The Role of Psychological and Cognitive Factors in the Psychological and Physical Recovery from Acute Stroke: A Longitudinal Study.

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Declaration

I, Parminder Dhiman, confirm that the work presented in this thesis is my own. Where information has been taken from other sources, I confirm that this has been referenced in the thesis.

Printed Name: Signature:

Abstract

Background: Stroke is the second leading cause of disability and mortality in the U.K., therefore research investigating stroke has been highlighted by the National Stroke Strategy to develop studies which are longitudinal and focus on outcome. A comprehensive systematic review (Study One) was undertaken to investigate the role of psychological factors on stroke recovery. This informed the development of the research study (Study Two). The aim of this study was to investigate the role of psychological and cognitive factors on psychological and physical recovery from acute stroke, in a longitudinal study as directed by the National Stroke Strategy. The current study additionally incorporates cognitive neuropsychological elements along with measures of mood, personality and coping. This is the first study to the authors' knowledge which has investigated repressive coping and Type D personality with stroke.

Method: Longitudinal data collection was conducted in two NHS hospitals, with a clinical sample at Time 1 (0-6 weeks post stroke), followed up at Time 2 (3 months post stroke) and Time 3 (6 months post stroke), in the participants' homes or in nursing homes.

Measures used to test independent variables were: Centre for Epidemiologic Studies Short Depression Scale (CES-D 10), Perceived Stress Scale (PSS), Multidimensional Scale of Perceived Social Support (MPSS), Standard Assessment of Negative Affectivity, Social Inhibition, and Type D Personality （DS 14, Type D personality), Marlowe-Crowne Form B & 6 Item STAI (for repressive coping), 3 item Sense of Coherence (SoC) scale, line bi-section & Bells cancellation task (visual neglect), forward digit span (verbal short term memory), Rivermead Behavioural Memory Test (visual short term memory) and the colour word Stroop test (executive function), along with demographic data, stroke markers and health behaviours.

Dependent variables were: Quality of life (measured by the SF-36) and physical recovery (modified Rankin Scale).

Results: The main analysis used hierarchical multiple regression analyses and mediation analysis to test a series of hypotheses.

Physical recovery outcome was predicted by stroke severity, age, stress, repressive coping, social support and visual neglect at different time points. Depression and visual memory were reported as mediators at Time 2.

Quality of life outcome was predicted by stroke severity, age, stress, social support, depression and visual neglect at different time points.

Conclusions: The results of this study indicate that psychological factors do have an impact on both physical and psychological outcome from stroke. Stress, repressive coping and visual neglect were the most consistent predictors of outcome. Depression and social support played a smaller role, whereas Type D personality was non-significant across analyses.

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In Remembrance

In memory of my supervisor Professor Lynn Myers, who sadly passed away after the acceptance of my Ph.D.

And in memory of the eleven participants who passed away during the course of this research.

Rest In Peace.

This thesis is dedicated to my Grandparents

Mrs. Sarjit Kaur Dhiman (née Ghataure) (Late)

Mrs. Kulwant Kaur Bhogal (née Sian) (Late)

Mr. Mehnga Singh Bhogal (Late)

Mr. Pritam Chand Dhiman (Late)

And was inspired by my Father

Mr. Mohan Singh Dhiman

Chapter 1

Introduction

1.1 Summary

This thesis aims to investigate the role of psychological and cognitive factors on psychological and physical stroke recovery. This chapter introduces stroke, including a rationale based on the National Stroke Strategy and the UK Government campaign to raise stroke awareness. This will be followed by background of stroke, aetiology, risk factors, signs and symptoms, effects of stroke, diagnosis, classification, treatment, incidence, mortality, recurrence of stroke and social inequalities.

1.2 Rationale

Stroke is the second leading cause of mortality and disability in the UK and worldwide after heart disease (World Health Organisation, 2002; Beswick, 2004; Feigin, 2007, Strong, Mathers, & Bonita, 2007). However, until recently the general public were unaware of the incidence and consequences of stroke. In 2004 the **F**ace **A**rm **S**peech **T**ime (FAST) test was developed which was taught to paramedics in order to quickly and successfully diagnose strokes (Nor et al., 2004). In 2008 this prompted the UK Government to fund advertising campaigns to educate and generate awareness for the British public on the dangers of stroke (see figures 1.1 and 1.2).

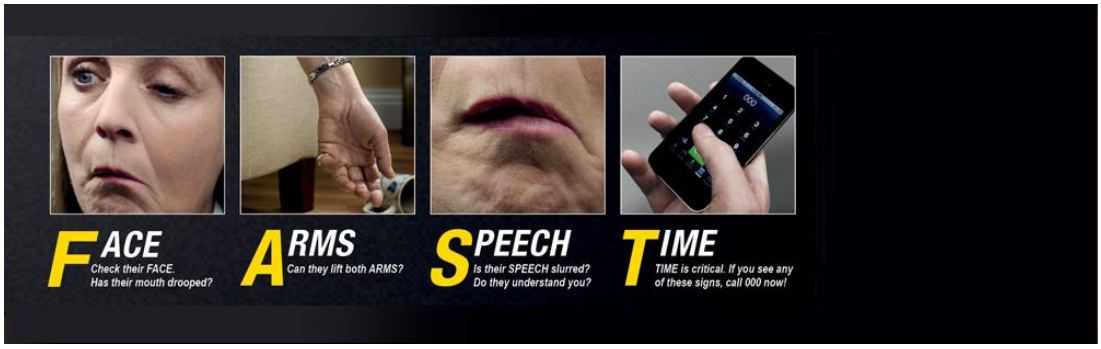


Figure 1.1

The FAST media campaign

http://www.stroke.org.uk/research/achievements/fast (2015)

In order to communicate a stroke with the general public the advert explains "When a stroke strikes it spreads like a fire in the brain".

Figure 1.2

FAST media campaign comparing a stroke to a fire in the brain

http://www.englemed.co.uk/09/09nov092_stroke_ad.php (2015)

This campaign has been very successful in raising stroke awareness and has resulted in an increase in ambulance requests for stroke by 55% (Stroke Association 2015). With more health awareness surrounding stroke, the hope is to reduce stroke incidence and post stroke disability and mortality. Stroke has normally been overshadowed by campaigns for heart disease and cancer. This has prompted the National Stroke Strategy to address issues of raising awareness of stroke, assessment and treatment, rehabilitation, participation in community life, returning to work, end

of life and research needs. One of the research needs identified by this strategy is the *"Estimation of the longer-term needs of patients (impairment, activity, participation, quality of life) at different time points post-stroke to help direct intervention studies to improve outcomes"* (DoH, 2007, p.66). A major motivation of the research in the current thesis is to address the poverty of research within this area.

The next section will explain the definition and background of stroke.

1.3 Definition and Background

Stroke is defined by the World Health Organization (WHO) as 'a clinical syndrome consisting of rapidly developing clinical signs of focal (or global in case of coma) disturbance of cerebral function lasting more than 24 hours or leading to death with no apparent cause other than a vascular origin' (Hatano, 1976, p3550). WHO have also developed the International Classification of Diseases (ICD), which is in its 10th revision since 1992. These classifications are used to code diseases so mortality statistics can be internationally compared (WHO, 1992). It has been estimated that by 2030 in the developed world stroke will be the fourth highest cause of Disability Adjusted Life Years (DALY's). DALY's are the sum of life-years lost due to years lived with disability and premature death (Lopez, Mathers, Ezzati, Jamison, & Murray, 2001; WHO, 2009). Stroke has been reported to be fatal for 2 out of 10 strokes, disabling 6 out of 10, and some degree of recovery being attained for 2 out of 10 patients (Kolb & Wishaw, 2009).

