Broad, 1997; Carod-Artal et al., 2002).

After the acute period post-stroke, improvement in functioning appears to plateau (Leonard, Miller, Griffiths, McClatchie, & Wherry, 1998) as evidenced by studies that have measured the proportion of stroke survivors achieving independence in basic ADLs at 2 (76%; Ahlsio, Britton, Murray, & Theorell, 1984), 3 (74%; Patel et al., 2006), 5 (71%; Feigin et al., 2010) and even 21 (81%; Anderson et al., 2004) years post-stroke.

In terms of stroke survivor's independence in IADLs, researchers have often measured this using the Frenchay Activities Index (FAI; Hollbrook & Skilbeck, 1983), and it appears that there is a greater level of impairment in IADLs compared to basic ADLs. Hartman-Maier et al. (2007) found that the majority of 1-year stroke survivors had difficulty in IADLs; notably the proportion of the sample independent in meal preparation was 27%, housekeeping 30%, shopping 48%, and laundry 18%. Another study found that at 18 months post-stroke 84% of survivors were independent in IADLs, which is higher than the 57% of survivors who were independent in basic ADLs (Hoffmann, McKenna, Cooke, & Tooth,

2003). As would be expected independence on IADLs correlates with independence in basic ADLs (Vykopal, 2010).

The literature available on differences in functional outcome according to stroke subtype is limited but tends to support differences in functional outcomes for different stroke subtypes. Studies have found a significant difference in functional outcome among the four major aetiological subtypes of ischaemic stroke (Grau et al., 2001; Petty et al., 2000; Sumer & Erterk, 2002) and between haemorrhagic and ischaemic stroke upon admission to hospital (Li et al., 1997). Further, research has found differences in participation (mobility, economic handicap, and orientation handicap) among stroke subtypes (Ischaemic, ICH, and SAH) at 5 years post-stroke (Feigin et al., 2010). Other findings, however, do not support a link between stroke subtype and independence in ADLs (Feigin et al., 2010; Li et al., 1997). However, Feigin et al.'s (2010) study examined outcomes at 5 years post-stroke, so the finding that there is no difference in ADLs according to stroke subtype at this time of measure does not rule out the possibility of a short-term difference post-stroke. It should also be noted that the findings of Li et al. (1997) should be treated with caution because the study was limited in that it compared functional outcomes of haemorrhagic and ischaemic stroke survivors upon discharge from hospital without accounting for the longer stay in hospital of haemorrhagic stroke survivors. Therefore, the comparison between the two groups was not relative.

Research that has looked at ICH specifically has found mixed prevalence rates of independence in basic ADLs; at discharge a prevalence rate of 2% has been reported (in a sample of survivors of thalamic ICH; Arboix et al., 2007), at 3 months post-ICH prevalence rates of 71% (Wang et al., 2012) and 52% (if first ever ICH) or 31% (if recurrent ICH; Kim, Kim, & Kim, 2012) have been reported, while at 6 months post-ICH prevalence rates of 75% (Wang et al., 2012), 48% (Counsell et al., 1995), and 43% (in a sample of survivors of

putaminal haemorrhage; Misra & Kalita, 1995) have been reported, and at 1 year post-ICH a prevalence of 80% has been reported (Wang et al., 2012). It must be noted that Wang et al. (2012) used the modified Rankin Scale (mRS) to assess functional outcome; classing independence as anything from no symptoms to slight disability which may explain why the results of this study indicate a greater level of independence compared to other studies that have used the BI and class independence as being able to complete a range of basic ADLs with no assistance. The results of Arboix et al. (2007) and Misra and Kalita (1995) relate to thalamic haemorrhage and putaminal haemorrhage respectively thus may not be generalizable to ICH more widely. If these results and the findings of Wang et al. (2012) are not considered then it appears as though the prevalence of ICH survivors who will be functionally independent over the first 6 months post-ICH is between 48 to 52% if it is a first ever ICH (Counsell, et al., 1995; Kim et al., 2012) and approximately 31% if it is a recurrent ICH (Kim et al., 2012). A study at 5 years post-ICH has noted that functional outcomes (both basic ADLs, measured by both mRS and BI, and IADLs, measure by the FAI) continue to be significantly worse than those reported for matched controls (Vypokal, 2010).

Factors associated with post-stroke general functional outcome.

In many studies older age has been associated with worse functional outcome after both stroke in general (Abanto et al., 2013; Demchuk & Buchan, 2000; Fiorelli et al., 1995; Hanun, Azidah, & Monniaty, 2012; Macciocchi, Diamond, Alves, & Mertz, 1998; Turhan, Atalay, & Muderrisoglu, 2009; Woo, Kay, Yuen, & Nicholls, 1992) and ICH specifically (Jeng, Huang, Tang, & Yip, 2008; Wang et al., 2012). Other studies are more mixed on this relationship. Black-Schaffer and Winston (2004) reported that age was only related to outcome for those patients who had low functioning at the time of stroke onset. Another study claims that age alone can only account for 3% of the variance in acute functional outcome of stroke survivors (Bagg, Pombo, & Hopman, 2002).

The impact of gender on functional outcomes post-stroke is unclear (Demchuk & Buchan, 2000). Whilst the majority of studies have found female sex to correlate with worse outcome (Abanto et al., 2013; Carlo et al., 2003; Glader et al., 2003; Kimberly et al., 2013; Lasek-Bal, Szymszal, & Kazibutowska, 2014; Nurten, Yurt, Meltem, Bolukbafi, & Çefime, 2005; Synhaeve et al., 2016; Wyller et al., 1997), two studies have found male sex to correlate with worse outcome (Arboix, García-Eroles, Massons, & Oliveres, 1998; Moulin et al., 1997), and other studies, including one looking at ICH specifically, have found no gender difference (Lefkovits et al., 1992; Vykopal, 2010). A recent review of the literature on gender differences in stroke outcomes concluded that females generally have worse functional outcomes than males (Gall, Tran, Martin, Blizzard, & Srikanth, 2012). This may in part be due to the fact that women tend to be older at onset of stroke than males and tend to sustain more severe strokes; it is unclear whether there is a gender difference when factors such as these are controlled for (Gall et al., 2012).

Previous stroke is a risk factor for poorer functional outcome after subsequent stroke (Demchuk & Buchan, 2000; Henon et al., 1995). Greater stroke severity is also known to result in worse functional outcome post-stroke (Abanto et al., 2013; Demchuk & Buchan, 2000; Henon et al., 1995; Macciocchi et al., 1998), and post-ICH (Jeng et al., 2008; Wang et al., 2012). Physical impairments limit stroke survivors' ability to engage in the same daily activities as they had previously. In fact it has been found that motor functioning, including balance, is the factor that explains the most variance in functional independence, followed by perceptual ability, and cognitive functioning (Mercier, Audet, Hébert, Rochette, & Dubois, 2001). These three factors jointly explained 83% of the variance in functional independence and 53% of the variance in quality of performance in ADLs (Mercier, et al., 2001).

Some research indicates that people who are married (compared to single or divorced) tend to have worse functional outcomes (Abanto et al., 2013), possibly because it is less of a

necessity to relearn doing things for themselves due to their partners' assistance. However, having a larger social network (Colantonio, Kasl, Ostfeld, & Berkman, 1993) and greater family functioning pre-stroke (e.g., family communication and problem-solving; Evans et al., 1987) are two factors that lead to less chance of institutionalization. Positive motivation towards rehabilitation also relates to better functional outcome (Tupper & Henley, 1987).

The literature considering the correlation between education level and functional outcomes post-stroke is scarce. Higher education was found to correlate with better functional outcome post-stroke in a study by Nyrkkoe (1999). It has been suggested that a higher education might give strokes survivors a better ability or a greater variety of strategies to compensate for some of the difficulties they were experiencing (Nyrkkoe, 1999). In contrast to this finding, however, Nurten et al. (2005) found that educational status and occupation had no impact on post-stroke functional outcome. Given Nyrkkoe's (1999) study was set in a developed country (Finland), whereas Nurten et al.'s (2005) study used a sample from a developing country (Turkey; the sample therefore had less variation in education level), it is possible that it was more difficult to detect a significant difference in functional outcomes in Nurten's study. No other studies could be found examining this relationship, which makes it difficult to draw conclusions on whether or not education is in fact a risk factor for poor functioning post-stroke.

Socioeconomically disadvantaged populations seem to be at higher risk of persisting poor functional outcomes (Addo et al., 2012; Cox, McKevitt, Rudd, & Wolfe, 2006; De Villiers et al., 2011; Heikinheimo et al., 2012; Shen et al., 2011; van den Bos, Smits, Westert, & van Straten, 2002). Shack housing (De Villiers et al., 2011) and malnutrition (Shen et al., 2011) specifically have been found to be independently associated with poor post-stroke functional outcomes. This is not surprising given that people who are socioeconomically disadvantaged tend to have little access to rehabilitation post-stroke (Heikinheimo et al.,

2012). Specifically, research has found that stroke survivors with a lower socioeconomic status are less likely to receive evidence based stroke services (Addo et al., 2012), physiotherapy, occupational therapy, and speech therapy (Kapral et al., 2002). Those ethnic groups that have a lower socioeconomic status than other groups in society are therefore likely at an increased risk of poorer functional outcomes (Bravata et al., 2005). In a NZ study found that that non-European NZers had significantly poorer functional outcomes at 1 year post-stroke compared to European NZers (McNaughton, Weatherall, McPherson, Taylor, & Harwood, 2002).

Wider Functional Outcome Post-Stroke: HRQoL.

As noted previously wider functional outcome has primarily been measured by HRQoL in the stroke outcomes literature; the available literature on HRQoL in stroke and ICH survivors will now be reviewed.

Average post-stroke HRQoL.

Studies at 3 months (Jonkman et al., 2009), 6 months (Gokkaya, Aras, & Cakci, 2005; Jonkman et al., 2009), 1 year (Jonkman et al., 2009; Suenkeler et al., 2002), 4 years (Niemi, Laaksonen, Kotila, & Waltimo, 1988), and 5 years (Gill 2011; Paul et al., 2005) post-stroke have found that stroke survivors have worse HRQoL than controls. In a study undertaken by Suenkeler et al. (2002), 66% of survivors at 1 year subjectively reported having worse HRQoL than their pre-stroke level. Paul et al., 2005 assessed survivors at 5 years post-stroke and noted that 50% of survivors had ‘low’ HRQoL and 20% had ‘very low’ HRQoL (Paul et al., 2005).

In contrast, other research, all of which has been conducted in NZ, has found no significant difference between HRQoL of stroke survivors and controls at 5 years (Vykopal, 2010), 6 years (Hackett, Duncan, Anderson, Broad, & Bonita, 2000), and 21 years (Anderson et al., 2004) post-stroke. Notably, the study by Vykopal (2010) exclusively assessed ICH

survivors; she found that ICH survivors had a relatively similar HRQoL to that of age, gender, and ethnicity matched controls despite poorer cognitive, emotional, and everyday functioning; with the only area of HRQoL that the ICH survivors differed significantly on being those areas in which physical functioning had a role (Vykopal, 2010).

The literature is also unclear on whether stroke survivors experience improvement in HRQoL over time, with some research noting a significant improvement from 3 to 12 months post-stroke (Jonkman et al., 2009) and other studies noting no significant difference between HRQoL in the acute stage and at 5 years (Feigin et al., 2010) and even ten years (Wolfe et al., 2011) post-stroke.

Some of this variability in results likely relates to how HRQoL is measured; whether this be via patient self-report, caregiver-report, or clinician-rated (Klocek, 2013). Overall there does not seem to be a consensus on whether or not stroke survivors can adjust well to living with a stroke and manage to maintain a good quality of life in the face of living with physical and mental disabilities.

Factors associated with post-stroke HRQoL outcome.

Factors found to be predict worse HRQoL post-stroke are older age (Nys et al., 2006; Paul et al., 2005), female sex (Gall et al., 2012; Gokkaya et al., 2005; Sturm et al., 2004; Suenkeler et al., 2002), more severe stroke (Paul et al., 2005; Sturm et al., 2004), low socioeconomic status (Paul et al., 2005; Sturm et al., 2004), low education (Gokkaya et al., 2005), incontinence (Haacke et al., 2006; Patel, McKevitt, Lawrence, Rudd, & Wolfe, 2007), and cognitive impairment (Patel et al., 2007). Some research indicates good quality relationships, integration in the community, and adaptive coping styles promote resilience in terms of maintaining a high HRQoL in the face of adversity caused by stroke (Hildon, Montgomery, Blane, Wiggins, & Netuveli, 2010).

When ICH has been looked at specifically it has been found that both age and gender do not correlate with HRQoL at 5 years post-ICH (Vykopal, 2010). Additionally, a relationship has been found between ADLs and HRQoL; with those who are more independent in ADLs generally having a higher HRQoL (Vykopal, 2010).

Section 3: The Interaction of Post-Stroke Outcomes

So far this literature review has looked at the outcomes of interest to this study (i.e. mood, cognitive, and functional) in isolation from each other. Given it is believed that these outcomes likely interact with each other in dynamic ways (Gresham, Duncan, & Stason, 1997), it is important to consider these interactions to better understand and predict outcomes for individual stroke survivors.

The interaction of post-stroke cognitive and functional outcomes.

Whilst there is a lack of comprehensive data on the relationship of neuropsychological outcomes of stroke to functional outcomes (Feigin, Barker-Collo, McNaughton, Brown, & Kerse, 2008), the available research indicates cognitive functioning appears to have a significant impact on the functional outcomes of stroke survivors (Patel et al., 2002); with greater cognitive impairment being associated with increased likelihood of general functional impairment (Feigin et al., 2010; Hama et al., 2007; Ng et al., 2007; Öneş, Yalçinkaya, Toklu, & Çağlar, 2009; Patel et al., 2002; Portney, Flak, Michalek, Moore, & Savidge, 2001; Van Zandvoort et al., 2005), even when factors including age, gender, and severity of stroke are controlled for (Heruti et al., 2002). This relationship appears to span both the acute and long-term phase post-stroke. Patel et al. (2002) found that stroke survivors who were cognitively impaired at 3 months post-stroke were significantly more likely to be dependent in ADLs at 6 months and 1, 3, and 4 years post-stroke. Improving cognitive functioning, through cognitive skills remediation training, has been found to lead to an increase in independence in basic ADLs (Carter et al., 1988).

Specific cognitive domains which have been found to correlate with general functional outcome include processing speed (Barker-Collo et al., 2010), visuospatial abilities (Barker-Collo et al., 2010; Feigin et al., 2010b; Nys et al., 2005c; Tupper & Henley, 1987; Van Zandvoort et al., 2005), executive functioning (Feigin et al., 2010b; Leśniak et al., 2008; Mok et al., 2004), verbal comprehension (Carter, Oliveira, Duponte, & Lynch, 1988), language (Feigin et al., 2010b), and visual neglect (Jehkonen et al., 2001; Louie, Wong, & Wong, 2009; Nys et al., 2005c). In Barker-Collo et al's (2010) research visuoperceptual abilities, visual memory, and information processing speed made significant independent contributions to functional outcomes over and above age, gender, education, and depression. Interestingly even an apparently simple cognitive task, such as a clock drawing task (which draws upon visuospatial abilities, attention, and executive functioning), administered when a stroke survivor is admitted to hospital has been found to be predictive of functional independence upon discharge (Adunsky et al., 2002; Suhr & Grace, 1999).

Acute cognitive impairment has also been found to predict poorer HRQoL at 6 to 10 months post-stroke, independent of demographic and neurological predictors (Nys et al., 2006) and at 4 years post-stroke (Haacke et al., 2006). Visuoperceptual abilities (Hochstenbach, Anderson, van Limbeek, & Mulder, 2001), executive functioning (Feigin et al., 2010; Hochstenbach, et al., 2001), visual neglect (Sturm et al., 2004), aphasia (Kwa, Limburg, & de Haan, 1996), and visual memory (Feigin et al., 2010) are cognitive domains that have been found to be independently associated with HRQoL.

Whilst there has been limited research to date looking at the relationship between cognitive and functional outcome in ICH in detail, a few studies have touched on this. Maeshima et al. (1997) found that, in a sample of survivors of thalamic haemorrhage, those that had neglect or aphasia were more likely to lack independence in basic ADLs; however, performance on a brief test of more general cognitive functioning (MMSE) and other tests of

cognitive function (digit span and word fluency) had no correlation to independence in ADLs. In her study of 5 year ICH survivors Vykopal (2010) did not find any significant correlations between cognitive domain summary scores and independence in ADLs. However, she did find significant correlations between information processing and executive functioning summary scores and some sub-scales of the Short Form-36 (Ware, Snow, Kosinski, & Gandez, 1993), which is a measure designed to assess HRQoL (Vykopal, 2010).

The interaction of post-stroke cognitive and emotional outcomes.

The relationship between cognitive difficulties and emotional problems is hard to tease out, because it is often the case that people who are depressed have difficulties on cognitive tests because of factors like lack of motivation and fatigue (Palsson, Johansson, Berg, & Skoog, 2000). Despite these difficulties the research indicates that both mild cognitive impairment (Nys et al., 2006) and dementia (Brodaty et al., 2007) in the acute stages post-stroke predict PSD in the long-term.

A study which looked at the relationship between depression and cognitive functioning very soon after stroke (within 3 weeks) found a distinct neuropsychological profile associated with moderate to severely depressed acute stroke patients, characterised by impairment in memory, visual perception, and language (Nys et al., 2005). Other studies have indicated that the areas of cognitive functioning most likely to be impaired in those with post-stroke depression are: cognitive speed, verbal memory (Barker-Collo, 2007), nonverbal problem solving, attention, and psychomotor speed (Kauhanen et al., 1999). Vykopal (2010) looked at ICH specifically and found that poorer visuoperception and increased overall cognitive impairment are associated with increased symptoms of depression.

There is a shortage of studies examining the relationship between post-stroke cognitive functioning and PSA. Some studies that have been conducted, including one looking at ICH specifically, have found no significant association between cognitive

functioning and PSA (Castillo et al., 1993, 1995; Shimoda & Robinson, 1998; Starkstein et al., 1990; Vypokal, 2010). Barker-Collo (2007), however, found that cognitive variables explained 39% of the variance in PSA symptoms with reduced cognitive speed being a particularly important factor found to be correlated with PSA suggesting that this is an area that deserves further investigation.

The Interaction of post-stroke functional and emotional outcomes.