In purely financial terms stroke presents a significant drain on economic resources. For example, the total direct and indirect cost of cardiovascular disease and stroke in the USA for 2007 was estimated to be $286 billion including the cost of professionals, hospital services, medications, home health care and lost productivity resulting from mortality (American Heart Association, 2011). In England the cost of stroke to the economy is high with an estimated £7 billion per year in 2005 (£2.8 billion to the NHS, £2.4 billion in care costs and £1.8 billion due to lost productivity and disability) (Department of Health (DoH), 2005; National Audit Office, 2005 – 2006). Nonetheless until recently stroke was not perceived as a high priority within the National Health Service (NHS), which left sufferers of stroke unable to receive adequate treatment to maximise the extent of recovery from stroke

(DoH, 2007). However, following the 2005 publication from the National Audit Office, the DoH developed the National Stroke Strategy (DoH, 2007).

The next section will explain the aetiology of stroke and the vascular system.

1.4 Aetiology of Stroke

Stroke is a disease caused by weakness in the vascular system (also referred to as the circulatory system), which is made up of the vessels (arteries, veins and capillaries) that carry blood throughout the body. These vessels deliver oxygen to the body tissues (see figure 1.3). Stroke is a *cerebrovascular disease* as disturbed blood flow affects the brain. If blood flow to the heart is affected this causes *cardiovascular disease* (for example, heart attack). Blood travels to the brain via three arteries (the two Carotid arteries and the Basilar artery). Blood flows into a circular artery at the base of the brain – "The Circle of Willis" (see figure 1.4). If one artery is blocked blood can still travel to the brain via the other two arteries but if there is a blockage in the blood vessels above the circle then it is more difficult for the blood to reach the brain. This can cause tissue death to the brain, some of it beyond repair (Smith, 2000; Stroke Association, 2013).

Researching strokes is important because of its prevalence and debilitating effects. The risk factors for strokes also predict other physical problems; for example, the possibility of heart problems (as they are both vascular diseases) and hypertension which can cause vascular diseases. This could be avoided with blood pressure being controlled and being kept within a healthy range (Stroke Association, 2007).

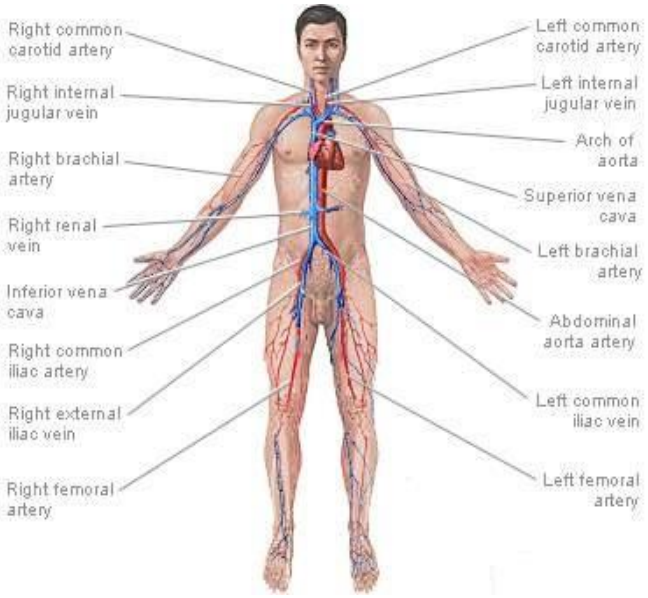


Figure 1.3

The vascular system around the whole body

(http://probaway.wordpress.com/2012/03/22arterial-disease-embolisms-heart-attacks-what-to-d/ 2012)

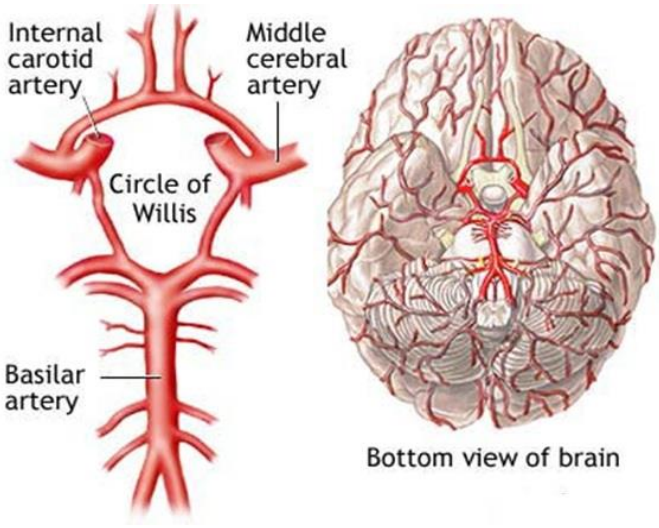


Figure 1.4

The vascular system of the brain

(http://health.allrefer.com/health/stroke-circle-of-willis.html 2012)

The next section will discuss the two different types of stroke that occur: Ischemic and haemorrhagic.

1.4.1 Types of Stroke

There are two causes of strokes: Ischaemic strokes (approximately 83%) and haemorrhagic stroke (approximately 17%) (Smith, 2000; Stroke Association, 2007).

1.4.1.1 Ischaemic Stroke

Ischaemic stroke is caused by disturbed blood flow to the brain by thrombosis (blood clot), embolism (debris from elsewhere in the body blocking vessels to the brain), or atherosclerotic clot (build-up of fatty deposits in the arteries) which causes brain tissue to die (infarction) (see figure 1.5). The clot size will control how much of the vessel is occluded and influence how much of the brain is affected (National Collaborating Centre for Chronic Conditions, 2008). The carotid arteries provide the main supply of blood to the brain and therefore occlusions in these arteries are likely to result in neurological damage (Jamrozik, 2005). The location of the stroke will depend on where the clot has formed, which will also determine the effect on brain function for example, speech disturbance, limb weakening, somatosensory loss, vision problems, memory decrease and decrease in executive function (Barnett, Mohr, Stein, & Yatsu, 1998; Kolb & Wishaw, 2009; Stroke Association, 2015a) (see Section 1.5.2).

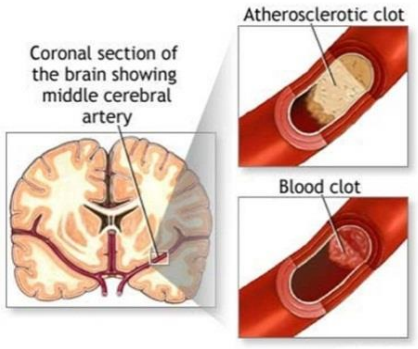


Figure 1.5

Diagram of Ischaemic stroke which illustrates two causes, atherosclerotic clot and blood clot.

(http://health.allrefer.com/health/stroke-stroke.html 2012)

1.4.1.2 Haemorrhagic Stroke

Haemorrhagic strokes result from bleeding in the brain following the rupture of an artery or vein. There are two main types of haemorrhagic stroke. First, an intracerebral haemorrhage is bleeding from an artery inside the brain (Saccro et al., 1984) and second, subarachnoid haemorrhage is an intracranial aneurysm in the space between the brain and the membranes around it. When an aneurysm in this area bursts blood is spread around the surface of the brain putting pressure on the brain and raising the pressure inside the head because blood cannot permeate the meninges. In both cases a rupture results in brain tissue death (Rinkle, Wijdicks, & Vermeulen, 1991; Smith, 2000; Stroke Association, 2013) (see figure 1.6).