Emotional and functional outcomes also interact. Multiple studies have found that both stroke survivors in general and ICH survivors who are depressed are significantly more likely to have poorer functional outcomes in terms of independence in ADLs (Brodaty et al., 2007; Dafer et al., 2008; Dennis et al., 2000; Kauhanen et al., 1999; Kotila et al., 2003; Pohjasvaara et al., 2001; Vypokal, 2010) and HRQoL (Haacke et al., 2006; Suenkeler et al., 2002; Wulsin et al., 2012), even up to 5 years post-stroke (Barker-Collo et al., 2010; Gill, 2011). It has been found that stroke patients who had an improvement in their mood in the acute period post-stroke gained back their functional independence quicker than those who showed no improvement in mood over this time (Chemerinski, Robinson, & Kossier, 2001). This suggests that reductions in depressive symptomology may accelerate functional recovery in stroke survivors (Saxena, Ng, Koh, Yong, & Fong, 2007); however, it is possible that improved functional independence would also have a positive effect on depression.

PSA has also been found to be associated with reduced independence in ADLs in some studies of stroke survivors, including ICH survivors; but the association is not as strong as that noted between PSD and independence in ADLs (Dennis et al., 2000; Schultz et al., 1997; Vypokal, 2010). Other studies have not found an association between PSA and functional impairment (Park, Jang, Lee, & Park, 1999; Shimoda & Robinson, 1998). However, in Shimoda and Robinson's (1998) study, stroke survivors with co-morbid PSA and PSD had greater functional impairment compared to those with PSD alone, suggesting

PSA does negatively impact functional outcome when paired with PSD (Shimoda & Robinson, 1998).

PSA has been associated with poorer HRQoL in survivors of ischaemic stroke (Huang, Li, & Wang, 2010) and ICH (Vypokal, 2010). Data on both PSA and PSD has been found to predict 49% of the overall HRQoL in stroke survivors at 6 months post-stroke (Huang et al., 2010).

Section 4: Summary and Implications for This Study

Currently there is a lack of understanding of the cognitive and mood outcomes of stroke despite these being common sequelae which have a significant and long-lasting impact on stroke survivors. The mood and cognitive outcomes of stroke have only been measured in a small proportion of stroke outcomes studies and those studies that do assess these outcomes show wide variability in their definition of what constitutes impairment or a disorder, the type of sample assessed, and the method of assessment. This has led to conflicting results which, combined with the lack of transparency of many studies, means it is hard to reconcile the findings. The infancy of the literature on neuropsychological outcomes of stroke, and ICH particularly, highlighted the need for exploratory research, such as the current study, to be undertaken.

The literature illuminated not only a need for further research investigating neuropsychological outcomes, but also for this research to be conducted in a way which promotes transparency and accessibility of findings and utilises thorough and scientifically-validated assessment measures. Thoroughness of cognitive assessment seems especially important to ensure that cognitive profiles of the various stroke subtypes can be created. This study will therefore collect data across the major domains of cognitive functioning (i.e., executive functioning, memory, visuospatial/visuoperceptual functioning and processing

speed), rather than limiting its assessment to the screening tools utilized in many previous studies.

Another trend in the current literature on stroke outcomes is to only assess one domain of functioning. This is contrary to the WHO's (2002) view of the importance of being able to get a holistic view of the outcome of a health condition for the individual. For stroke, this requires research to look at a host of factors that influence post-stroke outcome; allowing a better picture of the individual's outcome as a whole. This study will attempt to get a more holistic view of outcomes by including measures to assess cognitive functioning, mood, and functional outcomes.

A further point of interest for the current stroke outcomes literature is the type of sample being used to assess stroke outcomes; with clinical studies far outweighing population-based studies. Clinical studies of stroke outcomes involve a sample of people that is often not representative of the stroke population as a whole. For instance, all participants may be referred to a certain rehabilitation service so are selected based on convenience and impacted by criteria used in allocating services. In this example those people not attending rehabilitation, possibly because they are functioning well after their stroke, are missed out and the findings will therefore overestimate deficits if generalised to all stroke survivors. A population-based study on the other hand, which tries to identify all strokes (whether the individual was hospitalised or not), should give a less restricted view of outcomes and reduce the chance of selection bias (Hackett & Anderson, 2000; Feign et al., 2008). This promotes generalizability of the results (Hackett, Duncan, Anderson, Broad, & Bonita, 2000) and allows a more accurate estimate of the full range of long-term outcomes of stroke, which is useful for stroke survivors, their families and the health-care system to be able to plan for the future and allocate resources optimally (Feigin et al., 2008). Despite this advantage there has only been a very limited number of population-based outcome studies on stroke (Barker-

Collo & Feigin, 2006). The number of population-based studies that include some data on neuropsychological outcomes is even more limited as can be seen in Tables 2 and 3. This study will therefore aim to contribute to this gap in the literature by drawing the sample of ICH participants from a larger population-based study (Auckland Regional Community Stroke Study IV).

Differentiating between short-term and long-term outcomes post-stroke is also important because many outcomes show change over time (Woo et al., 1992). The data on long-term stroke outcomes is currently limited; this study will therefore examine both short-term (6 months) and long-term (1 year) outcomes. If the true impact of stroke is to be determined it is important to compare stroke survivors to a control group who have not experienced a stroke. It has been relatively well established in the literature the burden of stroke in the acute stages post-stroke; what is less clear is the extent to which these differences persist over time. Therefore, it is considered to be of greater value to compare controls to stroke survivors at 6 and 12 months rather than at baseline.

A further aspect of the current literature is that it has mainly focused on general stroke outcomes (rather than breaking outcomes down by stroke subtype). It seems justified to study outcomes specific to stroke subtypes given they have different aetiologies and mechanisms of injury, so they may also have different outcomes. In the case of ICH it appears that cognitive deficits are more common post-stroke compared to the other stroke subtypes (Makin, Turpin, Dennis, & Wardlaw, 2013). Those studies that have looked at one subtype of stroke have often selected ischaemic stroke, which is likely due to this being the most prevalent type of stroke so larger sample sizes are possible. ICH is, however, a particularly important subtype to study because despite recent advances in the morbidity and mortality for other types of stroke, there has been no such improvement for ICH (Ciccone, Pozzi, Motto, Tiraboschi, & Sterzi, 2008). ICH has also been described as the least treatable form of stroke (Ciccone et

al., 2008) and on average, places a greater financial burden on society than the more common ischaemic stroke (Dewey et al., 2003).Therefore data on the outcomes of ICH, which can potentially lead to improvement in services provided to patients post-ICH, is crucial to collect; justifying why the current study will focus on ICH outcomes exclusively. If a distinct profile of long-term neuropsychological deficits are established for ICH (which this study aims to contribute to), then differing community and rehabilitation services, educational programs and interventions may be warranted (Sturm et al., 2002).

A final facet of the literature that helped inform the aims of the current study is the lack of information about factors which can predict outcomes post-stroke. Prominent researchers in the field (Petterson, Dahl, & Wyller, 2002; Feigin et al., 2008) have commented on the limited and often conflicting information that is known about predictive factors of long-term post-stroke outcomes. The current study therefore aimed to contribute to this gap.

The overall objective of this exploratory research was to examine 6 month and 1 year mood, cognitive, and functional outcomes of ICH in a population-based sample. Specifically the aims are:

1. To compare 6 month and 1 year outcomes of ICH survivors to a group of healthy age, gender, and ethnicity matched controls,
2. To describe the natural course of recovery of mood, cognitive, and functional outcomes over the first year post-ICH,
3. To examine the interrelationships between mood, cognitive, and functional outcomes of ICH, and
4. To determine if there are characteristics of ICH or the ICH survivor at baseline that predicts good versus poor functional and cognitive outcomes at 1 year.

Chapter 2: Methodology

This chapter outlines the methodology used in the current study, beginning with a description of the participants and measures used. This is followed by a description of the procedure for data collection, data management, and data preparation for analyses.

Section 1: Participants

There were two groups of participants involved in this study; an ICH group and a matched control group. The ICH group was drawn from existing outcomes data from the Auckland Regional Community Stroke (ARCOS) IV Study; therefore the researcher was not involved in collection of this data. The ARCOS-IV aimed to collect data on all adults (aged >16) in the Auckland region who had a stroke of any subtype from March 2011-February 2012. The sample used for the current study was made up of all people in the ARCOS-IV study who: (1) survived an ICH, (2) gave informed consent to participate in the study, and (3) had outcomes data collected at 6- and/or 12-month follow-up post-ICH. In total 251 people had an ICH over the 1-year period of the study, of which 72 consented to be in the study; 110 had died, 15 were too unwell, 47 declined, 5 had a language barrier, 1 was unable to be contacted, and 1 lived too far away to take part in the study. Of the 72 who consented to be in the study, only 35 had data at 6 and/or 12 months. This gave a sample size of 35 people. The 35 participants in the ICH sample were 22 (62.9%) females and 13 (37.1%) males with a mean age of 65.5 years (S.D. = 14.19). Most participants (n= 24, 68.6%) self-identified as European, 6 (17.1%) as Asian, 4 (11.4%) as Pacific Islanders, and 1 (2.9%) as Latin American.

A healthy control sample matched for gender, age (+/- *1 year*), and ethnicity was recruited by the researcher from the general population. The researcher carried out all assessments with the controls. Gender, age, and ethnicity were the variables selected for matching because they have been found to significantly contribute to variance in

neuropsychological test performance; if they were not matched, then power would be lost during analysis by having to factor them in (Lezak, 2006). Consideration was given to matching the samples on education as well, however, matching on four variables would have required that the timeframe of data collection be extended, which was not possible within the constraints of the current thesis. Information on education level was still collected, however, so that it could be considered during analysis. Further inclusion criteria for controls were; (1) participants were not currently diagnosed as depressed, (2) did not have a previous diagnosis of psychosis, personality disorder or substance abuse disorder, (3) had not suffered a traumatic brain injury, (4) did not have any current neuropsychological or neurological problems, and (5) were not suffering from a chronic health problem that was likely to impact on performance. These criteria were not applied to participants in the ICH group given that they were not expected to be 'healthy'.

Matched controls could be found for all but one of the participants. It was not possible within the data collection timeframe to find a 73-year-old woman from South America who was willing to participate and able to speak English to the level required for the assessment. This meant that the ICH group consisted of 35 participants, while the matched control group consisted of 34 participants. The 34 matched controls were 21 (61.8%) females and 13 (38.2%) males with a mean age of 65.3 years (S.D. = 14.56). As expected, most participants (n= 24, 70.6%) self-identified as European, 6 (17.6%) as Asian, and 4 (11.8%) as Pacific Islanders.

The demographic characteristics of both the ICH and matched control group are summarised in Table 4. As expected, Table 4 shows that no significant differences were found between the two groups on the matching criteria of age, gender, and ethnicity. Relationship status did, however, differ significantly. A greater number of the ICH group had

Table 4

Demographics of ICH and Matched Control Groups

Demographic		**ICH Group** n=35		**Matched Control Group** n=34		**Significance of between group differences**	
		Mean	**SD**	**Mean**	**SD**	**T**	**p**
Age		65.5	14.19	65.3	14.56	0.55	0.956
		%		**%**		**Chi-Square**	**p**
Gender	Female	62.9		61.8		.009	.925
	Male	37.1		38.2			
Ethnicity	European	68.6		70.6		.986	.805
	Asian	17.1		17.6			
	Pacific Island	11.4		11.8			
	Latin American	2.9		0			
Relationship Status	Married, civil union, or living with partner	71.4		64.7			
	Separated, divorced, or widowed	2.9		29.4		11.998	**.002**
	Never married (single)	25.7		5.9			
Dwelling	Own home	51.4		82.4			
	Rented	34.3		14.7			
	Living at friend or family member's place	11.4		0			
	Rest home/ private hospital/ boarding House	0		0		14.007	**.003**
	Retirement village or similar	0		2.9			
	Other	2.9		0			
Living	Living with partner/family	77.1		70.6		8.063	**.045**
	Living with others	0		8.8			
	Living alone	22.9		20.6			
High school	Completed	80.0		73.5		.405	.524
	Did not complete	20.0		26.5			
If completed high school, further education?	Yes	71.4		91.7		3.895	**.048**
	No	28.6		8.3			

If further education, highest qualification?	Degree	50.0	36.4	7.282	.063
	Diploma or Certificate	20.0	50.0		
	Trade or Technical Qualification	25.0	13.6		
	Other	5.0	0		
Socioeconomic Status	Professional	5.7	8.8		
	Managerial/technical	17.1	23.6		
	Skilled non-manual	2.9	17.6		
	Partly Skilled	5.7	5.9	33.898	**.000**
	Unskilled	2.9	5.9		
	Armed Forces	5.7	2.9		
	Unemployed/ Retired	0	32.4		
	Other	60.0	2.9		

Note: bold typeface highlights significant difference. n= sample size

never been married at the time of their stroke compared to the control group. Significant differences can also be seen between groups in relation to their place of dwelling (before the ICH for the ICH group); the ICH group were less likely to be living in their own home and more likely to be renting or living with family or friends. There was also a significant difference between who the ICH group lived with compared to the control group; the ICH group were slightly more likely to be living with family or friends than the control group and the control group was more likely to live with others compared to the ICH group.

High school completion did not differ significantly between the two groups; however, those in the control group who completed high school were more likely to complete further education compared to the ICH group. Further, socioeconomic status (SES) varied significantly between the two groups; of particular note is that whilst 32.4% of the control group were retired or unemployed, none of the ICH group were retired or unemployed before their ICH. A limitation with the SES data is that the majority of participants in the ICH group (60.0%) were assigned the “other” category, indicating that the measure was perhaps not adequately designed to capture relevant socioeconomic classes.

Section 2: Measures

All ICH participants completed each of the assessments at baseline (within 2 weeks of stroke), and at 6-month and 12-month follow-ups; whereas control participants completed the assessment on only one occasion. Given the comprehensiveness of the assessment and to reduce burden, demographic information and surveys of HRQoL, mood and participation were conducted via telephone, whilst neuropsychological assessment and measures of disability were conducted face-to-face. All measures, excluding those computerised ones, are contained within Appendix B. Testing of controls on more than one occasion was considered; however, it was considered impracticable due to the possible increased difficulty of recruiting controls (due to greater burden) and the limited time period available for data collection (12-14 months due to the degree structure).

Demographic information.

Demographic information was collected at the outset of the telephone assessment and included age, gender, ethnicity, education, relationship status, living arrangement, socioeconomic status, and reviewing inclusion/exclusion criteria.

Measures of Mood.

Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983)

The HADS is a 14 item forced-choice questionnaire assessing anxiety and depression over the previous week. Each item can be scored from 0-3. For example, the item "I feel cheerful" can be answered "not at all (3), not often (2), sometimes (1), or most of the time (0)". Seven of the items total to give an overall depression score and the other seven give an overall anxiety score. A score of 0-7 is considered normal, 8-10 is the borderline clinical range, and 11-21 indicates clinical level anxiety or depression (Zigmond & Snaith, 1983). The HADS is commonly used and well validated among stroke populations (Campbell Burton et al., 2012; Sagen et al., 2009) and the general population (Bjelland et al., 2002). The

HADS has been found to have good sensitivity and specificity (approximately .80) and correlate well with other commonly used depression and anxiety questionnaires (.49 to .83; Bjelland et al., 2002). In addition to the reliability of the HADS, it was selected here both because it assesses both anxiety and depression but also as it places less emphasis on physiological aspects of depression and anxiety that may be impacted by a health condition such as stroke. A limitation of the measure is that it assesses primarily Generalised Anxiety Disorder symptoms. It does not capture symptoms of other possible anxiety disorders post-ICH including Social Anxiety Disorder, Phobic Disorders, and Agoraphobia.

General Health Questionnaire (GHQ-28; Goldberg & Hillier, 1979)

The GHQ-28 is a shorter version of the original 60 item General Health Questionnaire. It comprises 28 self-administered items which are equally divided between four subscales: somatic symptoms, anxiety and insomnia, social dysfunction, and severe depression (Goldberg & Hillier, 1979). Items are to be answered based on functioning in the previous few weeks; there are four answers to choose from for each item. For example, to the item "Have you felt that life isn't worth living?" individuals can respond: "Not at all", "No more than usual", "Rather more than usual", or "Much more than usual". The first two answers indicate that there has been little or no change in distress in the previous weeks (therefore a score of zero is given) and the third and fourth answers indicate an increase in distress (therefore a score of one is given; Goldberg & Hillier, 1979). The social dysfunction subscale more accurately captures general functional outcome (HRQoL) rather than mood so will be used as such in this study. This measure was selected in the current study because of its sound psychometric properties, including a high level of sensitivity (.85) and specificity (.94), and the fact it has been established as a suitable measure for large scale epidemiological studies (Hakim, 2009). Additionally, the measure has a simple scoring method which reduces chance of administrator error. A limitation of the measure is that the

response format is reasonably complex, thus the demands on attention and concentration could be a challenge for cognitively impaired participants and prove a barrier to them completing the measure.

Measures of cognitive functioning.

CNS-Vital Signs computerised test battery

CNS-Vital Signs is a computerised battery of 10 age-normed neuropsychological tests which assess major areas of cognitive functioning including: working memory and attention, immediate and delayed verbal and visual memory, processing speed, executive functioning, psychomotor speed, and perception of emotions. Table 5 gives a brief description of the individual tests within the CNS-Vital Signs battery. The tests have good psychometric properties; having very similar reliability and validity to the conventional neuropsychological tests upon which they are based (Gualtieri & Johnson, 2006). Gualteri and Johnson (2006) evaluated test-retest reliability of the CNS-Vital Signs by examining the performance of neuropsychiatric patients and healthy controls who completed the measure on two separate occasions (on average 62 days apart). Neither age nor clinical status had any bearing on reliability. In the domains of memory, psychomotor speed, reaction time, and cognitive flexibility a *high* level of correlation was found and a *moderate* correlation was found for complex attention. Thus, the measure appears to have good test-retest reliability. Gualteri and Johnson (2006) also investigated concurrent validity by comparing performance on the CNS-Vital Signs to conventional neuropsychological tests. The conventional tests were the Rey Auditory Verbal Learning Test, Logical Memory and Facial Recognition from the Wechsler Memory Test, a mechanical finger tapper, the Stroop Test, Trails B and the Verbal Fluency Test. It was determined that there was *moderate* correlation between CNS-Vital Signs and conventional tests. Further, Gualteri and Johnson (2006) established a reasonable level of discriminant validity when CNS-Vital signs was used to differentiate between Attention

Deficit Hyperactivity Disorder, mild traumatic brain injury, early dementia, malingering, and healthy controls, although the measure is not designed to be a diagnostic tool.