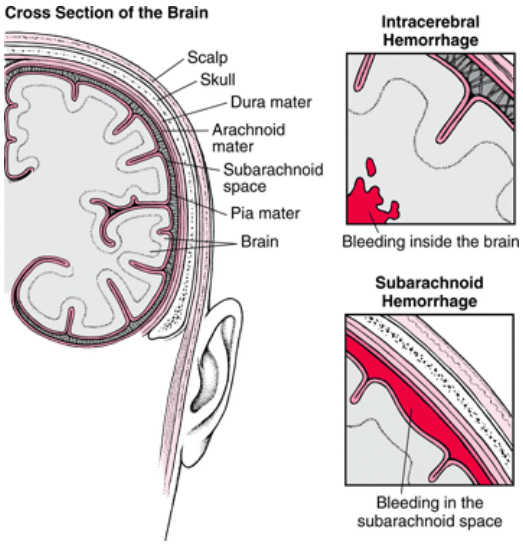


Figure 1.6

Diagram of a Haemorrhagic Stroke and a Subarachnoid Haemorrhagic Stroke.

(http://www.merckmanuals.com/home/brain_spinal_cord_and_nerve_disorders/stroke_cva/hemorrhagic_stroke.html 2012).

The next section will give an overview of brain anatomy and describe the functions of the cortical and subcortical regions.

1.5 Brain Anatomy

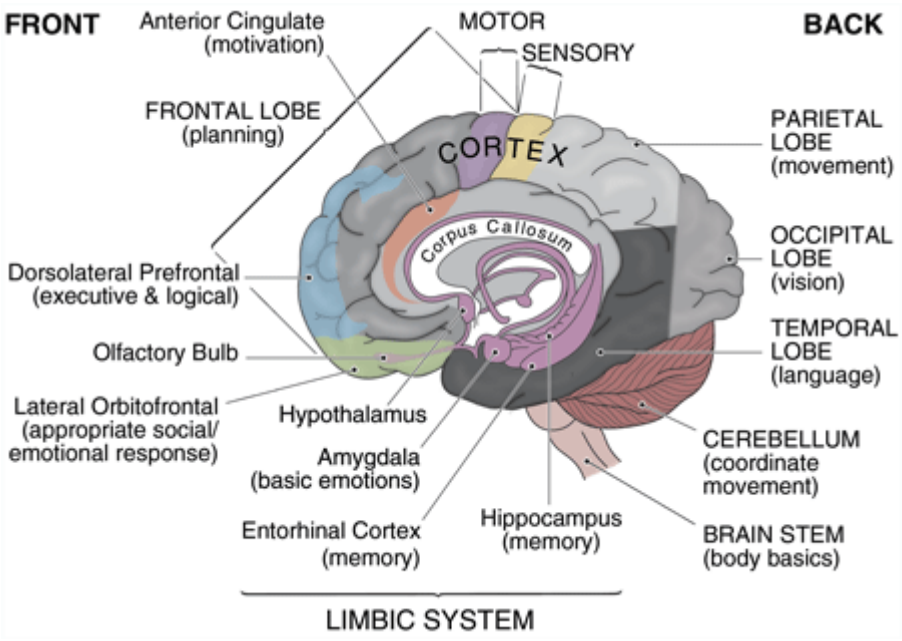


Figure 1.7

Diagram of the Human Brain showing the major anatomical areas and some of their functions

(http://www.brainwaves.com/ 2012)

The brain can be sub-divided into distinct anatomical and functional areas. It is divided into two hemispheres; the left and right hemisphere. The left hemisphere controls the right side of the body and the right hemisphere controls the left side of the body, but there are also differences in general function between the two hemispheres (e.g. language is generally left lateralised). The two halves of the brain are connected by the corpus collosum which is part of a network of pathways.

The brain is also divided into two regions; the cortex and the sub cortex. The brain consists of four major lobes in the cortical area which controls higher functioning; the frontal lobe, parietal lobe, temporal lobe and occipital lobe. The frontal lobe is primarily involved in motor function, understanding social rules, executive function, speech and short term memory. The parietal lobe controls somatosensory processing, spatial information, attention, guidance of movement, spatial navigation and language. In stroke visual neglect (neglecting one side of

vision) is common and is classically associated by damage to the parietal lobe. The temporal lobe controls auditory processing, object and face recognition, memory and emotion. The occipital lobe processes early visual representations.

Subcorticol areas are the phylogenetically older regions of the brain and mediate basic bodily functions (e.g. respiration), rapid motor responses and certain cognitive functions (e.g. emotion and memory). They can be sub-divided into the base forebrain, the mid brain and the hind brain. The basal ganglia is situated at the base of the forebrain and it primarily modulates movement. Lesions to this area of the brain, causes tremors and impairment in producing smooth movement (Cabeza & Kingstone, 2006; Kolb & Wishaw, 2009). However there is increasing evidence for its cognitive role especially in implicit memory and learning (Seger, 2006; Yang & Ping, 2012). The diencephalon resides at the back of the forebrain and this component borders the older brain (the subcortical areas) and the newer brain (the cortex) and it includes the thalamic structures; the hypothalamus (which processes motivated behaviour for example, emotional and sexual behaviour, feeding and sleeping) and the thalamus (which processes visual, auditory and temperature projections, touch, pressure and pain). Information passes through the thalamus and into the cortex.

The midbrain processes vision, hearing, alertness, temperature regulation and motor control. Finally the hind brain houses the brain stem and the cerebellum. The brain stem connects to the spinal cord allowing messages to be sent to the whole body, it mediates regulatory functions such as moving, eating and sleeping. The cerebellum is traditionally characterised as an area responsible for the control of motor movement but is also increasingly implicated in cognitive processing (Purves et al., 2008; Kolb & Wishaw, 2009).

The limbic system spans both cortical and subcortical areas and groups together functions which reinforce and motivate behaviour, such as emotional reactions and memory and has been described as the feeling and reacting brain. The cortical areas of the limbic system include the hippocampus (memory in the temporal lobe) and amygdala (emotional reactions). The subcortical areas would include the hypothalamus (Martin, 1998; Kolb & Wishaw, 2009) (see figure 1.7).

The main consequences of stroke include physical, cognitive and psychological effects. These will be discussed in the next section.

1.6 Effect of Stroke

Stroke can have many functional consequences which include physical, cognitive and psychological effects. Although these sub-categories are a convenient way to classify the consequences of stroke they do overlap with each other; for example, low physical functioning can cause depression (Chemerenski, Robinson, & Kosier, 2001; Jaracz, Jaracz, Kozubski, & Rybakowski, 2002) and depression can modulate cognition (Murphy, Michael, & Sahakian, 2012; Murphy et al., 1999; Gotlib & Joormann, 2009). The precise combination of such effects will depend upon the extent and location of the damage. Some effects may be transitory as they are caused by temporary impairment e.g., hypoperfusion (reduced blood flow) or oedema (swelling that causes pressure on the brain). Cortical reorganisation may also occur (where the brain uses different pathways to compensate for affected areas) (Enatsu et al., 2012; Michielsen et al., 2011). Different strokes affect different parts of the brain and therefore cause a variety of cognitive, psychological and physical problems.

1.6.1 Physical Effects

Stroke can cause impairment to areas of the brain responsible for basic muscular control (e.g., brain stem), higher cognitive areas involved in more abstract action planning (e.g., frontal motor areas) or sub-cortical areas lying between these two extremes. Hemiplegia is paralysis on one side of the body resulting from stroke damage to the opposite side of the brain. Paralysis can be localised to the face, arm, hand, trunk, leg, foot, or any combination of these. Hemiparesis describes a physical effect of stroke where the control of muscles on one side of the body is weakened but not fully paralysed. Again this affects the face, arm, hand, trunk, leg or foot, or a combination of these effectors. Numbness is a side effect which causes patients to have a lateralised reduction in somatosensory feeling (perception of sensation) in their skin. Strokes that affect the cerebellum may affect walking, movement and coordination and balance. Other physical effects of stroke include muscle spasticity, loss of control of bodily functions, dysphagia (swallowing problems) and dysarthria (problems with the muscles that help one to speak resulting in slurred speech). These

physical effects of stroke produce the greatest cause of disability worldwide (Barnett, Mohr, Stein, & Yatsu, 1998; Kolb & Wishaw, 2009).