Table 5

CNS-Vital Signs Tests

Verbal Memory Test	A list of 15 words must be remembered and then recognised in a field of distractor words, both immediately after presentation of the list and after a 30 minute delay.
Visual Memory Test	A series of 15 geometric figures must be remembered and then recognised in a field of distractor figures, both immediately after their initial presentation and after a 30 minute delay.
Finger Tapping Test	A test of fine motor control ability and motor speed. There are 3 rounds each of tapping a key with the left and right index finger.
Symbol Digit Coding	Using a provided key/legend, test-users are required to type in the number that corresponds with the symbol they are presented with. The test aims to measure ability to recognise and process information.
Stroop Test	Aims to measure simple and complex reaction time, inhibition/disinhibition, and directed attention. The test assesses how quickly test-users can both read words and name the colour of ink those words are printed in.
Shifting Attention Test	Test-users must shift their responses based on randomly changing rules. The test assesses aspects of executive functioning, including set shifting and abstraction.
Continuous Performance Test	A measure of sustained attention and choice reaction time. Test-users must track letters of the alphabet which are serially presented on the computer screen for a 5 minute period; they are only to respond when they see the defined stimulus (the letter 'B').
Perception of Emotions Test	Measures how well test-users can perceive and identify specific emotions (happy, sad, angry, and calm) by rating the expressions of people in photographs.
Non-Verbal Reasoning Test	Consists of 15 matrices or visual analogies which get progressively more difficult. It measures how well test-users can understand the relationship between visual-abstract concepts and make decisions based on these relationships.
Four Part Continuous Performance Test	A measure of working memory and sustained attention. It requires storing information in working memory in order to be able to respond to only certain stimuli that are presented.

Neuropsychological test scores on the CNS-Vital Signs battery were automatically converted from raw scores to standardised scores by the programme software using age-

relevant normative data; an overall score for cognitive function (neurocognition index) is also generated by the programme along with domain scores for composite memory, visual memory, verbal memory, working memory, processing speed, executive functioning, psychomotor speed, reaction time, complex attention, sustained attention, and social acuity. These scores are generated by the CNS-Vital Signs Programme software by combining data across all ten tests. A score ranging from 110-119 is considered to be in the high average range; 90-109 is average; 80-89 is low average; 70-79 is borderline; and 69 or below is extremely low. CNS-Vital Signs was selected over more typical neuropsychological tests due to the benefit of greater consistency across administration and the quicker administration time. A possible limitation to this measure is that the computerised format could be off-putting to the older adults in the study who may be unfamiliar with computer technology.

Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005),

The MoCA is a well-validated screening measure of cognitive function which was chosen for the current study because of the greater sensitivity to cognitive impairment post-stroke than the Mini Mental State Examination (MMSE) and the fact it is less influenced by cultural differences than the MMSE (Dong et al., 2010; Pendlebury, Cuthbertson, Welch, Mehta, & Rothwell, 2010, Pendlebury et al. 2012). The MoCA consists of 13 tasks which briefly assess visuospatial and executive functioning, naming, memory and delayed recall, attention, language, abstraction, and orientation (Nasreddine et al., 2005). Item scores are totalled to give an overall maximum possible score of 30. One point is added to the score for people who have 12 years or fewer of formal education. A score of 26 and above is considered normal (Nasreddine et al., 2005). The MoCA was included in addition to CNS-Vital Signs in case some participants were too impaired to complete CNS-Vital Signs, to allow comparison of the findings to existing literature using similar measures, and because MoCA scores have been found to detect post-stroke cognitive decline determined by formal

neuropsychological examination (Xu, Dong, & Chen, 2015). Additionally, the MoCA has been found to have a high level of test-retest reliability (.79) making it suitable for repeated administration and a high level of interrater reliability (.81) which makes it appropriate for the current study in which multiple researchers were anticipated to administer the measure (Gill et al., 2008).

Behavioural Dyscontrol Scale-Second Edition (BDS-2nd Ed, Grisby & Kaye, 1996)

The BDS-2nd Ed is a standardised well-validated nine item measure of behavioural self-regulation and executive function (Suchy, Blint, & Osmon, 1997). Seven of the items require motor functioning (e.g., if the examiner taps twice, participant taps once and if the examiner taps once, participant taps twice), one is a working memory and set shifting task (alphanumeric sequencing), and the final item is the examiners rating of the participants insight into accuracy of his or her performance (Grigsby & Kaye, 1996). Each item is scored from 0-3; the total score on the BDS is the sum of the scores on all items, which gives a total possible score of 27 (Grigsby & Kaye, 1996). The BDS was included in the study because of previous research findings indicating executive dysfunction may feature in the expected cognitive profile post-ICH (Su et al., 2007; Vykopal, 2010). However, it should be noted that given motor functioning is required for the majority of items within this measure it is difficult to distinguish between the impact of any deficits in motor abilities due to ICH and cognitive deficits post-ICH.

Measures of general functional outcome (Impairments in ADLs).

Barthel Index (BI; Mahoney & Barthel, 1965)

The BI is a commonly used test of independence in ADLs. It comprises ten self-report items which assess feeding, bathing, grooming, dressing, bowels, bladder, toilet use, bed-to-chair transfers, mobility, and stair use; the maximum overall score of 100 indicates independence in all the listed activities (Mahoney & Barthel, 1965). The BI was selected for

inclusion in the current study as it has been found to have good reliability and validity with older adult populations (Sainsbury, Seebass, Bansal, & Young, 2005) and stroke survivors (Sulter, Steen, & De Keyser, 1999). Further, it is the most commonly reported measure of ADLs in the post-stroke literature and is brief and easy to administer, which likely makes it accessible to a greater number of study participants.

Measures of wider functional outcome (HRQoL).

Short Form-36 (SF-36; Ware, Snow, Kosinski, & Gandek, 1993)

The SF-36 is a self-rated 36 item measure of HRQoL over the previous month. It was chosen for inclusion in the current study because it is well-validated both with stroke samples (Anderson, Laubscher, & Burns, 1996) and the general population (Jenkinson, Wright, & Coulter, 1994). It has also been found to be a reliable measure (.85) that is easy to use and viewed as acceptable by patients (Brazier et al., 1992). Additionally, it was chosen for the current study because of the benefit of an online score calculation tool which reduces the time taken to score the measure and also the chance of error within scoring. The items on the SF-36 are arranged in eight scales: physical functioning, role limitations due to physical health problems, bodily pain, social functioning, mental health, role limitations due to emotional problems, vitality, and general health perceptions. The scales overall combine to give two summary scores: physical and mental health. A limitation of the measure to consider is that it relies solely on self-report; given the high level of cognitive deficits expected post-ICH, it is possible that participants' insight and capacity to reflect on their own wider functional impairments might be limited.

Section 3: Procedure

With regards to collection of the ICH group data, NZ Health and Disability Northern Region Ethics Committee approval had been obtained by the ARCOS-IV study. Approval to collect data for the matched controls was gained from the University of Auckland Human

Participants Ethics Committee. The procedure for identification and recruitment of the ICH sample will firstly be described followed by data collection procedures for the control group.

The following account of recruitment for the ICH group describes the procedure carried out during the ARCOS-IV study for all stroke subtypes. Identifying the ICH group involved collecting a prospective register of all new ICH cases (in people aged 16 or over) in the Auckland region over a 1 year period from March 2011 to Feb 2012. Searches were made of presentations/admissions recording any diagnoses suggesting stroke or transient ischaemic attack. Searches involved all public hospitals and emergency departments' records; CT/MRI records; the hospital discharge register; all private hospitals, rest homes, and community health services (GPs, rehabilitation centres, outpatient clinics); coroner/autopsy records; checks of death certificates from Registrar of Births, Deaths and Marriages to identify people who have died with any mention of stroke, and NZ Health Information Service data of all fatal and non-fatal stroke cases.

As soon as possible after notification of a potential stroke a trained nurse made contact with the patient or next of kin (if the patient was too unwell or deceased) to provide information about the ARCOS-IV study and gain consent if they were willing to take part. An interview time was then made to administer the baseline assessment which included the measures listed above. Only those whose medical records indicated the stroke was an ICH were included in the current study. Stroke subtypes were verified via CT and MRI scans in some cases in which subtype was unclear within the records.The ICH group were assessed at multiple timeframes post-stroke (baseline, 6 months, and 1 year post-stroke), whereas the matched control group was only assessed on one occasion.

Control participants were recruited and assessed by the researcher. Controls were recruited by advertising using the Participant Information Sheet (see Appendix C), which was

distributed within the University of Auckland, on local community noticeboards, within local community organisations, groups, and churches, within the district health boards in Auckland, and retirement villages in the Auckland region. In addition, an advert was also placed in local newspapers. Advertisements provided potential participants with researcher contact details; they were to contact the researcher if interested.

Once a participant had contacted the researcher, they were screened to determine if they matched an ICH group participant and the criteria for inclusion. If this was not the case, then the individual was thanked for their interest in the study but informed that they could not participate. If the individual was eligible they were then asked to provide verbal consent to participate. If this was provided two measures were then administered over the phone (HADS and SF-36) which took up to 20 minutes. At this time an appointment was also made for a face-to-face interview at the person's place of residence or in a bookable room at the University (whichever was most convenient to the participant). During this interview a written consent form and all other measures were administered, which took on average 90 minutes.

All measures were administered and scored according to standard procedures. All data was then entered into statistical software programme IBM SPSS statistics 20.0 for analysis. Each participant was given a code and identifying information was not included in the data file. Hard copies of the data were kept in a locked filing cabinet in a locked room on university premises; the data will remain there for 10 years, at which time it will be destroyed. It was specified to the participants when information would be released; specifically their GP may contact the researcher and request a summary of neuropsychological results (provided as a summary report by the research team neuropsychologist who is the primary research supervisor) should they wish to within the next 10 years.

Chapter 3: Results

In this chapter the findings of the research are presented across four sections. The first section describes the preliminary analyses performed on the data such as inspection of the data set for accuracy of input, management of missing data, the testing of the statistical assumptions of normality, linearity, and homogeneity, and a power analysis. The second section presents descriptive statistics for the control and ICH group at 6 and 12 months across measures. Performance of the two groups on measures of mood, cognitive functioning, and functional outcomes are then compared using one-way ANOVA. The third section focusses on reporting the findings concerning change over time using the data available for measures taken at baseline, and 6 months and 12 months post ICH. Section four is concerned with the relationships between measures of outcomes at 12-months; with correlation analyses exploring the relationships between mood, cognitive impairment, and functional outcome, including quality of life, at 12-months post ICH, as well as the correlations between these and demographic characteristics at baseline. Given the exploratory nature of the study, results should not be interpreted as conclusive, rather they should be utilised to inform further research.

Section 1: Preliminary Analyses

Inspection of the data set for accuracy and management of missing data.

Inspection of the data set for accuracy of input involved checking that all data were within expected ranges by checking the minimum and maximum values of all variables, and also randomly checking accuracy of entry for 10% of the data. When examining the range of scores on measures two data entries were found to be out of the expected range and these were corrected. In regards to management of missing data, whilst there was no missing data for the control group, for the ICH group data was missing for many of the participants, particularly on the CNS-Vital Signs. In the majority of cases, measures were missing as

participants declined to participate in an assessment (e.g., declining to participate in the 6-month assessment but agreeing to participate at 12 months, often citing they did not want to use the computer to complete CNS-Vital Signs or did not have time to complete the assessment; 33%). Other reasons recorded for the missing data were that the person's cognitive functioning was judged as being too poor to complete cognitive tests (28%) or that there was a language barrier (8%). For 31% of cases, reasons were unfortunately not recorded.

The percentage of the ICH group that completed all measures other than the CNS-Vital Signs battery at baseline, 6 months, and 12 months were 43%, 46%, and 54% respectively. The proportion of missing data for each measure for the ICH group is presented in Table 6 below. Where an entire measure was missing, cases were excluded list-wise from analyses. There were no instances where single items were missing from a particular scale.

Table 6

Proportion of Cases Missing for Each Measure from the ICH Group at Each Time Period

Measure	Proportion of Missing Values Baseline		Proportion of Missing Values 6 months		Proportion of Missing Values 12 months	
	N	%	N	%	N	%
Mood						
Hospital Anxiety and Depression Scale	14	40	13	37.1	12	34.3
General Health Questionnaire-28	14	40	14	40	12	34.3
Cognitive Functioning						
CNS-Vital Signs	29	82.9	31	88.6	32	91.4
Behavioural Dyscontrol Scale	16	45.7	16	45.7	15	42.9
Montreal Cognitive Assessment	19	54.3	18	51.4	15	42.9
Functional Outcome						
Barthel Index	1	2.9	1	2.9	0	0
Short Form-36	15	42.9	13	37.1	12	34.3

Testing the statistical assumptions of normality, linearity, and homogeneity.

The statistical assumption of normality is the presumption that data collected for a variable is normally distributed (i.e., the data is distributed over a symmetrical bell-shaped curve; Gordon, 2012). If data is to undergo multivariate analysis it is preferable for it to be normally distributed (Tabachnick & Fidell, 1989). Given that there were less than 50 cases in each group, the Shapiro-Wilk test of normality (Shapiro & Wilk, 1965) was used here. When using this test, a significant p-value ($p<0.05$) indicates that the data distribution is significantly different from a normal distribution (i.e., the assumption of normality is violated; Shapiro & Wilk, 1965). The normality of the data for all measures at baseline, 6 months, and 12 months was explored for the ICH group and matched controls separately. Those measures that were found to violate the assumption of normality are listed in Table 7. Measures not listed in the table were approximately normally distributed. Data transformations to remedy failures of normality (e.g., square root, logarithmic, and inverse transformations) were undertaken as outlined in Tabachnick and Fidell (1989). Unfortunately, these did not improve the normality for any of the variables. Therefore, in order to maintain interpretability of the data, the data were left as is. Whilst multivariate procedures are considered fairly robust to violations of normality when groups are of relatively equal size (Tabachnick & Fidell, 1989), caution will be used in interpretation.

The second statistical assumption to be tested was that of linearity; the assumption that the relationship between variables is linear (i.e., that the amount of change between scores on two variables are constant for the whole range of scores on those variables; Gordon, 2012). To investigate this assumption bivariate scatterplots can be examined for bivariate combinations of measures within each time frame to check for a straight line relationship (Tabachnick & Fidell, 1989). When there are many variables to examine, such as in the current study, it is recommended that statistics for skewness and kurtosis are used to indicate

pairs most likely to depart from linearity and then screen only those pairs (Tabachnick & Fidell, 1989). This procedure was used in the current study and there was no evidence that the assumption had been violated for any of the relationships examined.

Table 7

Measures Violating the Assumption of Normality

ICH Group	Control Group
Measures of mood	***Measures of mood***
HADS Depression Scale (Baseline)	HADS Anxiety Scale
Measures of cognitive functioning	HADS Depression Scale
Behavioural Dyscontrol Scale (Baseline)	Short Form-36
CNS-Vital Signs	***Measures of cognitive functioning***
Composite Memory (Baseline)	Behavioural Dyscontrol Scale
Neurocognition Index (6 months)	Montreal Cognitive Assessment
Reaction Time (6 months)	*CNS-Vital Signs*
Reasoning (6 months)	Processing Speed
Complex Attention (12 months)	Reaction Time
Measures of functional outcome	Complex Attention
Barthel Index (Baseline, 6 & 12 months)	***Measures of functional outcome***
	Barthel Index
	General Health Questionnaire-28

HADS= Hospital Anxiety and Depression Scale

The third statistical assumption to be tested was homogeneity of variance. This is the assumption that the variance (i.e., spread of scores across measures) in both of the populations sampled from (in the current study- general population and ICH survivors) is equal (Gordon, 2012). Levene's test of equality (Levene, 1960) was used to establish whether there was a significant difference between the ICH group and matched control group across the measures used in this study. A p-value less than 0.05 would indicate that the variances are significantly different, whereas a p-value greater than 0.05 would indicate that the variances can be assumed to be relatively similar. As expected, given a clinical population was being compared to the general population, the variance for the two groups differed significantly on many of the measures. Table 8 outlines those measures for which the assumption of homogeneity of variance was violated. Despite these violations data analysis went ahead as

Tabachnick and Fidell (1989) posit that multivariate analyses are quite robust to violations of homogeneity of variance; however, caution has been taken when interpreting the results.

Table 8

Measures Violating the Assumption of Homogeneity of Variance between the ICH and Control Group

Measures of Mood
Hospital Anxiety and Depression Scale- Depression Score (Baseline, 6 months, 12 months)
Short-Form 36 (Baseline, 6 months, 12 months)
Measures of Cognitive Functioning
Behavioural Dyscontrol Scale (Baseline, 6 months, 12 months)
Montreal Cognitive Assessment (Baseline, 6 months, 12 months)
CNS-Vital Signs
Reasoning (Baseline)
Neurocognition Index (12 months)
Psychomotor Speed (12 months)
Reaction Time (12 months)
Complex Attention
Measures of Functional Outcome
General Health Questionnaire-28 (Baseline, 12 months)
Barthel Index (Baseline, 6 months, 12 months)

Power analysis.

A power analysis was undertaken to determine what sample size would be needed to have an 80% probability (i.e. .80 power) of detecting a one standard deviation difference between the ICH group and control group with a two-sided 0.05 significance level. Table 9 presents the results of this analysis for a selection of measures used in this study, spanning the three outcomes of interest (mood, cognitive, and functional outcomes). As can be seen, on the measures of mood and cognitive functioning, the current study does not have the required sample size to gain .80 power. However, on the functional outcome measure (SF-36) the current study did meet the required minimum sample size to reach power at .80. Overall, the

results of the power analysis confirm that the current study should be considered exploratory due to limited power.

Table 9

Power Analysis: Sample Size Required for 80% Probability of Detecting One Standard Deviation Difference between the ICH Group and Control Group

Measure	**Sample Size (both groups combined) Required for .80 Power**
CNS-Vital Signs-Neurocognition Index	82
Montreal Cognitive Assessment	152
Hospital Anxiety and Depression Scale (HADS)- Depression Scale	154
Hospital Anxiety and Depression Scale (HADS)- Anxiety Scale	66
Short Form-36	30

Section 2: Group Comparisons

In this section the means and standard deviations of the ICH group at each time of measure as well as the means and standard deviations of the control group are presented for each type of assessment completed (i.e., mood, cognitive, and functional outcome). Comparisons of the performance of the control group on each measure to that of the ICH group at 6 and 12 month follow-ups are then presented.

Measures of mood.

Table 10 presents the means and SDs of the control group and the ICH group at each time of measure as well as the results of one-way ANOVAs to compare the two groups in

Table 10

Baseline, 6- and 12-Month Performances of ICH Group with Comparison to Control Group on Mood Outcomes

Measures	ICH Group									Control Group			Differences between groups 6 months			Differences between groups 12 months		
	Baseline			6 months			12 months											
	n	Mean	SD	n	Mean	SD	n	Mean	SD	N	Mean	SD	F	p	Eta^2	F	p	Eta^2
Hospital Anxiety Depression Scale																		
Anxiety	21	3.71	3.85	22	4.18	3.42	23	3.30	3.64	34	2.88	2.71	2.317	.134	.041	.190	.665	.003
Depression	21	4.10	4.23	22	4.45	3.58	23	5.33	8.07	34	1.68	1.93	14.168	**.000**	.210	9.472	**.003**	.150
General Health Questionnaire-28																		
Somatic symptoms	21	1.48	1.81	21	1.33	1.96	23	1.52	1.73	34	0.50	1.48	3.203	.079	.057	5.698	**.020**	.094
Anxiety and insomnia	21	1.38	1.83	21	1.24	1.95	23	0.87	1.69	34	0.68	1.41	1.536	.221	.028	.220	.641	.004
Social dysfunction	21	1.81	1.81	21	1.29	1.27	23	1.22	1.68	34	0.53	0.96	6.271	**.015**	.110	3.866	.054	.066
Severe depression	21	0.48	1.33	21	0.05	.22	23	0.17	0.49	34	0.03	0.17	.119	.732	.002	2.511	.119	.044
Total score	21	6.00	5.55	21	3.90	4.02	23	3.78	4.51	34	1.74	3.32	4.718	**.034**	.080	3.902	.053	.066

Note: Bold typeface highlights significant difference.

terms of the ICH group performances at 6 and 12 months. The means for all measures of mood were higher for the ICH group at all the time points compared to controls, indicating the ICH group experienced more somatic symptoms, social dysfunction, anxiety, and depression symptoms than the controls at baseline, 6 months, and 12 months post-ICH. All means for the HADS were, however, in the normal range of anxiety and depression expected of the general population (i.e., these means are not reflective of a clinically significant syndrome).

As can be seen in Table 10 the one-way ANOVA indicates that differences between the control group and ICH group at both 6 and 12 months on the depression scale of the HADS were significant (ICH group scored significantly higher indicating greater symptoms of depression), but differences on the anxiety scale were not significant. In regards to the effect size of these differences Cohen, Cohen, West, and Aiken's (2013) criteria for one-way ANOVAs was used to interpret eta squared which states that an effect size is small if >0.01, medium if >0.06, and large if >0.14. The difference in depression scores at both 6 and 12 months is therefore large.

On the GHQ-28, the ICH group reported significantly more social dysfunction at 6 months post-ICH (medium effect size) and significantly more somatic symptoms at 12 months post-ICH compared to the control group (medium effect size).

Measures of cognitive functioning.

Table 11 presents descriptive statistics and the results of one-way ANOVAs comparing the performance of the ICH group and control group on measures of cognitive functioning. As would be expected, on the CNS-Vital Signs battery the controls scored in the average range on all measures. Compared to the control group, the ICH group means were lower on all measures, at all times of measure, with the exception of Social Acuity at baseline where the two groups were similar. Whilst there was a very small sample size of ICH Group participants who could complete the CNS-VS measure, which limits interpretability of the

Table 11

Baseline, 6- and 12-Month Performances of ICH Group with Comparison to Control Group on Cognitive Outcomes

	ICH Group Baseline			ICH Group 6 months			ICH Group 12 months			Control Group			Differences between groups 6 months			Differences between groups 12 months		
Measures	N	Mean	SD	n	Mean	SD	n	Mean	SD	n	Mean	SD	F	P	Eta^2	F	p	Eta^2
CNS-Vital Signs Domains																		
NCI	6	85.83	15.92	4	80.00	20.05	3	57.67	27.21	34	101.94	11.61	10.971	**.002**	0.23	31.903	**.000**	0.48
CM	6	88.50	21.49	5	90.20	18.07	5	79.6	19.97	34	104.41	16.12	3.294	.078	.082	9.756	**.003**	0.21
Verbal Memory	6	85.80	20.27	5	93.00	21.48	5	86.20	22.53	34	106.03	16.39	2.555	.118	.065	5.818	**.021**	0.14
Visual Memory	6	95.83	16.93	5	90.20	14.75	6	83.67	24.79	34	101.97	18.44	1.848	.182	.048	4.543	**.040**	0.11
PS	7	91.00	24.50	5	75.20	23.26	6	76.83	17.38	34	107.65	18.20	12.961	**.001**	0.26	14.784	**.000**	0.28
EF	7	81.43	19.31	5	80.40	17.27	6	73.17	29.86	34	100.06	17.08	5.762	**.022**	0.13	9.952	**.003**	0.21
PMS	7	82.71	20.52	5	78.20	19.72	6	73.67	36.50	34	104.12	15.80	11.068	**.002**	0.23	12.068	**.001**	0.24
RT	7	89.43	19.29	5	70.00	24.70	6	68.17	42.50	34	101.71	17.25	13.234	**.001**	0.26	11.566	**.002**	0.23
CA	7	79.71	22.31	4	87.75	12.61	3	53.0	48.51	34	100.32	14.15	2.877	.098	.074	19.106	**.000**	0.35
CF	7	79.14	21.31	5	83.20	16.18	6	72.67	34.06	34	99.50	16.87	4.106	**.050**	0.10	9.185	**.004**	0.19
WM	6	96.00	8.60	4	101.00	18.13	4	89.75	17.08	34	104.18	11.22	.253	.618	.007	5.331	**.027**	0.13
Sus At	6	97.33	6.59	4	96.00	18.89	4	85.25	25.93	34	104.38	11.79	1.601	.214	.043	7.145	**.011**	0.17
SA	7	101.43	13.53	5	86.40	12.22	6	99.17	10.03	34	101.15	16.81	3.537	.068	.087	.077	.782	.002
Behavioural Dyscontrol Scale																		
ANS	19	46.28	31.76	18	38.33	15.98	20	44.00	27.37	34	19.47	4.70	41.310	**.000**	0.45	26.329	**.000**	0.34
Total Score	19	18.6	6.64	19	19.89	4.94	20	19.80	5.42	34	25.44	2.31	31.035	**.000**	0.38	28.406	**.000**	0.35
Montreal Cognitive Assessment																		
Total Score	16	23.81	4.79	17	23.65	7.06	20	25.20	4.12	34	28.56	1.86	14.686	**.000**	0.23	16.882	**.000**	0.25

Note: Bold typeface highlights significant difference. ANS=Alphanumeric Sequencing; CA=Complex Attention; CF=Cognitive Flexibility; CM=Composite Memory; EF= Executive Functioning; NCI = Neurocognitive Index; PMS-Psychomotor Speed; PS=Processing Speed; RT=Reaction Time; SA=Social Acuity; Sus At= Sustained Attention; WM=Working Memory.

results, it can be seen that on the majority of the other CNS-Vital Signs scales the ICH group performed in the low average range. As seen in Table 11, the results of the ANOVAs show that at 6 months the ICH group scored significantly worse than the controls on 9 of the 16 measures of cognitive functioning (7 of these effect sizes are large with the other 2 being medium); whilst at 12 months the ICH group scored significantly worse than the control group on 15 of the 16 measures of cognitive functioning (13 of these effect sizes were large with the other 2 being medium).

On the Behavioural Dyscontrol Scale total score and Alphanumeric Sequencing score it can be seen that the ICH group scored significantly worse than the control group at both 6 and 12 months, with a large effect size. On the MoCA a score of 26 or above is considered to be in the normal range. The mean score for the control group on the MoCA was in the normal range as expected. For the ICH group, however, the mean score at all 3 time points is in the clinically significant range indicating cognitive impairment; the means at baseline and 6 months were similar and then an improvement was made at 12 months, with the mean score approaching the normal range. At both 6 and 12 months the ICH group scored significantly worse on the MoCA compared to the control group, with a large effect size found.

Measures of functional outcome.

Table 12 presents the performance of the control group and the ICH group across measures of functional outcomes including general functional outcome and HRQoL. As can be seen in Table 12, the ICH group scored significantly lower on the Barthel Index than the control group at each point of comparison, suggesting that the ICH group had less independence in activities of daily living than the control group.

The average normed score on the SF-36 is 50 (SD=10), hence scores lower than this indicate poorer HRQoL. As expected the control group scored in the average range on all SF-36 domains. The ICH group means were lower than the control group means on all scales of

Table 12

Baseline, 6- and 12-Month Performances of ICH Group with Comparison to Control Group on Functional Outcomes

	ICH Group Baseline			ICH Group 6 months			ICH Group 12 months			Control Group			Differences between groups 6 months			Differences between groups 12 months		
Measures	N	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD	F	P	Eta^2	F	P	Eta^2
Barthel Index																		
Total score	34	15	6.85	34	17.71	4.80	35	18.00	4.43	34	19.97	0.17	7.572	**.008**	0.10	6.728	**.012**	0.09
Short Form-36																		
PF	20	36.37	15.09	22	39.23	14.37	23	45.20	8.54	34	51.75	9.20	11.530	**.001**	0.21	4.034	.051	.211
RP	20	40.92	11.19	22	39.53	11.92	23	42.80	12.64	34	50.60	9.16	10.471	**.002**	0.20	4.700	**.036**	0.10
BP	20	47.22	13.46	22	49.50	14.81	23	48.33	15.78	34	52.44	8.27	.694	.410	.016	1.218	.276	.016
GH	20	49.65	10.17	22	48.48	12.31	23	52.61	9.34	34	51.29	6.38	.988	.326	.022	.275	.603	.022
Vitality	20	51.63	10.41	22	45.18	10.96	23	50.49	9.80	34	55.69	7.49	12.916	**.001**	0.23	3.226	.080	.231
SF	20	42.20	14.47	22	45.77	13.82	23	50.06	11.15	34	54.56	6.28	8.585	**.005**	0.17	2.710	.107	.166
RE	20	49.17	11.40	22	49.56	10.90	23	52.15	7.09	34	53.45	5.47	2.480	.123	.055	.380	.541	.055
MH	20	55.08	8.94	22	53.35	5.67	23	55.54	8.49	34	54.32	6.01	.226	.637	.005	.008	.928	.005
PCS	20	37.00	9.29	22	41.47	12.11	23	42.30	10.79	34	48.89	7.84	7.772	**.007**	0.13	7.132	**.010**	0.11
MCS	20	52.61	9.29	22	50.83	11.16	23	53.28	8.25	34	55.72	6.39	4.357	**.042**	0.07	1.577	.214	.028

Note: Bold typeface highlights significant difference. BP= Bodily Pain; GH=General Health; MCS=Mental Component Summary; MH=Mental Health; PCS=Physical Component Summary; PF= Physical Functioning; RE- Role Emotional RP=Role Physical; SF=Social Functioning.

the SF-36 at all times of measure, except for General Health at 12 months and Mental Health at baseline and 12 months. The ICH group scored more than 1 SD below the mean on Social Functioning, Physical Functioning, and the Physical Component Summary at baseline and on Role Physical and Physical Functioning at 6 months. All scores for the ICH group on the SF-36 were within 1 SD of the mean at 12 months.

Table 12 demonstrates that the ICH group had significantly worse functional outcome than controls on 7 out of 11 measures at 6 months. A large effect size was found for four of the measures; specifically the SF-36 Social Functioning, Vitality, Role Physical, and Physical Functioning scales. The ICH group scored significantly worse than controls on 3 of the 11 measures at 12 months post-ICH. The ICH group was worse off specifically in regards to physical HRQoL (Role Physical and Physical Component Summary score from SF-36) and basic ADLs (Barthel Index). Though, as noted above, their performance on SF-36 subscales all fell within 1 SD of the normative mean and the effect sizes are in the medium range.

Section 3: The Natural Course of Recovery over the First Year Post-ICH

In order to examine the natural course of recovery over time for the ICH group, a series of repeated measure within subject contrasts (ANOVA) were performed. These are presented for measures of mood, followed by measures of cognitive functioning and measures of functional outcome.

Measures of mood.

Table 13 presents change over time for the ICH group across measures of mood. As can be seen in Table 13, on the HADS Anxiety Scale the ICH group on average show a slight increase in anxiety symptoms from baseline to 6 months although this was not significant. There was then an improvement on the HADS Anxiety Scale from 6 to 12 months which was statistically significant and indicates fewer symptoms of anxiety. On the HADS Depression

Table 13

Change Over Time for ICH Group on Measures of Mood

	Time of measure									Significance of change					
	Baseline			6 months			12 months			Baseline 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	21	3.71	3.85	22	4.18	3.42	23	3.30	3.64	21	.564	.461	21	9.633	**.006**
Depression	21	4.10	4.23	22	4.45	3.58	23	5.33	8.07	21	.697	.414	21	2.424	.135
General Health Questionnaire-28															
Somatic Symptoms	21	1.48	1.81	21	1.33	1.96	23	1.52	1.73	20	.157	.697	21	.031	.863
Anxiety and Insomnia	21	1.38	1.83	21	1.24	1.95	23	0.87	1.69	20	.094	.762	21	.856	.366
Social Dysfunction	21	1.81	1.81	21	1.29	1.27	23	1.22	1.68	20	3.353	.083	21	.023	.880
Severe Depression	21	0.48	1.33	21	0.05	.22	23	0.17	0.49	20	2.202	.154	21	1.875	.186
Total Score	21	6.00	5.55	21	3.90	4.02	23	3.78	4.51	20	1.639	.216	21	.240	.629

Note: Bold typeface highlights significant change.

Scale the mean indicates an increase in depression symptoms over the 12 months, although the differences from baseline to 6 months, and 6 months to 12 months were not significant.

On the GHQ-28 Somatic Symptoms Scale the mean is similar at baseline and 12 months, with a small decline (i.e., fewer symptoms) at 6 months which was not significant. On both the Anxiety and Insomnia Scales and the Social Dysfunction Scale there is a decrease in the score over time, indicating fewer symptoms, although this change was not significant. On the Severe Depression Scale the mean at baseline is higher than the mean at 6 months indicating some improvement over time, with the mean at 12 months being slightly higher than that at 6 months. Again, these differences were not significant. Overall on the GHQ-28 the total scores indicate the ICH group improved over the first 12 months, although the differences from baseline to 6 months and 6 months to 12 months were not significant.

Measures of cognitive functioning.

Table 14 presents change over time for the ICH group across measures of cognitive functioning. As can be seen in Table 14, the sample size of people who repeated the CNS-Vital Signs measures is very small, so caution should be taken in interpreting these results, which should not be considered conclusive. On the Neurocognition Index the mean for the ICH group is in the low average range at baseline and 6 months, but drops to the extremely low range at 12 months. This may be explained by the very small group size rather than reflecting a true drop in cognitive functioning. In examining the data, it was found that those people who completed the measure only at baseline had higher scores and then did not always participate in later assessments, whilst those not participating in the baseline assessment tended to produce lower scores when assessed at later time periods. The participants assessed only at baseline tended to report that they were too busy to complete the

Table 14

Within-Subject Change Over Time for ICH Group on Measures of Cognitive Outcomes

	Time of measure									Significance of change					
	Baseline			6 months			12 months			Baseline 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	6	85.83	15.92	4	80.00	20.05	3	57.67	27.21	2	.250	.705	2	1.000	.500
CM	6	88.50	21.49	5	90.20	18.07	5	79.6	19.97	2	.405	.639	2	1.000	.500
Verbal Memory	6	85.80	20.27	5	93.00	21.48	5	86.20	22.53	2	625.000	**.025**	2	1.731	.414
Visual Memory	6	95.83	16.93	5	90.20	14.75	6	83.67	24.79	2	.490	.611	3	.771	.473
PS	7	91.00	24.50	5	75.20	23.26	6	76.83	17.38	3	.100	.782	3	.287	.646
EF	7	81.43	19.31	5	80.40	17.27	6	73.17	29.86	3	5.846	.137	3	.132	.751
PMS	7	82.71	20.52	5	78.20	19.72	6	73.67	36.50	3	.451	.571	3	1.548	.339
RT	7	89.43	19.29	5	70.00	24.70	6	68.17	42.50	3	4.585	.166	3	.747	.478
CA	7	79.71	22.31	4	87.75	12.61	3	53.0	48.51	3	.399	.592	2	.207	.728
CF	7	79.14	21.31	5	83.20	16.18	6	72.67	34.06	3	6.462	.126	3	.033	.873
WM	6	96.00	8.60	4	101.00	18.13	4	89.75	17.08	2	56.250	.084	2	49.000	.090
Sus At	6	97.33	6.59	4	96.00	18.89	4	85.25	25.93	2	49.000	.090	2	.040	.874
SA	7	101.43	13.53	5	86.40	12.22	6	99.17	10.03	3	.231	.678	3	.090	.792
Behavioural Dyscontrol Scale															
ANS (s)	19	46.28	31.76	18	38.33	15.98	20	44.00	27.37	16	.181	.677	17	.052	.823
Total Score	19	18.6	6.64	19	19.89	4.94	20	19.80	5.42	17	2.119	.165	18	.512	.484
Montreal Cognitive Assessment															
Total Score	16	23.81	4.79	17	23.65	7.06	20	25.20	4.12	15	.053	.821	16	.732	.406

Note: Bold typeface highlights significant change. ANS=Alphanumeric Sequencing; CA=Complex Attention; CF=Cognitive Flexibility; CM=Composite Memory; EF= Executive Functioning; NCI = Neurocognitive Index; PMS-Psychomotor Speed; PS=Processing Speed; RT=Reaction Time; SA=Social Acuity; Sus At= Sustained Attention; WM=Working Memory.

CNS-Vital Signs battery again at the later time periods. There was only one difference over time found to be significant on the CNS-Vital Signs and that was the improvement in Verbal Memory from baseline to 6 months.