1.6.2 Cognitive Effects

Cognitive neuropsychology examines mental function. Cognitive neuropsychology attempts to understand the way that information is acquired and manipulated within the brain and how these processes can be affected by damage to the brain. There are four main cognitive neuropsychological domains; sensory perception, memory, executive function and language. Memory is the process by which we are able to retain, manipulate and retrieve information to guide our behaviour in daily situations (Fuster, 1995). Memory itself is a complex set of different processes which can be broken down into a variety of sub-processes, for example, sensory memory, verbal memory, prospective memory, remote memory, long term memory, short term memory, episodic memory and autobiographical memory (Squire & Schacter, 2003; Kolb & Wishaw, 2009). Impairment to this domain can lead to a variety of different dysfunctions in retaining or retrieving information. Sensory perception refers to how the brain extracts and encodes information about the world from the different sensory systems. Following stroke common impairments to visual perception include deficits in both basic visual perception - including hemianopia (loss of vision in half of the visual field) and quadrantonopia (loss of vision in a quarter of the visual field), and higher-cognitive representations, including attentional deficits (e.g. visual neglect) and agnosias (impaired objects recognition). These deficits occur in the visual field contralateral (opposite) to the hemisphere damaged (Kolb & Wishaw, 2009). Executive functions include planning actions, volition, self-monitoring of action outcomes, self-regulation, initiation and purposive action. Impairment to this domain can result in impairment of motor function (selecting movements before we act on them), responding to internal cues (memory), external cues (stimulus from the environment or people), context cues (rules of social behaviour), speech (selection of words), difficulties in forming strategies and response inhibition (inhibiting one piece of information and concentrating on another) (Lezak, Howieson, & Loring, 2004; Kolb & Wishaw, 2009). The language domain is concerned with how we learn, process and produce language. Impairment in language can lead to aphasia (a complete

inability to utter or understand comprehensible speech) or dysphasia (impaired speech) which are both common following a left hemisphere stroke. Dysphasia has two categories, receptive and expressive dysphasia. Receptive dysphasia is when a person has difficulty in *understanding* information. Expressive dysphasia is when a person has difficulty in *expressing* information. Stroke can affect any of these cognitive domains and commonly will result in a large combination of cognitive effects across domains (Barnett et al., 1998).

1.6.3 Psychological Effects

As with any disease patients who have had a stroke may suffer from strong psychological symptoms which are not necessarily specifically related to the functions of the areas damaged. Psychological responses could include stress, personality changes and depression (Miller & Blackwell, 2006; Denollet, 1999; Bilge, Kocer, Kocer, & Turk Boru, 2008) (See Chapter 3). Importantly as psychological effects are a product of brain function they may directly follow, or be exacerbated by damage to emotional centres of the brain (for example, the amygdala and medial temporal lobe). People may find they have less emotional self-control, e.g., crying or laughing easily (Smith, 2000). The most common emotional reaction after stroke is depression (Piber et al., 2012; Altieri et al., 2012; Pascoe, Crewther, Carey, & Crewther, 2011) but euphoria can also occur (Kotila, Waltimo, Niemi, Laaksonen, & Lempinen, 1984; Turecki, Mari, & Del Porto, 1993) and other psychological factors such as coping and social support are also important to stroke (Surtees 2006; Surtees et al., 2008) .

These psychological factors are also considered to be risk factors and they will be discussed in the next section alongside other traditional risk factors such as, age, gender, hypertension, cholesterol, cardiac factors, diabetes mellitus, cigarette smoking, diet, abdominal obesity, lack of exercise, alcohol intake, ethnicity, family history of stroke and social inequalities.

1.7 What are the Major Risk Factors and Causes of Stroke?

There are differing risk factors and causes of stroke that can be broadly classified into biomedical, lifestyle and psychological factors. However it is important to note that these factors potentially overlap and interact, i.e., the effects of lifestyle (e.g. smoking) can induce biological changes (e.g. restricted blood flow).

1.7.1 Biomedical risk factors

a) Age

Ageing causes the arteries in the body to weaken and for the arteries to become stiff. Fatty deposits cause weak spots in the artery walls which results in the elderly population being susceptible to vascular disease (Smith, 2000; Stroke Association, 2013; Stork et al., 2004; Mattace-Raso et al., 2006). Hence stroke risk increases with age. The British Heart Foundation has reported the age group 75+ has accrued 40,770 deaths in 2010, the age group 65-74 years accrued 5,209 deaths, the age groups 55-64, accrued 1,939, the age group 45-54 years accrued 940 deaths, the age group 35-44 years accrued 355 deaths and under 35's accrued 153 deaths (Townsend et al., 2012). Comorbidities also increase with age (Giaquinto, 2003; Bushnell, Lee, Duncan, Newby, & Goldstein, 2008; Karatepe, Gunaydin, Kaya, & Turkmen, 2008).

b) Gender

There is conflicting evidence in regard to the issue of gender and stroke. Men have been reported to have a higher likelihood of experiencing vascular disease compared to premenopausal women. However after menopause it has been reported that men and women experience similar levels of vascular disease. This has been hypothesised perhaps due to the loss of the hormone oestrogen in women which may have protective properties (Moosmann & Behl, 1999; Cordey & Pike, 2005; Kumar & Clarke, 2009). In contrast men have been reported to have higher rates of stroke occurrence compared to women from the ages of 18 -74. However from 75+, it has been reported the trend changes, with women experiencing higher rates of stroke, with 26,322 reported cases in 2010 compared with 14,448 cases in men (Townsend et al., 2012). This reversal in trends however is not explained by Townsend et al.

However the Stroke Association (2013) report that in 2010 in the UK more women suffered a stroke compared to men (30,079 in women compared with 19,287 in men) and The American Heart Association (2013) has reported that approximately 55,000 more women experience stroke compared to men (Go et al., 2013).

In 2012/2013 approximately 404,000 hospital admissions for coronary heart disease were recorded in the U.K but no specific number of admissions were clarified due to stroke only (Townsend, Williams, Bhatnagar, Wickramasinghe, & Rayner, 2014, p.52). Including coronary heart disease, stroke, other cardiovascular disease, nervous system disease, respiratory disease, cancer, digestive system disease, genitourinary disease, injury & poisoning and all other causes, 115, 013 (1.2%) men were reported to have had experienced a stroke and 119, 484 (1%) women. Prevalence of stroke in percentages for the UK, are reported as 2.53% for men (from a sample of 47,888) and 1.99% for women (from a sample of 46,549) for all ages. According to these statistics, men experience a higher rate of stroke compared with women in the U.K.

The Quality and Outcome framework (QOF) encourages GPs to keep records on patients suffering specific illnesses. Stroke has been reported in 2012/2013 to have been experienced by 1.17 million people, however it is important to bear in mind the difficulty in gaining accurate statistics due to misdiagnoses (Townsend, Williams, Bhatnagar, Wickramasinghe, & Rayner, 2014).

A Canadian study by Reid, Dai, Gubitz, Kapral, Christian, & Phillips, (2008) concluded the majority of gender differences in stroke could be explained by confounds and more research should be conducted in this area.

c) Hypertension (High Blood Pressure)

Normal blood pressure should fall below 120 (systolic blood pressure, the highest pressure when the heart beats) over 80 (diastolic blood pressure, the lowest level of pressure as the heart is between beats) mmHg. Consistent high blood pressure can cause problems to the vascular system because arterial blood pressure (the pressure of the blood being circulated on the vessel walls) can weaken vessels. Elevated blood pressure leads to stroke, ischaemic heart disease and other diseases of the vascular system (for instance peripheral vascular disease) (MacMahon et al., 1990; Kumar & Clarke, 2009).