On the Behavioural Dyscontrol Scale and the MoCA, however, there is a more robust sample who completed measures over time. The means for the total score on the Behavioural Dyscontrol Scale and Alphanumeric Sequencing score improve from baseline to 6 months (they took less time to complete the alphanumeric sequence and scored better on other cognitive tasks), and then decrease slightly from 6 to 12 months, although none of these were statistically significant. On the MoCA, there is an improvement in the mean scores over time, although this is again not significant.

Measures of functional outcome.

Table 15 presents change over time for the ICH group across measures of functional outcome. As can be seen in Table 14, the mean score on the Barthel Index increased over time. The differences on this score from baseline to 6 months and from 6 months to 12 months are significant, indicating that there was an improvement in basic activities of daily living over the first year post-ICH. On the SF-36, there was an improvement on the Physical Functioning Scale over time, with the improvement from 6 to 12 months being significant. On the Role Physical and Bodily Pain Scales there is little variation in scores over time, with no significant differences noted. On the General Health Scale there is little difference between the means at baseline and 6 months, but there is a significant difference between the mean at 6 and 12 months, with ICH survivors perceiving their general HRQoL to be better at 12 months. On the Vitality Scale the mean at baseline and 12 months are similar; however there is a decrease in the mean (indicating a decrease in felt sense of vitality) at 6 months. The increase from 6 to 12 months is significant. On both the Social Functioning and Role

Table 15

Change Over Time for ICH Group on Measures of Functional Outcomes

Measures	Time of measure									Significance of change					
	Baseline			6 months			12 months			Baseline to 6 months			6 to 12 months		
	N	Mean	SD	n	Mean	SD	n	Mean	SD	n	F	P	n	F	p
Barthel Index Total score	34	15	6.85	34	17.71	4.80	35	18.00	4.43	33	8.606	**.006**	34	6.926	**.013**
Short Form-36															
Physical Functioning	20	36.37	15.09	22	39.23	14.37	23	45.20	8.54	20	.271	.609	22	7.524	**.013**
Role Physical	20	40.92	11.19	22	39.53	11.92	23	42.80	12.64	20	1.348	.259	22	.854	.367
Bodily Pain	20	47.22	13.46	22	49.50	14.81	23	48.33	15.78	20	2.692	.116	22	.002	.967
General Health	20	49.65	10.17	22	48.48	12.31	23	52.61	9.34	20	.163	.691	22	7.324	**.014**
Vitality	20	51.63	10.41	22	45.18	10.96	23	50.49	9.80	20	3.162	.090	22	6.022	**.024**
Social Functioning	20	42.20	14.47	22	45.77	13.82	23	50.06	11.15	20	.224	.641	22	3.165	.091
Role Emotional	20	49.17	11.40	22	49.56	10.90	23	52.15	7.09	20	.062	.806	22	.948	.342
Mental Health	20	55.08	8.94	22	53.35	5.67	23	55.54	8.49	20	.299	.590	22	.271	.609
Physical Component Summary	20	37.00	9.29	22	41.47	12.11	23	42.30	10.79	20	3.506	.077	22	.025	.876
Mental Component Summary	20	52.61	9.29	22	50.83	11.16	23	53.28	8.25	20	.512	.483	22	4.635	**.044**

Note: Bold typeface highlights significant change.

Emotional Scales means improve over time, although not significantly so. On the Mental Health Scale the mean is similar at baseline and 12 months, but there is a dip at 6 months although this is not significant. The Physical Component Summary score of the SF-36 indicates an improvement in the means over time although this is not significant. The Mental Component Summary Score mean is similar at baseline and 12 months, but there is a dip at 6 months, resulting in the increase from 6 to 12 months being significant.

Section 4: Interrelationships between Mood, Cognitive Functioning, and Functional Outcomes of ICH at 12 Months Post-ICH

In this section bivariate correlations between the various outcomes of interest (i.e., cognition, mood, functional outcomes) at 12 months are examined. In addition, the correlations between each of these areas and demographic related characteristics at baseline will be examined. Interrelationships found could give a better overall picture of how stroke outcomes relate to each other and what variables may relate to good versus poor outcome. Pearson's correlation coefficients and Spearman's Rho will be used as appropriate to the data. Due to the small sample size and large number of variables $p>0.1$ will be used to determine significance; those correlations with $p>0.5$ will be discussed as trends.

Correlations between measure of mood and cognitive outcomes.

As can be seen in Table 16, there were no correlations significant at the $p>0.1$ level. However, a trend to positive correlation was found between the Neurocognition Index score and the Severe Depression Scale of the GHQ-28, indicating that the higher the depression score, the higher the score of overall cognitive functioning; this was, however, based on a sample size of only 3 people. Negative trends were also found between the HADS Anxiety Scale and CNS-VS scales for Cognitive Flexibility and Executive Functioning, indicating the more anxiety symptoms someone had the worse they did on tasks requiring cognitive flexibility and executive functioning. Similarly, negative trends were found between the

Table 16

Correlations between Measures of Mood and Cognitive Outcome

		HADS						General Health Questionnaire-28							
Measure		Anxiety		Depression		Somatic Symptoms		Anxiety and Insomnia		Social Dysfunction		Severe Depression		Total Score	
	n	r	p	r	P	r	p	R	P	r	P	R	p	r	p
CNS-Vital Signs Domains															
NCI	3	-.041	.974	-.290	.813	.074	.953	.562	.620	.918	.259	.997	**.047**	.779	.431
CM	5	-.207	.738	-.468	.426	-.167	.789	.064	.919	.552	.335	.787	.114	.241	.696
Verbal Memory	5	-.353	.560	-.533	.355	-.278	.651	.053	.933	.725	.166	.836	.078	.244	.932
Visual Memory	6	.060	.909	-.424	.402	-.184	.727	-.068	.899	-.329	.525	.164	.756	-.157	.767
PS	6	.056	.916	-.406	.424	-.327	.527	-.438	.385	-.187	.722	-.516	.295	-.608	.201
EF	6	-.815	**.048**	-.054	**.031**	-.707	.116	-.562	.246	.188	.721	.404	.427	-.375	.464
PMS	6	.483	.332	-.046	.424	.163	.758	.283	.587	-.187	.722	.197	.709	.157	.767
RT	6	.259	.621	.091	.863	.190	.719	.252	.630	.056	.916	.498	.315	.268	.608
CA	3	-.926	.247	-.991	.086	-.876	.320	-.518	.654	.742	.467	.482	.680	-.245	.842
CF	6	-.880	**.048**	-.854	**.031**	-.745	.089	-.606	.202	.208	.693	.365	.477	-.409	.421
WM	4	.038	.962	-.141	.859	.123	.877	.558	.442	.919	.081	.933	.067	.776	.224
Sus At	4	.389	.611	.147	.853	.514	.486	.679	.321	.570	.430	.822	.178	.864	.136
SA	6	-.237	.651	-.427	.399	-.368	.473	-.592	.216	-.375	.464	-.226	.666	-.511	.300
Behavioural Dyscontrol Scale															
ANS (s)	20	.111	.642	.037	.878	.194	.412	-.070	.770	.269	.252	-.055	.817	.137	.565
Total score	20	-.063	.792	-.003	.990	.090	.706	-.001	.996	-.005	.982	.238	.313	.055	.818
Montreal Cognitive Assessment															
Total score	20	-.084	.725	-.011	.962	-.092	.700	.087	.716	-.195	.410	.176	.459	-.053	.824

Note: Bold typeface highlights a trend. ANS=Alphanumeric Sequencing; CA=Complex Attention; CF=Cognitive Flexibility; CM=Composite Memory; EF= Executive Functioning; NCI = Neurocognitive Index; PMS-Psychomotor Speed; PS=Processing Speed; RT=Reaction Time; SA=Social Acuity; Sus At= Sustained Attention; WM=Working Memory.

HADS Depression Scale and CNS-VS Scales for Cognitive Flexibility and Executive Functioning. No other trends ($p<0.05$) were found on measures of mood and cognition.

Correlations between measures of cognitive and functional outcomes.

As Table 17 demonstrates, there were no significant correlations at the $p>.01$ level. There was, however, a negative trend between the CNS-VS Neurocognition Index and the Social Functioning Scale of the SF-36, indicating that overall poorer cognitive functioning was associated with greater social dysfunction. There was also a negative trend in the correlations between Complex Attention on the CNS-VS and both the Role Physical Scale and the General Health Scale of the SF-36, indicating those people with poorer complex attention skills rated their general health as being poorer and felt their physical roles had been compromised more than those who scored better on complex attention tasks. No other trends ($p<0.05$) were found between measures of cognitive and functional outcomes.

Correlations between measures of mood and functional outcomes.

As can be seen in Table 18, there was a negative trend in the correlation between the SF-36 Social Functioning Scale and the Somatic Symptoms Scale of the GHQ-28, indicating those with greater Somatic Symptoms experienced greater social dysfunction. The Physical Component Summary Score of the SF-36 was significantly negatively correlated with Somatic Symptoms and Total Score on the GHQ-28, and there was a trend towards negative correlation with the Physical Component Summary Score of the HADS Depression Scale and Social Dysfunction on the GHQ-28. This indicated that the lower participants ranked their physical HRQoL the more mood symptoms they reported. The Mental Component Summary Score of the SF-36 significantly negatively correlated with most measures of mood (HADS Anxiety Scale and Somatic Symptoms, Anxiety and Insomnia, Severe Depression, and Total Score on the GHQ-28); and had a trend towards negative correlation with the remaining

Table 17

Correlations between Measures of Cognitive and Functional Outcome

		Barthel Index		Short Form-36																			
Measures		Total Score		Physical Functioning		Role Physical		Bodily Pain		General Health		Vitality		Social Functioning		Role Emotional		Mental Health		Physical Component Summary		Mental Component Summary	
	n	r	p	R	p	r	p	R	P	r	p	R	P	r	p	R	P	r	p	r	p	r	p
CNS-Vital Signs																							
NCI	3	.000	1.000	.710	.498	-.435	.714	-.562	.620	-.435	.714	.562	.620	-.997	**.047**	.000	1.000	-.777	.433	-.789	.421	-.749	.461
CM	5	-.207	.738	.051	.935	-.590	.295	-.235	.703	-.479	.415	.056	.929	-.760	.136	-.375	.534	-.556	.331	-.354	.559	-.536	.351
VM	5	-.169	.786	.099	.874	-.554	.332	-.192	.757	-.660	.225	-.019	.975	-.606	.279	-.194	.755	-.629	.255	-.329	.589	.409	.494
Vis M	6	-.046	.931	. 207	.695	.099	.852	.028	.957	.247	.637	.101	.849	-.447	.374	-.742	.091	.022	.967	.369	.471	-.192	.715
PS	6	-.230	.661	-.220	.676	.116	.827	.236	.653	.443	.379	-.248	635	-.208	.692	-.722	.105	.162	.759	.672	.144	.284	.585
EF	6	-.112	.833	-.324	.531	-.502	.311	.441	.382	-.690	.129	-.481	.334	-.630	.180	-.500	.312	-.454	.366	.365	.477	.111	.834
PMS	6	-.327	.527	.254	.628	-.025	.963	-.512	.299	.310	.550	.244	.641	-.380	.458	-.236	.653	-.174	.741	-.067	.900	-.417	.411
RT	6	-.021	.968	.257	.623	-.110	.835	-.252	.630	.181	.731	.185	.726	-.645	.167	-.385	.451	-.059	.911	-.145	.784	-.627	.183
CA	3	.000	1.000	-.345	.776	-1.000	**.013**	.518	.654	-1.000	**.013**	-.518	.654	-.482	.680	.000	1.000	-.896	.293	.230	.852	.291	.812
CF	6	-.105	.842	-.370	.471	-.512	.299	.487	.327	-.748	.087	-.522	.289	-.568	.240	-.449	.372	-.463	.355	.377	.461	.187	.722
WM	4	.000	1.000	.637	.363	-.096	.904	-.558	.442	-.386	.614	.514	.486	-.219	.781	.185	.815	-.752	.248	-.797	.203	-.654	.346
Sus At	4	.000	1.000	.641	.359	-.031	.969	-.679	.321	.062	.938	.548	.452	-.414	.586	-.096	.904	-.354	.646	-.929	.071	-.956	**.035**
SA	6	-.578	.229	-.778	.068	-.691	.128	.116	.827	-.193	.714	-.770	.073	-.679	.138	-.203	.700	-.390	.445	.220	.675	.117	.825
Behavioural Dyscontrol Scale																							
ANS (s)	16	-.095	.692	.078	.775	-.255	.340	.020	.940	-.338	.200	-.168	.535	-.179	.507	.128	.637	-.029	.914	-.211	.373	.053	.823
Total	16	.122	.609	.037	.891	.141	.603	-.040	.883	-.049	.857	.083	.759	-.100	.713	-.341	.197	.085	.754	-.107	.654	-.171	.470
Montreal Cognitive Assessment																							
Total	15	-.205	.385	.150	.595	.344	.209	-.042	.881	.344	.209	.394	.147	.163	.563	-.121	.668	.033	.906	-.114	.634	-.038	.874

Note: Bold typeface highlights a trend. ANS=Alphanumeric Sequencing; CA=Complex Attention; CF=Cognitive Flexibility; CM=Composite Memory; EF= Executive Functioning; NCI = Neurocognitive Index; PMS-Psychomotor Speed; PS=Processing Speed; RT=Reaction Time; SA=Social Acuity; Sus At= Sustained Attention; WM=Working Memory.

Table 18

Correlations between Measures of Mood and Functional Outcome

	HADS						General Health Questionnaire-28														
Measures	Anxiety Scale			Depression Scale			Somatic Symptoms			Anxiety and Insomnia			Social Dysfunction			Severe Depression			Total Score		
	n	r	P	n	r	p	n	r	p	N	r	P	n	r	P	N	r	p	N	r	p
Barthel Index																					
Total Score	23	-.046	.834	23	.187	.394	23	.181	.410	23	.175	.426	23	.044	.842	23	.120	.586	23	.164	.455
Short Form-36																					
PF	17	-.040	.878	17	-.236	.363	17	-.048	.854	17	-.058	.824	17	-.204	.431	17	. 055	.833	17	-.109	.676
RP	17	.059	.822	17	-.037	.889	17	-.077	.768	17	.170	.514	17	-.165	.526	17	-.062	.813	17	-.032	.901
BP	17	-.063	.810	17	-.163	.533	17	-.366	.149	17	.051	.846	17	-.219	.398	17	.162	.536	17	-.178	.495
GH	17	.242	.350	17	.190	.464	17	-.291	.257	17	.214	.410	17	-.322	.193	17	.016	.951	17	-.147	.573
Vit	17	.066	.802	17	.012	.964	17	-.187	.472	17	.117	.655	17	-.264	.306	17	.103	.695	17	-.111	.671
SF	17	.052	.842	17	.204	.431	17	-.490	**.046**	17	.092	.725	17	-.413	.100	17	-.209	.420	17	-.320	.211
RE	17	.096	.715	17	.343	.178	17	.124	.636	17	.251	.331	17	.228	.378	17	.029	.913	17	.227	.381
MH	17	-.095	.716	17	-.023	.929	17	.223	.369	17	-.023	.931	17	.071	.787	17	-.096	.714	17	-.092	.726
PCS	17	-.234	.282	17	-.446	**.033**	17	-.642	.001*	17	-.290	.179	17	-.496	**.016**	17	-.234	.284	17	-.564	.005*
MCS	17	-.689	.000*	17	-.472	**.023**	17	-.640	.001*	17	-.813	.000*	17	-.476	**.022**	17	-.582	.004*	17	-.790	.000*

Note: Bold typeface highlights a trend; * indicates significant correlation at p>.05. BP=Bodily Pain; GH=General Health; MCS=Mental Component Summary; MH=Mental Health; PCS=Physical Component Summary; PF=Physical Functioning; RE=Role Emotional; RP=Role Physical; SF=Social Functioning; Vit=Vitality.

measures (HADS Depression Scale and Social Dysfunction on the GHQ-28). This indicates that the more mood symptoms participants reported the worse mental HRQoL they reported. No other trends or significant correlations were found on measures of mood and functional outcome.

Correlations between 12-month outcomes and baseline demographic characteristics.

It is useful to examine factors at baseline that may relate to outcome post-ICH so as to be able to better understand who is vulnerable to negative outcomes. The current study therefore utilised available baseline information on age, sex, and gender (characteristics that other research suggest may have an impact on post-stroke outcomes) to look for significant correlations ($p>.01$) or trends ($p>.05$) with mood, cognitive, and functional outcomes 12 months post-ICH.

As can be seen in Table 19, none of the measures of cognitive functioning were found to correlate significantly with the demographic characteristics. A correlation could not be calculated for ethnicity and CNS-VS measures as all ICH group participants who completed CNS-VS were of the same ethnicity (European). In regards to measures of mood, it was found that there was a significant negative correlation between age and HADS Anxiety Scale, and the GHQ-28 Anxiety and Insomnia and Severe Depression scales. There was also a trend towards a negative correlation for age and Total Score on the GHQ-28. This indicates those who are younger when they have an ICH experienced greater mood symptoms.

In terms of functional outcomes, it can be seen that there was a trend towards a correlation between the Barthel Index and ethnicity. The Mental Component Summary score of the SF-36 significantly positively correlated with age; indicating that people who are older when they have a stroke experience better mental health related quality of life.