Research has reported diastolic blood pressure between 75 and 102 mm Hg produces a fivefold increase in stroke risk from people with and without pre-existing symptoms of cardiovascular disease. It has been reported that with every 10 mmHg increase in the usual diastolic blood pressure (minimum blood pressure) there is an 80% increase in stroke risk (Qizilbash, Lewington, Duffy, & Peto, 1995) and also with every 7.5 mmHg increment, the risk of stroke doubles (Eastern Stroke and Coronary heart Disease Collaborative Group, 1998).

However recording blood pressure in research can be problematic, as blood pressure can be high after stroke but can also be manipulated to be lower afterwards with medication and lifestyle changes (O'Donnell et al., 2010). Also the White Coat Effect (WCE) can occur when blood pressure readings are taken. This can be a problem for research as when blood pressure readings are taken by a doctor or nurse, by the very nature of taking the reading the patient can have an increase in blood pressure in reaction to the test (Saladini, Benetti, Malipiero, Casiglia, & Palatini, 2012; Garcia-Donaire et al., 2012). In Lee et al's (2011) study 65% of stroke participants had hypertension and 67% of those were treated with antihypertensive medication in the year prior to stroke, which illustrates the importance of hypertension and stroke.

d) Cholesterol

The accepted level of healthy total cholesterol is less than 5.0mmol/l. Low Density Cholesterol (LDL) is the cholesterol type which can cause atherosclerosis and High Density Cholesterol (HDL) is the "good" cholesterol which removes the LDL from the blood. Too much LDL cholesterol can cause vascular disease (National Institute for Health and Clinical Excellence, 2010). Cholesterol is a lipid based protein which is produced in the liver. It serves many functions of the body such as facilitating hormone production and making healthy cell walls. Cholesterol is a fatty substance that travels via the blood. Too much cholesterol can accumulate through dietary fat which can cause a build up of fat in the arteries and blood vessels (atherosclerosis). Atherosclerosis can block the flow of blood to the heart (cardiovascular disease) and to the brain (cerebrovascular disease). Angiographic data has illustrated lowering cholesterol can both reduce atherosclerosis and revert the build-up of fatty deposits (Rizzo et al., 2009; Kumar & Clarke, 2009). Cholesterol has caused some controversy in that there are conflicting research

findings on its relationship with stroke. Some studies suggest there is an association between cholesterol and stroke (Lindenstrom, Boysen, & Nyboe, 1994; Horenstein, Smith, & Mosca, 2002; Prospective Studies Collaboration, 2007), some studies report inconclusive results (Sacco et al., 1997; Lewington, Clarke, Qizilbash, Peto, & Collins, 2002; Larsson, 2013) and some studies report no relationship (Oliver, 2000; Varbo et al., 2011).

e) Cardiac Factors (Atrial Fibrillation, Myocardial Infarction)

Atrial fibrillation is the irregular rapid beating of the heart. This causes slow blood flow in the left chamber of the heart. This blood pools and can cause blood clots. Clots can travel around the body causing blockages to arteries that can cause strokes and heart attacks (National Collaborating Centre for Chronic Conditions, 2006).

Stroke mortality has been reported as higher in patients with atrial fibrillation than for those without (Lee, Shafe, & Cowie, 2011). It is suggested it causes a five-fold increase in likelihood and also produces more severe strokes (Camm et al., 2010). Atrial fibrillation is an important risk factor for stroke but recent reports indicate that it is not treated as a serious risk factor (National Institute for Health and Clinical Excellence, 2010; DoH, 2011).

A myocardial infarction (MI) is commonly referred to as a heart attack. A heart attack occurs when a clot forms disturbing blood flow from reaching the heart, causing the heart to be starved of oxygen (Brown, Jacobsen, Weston, Yawn, & Roger, 2005; Kumar & Clarke, 2009). In a meta-analysis conducted by Camm et al., (2010), previous MI was predictive of increased stroke risk and these patients had a higher mortality rate compared with patients without previous MI.

f) Diabetes mellitus

Diabetes is a metabolic disease and affects blood circulation because of abnormal glucose intolerance. Type 1 diabetes develops when the immune system attacks the cells that produce insulin, which leads to increased blood glucose levels. Type 2 diabetes develops when the body does not produce enough insulin. This can cause serious damage to all organ systems in the body if the condition is not controlled.

Diabetes increases the risk of vascular disease, hypertension, cholesterol and obesity. High blood glucose levels can cause a higher mass of fatty substances inside the blood vessel walls. The fatty substances may affect blood flow increasing the chance of atherosclerosis (Kumar & Clarke, 2009; National Diabetes Information Clearinghouse, 2012).

1.7.2 Lifestyle and Behavioural Factors

a) Cigarette Smoking

Cigarettes are addictive due to their nicotine content but also contain components (e.g. carcinogens) that can cause a thickening of the lining in the carotid arteries in active and passive smokers and smoking accelerates the process of degeneration of the cerebral arteries (Howard et al., 1994; Smith, 2000; Stroke Association 2013). This thickening layer leads to atherosclerosis, the fatty build up in the arteries which obstructs the flow of blood to the brain. Additionally blood carries oxygen but smoking produces carboxyhaemoglobin in the blood, which affects the transport of oxygen around the body. Haemoglobin prefers to bind with carbon monoxide over oxygen, thus transporting carbon monoxide around the body instead of oxygen. Smoking also makes the blood susceptible to clotting. These clots can travel around the body causing blockages (causing an embolism clot, which can lead to an ischaemic stroke).

Active smoking is a risk factor for all stroke types (Jamrozik, Broadhurst, Anderson, & Stewart-Wynne, 1994; Jamozik, 2005). It has been estimated that 10% of mortality from stroke are attributable to smoking (Health Committee second report, 2000). The risk of stroke in smokers has been reported as approximately two to four times the risk in non-smokers (American Heart Association Scientific Statement, 2001; Bonita, 1999) with current smoking leading to earlier stroke onset (Adib-Samii, Brice, Martin, & Markus, 2010). However it has been reported that the effects of smoking on the vascular system can be reversed if smoking is ceased and the risk can completely disappear by ten years of smoking cessation (Kawachi et al., 1993; Jamozik, 2005; Kumar & Clarke, 2009).

b) Diet

Fatty diets low in antioxidants, high in salt and low in carbohydrates are associated with higher levels of vascular disease. The fat from unhealthy diets causes

obesity and atherosclerotic clots which can lead to an ischaemic stroke (see Figure 2). Food stuffs such as fish, lean meats, fruits and vegetables make up a healthy diet. Sugar, fat, protein, refined grains and starch contribute to an unhealthy diet which can lead to abdominal obesity (Drewnowski & Darmon, 2005; Kumar & Clarke, 2009).

c) Abdominal obesity

Abdominal obesity is a risk factor for stroke due to the increased risk of atherosclerosis and diabetes which can lead to ischaemic stroke (Kumar & Clarke, 2009; O'Donnell et al., 2010). Obesity causes increased risk of mortality and morbidity in cases of diabetes mellitus, coronary heart disease, stroke, heart failure, asthma, cancer, degenerative joint disease, and many others (American Heart Association, 2011).

d) Lack of Exercise

Lack of exercise is an independent risk factor for vascular disease. Regular exercise prior to stroke can affect glutamate receptors (which are chemical neurotransmitters which passes information between neurons), which may facilitate resistance to ischaemic stroke (Zhang, Jia, Wu, Hu, & Wang, 2010). Exercise lowers the risk of vascular disease especially aerobic exercises which use large muscles in the back, legs and arms which promote increased heart rate and breathing (Leung et al., 2008; Kumar & Clarke, 2009).

e) Alcohol intake

Moderate alcohol consumption may protect against strokes but excessive alcohol consumption can affect vascular disease by increasing hypertension and impairing clotting mechanisms. This is because alcohol reduces the functioning of the liver which produces proteins that controls spontaneous bleeding (Klatsky 2008; Kumar & Clarke, 2009).

1.7.3 Psychosocial Factors

a) Mood, Personality & Social Support

Five main areas of psychosocial well-being have been linked to vascular disease: depression, stress, social support, personality, and coping styles (Steptoe & Brydon,

2009; Kumar & Clarke, 2009; Buckley, McKinley, Tofler, & Bartrop, 2010; Menezes, Lavie, Milani, O'Keefe, & Lavie, 2011; Glozier et al., 2013).

(i) **Depression.** The clinical definition of depression is described using the Diagnostic and Statistical Manual of Mental Disorders (DSM-V, 2013) criteria which includes depressed mood, weight loss, loss of interest in pleasure, insomnia, fatigue, feelings of worthlessness, inability to concentrate and thoughts of death.

(ii) **Stress.** Stress is a multifaceted factor. It covers the psychological, behavioural, physiological, or all three, changes to stressors. Stress depends upon how it is perceived, responses to change the situation and the appraisal of outcomes (Lazarus and Folkman, 1984).

(iii) **Social Support.** Social support has been defined by Sarason, Sarason, Shearin, & Pierce, (1983) as the number of friends that supply social support and also the satisfaction the individual in question, has with this support. This support buffers against stress.

(iv) **Personality Style.** A recent interest in Type D personality (distressed personality) and vascular disease has emerged. Type D Personality is characterised as individuals who experience negative emotions and inhibit the expression of these emotions in social situations (Denollet, Pedersen, Vrints, & Conraads, 2006).

(v) **Coping Styles.** Coping styles such as Repressive coping and Sense of Coherence (SoC) may be important in managing recovery from disease. People with a repressive coping style are identified by showing high defensiveness and low trait anxiety. Repressors report low levels of distress whilst showing high physiological signs of stress therefore repressors may appear psychologically healthy but are prone to suffer from physical health problems (Myers et al., 2008). SoC measures coping with adverse experiences using stress adaptive coping, which has been demonstrated to be an important factor in previous stroke research (Surtees et al., 2006).

These factors will be discussed more thoroughly in Chapter 3.

1.7.4 Interaction Factors

a) Ethnicity

There have been studies to suggest there is an ethnic difference in stroke, implying there are ethnic risk factors between Caucasians in East and Central Europe (Sudlow & Warlow, 1997), Taiwanese, Japanese, Chinese, people from Hong Kong (Hu et al., 1992; Suzuki et al., 1987; Kay et al., 1992; Asian Acute Stroke Advisory Panel, 2000; Shi, Hart, Sherman, & Tegeler, 1989), African Americans (Kleindorfer, 2009; Waddy et al., 2009; American Heart Association, 2011) and Indian and Sri Lankans (Anand et al., 2000).

Although ethnic background may be reported as a potential risk factor for stroke, no known reliable blood biomarkers or genes have been identified to predict the risk of developing stroke (Anand et al 2000; Ariyaratnam et al., 2007; Bondarenko et al., 2011), although some studies have shown a potential link (Wei et al., 2011). The two most extensively studied candidate genes are angiotensin I converting enzyme (ACE) and 5,10-methylenetetrahydrofolate reductase (MTHFR). The number of susceptibility genes that could be important in this case has not been determined (Bondarenko et al., 2011).

The BRAINS study is a project which currently aims to recruit 3000 participants from the UK, India and Sri Lanka. This is to develop a repository bank to investigate the differences in genes between Caucasians and South Indians to establish if there are genetic differences and/or environmental differences in stroke incidence. This could facilitate the understanding of the role of ethnicity as a risk factor for stroke (Yadav et al., 2011).

Cultural factors may additionally influence stroke incidence aside from genetic factors. In the most up to date British Heart Foundations Statistics Database on ethnicity (Scarborough et al., 2010), people of African Caribbean background have the highest stroke incidence compared to people of a Caucasian background. However no explanation was given as to why this may be the case. People with an African Caribbean background have double the risk of stroke and experience stroke a decade earlier compared to Caucasians (Balarajan, 1991; Stewart, Dundas, Howard, Rudd, & Wolfe, 1999). Upon further investigation the Stroke Association (2015) have declared the reasons for this ethnic difference as complicated and currently unknown.

African Caribbean's also have a higher degree of diabetes incidence (Lemic-Stojcevic, Dundas, Jenkins, Rudd & Wolfe 2001). This is due to dietary habits which are rich in sugar. Changes in diet and exercising are ways to combat this. For example, eating more fruit, veg and fibre, (such as yams, plantain and sweet potato), protein (lentils and fish), to cut down on high sugar (which can be present in coconut and palm oil) and reducing intake of fried foods (jerk chicken, fritters). Other methods of cooking such as steaming and grilling would be beneficial to healthy eating.

Additionally other cultural factors may contribute to this ethnic trend such as smoking and BMI. Smoking has been reported as being high in the African Caribbean group and African Caribbean women have a higher BMI than Caucasian women and are therefore more vulnerable to stroke. Obesity and hypertension are major risk factors which are prevalent in African Caribbean people (Hajat, Tilling, Stewart, Lemic-Stojcevic, & Wolfe, 2004). More targeted interventions should be given to African Caribbean people due to this (Dundas, Morgan, Redfern, Lemic-Stojcevic & Wolfe 2001).

b) Family History

Family history combines lifestyle and biology, however the roles of genetic factors and lifestyle factors can be difficult to separate as this is open to interpretations. Family history is clinically used as a risk factor for stroke.

However many risk factors are viable and family history incorporates many of them. The medical profession believe vascular disease before 50 years of age is more indicative of family history effects (Kumar & Clarke, 2009). Studies have shown there is an increased risk of offspring experiencing stroke if their parents have suffered stroke, with ischaemic stroke (Seshadri et al., 2010), and intracerebral haemorrhage (Woo et al., 2002) and subarachnoid haemorrhage (Kissela et al., 2002).

c) Social Inequalities

Socioeconomic status may also be a risk factor for stroke. Examining socioeconomic status and stroke mortality in the 1980s for men aged 30 to 64 years

of age (from England and Wales, Ireland, Finland, Sweden, Norway, Denmark, Italy, Spain, United States, France, Switzerland, and Portugal) it was reported that manual classes had higher stroke mortality rates than non-manual classes (Kunst, del Rios, Groenhof, & Mackenbach, 1998).

Important factors in explaining such differences include employment and education. The highest prevalence of stroke (69.3%) was related to adults that were unable to work. Forty five percent of strokes were experienced by retired people, unemployed adults (43.4%), homemakers (34.3%), and employed people (34.0%) (American Heart Association, 2011). However these statistics could show a cohort bias as those unable to work may already have morbidity and be older compared to those who are employed as they may be younger and healthier.

Educational level may determine the level of knowledge regarding the causes of stroke. Hispanic women were more likely than American Caucasian women to report they did not know the risk factors for stroke (Christian, Rosamond, White, & Mosca, 2007) and 25.9% of college graduates did not know about risk factors for stroke compared with 52.5% with no education (American Heart Association, 2011).

In this section risk factors for stroke have been detailed. It is also important to correctly diagnose a stroke. This is achieved through investigating clinical signs and images of the brain. This will be discussed in the next section.

1.8 Diagnosis of Stroke: Clinical Signs and Brain Imaging

Strokes are diagnosed based upon clinical features and the use of brain imaging. The most common clinical symptoms of a stroke are weakness or paralysis on one side of the body (affecting the arms, legs, trunk and/or face) and dizziness, loss of balance and coordination and possible blacking out. Speech and swallowing may be affected and changes or loss of vision and severe headaches are also sign of stroke (Stroke Association, 2013).