Table 19

Correlations between Demographic Characteristics and Measures of Mood, Cognition, and Functional Outcome at 12 Months

	Age			Gender			Ethnicity		
	N	r	P	n	R	P	N	r	P
Cognitive Outcome Measures									
CNS-Vital Signs Domains									
Neurocognition Index	3	-.747	.463	3	.000	1.00			
Composite Memory	5	-.513	.377	5	-.354	.559			
Verbal Memory	5	-.435	.464	5	-.707	.182			
Visual Memory	6	-.401	.430	6	-.399	.434			
Processing Speed	6	.161	.761	6	-.133	.802			
Executive Function	6	.157	.766	6	-.655	.158			
Psychomotor Speed	6	-.634	.176	6	.393	.441			
Reaction Time	6	-.689	.130	6	-.266	.611			
Complex Attention	3	.293	.811	3	-.866	.333			
Cognitive Flexibility	6	.252	.630	6	-.655	.158			
Working Memory	4	-.755	.245	4	-.258	.742			
Sustained Attention	4	-.948	.052	4	-.258	.742			
Social Acuity	6	.329	.524	6	-.131	.805			
BDS									
Alphanumeric Sequencing (s)	20	-.407	.075	20	.009	.970	20	-.116	.627
Total Score	20	-.420	.065	20	.101	.673	20	-.306	.190
MoCA									
MoCA Total Score	20	-.398	.082	20	.083	.729	20	-.234	.321
Mood Outcome Measures									
HADS									
Anxiety	23	-.562	.005*	23	.109	.622	23	.237	.277

Depression	23	-.393	.063	23	.332	.122	23	.259	.233
GHQ-28									
Somatic Symptoms	23	-.302	.161	23	.015	.946	23	-.073	.740
Anxiety and Insomnia	23	-.670	.000*	23	.333	.121	23	.129	.558
Social Dysfunction	23	-.134	.542	23	-.302	.161	23	-.321	.136
Severe Depression	23	-.591	.003*	23	-.256	.239	23	-.119	.588
Total Score	23	-.480	**.020**	23	.014	.948	23	-.071	.749
Functional Outcome Measures									
Barthel Index									
Total Score	35	-.316	.065	35	-.250	.147	35	-.400	**.017**
Short Form-36									
Physical Functioning	23	.081	.720	23	-.041	.857	23	-.016	.943
Role Physical	23	-.075	.741	23	-.195	.385	23	-.310	.160
Bodily Pain	22	-.108	.631	23	-.250	.262	22	-.159	.478
General Health	23	-.277	.211	23	.049	.827	23	-.105	.641
Vitality	23	-.076	.738	23	-.049	.828	23	-.059	.794
Social Functioning	23	-.116	.608	23	.064	.779	23	.001	.995
Role Emotional	23	.233	.296	23	.048	.832	23	.012	.959
Mental Health	23	.132	.557	23	-.106	.640	23	-.325	.141
PCS	23	-.072	.743	23	-.228	.295	23	.140	.525
MCS	23	.641	.001*	23	-.007	.974	23	.000	1.000

Note: Bold typeface highlights significant change; * highlights a trend. MCS=Mental Component Summary; MoCA=Montreal Cognitive Assessment; PCS=Physical Component Summary.

Chapter 4: Discussion

This exploratory study compared short-term (baseline and 6-month) and long-term (12-month) mood, cognitive, and functional outcomes of ICH survivors to a healthy control group matched on age, gender, and ethnicity. Change within the ICH group was also tracked over time. Specific aims of the study were to identify measures on which the ICH group differed from the control group; to describe the natural course of recovery of mood, cognitive, and functional outcomes over the first year post-ICH; to examine the relationships between mood, cognitive, and functional outcomes of ICH at 12 months; and to determine if there are any demographic characteristics of the ICH survivors at baseline that correlate with good versus poor functional and neuropsychological outcomes at 1 year. Each of these is examined, in relation to the literature, below.

Section 1: Comparing the ICH group with matched controls

Whilst there was variability within the performance of the ICH group across measures, in general they scored worse than the control group at baseline, 6 months, and 12 months on measures of mood, cognition, and functional outcome. Each of these areas is discussed below with reference to relevant literature.

Mood

Across all measures of mood, at all times of measure, the ICH group mean was higher than the control group indicating they experienced greater mood difficulties overall. This makes sense given that having a stroke can be a serious shock for survivors and their family and bring about many changes and challenges for people. Further, the control group was a healthy sample so it was expected they would score in the normal range on measures of mood.

When examining only those differences between groups found to be statistically significant, symptoms of depression as measured by the HADS were greater among the ICH

group than controls at 6 and 12 months post-ICH. This is consistent with other studies finding greater depression among survivors of ICH and stroke more generally in the short-term (Hackett et al., 2005) and long-term post-stroke (Gill, 2011; Vykopal, 2011).

There was also a trend for scores to increase over the first year which may suggest somewhat of a delayed onset of depression symptoms after ICH. Delayed onset depression after stroke has been linked to psychosocial causes such as social isolation (Whyte & Mulsant, 2002). It is of note, however, that the mean scores noted in the ICH group were within the range of scores considered normal, though at the top end of the range (therefore meaning the symptoms were subclinical). This, taken together with the fact that there was no significant difference on the GHQ-28 severe depression scale, indicates that the level of depression typically experienced by ICH survivors may be mild. A key difference between the GHQ-28 and HADS is that the GHQ-28 is thought to measure severe depression (e.g., it includes questions on the presence of suicidal ideation whereas the HADS does not). Therefore, these results may indicate that the typical depression experienced by ICH survivors does not include suicidal ideation, which is consistent with the findings of Vykopal's (2011) study of ICH survivors.

The prevalence of clinically significant depression (according to the cut-off scores in the HADS) among the ICH group ranged from 14-19% over the first year post-ICH; which is lower than estimates from previous stroke studies which have used the HADS (24-30%; De Wit et al., 2008). However, other prevalence rates are typically from hospital-based studies, which tend to report higher rates of PSD than population-based studies (Hackett et al., 2005); likely because population-based studies also capture people who have had a milder stroke that did not require hospitalisation. Another population-based study of ICH at 5 year follow-up found a prevalence rate of depression of 15% using the HADS (Vykopal, 2011).

The other significant differences between groups, which were noted on the GHQ-28, were in relation to somatic symptoms (e.g., hot or cold spells, headaches, feeling run down) at 12 months, and social functioning (e.g., feeling you are playing a useful part in things, keeping busy and occupied) at 6 months. The ICH group reported greater somatic symptoms and social dysfunction compared to the controls. The difference in social dysfunction is consistent with research indicating that whilst stroke survivors tend to maintain family relationships they have less social contact with friends and neighbours directly after a stroke, but that this resumes throughout the first year post-stroke (Astrom et al., 1992). In terms of social dysfunction it appears there is a trend for this to decrease over the first year post-ICH, explaining why there was not a significant difference at 12 months. Somatic symptoms as a representation of mood disturbance are more common among older adults compared to the general adult population (Berry, Storandt, & Coyne, 1984), so it is possible that the greater rate of depression seen at 12 months in the ICH group could account for their greater reports of somatic symptoms. Vykopal (2011), however, did not find a difference between groups in regards to somatic symptoms, despite having found a greater prevalence of depression.

Interestingly, the difference between the ICH and controls on measures of anxiety (both the HADS and anxiety/insomnia scale of the GHQ-28) were not significant. The mean anxiety level on the ICH group on the HADS was within the low-mid portion of the normal range. This is inconsistent with the general stroke literature which does find higher rates of anxiety among stroke survivors compared to the general population (Campbell Burton et al., 2012). In the current study the prevalence of meeting criteria for clinically significant anxiety in the ICH group of this study fluctuated between 5-18% over the first year which is lower than the average of 25% found in a review of studies post-stroke (Campbell-Burton et al., 2012) and more consistent with the prevalence of anxiety in the general population which tends to range from 10-15% (Henderson, Andrews, and Hall, 2000; Wells et al., 2006). Given

that both the HADS and the GHQ-28 were used to assess anxiety, which are commonly used in the stroke literature and designed to assess both emotional and physical aspects of anxiety across different levels (i.e., from mild to severe), it does not seem as if the measures used can explain the lower than expected anxiety levels. It may be the case that, as with rates of PSD, PSA is lower in samples that are population-based compared to hospital-based. This is consistent with the fact other population-based studies of ICH and SAH have found no difference in anxiety between stroke survivors and matched controls using a clinical interview at 12 months (House et al., 1991) and using the GHQ-28 at 5 years (Gill, 2011; Vykopal, 2011). The findings of the current study therefore reflect that anxiety is a concern for some survivors of ICH, but perhaps the prevalence levels are not as unfavourable as other hospital-based stroke outcome studies would imply.

Cognitive functioning

Overall, the ICH group performed more poorly on cognitive measures than the control group at baseline, 6 months, and 12 months. Impaired performance across measures of cognitive functioning is commonly found in the ICH and stroke literature (Gill, 2011; Patel et al., 2002, 2003; Vykopal, 2011). When comparing their performance at 12 months on CNS Vital Signs, the ICH group appear to have widespread impairment across cognitive domains including: working memory, verbal memory, visual memory, processing speed, executive functioning, cognitive flexibility, psychomotor speed, reaction time, complex attention, and sustained attention. Throughout these domains the control group scored on average in the average range while the ICH group scored anywhere from low average to extremely low. This is consistent with other studies at 6 months, 1 year, and 5 years post-ICH that have found deficits in the domains of processing speed, executive functioning, attention, and memory (Fuh & Wang, 1995; Su et al., 2007;Vykopal, 2011). It is likely that the results of the current research underestimate the extent of cognitive dysfunction post-ICH given that

many participants could not complete CNS-Vital Signs and many of the larger sample were too unwell to participate all together.

Social acuity was the only cognitive domain in which the ICH group scored the same as the controls at 12 months, and the only domain in which they scored in the average range, suggesting this domain may typically be spared post-ICH. This could have important implications for continued social relationships among ICH survivors as social acuity is associated with interpersonal effectiveness (e.g., being able to empathise with others promotes social relationships; Funder & Harris, 1986). Other research looking at stroke more generally, however, has found impairments in social acuity in stroke survivors, which is, in turn, correlated with deficits in social participation and reduced mental HRQOL (Cooper et al., 2014).

Differences on CNS-Vital Signs domains were not as pronounced at 6 months as they were at baseline with the ICH group scoring in the average range for memory and attention tasks. Again, as stated in the results section, this may be explained by the very small group size rather than reflecting a true drop in cognitive functioning. Visual examination of the data revealed that participants who completed the measure only at baseline appear to have produced higher scores, whilst those with lower scores were assessed at later time periods. This may reflect greater disability during the acute stage, precluding participation in the neuropsychological aspect of the assessment.

Given the exploratory nature of the study and power limitations noted, it is of importance to consider patterns within the findings rather than looking at scores in isolation. Therefore, of note is that the cognitive impairment suggested by the CNS-Vital Signs is supported by BDS and MoCA results, which are more robust given the greater sample size; and given that the MoCA is designed only as a screening tool and therefore only detects more obvious impairment. At both 6 and 12 months the ICH group scored significantly worse than

controls on both measures. Examination of the data shows that according to the MoCA the prevalence of cognitive impairment in the ICH group participants who could complete the measure was 50%, 47%, and 45% at baseline, 6 months, and 12 months, respectively. This is higher than the prevalence found in general stroke studies using screening measures over the first year (22-39%; Douiri et al., 2013; Patel et al., 2002; Patel et al., 2003). Further, it is likely that given many people were too impaired for cognitive testing in the current study, including the MoCA, the true rate of cognitive impairment post-ICH has likely been underestimated. This would be consistent with other researcher's findings that ICH has the poorest cognitive outcomes of all the stroke subtypes; with some studies finding deficit rates of 80-96% in the acute phase post-ICH (Nys et al., 2007 & Su et al., 2007).

The BDS assesses executive functioning and self-regulation. The poor scores of the ICH group on the BDS, in combination with the poor performance of the ICH group on the executive functioning and cognitive flexibility scale on the CNS-Vital Signs, suggests that at both 6 and 12 months executive dysfunction is an issue for ICH survivors. Executive functioning was also found to be a significant area of impairment in a NZ study 5 years post-ICH (Vykopal. 2011) which is consistent with the wider stroke literature (Barker-Collo et al., 2010; Pohjasavaara et al., 2002) but not in other studies of ICH which look at haemorrhages in the basal ganglia specifically (Fuh & Wang, 1995; Liang et al., 2001, Su et al., 2007). Thus, the findings here may reflect the more varied area of damage related to the ICH.

Whilst the findings regarding cognitive outcomes in the current study should not be considered conclusive, it does appear that when comparing cognitive functioning in the ICH group versus the control group, ICH survivors experience cognitive impairment in both the short-term (6 months) and long-term (12 months). Whilst the exact profile of cognitive impairment post-ICH is not clear given the small sample size who could complete cognitive measures in the current study it does seem executive functioning is a particular area of deficit,

a finding which will be addressed further in the clinical implications section. It is also likely, given those within the ICH group with the worst cognitive functioning were excluded from taking part in the tests, that these findings underestimate the true extent of cognitive difficulties in ICH survivors.

Functional outcome

In regards to general functional outcome (i.e., Barthel Index) the ICH group performed significantly worse than controls at 6 and 12 months post-stroke. This indicates that ICH survivors have less independence in ADLs in the short-term and long-term compared to matched controls, which is consistent with the findings of other stroke (Chausson et al., 2010; Hankey et al., 2002) and ICH studies (Vykopal, 2011).

In regards to wider functional outcome (HRQoL) the picture is not so straightforward. Overall, ICH survivors experience poorer physical HRQoL at 6 and 12 months. It also appears that at 6 months, but not 12 months, ICH survivors perceive that their HRQoL in regards to social functioning and vitality was poorer than controls. Interestingly, ICH survivors did not report worse general health or bodily pain at any time point measured. The literature on HRQoL post-stroke is mixed. Many studies report poorer HRQoL among stroke survivors compared to the general population or matched controls from 3 months to 5 years post-stroke (Gill, 2011; Gokkaya et al., 2005; Jonkman et al., 2009; Niemi et al., 1988; Paul et al., 2005; Suenkeler et al., 2002). Whilst other studies looking at outcomes 5 years and beyond have found little difference in HRQoL (Anderson et al., 2004; Hackett et al., 2000). Studies specifically looking at ICH outcomes are also mixed; one study found ICH survivors at 3 months had poorer HRQoL (Christensen et al., 2009) and another found no difference between ICH survivors at 5 years and controls (Vykopal, 2011). It therefore seems that within the first year post-ICH poorer HRQoL, particularly physical HRQoL, is present among ICH survivors, though these disappear in the longer term. Given that ICH survivors experience

limitations in their basic ADLs post-stroke, it makes sense that they experience poorer quality of life in regards to their physical functioning and roles.

Section 2: Change over time in the ICH group

The natural course of recovery over the first year post-ICH was mixed. Generally it appears there was recovery of functioning in regards to independence in ADLs but limited spontaneous recovery in mood and cognition.

Mood

As noted above depression is a concern post-ICH. There was no significant change in the level of depression or social dysfunction experienced over the first year post-ICH, which was consistent with Vykopal's (2011) study of ICH survivors over 5 years. This suggests there was little spontaneous improvement in depression and that there is a need for interventions targeting low mood in post-stroke rehabilitation when depression is present.

Despite ICH survivors not experiencing more anxiety than the control group, the anxiety they did experience, showed a significant improvement from 6 to 12 months. This is consistent with some general stroke studies (Burvill et al., 1995a), yet inconsistent with others that found no change in anxiety over time (Vykopal, 2011), particularly from 6 months post-stroke onwards (Bergersen et al., 2010; Lincoln et al., 2013). The findings of this study in relation to anxiety suggest that in post-stroke rehabilitation in general there may be a need for prioritisation of interventions for depression over anxiety. However, there will be a proportion of ICH survivors for whom this does not hold true, hence, it should be assessed on a case-by-case basis, particularly within the shorter term.

Cognitive Outcomes

In regards to improvements made in cognitive functioning over time, the very limited sample that undertook the CNS-Vital Signs battery over time did not show any significant change, apart from improvement on the verbal memory scale from baseline to 6 months.

There would likely not be enough power with such a small sample size to be able to find significant differences but despite that, these results may point towards little spontaneous improvement in cognitive functioning over the first year post-ICH. This is consistent with the MoCA and BDS which had larger sample sizes and still found no significant improvement over time. The mean for the MoCA was in the clinically impaired range at all times of measure indicating that cognitive impairment was an ongoing issue for ICH survivors.

The wider stroke literature has mixed findings on the amount of spontaneous recovery over the first year post-stroke with some indicating little improvement over the first few months post-stroke (del Ser et al., 2005; Patel et al., 2003; Serrano et al., 2007), including one study of ICH specifically (Douriri et al., 2013); and others indicating gradual improvement (Ballard et al., 2003; Gottesman & Hillis, 2010; Rasquin et al., 2004; Rasquin, Lodder, & Verhey, 2005). The general literature therefore indicates recovery in cognitive functioning post-stroke is possible, with the acute phase potentially being the best time for recovery of function. The current study suggests that there is a need for intervention post-ICH that targets cognitive functioning; though further study with larger samples is required to validate this.

Functional Outcome

General functional outcome improved significantly over the first year post-ICH; reflected in an increase in independence in ADLs over time. The general stroke literature indicates an improvement in the acute stage post-stroke and a levelling off of this improvement, with it being a lot more gradual after the first few months post-stroke (Carod-Artal et al., 2002; Leonard et al., 1998). This is consistent with the results of the present study which indicated gradual improvement over the first year. An ICH study over 5 years found a good level of recovery of ADLs from 6 months to 5 years (Vykopal, 2011), suggesting that further recovery after the first year is possible; though an assessment at 12 months by Vykopal would have allowed better determination of this possibility. It makes sense that

survivors of ICH see improvements in their independence in daily life activities as they gain back physical functioning over time.

There were also some improvements seen in physical HRQoL, general health, and vitality from 6 to 12 months. This indicates that improvement can occur from short to long term in regards to HRQoL after ICH. It is possible that improvement will continue after the first 12 months, as Vykopal (2011) found improvement in physical HRQoL from 6 months to 5 years post-ICH. Whilst the current study found no significant change in mental HRQoL over time, other studies of ICH (Vykopal, 2011) and stroke in general (Kwok et al., 2006) have reported a significant worsening of mental HRQoL over the first 5 years. Studies looking at HRQoL overall are mixed; with some research noting a significant improvement between short and long-term outcomes (Jonkman et al., 2009) and other studies noting no significant difference between HRQoL in the acute stage and at 5 years (Feigin et al., 2010) and even 10 years (Wolfe et al., 2011) post-stroke. The findings of the current study indicate that it is worthwhile breaking down HRQoL outcomes and that some improvement in HRQoL can occur over the first year post-stroke.

Section 3: Relationships between outcomes 12 months post-ICH

Significant correlations between measures of mood, cognition, and functioning are discussed below, as are correlations which were expected given other literature, but not found in the current study.

Mood and cognitive outcomes

Symptoms of anxiety and depression were significantly correlated with executive functioning and cognitive flexibility tasks. The more symptoms of anxiety or depression someone had the poorer they performed on these tasks. Whilst these correlations are based on a small sample they make intuitive sense (e.g., distractibility caused by anxiety and lack of motivation associated with depression could hinder performance on tasks, or alternatively

being aware of cognitive decline could lead to depression) and are consistent with findings in the general stroke literature (Kauhanen et al., 1999; Pohjasavaara et al., 2002; Starkstein et al., 1990). Some studies, however, find no link between PSA and cognitive functioning (e.g., Shimoda & Robinson, 1998; Starkstein et al., 1990; Vypokal, 2010) although cognitive functioning, particularly cognitive speed, explained nearly 40% of the variance in PSA in another study (Barker-Collo, 2007).

The only other significant correlation within this category was a positive correlation between severe depression and an overall summary score for cognitive functioning. This finding would suggest that those who were more depressed performed better on cognitive tasks. This does not make intuitive sense and is not backed up by other literature. Given the small sample size and the relatively large number of correlations run this could be a spurious correlation. Other studies with ICH survivors (e.g., Vykopal, 2011) and with stroke in general (e.g., Robinson et al., 1983) have found the opposite; that depression correlates with poorer overall cognitive functioning which makes sense given that people who are depressed report feeling slowed down and lack motivation to give their full effort on cognitive tests.

The general stroke literature has also found an association between memory and attention dysfunction and depression (Kauhanen et al., 1999), which was not found here. This is a surprise, given that the link between poorer memory and depression is well established beyond just stroke literature. However, the very small sample size of people completing CNS-Vital Signs measures would again likely account for difficulty in being able to find significant correlations. A further possibility is that the lack of an association in the current study may relate to the tasks used to assess memory. Memory in the CNS-Vital Signs is only assessed as recognition memory; whereas the wider literature tends to also assess recall memory which may be more prone to being adversely affected by depression because recall tasks tend to be more difficult than recollection tasks.

Cognitive and functional outcomes

Poorer overall cognitive functioning was associated with poorer social HRQoL in this study, with cognitive impairment linked to less satisfaction in social roles. This makes intuitive sense as poorer cognition might act as a barrier to engaging in previously enjoyed social activities. Poorer complex attention was also associated with poorer physical and overall HRQoL. Complex attention is needed for many tasks people engage in daily, so it is intuitive that this could impact on the roles they can play in their life and the satisfaction they therefore feel with this. It would, however, be expected that other important cognitive functions would also impact on HRQoL for similar reasons. Other researchers, such as Vykopal (2011), report a relationship between processing speed and executive functioning impairments and poorer HRQoL; but no such correlations were found in the current study. This could be due to the limited sample size of people undertaking cognitive tests, which means power to detect real effects would be reduced.

It is of note that cognitive functioning was not significantly correlated with independence in ADLs in the current study. Some other studies in the stroke literature also failed to find a relationship (Cederfeldt et al., 2009; Maeshima et al., 1997); however, the majority of studies report that difficulties in overall cognitive functioning, as well as memory, attention, executive and visuospatial functioning more specifically, are associated with less independence in ADLs (Feigin et al., 2010; Hama et al., 2007; Mercier et al., 2001; Ng et al., 2007; Öneş et al., 2009; Patel et al., 2002; Portney et al., 2001; Stephens et al., 2005; Van Zandvoort et al., 2005). In the current study it is understandable that correlations may not be found between ADLs and the CNS-Vital Signs measures because of the small sample sizes. However, there was also no correlation found with the MoCA and BDS, for which bigger sample sizes were available. The current study only measured basic ADLs and not IADLs. IADLs (e.g., cooking, driving) are more complex than basic ADLs (e.g., eating, bathing);

hence it is feasible that IADLs may be more likely to be associated with cognitive functioning. It may therefore be worthwhile to include a measure of IADLs in future research.

Mood and functional outcomes

Depression and somatic symptoms correlated significantly with poor physical HRQoL. This is consistent with the finding that depression correlates with poor functional outcome (independence in ADLs and HRQol) in general stroke studies (Dafer et al., 2008; Dennis et al., 2000; Gill, 2011; Pohjasavaara et al., 2001; Starkstein et al., 1990; Sturm et al., 2004; Wulsin et al., 2012) and ICH studies specifically (Vykopal, 2011). This makes sense as individuals who are depressed often lack energy, feel tired, and lack motivation so may be left unsatisfied with what they are able to complete and accomplish on a day-to-day basis. People with somatic symptoms also might be limited in what physical tasks and roles they can fulfil.

Mood symptoms across the board correlated with the mental component of HRQoL which is consistent with other literature (Haacke et al., 2006; Huang et al., 2010; Suenkeler et al., 2002; Vykopal, 2011; Wulsin et al., 2012). This indicates that mood difficulties post-ICH do impact on quality of life. This is logical as it has been theorised that the loss of valued roles and social functioning is an important factor in explaining higher rates of depression among stroke survivors (Sienkiewicz-Jarosz et al., 2010). In fact, lack of activity and engagement in life activities is often a target when treating depression in the general population (Barlow, 2014). So if stroke related disability is causing a lack of ability to engage in valued roles then this poses a challenge for treating depression in stroke survivors. More creative means of carving out valued roles for stroke survivors may be necessary, even if they cannot mirror previous roles.

The current study did not find a correlation between ADLs and any measures of mood. This is unexpected given that many studies have found a correlation between independence in ADLs and PSD (Brodaty et al., 2007; Dafer et al., 2008; Dennis et al., 2000; Kauhanen et al., 1999; Kotila et al., 2003; Pohjasvaara et al., 2001; Vypokal, 2010). PSA also correlates with poorer ADLs in some studies, although the association is not as strong as with PSD (Dennis et al., 2000; Schultz et al., 1997; Vypokal, 2010). Other studies have found no association between PSA and functional impairment (Park, et al., 1999; Shimoda & Robinson, 1998). Again, as mentioned in the previous section, the lack of correlation found in the current study may be due to measuring only basic ADLs, with the lack of association possibly reflecting that mood difficulties are more of a hindrance when it comes to engaging in more complex functional tasks.

Baseline demographic factors related to 12-month post-ICH outcome.

Mood

In this study age was found to significantly correlate with mood outcomes, specifically severe depression, anxiety, and insomnia. The younger someone was when they had an ICH the more likely they were to develop mood difficulties post-ICH. The literature is mixed on this relationship with some studies finding no correlation (Starkstein et al., 1998; Chemerinski et al., 2001), others finding PSD (Barker-Collo, 2007; Robinson, 1983) and PSA (Schultz et al., 1997) is more prevalent among younger stroke survivors, and yet other studies finding that it is advanced age which is a risk factor for mood difficulties post-stroke (Fruehwald et al., 2001; Giaquinto et al., 1999). The findings of this study suggest that younger people seem to be more vulnerable to mood difficulties. An explanation for this finding might be that whilst stroke is a sudden and unexpected event for anyone, perhaps for younger people a stroke is more unexpected at their particular life stage. A stroke in someone younger may also have more implications for daily life, such as interfering with work and

parenting roles. Further, among older individuals stoke and other serious medical conditions may be more of a norm in their peer groups. This would be consistent with another finding from this study- that age was also significantly correlated with mental HRQoL; with those who are younger experiencing poorer HRQoL. Again, given that some questions on the SF-36 ask people to rank their quality of life compared to others they know; older individuals may not rank themselves as poorly given their peers are more likely to also be experiencing health conditions. The opposite association between HRQoL and age has been found in other studies of stroke (Nys et al., 2006; Paul et al., 2005) and ICH (Christensen et al., 2009), with older age associated with poorer HRQoL. Clearly further research is needed to clarify this relationship.

Gender was not significantly correlated with depression or anxiety in this study, which is in contrast to some studies in the general stroke literature which have found that women are slightly more likely to develop PSD (Carod-Artal et al., 2000; Kotila, Numminen, Waltimo, & Kaste, 2003; Nys et al., 2006) and PSA (Burvill et al., 1995a; Schultz et al., 1997), but consistent with other literature finding no gender difference in PSA or PSD (Burvill et al., 1995; Giaquinto et al., 1999; Hosking et al., 2000; Vykopal, 2011).

Cognition

In this study age, gender, and ethnicity did not significantly impact cognitive outcomes post-ICH. Whilst this is consistent with Vykopal (2011) who found no effect of age or gender on outcomes at 5 years post-ICH; it is inconsistent with general stroke literature that has found older age and ethnic minority group status to be associated with poorer cognitive outcomes post-stroke (Feigin et al., 2010; Patel et al., 2002). Potentially the limited sample size for many of the measures of cognition can account for the lack of findings in this area. Also a possibility is that there is a difference in how age and ethnicity relate to cognitive outcomes for ICH survivors and stroke survivors in general.

Functional outcomes

Previous studies have been mixed on the relationship between age and independence in ADLs post-stroke. Some studies have determined that advanced age related to poorer general functional outcomes post-stroke (Abanto et al., 2013; Demchuk & Buchan, 2000; Fiorelli et al., 1995; Hanun et al., 2012; Macciocchi et al., 1998; Turhan et al., 2009; Woo et al., 1992) and post-ICH specifically (Jeng et al., 2008; Wang et al., 2012). Although other studies have found age only accounts for a small amount of the variance in functional outcome (Black-Schaffer & Winston, 2004; Bagg et al., 2002). The current study did not find a significant relationship between independence in ADLs and age. Potentially, if this study had a larger sample size then it is possible a relationship might have been picked up. However, the results as they stand indicate that those people who had an ICH at an older age were not necessarily worse off in terms of their ability to engage in everyday basic activities.

In regards to the impact of gender on ADLs; in the stroke literature females reportedly have less independence in ADLs post-stroke (Gall et al., 2012), however, this has been attributed to them being older when they have strokes (Kelly-Hayes et al., 2003). Thus, in the current study, no significant gender difference was found, likely because the average age of females and males in this sample did not differ greatly.

A final finding was that there was a trend towards ethnicity correlating with impairment in basic ADLs. On examination of the data set it can be seen that on the Barthel Index Europeans and Pacific Islanders scored nearly the same and demonstrated the least impairment followed by the one Latin American participant. Asian peoples showed the most impairment, although one individual in the sample scored extremely low which had the effect of pulling down the group mean, so the finding could merely reflect outlier data. The results are unexpected, given that other studies have found people who have the poorest socioeconomic status tend to have poorer functional outcomes (Heikinheimo et al., 2012).

Pacific Peoples in this study would have therefore been expected to have poorer functioning than Europeans; a finding reported in other NZ studies (McNaughton et al., 2002). Given that age affects functional outcomes post-stroke in many studies then it is possible that the average age of people in the different ethnic groups in this study had an impact on the relationship found. Europeans were on average 66.5, Pacific Islanders 52.5, Asians 68.7, and the Latin American was 73. It seems then that the fact many Pacific Islanders were younger when they had a stroke may explain why they scored better than expected. Further clarification is needed on this relationship, preferably using a larger sample. Ideally, if the sample in the current research had been larger, the correlations between demographic characteristics and outcomes would have informed regression analyses.

Section 4: Clinical Implications

This is the first population-based study to examine the 6 and 12 month mood, cognitive, and functional outcomes of ICH, in contrast to a matched control group that the researcher is aware of. As such, the results are considered tentative. However, clinically, the findings of this study can begin to inform; 1) what areas should be assessed post-ICH and when, 2) what areas should be targeted for rehabilitation, and 3) who might be at greatest risk of poor outcomes post-ICH and therefore be targeted for assessment and treatment if resources are scarce. Each of these areas will now be discussed in turn.

This study can provide information for ICH survivors and their families, clinicians, and researchers as to the range of deficits that can occur post-ICH. It was clear from this study that not only do ICH survivors experience difficulties in functioning, consistent with the focus in post-stroke research and rehabilitation, but that significant cognitive deficits are very common and mood disturbance, particularly depression, is slightly elevated compared to controls, and may worsen over time. There is a current lack of information available as to

what neuropsychological profile is typical post-ICH, particularly in the long-term post-ICH. This study, along with other recent studies (Barker-Collo, Feigin, & Krishmnamurthi, 2013; Vykopal, 2011), can go some way towards informing what this profile may look like, which seems to be diffuse severe cognitive difficulties, with a particular area of likely difficulty being executive functioning. Barker-Collo et al. (2013) found that the cognitive profile of ICH also tends to be worse than SAH, particularly in the domains of visual attention, organisational/problem solving tasks, and visual memory. ICH survivors also commonly experience decreased independence in ADLs, although recovery of function appears to occur over the first year post-ICH. They further experience some impairment in areas of HRQoL. Therefore it is recommended that assessment post-ICH is done holistically- covering not only functional outcomes but also neuropsychological outcomes. This study shows that impairment is evident within the first few weeks post-ICH and is still present at 12 months post-ICH, suggesting assessment should not be a once-off event, but rather an ongoing process.

In regards to rehabilitation post-ICH, the findings of this study indicate that rehabilitation post-ICH should include a long-term plan, given that whilst improvement does occur over the short term, complete recovery is not achieved by 12 months. As part of this, interventions to rehabilitate cognitive functioning after ICH seem highly important given the severe, persisting, and widespread cognitive difficulties noted in this study. However, typical rehabilitation after a stroke focuses on physical, occupational, and speech and language therapy (Rohling, Faust, Beverly, & Demakis, 2009).Focusing on cognitive dysfunction, which may stop the individual re-entering the workforce and participating fully in their day-to-day life, seems to be uncommon (Gottesman & Hillis, 2010; Lees et al., 2012; Leifer, 2009).

Improvement in scores on a cognitive screening measure could be seen over the first year post-ICH in this study, indicating some spontaneous improvement in cognitive functioning is possible after ICH. Some of the spontaneous cognitive recovery may be due to the plasticity of the cerebral cortex. Although this process is not well understood it is possible that regions adjacent to those damaged may be able to take over functions impacted by the ICH to some extent (Gottesman & Hillis, 2010). Whilst the possible extent of potential recovery should cognitive rehabilitation not occur is not well established (Langhorne et al., 2011), a review of cognitive rehabilitation studies from 1998 to 2002 found that there is substantial evidence to support cognitive rehabilitation of visuospatial deficits, apraxia, and language difficulties post-stroke (Cicerone et al., 2005). Research does indicate that the brain is likely most responsive to rehabilitation efforts in the early stages post-stroke compared to later stages (Paolucci et al., 2000) hence why it would be recommended that rehabilitation start early.

Cognitive rehabilitation should also be tailored to individuals if possible; therefore early neuropsychological evaluation would be optimal to inform tailored rehabilitation strategies and help prepare stroke survivors for the difficulties they may face in everyday life because of cognitive deficits (Van Zandvoort, Kessels, Nys, De Haan, & Kappelle, 2005). Further, if through future research a distinct profile of long-term neuropsychological deficits can be established for ICH then differing community and rehabilitation services, educational programs and interventions may be warranted for this stroke subtype (Sturm et al., 2002). Collecting more data on neuropsychological outcomes is therefore an important step in the routinizing of neuropsychological rehabilitation post-stroke. Currently the data is scarce and often the results are inconsistent between studies (Barker-Collo & Feigin, 2006) as was the case with many of the findings of this study.

Options for cognitive rehabilitation have not yet been well researched (Langhorne et al., 2011). One option that has been explored is using virtual training programs to improve executive dysfunction, such as completing tasks in a virtual supermarket, but this has not yet been proven to be effective (Josman et al., 2006). One small study used a computerised battery of cognitive learning tasks in addition to the standard rehabilitation offered and compared this to a control group only getting the standard care. They found that over the 2 week study time, during which time(patients used the programme for 35 minutes a day, this group had improvements in cognitive functioning compared to the control group which showed no improvement (Prokopenko et al., 2013). Patients in the group also rated that they felt they had shown "considerable improvement" compared to controls who overall noted an "absence of improvement" (Prokopenko et al., 2013). Computerised cognitive tasks would certainly be a cost-effective means of offering cognitive rehabilitation should more studies find positive outcomes. However, as seen in the current study computerised tasks were off-putting for many of the older adults in the sample who were unfamiliar with computers; this may not be such an issue in the future as the 'technology savvy' generations reach older adult status.

Another method for cognitive rehabilitation that has been trialled is everyday listening to music. In a study conducted by Särkämö et al. (2008) stroke patients were assigned to either: a group that listened to self-selected music every day, a group that listened to audio books, or a control group that did neither of these things. Neuropsychological examinations were done at 3 and 6 months post-stroke and it was found that recovery in verbal memory and focused attention domains increased significantly more in the music group than in the control or language group (Särkämö et al., 2008). The music group also experienced less depressed mood (Särkämö et al., 2008).

Depressed mood was found to be elevated post-ICH in the current study, so interventions aimed at improving mood after ICH should be offered. Before interventions can be offered of course, there must be assessment of mood difficulties. Routine screening post-ICH would be indicated by the results of this study as mood difficulties may not be apparent amongst the many changes a stroke survivor experiences. Screening for depression post-stroke is not universally undertaken; with reports of only 50% of patients being screened (Morris, Jones, Wilcox, & Cole, 2012). Efforts to increase screening may involve education of the importance of screening for depression (and anxiety) post-stroke, putting protocols in place and increasing access to screening materials (Morris et al., 2012). Screening should be done beyond the acute stage and included in follow-up visits as depression was found to increase over the 12 months.

Cognitive behaviour therapy may be helpful for PSD. It is recommended that it be individually tailored to the person using motivational interviewing techniques, grief resolution, adaptations for cognitive deficits and executive dysfunction training (Broomfield et al., 2011). We know that a stroke survivor's felt confidence in their ability to recover from stroke and being provided with enough information from health professionals post-stroke accounts for variation in both PSD and PSA (Morrisson et al., 2000); these could therefore be targets of interventions. Given the reduced HRQoL seen in ICH survivors, interventions to improve survivor's roles and purpose on a day-to-day basis would likely also be beneficial.

Medication is also often used in the treatment of anxiety and depression. Ju, Liu, & Cai (2008) looked at the effectiveness of treatments for stroke patients with depression and anxiety and found that medication combined with psychotherapy was more effective than medication alone, which was significantly better than a control group. Burton et al. (2011) report a study in which treatment of anxiety with medication alone was compared to treatment involving both medication and psychotherapy. These two treatment options

resulted in a 58% and 71% lower anxiety level respectively in patients compared to the standard care they receive (Burton et al., 2011).

As well as informing us about the range of outcomes expected post-ICH, this study also provided some information on who may be most vulnerable to poorer outcomes post-ICH. Tupper and Henley (1987) note that in order to improve patient care we must know which patients are likely to do well and which are likely to do poorly so that a realistic prognosis can be given and plans can be made that are appropriate to the future needs of the patient. This study indicates that age is an important factor in regards to mood and anxiety outcomes, with younger people appearing to be more vulnerable, although as mentioned previously this result is in contrast to findings of some other studies.