Brain imaging following stroke is typically performed using Computed Tomography (CT) and Magnetic Resonance (MRI) Scanning. A CT scan is a radiation x-ray taken from multiple angles to generate a 3D image and it highlights the difference in density of bones, blood, brain and areas of infarctions. CT resolution is less detailed than MRI's but it is cheaper and more accessible. If

administered early it cannot identify infarcted cells but haemorrhages will be visible instantly. If haemorrhage has not occurred treatments such as thrombolysis and warfarin can be given.

An MRI scan can record finer detail of the damage to the soft tissue and the vasculature of the brain. This method uses a strong magnetic field to facilitate producing an image of the brain. MRI's do not expose the patient to radiation but are considerably more expensive (Barnett, Mohr, Stein, & Yatsu, 1998).

Investigating brain images aids in deciphering the classification of stroke. This will be discussed in the next section.

1.9 Classification of Stroke

There are two main taxonomies which are used to classify stroke; The Oxford Community Stroke Project classification (OCSP, also referred to as the Bamford Scale) (Bamford, Sandercock, Dennis, Burn, & Warlow, 1991) and the Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification (Adams et al., 1993).

The OCSP determines which *areas* of the brain have been affected and relies on the initial symptoms. There are four categories; total anterior circulation infarct (TACI), partial anterior circulation infarct (PACI), lacunar infarct (LACI) or posterior circulation infarct (POCI) (Bamford, Sandercock, Dennis, Burn, & Warlow, 1991).

The TOAST classification focuses on the underlying *cause* of the stroke and is based on clinical symptoms with 5 categories: (1) thrombosis or embolism due to atherosclerosis of a large artery, (2) embolism of cardiac origin, (3) occlusion of a small blood vessel, (4) other determined cause, (5) undetermined cause (two possible causes, no cause identified, or incomplete investigation) (Adams et al., 1993).

Once stroke has been confirmed, treatment must follow. This is typically achieved with the use of anticoagulation drugs, antihypertensive drugs, statins, thrombolysis, surgery and rehabilitative therapy. These will be discussed in the next section.

1.10 Treatment

There is no cure for stroke but there are various treatment options, which include drug administration, surgery and rehabilitative therapy. These interventions aim to limit the damage of strokes and decrease the probability of further events, which will increase recovery.

1.10.1 Drug Therapy

a) Antiplatelet drugs

Aspirin reduces platelet aggregation therefore blood platelets do not form a blockage which can lead to the formation of a clot, consequently thinning the blood. Clopidogrel and dipyridamole are other commonly used drug treatments for blood thinning (Albers & Amarenco, 2001; Sudlow, 2007; Kumar & Clarke, 2009). Aspirin was introduced in 1978 in stroke therapy for the prevention of stroke (The Canadian Cooperative Study Group, 1978). Evidence from 40,000 randomised patients showed the use of oral aspirin within 48 hours of ischaemic stroke reduces 14-day morbidity and mortality (The International Stroke Trial (IST), 1997; CAST (Chinese Acute Stroke Trial), 1997). The advantages of aspirin use are its simple administration and low cost (Gilligan et al., 2005). It is now regularly used for stroke prevention (Thomson & Anderson, 2013) and post stroke treatment to prevent recurrent stroke (Chen et al., 2000).

b) Anticoagulation drugs

Warfarin is the most common anticoagulant given after stroke. It inhibits vitamin K dependent synthesis of clotting factors and therefore prevents blood clots forming. Warfarin is administered if haemorrhage has been excluded and if cardiac problems are present (i.e., atrial fibrillation) (Hankey, 2002; Kumar & Clarke, 2009; Shah et al., 2014).

c) Antihypertensive drugs

Blood pressure abnormalities are common with stroke both before and after stroke onset (National Collaborating Centre for Chronic Conditions, 2008). Results

of the Perindopril Protection Against Recurrent Stroke Study (PROGRESS), tested Perindopril and Indapamide, and yielded a 30% reduction in recurrent stroke during 5 years (PROGRESS Collaborative Group, 2001). Ramipril was also proven to be effective in reducing hypertension in the Heart Outcomes Prevention Evaluation (HOPE) study (Yusuf et al., 2000).

d) Statins (for cholesterol treatment)

Statins inhibit enzymes in the liver that control the production of cholesterol. Statins have been reported to demonstrate a protective effect against strokes (Schwartz et al., 2001). Stroke incidence is reduced in patients with coronary artery disease who are given statins (Amarenco, 2005). Prescriptions for cholesterol lowering drugs have increased over the last 10 years (Lee, Shafe, & Cowie, 2011) with statins such as Simvastatin, Pravastain and Atorvastatin being successful in reducing stroke and coronary heart disease (Byington et al., 2001; Montaner et al., 2008; Kumar & Clarke, 2009; Moonis, 2012). However statin treatment may cause haemorrhages to occur in acute stroke and therefore caution is needed (Hankey, 2006).

e) Thrombolysis

Thrombolysis is the disintegration of blood clots by medical intervention (Kumar & Clarke, 2009; Robinson, Zaheer & Mistri, 2011). Thrombolysis in acute ischaemic stroke has been shown to significantly improve outcome in selected patients treated within 3 hours of onset of symptoms but is not applicable to haemorrhagic stroke because it would exacerbate bleeding. Thrombolysis in acute stroke is associated with an increased risk of haemorrhage (up to 6% of patients) and is to be used carefully (National Institute for Health and Clinical Excellence, 2007). Thrombolysis is effective in reducing disability but not mortality rates (Hacke, Donnan & Fieschi, 2004). In 2010, 5% of patients received thrombolysis in the UK (Royal College of Physicians, 2011). This low number could be due to the low number of Hyper Acute Stroke Units (HASU's). HASU's administer thrombolysis however HASU's are only in certain hospitals. For example in London UK, there are approximately 85 NHS hospitals, however an examination of their websites reveals only 8 have a HASU.

1.10.2 Surgery

Surgical procedures are also used to combat stroke. Procedures such as carotid endarterectomy can be used to remove significant atherosclerotic narrowing (stenosis) of the carotid arteries, which supplies blood to the brain whilst procedures such as thrombectomy removes the clot directly (Sylaja, Setiawan, Hill, Demchuk, & Wong, 2009; Kumar & Clarke, 2009; Hacke et al., 2015).

1.10.3 Rehabilitative Therapy

The aim of rehabilitative therapy is to maximise the extent of functional recovery by minimising cell loss due to infarction and to also promote cortical reorganisation (using other areas of the brain cortex to help perform tasks). It can reduce levels of infarction by increasing blood flow and activation of neurons in the ischaemic penumbra (the area around the infarct) which can also increase functional recovery (Fisher, 2004; Kumar & Clarke, 2009; Ramos-Cabrer, Campos, Sobrino, & Castillo, 2011). Effects of rehabilitative therapy may be permanent or temporary (Douglas, Edwards, & Goodyear, 2006).

Specialised treatment in Stroke Care Units, have been tailored to aid stroke recovery. As a consequence patients on a specified stroke ward have a 20% increase in functional outcome and reduction in mortality (Langhorne, Williams, Gilchrist, & Howie, 1993; Langhorne, Dey, & Woodman, 2005) with improved blood pressure control, early mobilisation, and better adherence to treatment (Indredavik, Bakke, Slordahl, Rokseth, & Haheim,1999; Cadilhac, Ibrahim, & Pearce, 2004).

Speech therapists help patients with dysphasia, aphasia and swallowing impairment by helping them to use their throat muscles and vocal chords. Physiotherapists help patients to use limbs which may have been affected by the stroke by teaching exercises to help strengthen muscle groups. Occupational therapists help patients to become independent in their daily living by helping them to learn how to cook and manage themselves on a daily basis (Kumar & Clarke, 2009; Rudd, 2012; Kelly, Godwin, & Enderby, 2012). However there are no current psychological health care policies to help patients to deal with the emotional consequences of suffering a stroke, despite the wealth of psychological research undertaken.