When the findings of the current study are taken together with other literature they can inform the planning, provision, and allocation of health services. It is strongly indicated that health services need to start focusing more resources on cognitive rehabilitation in particular.

Section 5: Strengths and limitations of this study

Strengths

One of the major strengths of this study is that it sourced participants from a population-based, rather than using a hospital-based sample. As a population-based study it can be expected that the findings are reflective of the reality for a broad range of ICH survivors, rather than just being representative of those ICH survivors who required hospitalisation post-ICH. An over-reliance on hospital-based studies may paint a far worse picture for outcomes post-ICH, which is likely to be unhelpful for ICH survivors and their families.

Another important strength of this study was that it looked at ICH specifically; as discussed in the introduction section of this report, there is a need for ICH specific studies.

This is because much of the literature for stroke excludes those with haemorrhagic stroke in order to remain more homogeneous, and also the sample numbers for ICH are often too small to allow comparisons. A further strength of this study is that matched controls were used. Controls were matched to major demographic factors known to impact on stroke outcomes, which means that these variables can be ruled out as factors when comparing the two groups. The time period over which stroke was assessed in this study is also a strength because there is sparse literature on long term outcomes; as can be seen in this study, comparing acute and long-term outcomes is helpful to get an insight into the pattern of recovery post-ICH.

A final and very important strength of this study is that it assessed neuropsychological outcomes using a battery approach. As stated earlier, the majority of studies fail to assess mood and cognitive outcomes post-ICH, or rely solely on brief screening measures, despite the very high prevalence of disturbance in these areas. Given that a stroke occurs in, and often damages, the brain it seems vital to assess cognitive functioning post-stroke. Further, as this study indicated, aspects of mood and cognition are related to functional outcome which provides further support for their inclusion in post-ICH assessment. This study tested cognitive functioning using a comprehensive battery of neuropsychological tests, as opposed to other studies who have relied on cognitive screening measures only which have been found to be a limited in the measurement of post-stroke cognitive deficit (Nys et al., 2005).

Limitations

Despite the strengths of this study it does have some limitations that should be noted. Firstly, due to the small sample size the study had limited power, particularly on measures of mood and cognition, which meant that the study was considered exploratory in nature only because of the reduced reliability of findings. One of the most serious limitations was in regards to the limited data on cognitive outcomes. Whilst the MoCA was administered to many of the participants and gave basic data on cognitive functioning for them, there was a

very small sample size of ICH survivors who completed the CNS-Vital Signs measures as well. As such, a detailed profile of neuropsychological functioning post-ICH could not be gained from this study. Also, individuals who have a stroke often have other comorbidities so it is hard to single out the exact cognitive deficits that result from the ICH itself; there is always going to be a certain amount of error associated with this (Gottesman & Hillis, 2010). Further, many patients with haemorrhagic stroke may have existing cognitive deficits. This may be the case in stroke survivors with cerebral amyloid angiopathy (which is a common cause of haemorrhagic bleed), who may at baseline have had cerebral microbleeds and neurodegenerative changes (Pfeifer, White, Ross, Petrovitch, & Launer, 2002). The study of the cognitive outcome of stroke is also limited by the lack of measurement of cognition before the stroke occurred (Gottesman & Hillis, 2010).

Additionally, it is of note that level of education, which has previously been found to be associated with cognitive functioning post-stroke (Rasquin, Verhey, Van Oostenbrugge, Lousberg, & Lodder, 2004), was not controlled for in the current study. Whilst there was no difference in whether the ICH and control groups had completed high school or not, the controls were more likely than the ICH group to have completed further education. The decision was made not to covary education within the analyses in the current study to save power. Thus, it should be considered a limitation of the current study that it is unknown to what extent a higher level of education might have accounted for the better performance of the controls on cognitive measures, compared to the ICH group.

A further issue to note in relation to a small sample size is that when multiple correlations are run, such as in this study, then there is an increased chance of finding a spurious result. For example, if 100 correlations are run using p of .05 then 5% of the significant correlations are only significant by chance (Gordon, 2012). This was addressed by approaching significant correlations with caution, looking for patterns in findings, and

findings that made intuitive sense and could be backed up by other literature. Additionally, when looking at the numerous correlations run between demographic characteristics and outcomes, significance was set at p>.01 to reduce the chance of finding a spurious result.

A further limitation of this study was that people with communication problems and severe cognitive impairments had to be excluded from the study due to the practicalities of being able to assess these participants. This may have led to an underestimation of the level of impairment post-ICH. We also cannot be sure if the sample is representative of all people with ICH or only those who are likely to consent to participate in such a study. Whilst the data was not available in the current study, it would have been helpful to do sensitivity analyses which compare the ICH group to those who did not consent in terms of things like age (t-test), ethnicity, and gender (chi square). This would have allowed a statement to be made as to whether the findings can be generalized to all people with ICH. It is of note that no Māori were included in the ICH sample, despite Māori making up nearly 15% of the NZ population. This was due to consent not being gained from Māori approached to take part in the ARCOS-IV study. This raises questions about what could be done to improve Māori participation in such studies in the future. Cultural consultation before recruitment would be an important first step in addressing this issue. Use of Māori language during recruitment advertising and a clear explanation as to the relevance of the research to Māori might be worth considering.

This study relied on self-report of participants on their level of functioning, HRQoL, and mood disturbance. Barrett (2009) has pointed out that outcomes based on patient report may give a significantly underestimated view of patient disability because of the failure to account for the effect of neuropsychological deficits on their self-report. These deficits may include impaired magnitude estimation and pathological alteration of self-awareness (Barrett, 2009). Barrett (2009) has likened this phenomenon to getting ‘rose-coloured answers’. The

collection of collateral information from family members or care-givers might therefore be warranted in such research, should resources allow (e.g., administration of the Vineland Adaptive Behaviour Scales-Caregiver Rating Form).

During discussion of the findings of this study it was also felt that the study was limited in looking at functional outcomes by only assessing basic ADLs and not IADLs. Some research indicates that the Barthel index significantly underestimates the impact of stroke (Lai, Studenski, Duncan, & Perera, 2002) suggesting that future research might broaden the assessment of functional outcome by incorporation of other measures.

Also of note is that some research indicates that a generic HRQOL scale, such as the SF-36 used in this study, correlates less with patient's subjective view of their QOL than QOL measures specific to stroke, such as the Stroke Specific Quality of Life Scale (SS-QOL) (Williams, Weinberger, Harris, & Biller, 1999). The current study, however, involved healthy controls who have not had a stroke so it was justified to have a more generic measure. The majority of other research in the stroke outcomes literature uses SF-36 also. The measures of anxiety used were also limited in this study as they were only geared towards generalised anxiety disorder despite specific phobias, agoraphobia, and PTSD being other common anxiety disorders post-stroke (Favrole et al., 2013). This may be one possible reason for why this study found fewer people with anxiety disorders than expected.

Finally, another limitation is that control participants were only tested on one occasion due to the impracticalities of testing them on more than one occasion during the period available for data collection in the current study. Therefore, had any improvement been seen in the ICH group on measures of cognitive functioning it could not have been ruled out that this might be down to practice effects. Additionally, it is difficult to determine to what extent test-retest variability might have played a factor in any changes over time. To address the latter point, measures were chosen for the current study that had sound test-retest

reliability. Additionally, should any significant changes have been found in cognitive scores over time they would have been given more weight should the difference have been greater than one standard deviation and if they were in keeping with a general pattern of findings.

Section 6: Future Directions for Research

The current study is considered exploratory in nature; therefore, the results can offer valuable insights into how to approach future research. One of the key issues highlighted in this research was the problem of a small sample size of people who were able to participate due to the severity of deficits experienced post-ICH. Given the small sample size, it was not possible to examine predictive relationships between variables at baseline and later stages of data collection as had been originally hoped. A larger sample size would allow these relationships to be better understood in future research. Predictive factors can provide valuable information about expected recovery and long term outcomes and provides a way of targeting specific individuals who may benefit from certain forms of rehabilitation, hence why further research in this area is warranted (Rohling et al., 2009).

One way of gaining a larger sample size for cognitive outcomes that is suggested for future research may be to use non-computerised and less demanding neuropsychological tests. As has already been mentioned, many participants did not want to use the computer or felt it would be too time-consuming to complete. The sample, overall, was capable of completing shorter paper and pencil or verbally administered measures, so more of a focus on these types of measures may give more results. Additionally, sampling over a longer time period or greater geographical area might be considered to get a larger sample size.

Clarification is needed on many of the results found in the current exploratory study; they are simply one set of findings among the wider stroke literature, which tends to be inconsistent and at times conflicting. The current study certainly posits that further research

into the neglected area of cognitive functioning post-ICH is warranted. Particularly, developing a picture of the typical neuropsychological profile post-ICH is important.

Section 7: Conclusion

This study acknowledged the uniqueness of different types of stroke by examining outcomes of ICH specifically. It examined outcomes in the acute period post-ICH as well as short-term (6 month) and long-term (12 month) outcomes. This study used a more holistic approach than the majority of previous research that appears to favour functional outcomes exclusively. Instead this research examined mood, cognitive, HRQoL, and basic functional outcomes. The current study used a population-based sample and compared survivors of ICH to gender, age, and ethnicity matched controls. Whilst the results of this study are limited by the small sample size and should be considered exploratory only, it does appear that cognitive impairment following ICH is common, widespread, and persisting. Mood difficulties, particularly mild depression, were also of concern for ICH survivors over the study period. The ICH group had significantly poorer independence in ADLs, although improvement over the 12 months was exhibited. There were mixed findings when it came to HRQoL; it does seem as though physical HRQoL post-ICH is impaired but not mental HRQoL. The results point to the need for a greater focus on cognition and mood in rehabilitation of ICH survivors and future research.

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Appendix A

International Classification of Functioning, Disability and Health (ICF) Categories Included in the Comprehensive ICF Core Set for Stroke, from Geyh et al. (2004)

ICF Code	ICF Category
Body Functions	
b110	Consciousness functions
b114	Orientation functions
b117	Intellectual functions
b126	Temperament and personality functions
b130	Energy and drive functions
b134	Sleep functions
b140	Attention functions
b144	Memory functions
b152	Emotional functions
b156	Perceptual functions
b164	Higher-level cognitive functions
b167	Mental functions of language
b172	Calculation functions
b176	Mental function of sequencing complex movements
b180	Experience of self and time functions
b210	Seeing functions
b215	Functions of structures adjoining the eye
b260	Proprioceptive function
b265	Touch function
b270	Sensory functions related to temperature and other stimuli
b280	Sensation of pain
b310	Voice functions
b320	Articulation functions
b330	Fluency and rhythm of speech functions
b410	Heart functions
b415	Blood vessel functions
b420	Blood pressure functions
b455	Exercise tolerance functions
b510	Ingestion functions
b525	Defecation functions
b620	Urination functions
b640	Sexual functions
b710	Mobility of joint functions
b715	Stability of joint functions
b730	Muscle power functions
b735	Muscle tone functions
b740	Muscle endurance functions
b750	Motor reflex functions
b755	Involuntary movement reaction functions
b760	Control of voluntary movement functions
b770	Gait pattern functions
Body Structure	
s110	Structure of brain
s410	Structure of cardiovascular system
s720	Structure of shoulder region
s730	Structure of upper extremity
s750	Structure of lower extremity
Activities and Participation	
d115	Listening
d155	Acquiring skills
d160	Focusing attention

d166	Reading
d170	Writing
d172	Calculating
d175	Solving problems
d210	Undertaking a single task
d220	Undertaking multiple tasks
d230	Carrying out daily routine
d240	Handling stress and other psychological demands
d310	Communicating with – receiving – spoken messages
d315	Communicating with – receiving – non-verbal messages
d325	Communicating with – receiving – written messages
d330	Speaking
d335	Producing non-verbal messages
d345	Writing messages
d350	Conversation
d360	Using communication devices and techniques
d410	Changing basic body position
d415	Maintaining a body position
d420	Transferring oneself
d430	Lifting and carrying objects
d440	Fine hand use
d445	Hand and arm use
d450	Walking
d455	Moving around
d460	Moving around in different locations
d465	Moving around using equipment
d470	Using transportation
d475	Driving
d510	Washing oneself
d520	Caring for body parts
d530	Toileting
d540	Dressing
d550	Eating
d570	Looking after one's health
d620	Acquisition of goods and services
d630	Preparing meals
d640	Doing housework
d710	Basic interpersonal interactions
d750	Informal social relationships
d760	Family relationships
d770	Intimate relationships
d845	Acquiring, keeping and terminating a job
d850	Remunerative employment
d855	Non-remunerative employment
d860	Basic economic transactions
d870	Economic self-sufficiency
d910	Community life
d920	Recreation and leisure
Environmental Factors	
e110	Products or substances for personal consumption
e115	Products and technology for personal use in daily living
e120	Products and technology for personal indoor and outdoor mobility and transportation
e125	Products and technology for communication
e135	Products and technology for employment
e150	Design, construction and building products and technology of buildings for public use

e155	Design, construction and building products and technology of buildings for private use
e165	Assets
e210	Physical geography
e310	Immediate family
e315	Extended family
e320	Friends
e325	Acquaintances, peers, colleagues, neighbours and community members
e340	Personal care providers and personal assistants
e355	Health professionals
e360	Health-related professionals
e410	Individual attitudes of immediate family members
e420	Individual attitudes of friends
e425	Individual attitudes of acquaintances, peers, colleagues, neighbours and community members
e440	Individual attitudes of personal care providers and personal assistants
e450	Individual attitudes of health professionals
e455	Individual attitudes of health-related professionals
e460	Societal attitudes
e515	Architecture and construction services, systems and policies
e525	Housing services, systems and policies
e535	Communication services, systems and policies
e540	Transportation services, systems and policies
e550	Legal services, systems and policies
e555	Associations and organizational services, systems and policies
e570	Social security services, systems and policies
e575	General social support services, systems and policies
e580	Health services, systems and policies
e590	Labour and employment services, systems and policies

Appendix B

Outcome Measures

Demographic information sheet

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)
□ 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

Measures of mood

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				

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?				

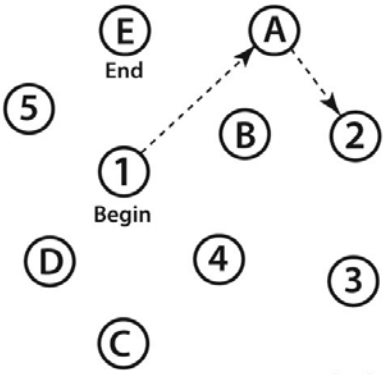
		Definitely not	I don't think so	Has crossed my mind	Definitely
28.	Found the idea of taking your own life kept coming into your mind?				

Measures of cognitive functioning (excluding computerised tests)

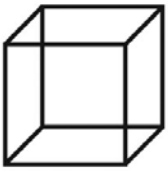
MONTREAL COGNITIVE ASSESSMENT (MOCA)
Version 7.1 Original Version

NAME :
Education : Date of birth :
Sex : DATE :

VISUOSPATIAL / EXECUTIVE



Copy cube



[ ] [ ]

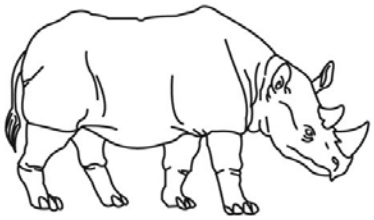
Draw CLOCK (Ten past eleven)
(3 points)

[ ] Contour [ ] Numbers [ ] Hands

POINTS ___/5

NAMING

[ ]



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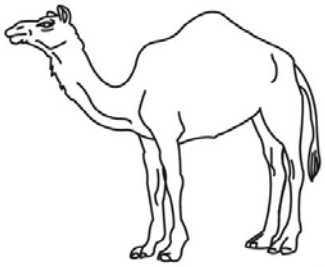

[ ]

___/3

MEMORY Read list of words, subject must repeat them. Do 2 trials, even if 1st trial is successful. Do a recall after 5 minutes.

	FACE	VELVET	CHURCH	DAISY	RED
1st trial					
2nd trial					

No points

ATTENTION Read list of digits (1 digit/ sec.).
Subject has to repeat them in the forward order [ ] 2 1 8 5 4
Subject has to repeat them in the backward order [ ] 7 4 2
___/2

Read list of letters. The subject must tap with his hand at each letter A. No points if ≥ 2 errors
[ ] F B A C M N A A J K L B A F A K D E A A A J A M O F A A B
___/1

Serial 7 subtraction starting at 100 [ ] 93 [ ] 86 [ ] 79 [ ] 72 [ ] 65
4 or 5 correct subtractions: **3 pts**, 2 or 3 correct: **2 pts**, 1 correct: **1 pt**, 0 correct: **0 pt**
___/3

LANGUAGE Repeat : I only know that John is the one to help today. [ ]
The cat always hid under the couch when dogs were in the room. [ ]
___/2

Fluency / Name maximum number of words in one minute that begin with the letter F [ ] _____ (N ≥ 11 words)
___/1

ABSTRACTION Similarity between e.g. banana - orange = fruit [ ] train – bicycle [ ] watch - ruler
___/2

DELAYED RECALL

	Has to recall words **WITH NO CUE**	FACE [ ]	VELVET [ ]	CHURCH [ ]	DAISY [ ]	RED [ ]	Points for UNCUED recall only
Optional	Category cue						
	Multiple choice cue						

___/5

ORIENTATION [ ] Date [ ] Month [ ] Year [ ] Day [ ] Place [ ] City
___/6

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TOTAL ___/30
Add 1 point if ≤ 12 yr edu

Administered by: _____

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	3 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	3 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

Measures of functional outcome

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/herslef
	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.

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					

Appendix C

Participant Information Sheet and Consent Form

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: Natasha Bauer

Dear potential participant,

My name is Natasha Bauer. 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 20 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 October 1st 2014. 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 $20 petrol or grocery 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:

Natasha Bauer
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Private Bag 92019
Auckland, New Zealand
Tel: (09) 373 7599 extn. 82458
Email: nbau005@aucklanduni.ac.nz

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Head of Department 2014:
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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 25 March 2013 for a period of 3 years, Reference Number 8289.

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

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: Natasha Bauer

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 2014).
- I wish / do not wish to receive the summary of findings (circle one). Contact the researcher at nbau005@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 petrol or grocery voucher for participation in this research
□ YES □ NO

APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN PARTICIPANTS ETHICS COMMITTEE on 25 March 2013 for a period of 3 years, Reference Number 8289.