Risk factors and treatment of stroke will influence the incidence and prevalence rates of stroke. These will be discussed in the following section.

1.11 Incidence and prevalence

Accurate data on stroke incidence and prevalence can be difficult to obtain. Incidence pertains to new cases of stroke and prevalence pertains to existing cases of stroke. When a patient is admitted to hospital, it may not be recorded if that person has had their first stroke or if it is a recurrent stroke (therefore if it is incidence or prevalence). However, stroke incidence has been reported to have fallen (Rothwell, Coull, & Giles, 2004; Heuschmann, Grieve, & Toschke, 2008; Feigin, Lawes, & Bennett, 2009). Between 1999 and 2008, the incidence of stroke in the UK has been reported to have decreased by 29% (Lee, Shafe, & Cowie, 2011). The Framingham Heart Study (USA) also reported decreasing incidence rates from 1950 to 1977, 1978 to 1989, and 1990 to 2004, (Carandang et al., 2006). The USA reports 795,000 new and recurrent strokes per year (Lloyd-Jones, 2010).

Prevalence of stroke in England has been reported to be 2.4% for men and 2.2% for women. In Northern Ireland, 2% and 1% have been reported respectively, in Wales 3% and 2% respectively, and for Scotland, 3.3% and 2.5% respectively, which is the highest in the UK (Townsend et al., 2012). In the U.S, 2.8% is the reported prevalence (2.7% of men and 2.4% of women) (Go et al., 2013).

With treatments available to aid stroke recovery, this will impact upon long-term survival, recurrence of stroke, morbidity and mortality. These will be the final sections covered in this Chapter and will be discussed next.

1.12 Long-term Survival

Alongside the declining incidence of stroke, survival rates have increased and hence the number of stroke survivors living with disability (Hackett, Duncan, Anderson, Broad, & Bonita, 2000; Gallien et al., 2005). This is due to improved diagnosis, control of risk factors, and rehabilitative gains (Murray & Lopez, 1997).

Survival at five years was 82% in men and 81% in women. Those free of recurrent stroke were 74% (Lee, Shafe, & Crowie, 2011). Approximately 60% to 83% of survivors are independent one year after a stroke (Appelros, Nydevik, &

Viitanen, 2002). In a research study of 2531 participants, variables linked to surviving to 85 years of age included absence of current smoking, low total cholesterol, low systolic blood pressure, good glucose tolerance, higher educational status and female sex (Terry et al., 2005). Additionally 30-40% of stroke patients survived for at least 3 years (Kumar & Clarke, 2009).

1.13 Recurrence of Stroke

Those that have experienced a stroke are at a 10% risk of a recurrent stroke in the first year and a 5% risk for every year thereafter (Burn et al., 1994; Kumar & Clarke, 2009).

In a longitudinal study where participants were followed up for 5 years, 24% had a second cardiovascular event, 75% of which were strokes and 16% of these were fatal (Lee, Shafe, & Crowie, 2011).

1.14 Morbidity

Stroke has been reported as the illness which results in the most complex disability effects (Adamson, Beswick, & Ebrahim, 2004) with a reported 12% experiencing very severe disability, 10% experience severe disability, 14% experience moderate disability, 22% experience mild disability and 42% reported recovering to pre-stroke level (Royal College of Physicians, 2011). There are more than 900,000 people who have had a stroke living in England. Approximately half of these people are left dependent on others for everyday activities (National Audit Office Report: DoH, 2005).

The Stroke Association (2013) has reported the most common effects of stroke morbidity below in Table 1.1:

Table 1.1:

The most common stroke morbidity effects reported by The Stroke Association (2013)

Morbidity	Percentage Affected
Movement	80%
Swallowing problems	40%
Somatosensory loss	80%
Aphasia	33%
Visual problems	66%
Bladder problems	50%
Bowel problems	33%
Dementia	20%
Depression	29%

www.stroke.org.uk/sites/default/files/Stroke%20statistics.pdf (2013)

1.15 Mortality

Stroke causes over 9,500 deaths in people under 75 years of age (1 in 20 deaths) in the UK. Scotland has the highest rates of mortality from stroke, followed by North England, Wales, Northern Ireland and South East England with the lowest rates (Scarborough et al., 2009).

Intracerebral haemorrhage has a mortality rate of 44% after 30 days which is higher than that for ischaemic stroke. However there are difficulties in producing precise statistics on mortality rates for subtypes of stroke because many are classified as "unspecified" (from the Oxford Classification) due in part to patients not being administered brain scans or the stroke lesion not being detected.

Approximately one third of stroke sufferers die within the first ten days, a third recover within one month and a third have chronic disabilities with a need for rehabilitation (Bosanquet, & Franks, 1998). The death rate after stroke is 20% - 25% within 2 years in the UK (Kumar & Clarke, 2009). In the USA, stroke death rates decreased by 44.8% and the actual number of stroke deaths fell by 14.7% (American Heart Association, 2011). India has reported 80% of stroke deaths in 2005 (WHO, 2005) and 80% of strokes have been estimated to occur in low and middle income countries (e.g. India) by 2050 (Feigin, 2007).

However official mortality data rely on the accuracy of death certificates, which can be questionable (Corwin, Wolf, Kannel, & McNamara, 1982). The decline in stroke mortality in many developed countries (Bonita, Stewart, & Beaglehole, 1990) may reflect changes in diagnostic tools or a real decrease. False positives and

false negatives can both occur in reporting. In the Northern Sweden MONICA study 91.7% of stroke deaths were reported correctly (Stegmayr & Asplund, 1992) but there have been other reported studies with higher false positives rates in mortality data (Szczesniewska, Kurjata, Broda, Polakowska, & Kupsc, 1990; Hasuo et al., 1989; Corwin, Wolf, Kannel, & McNamara, 1982). This should warn organising bodies to be vigilant when making conclusions about mortality from death certificate data and may also suggest that error rates can vary across countries.

1.16 Summary

In summary, stroke is a global burden causing mortality and morbidity, which strains the health service. Stroke affects physiological function (such as paralysis and weakening of the limbs, face and trunk), cognitive functions (such as memory, vision, language and executive function) and psychological factors (such as the ability to cope with life changes and mortality).

Unfortunately there is no cure for strokes but risk factors that have been shown to be related to stroke can be acknowledged and in some cases controlled. Risk factors include age, gender, hypertension, high cholesterol, atrial fibrillation, myocardial infarction, diabetes mellitus, cigarette smoking, diet, abdominal obesity, lack of exercise, alcohol consumption, depression, lack of social support, personality style, ethnicity, family history and social inequalities.

Stroke can be treated with drug therapies such as antiplatelet, anticoagulation and antihypertensive drugs, statins and thrombolysis. It can also be treated with surgeries, such as carotid endarterectomy, thrombectomy and so on, and with rehabilitative therapies, such as speech, occupational and physiotherapy.

The DoH has developed a National Stroke Strategy, which amongst other items addressed the needs of research. One of the research needs identified by this strategy is the *"Estimation of the longer-term needs of patients (impairment, activity, participation, quality of life) at different time points post-stroke to help direct intervention studies to improve outcomes*. (DoH, 2005, p.66)

The goal of this thesis is to contribute specifically to this identified research need. Therefore, psychological and cognitive variables will be longitudinally investigated in relation to outcome from stroke. The reverse of this relationship will

not be explored in this thesis, however this can be investigated for possible publication on a separate occasion.

1.17 Outline of Thesis

This thesis is divided into 8 chapters:

Chapter 2: A systematic review (Study One) explores the available literature in regards to longitudinal research designs which focus on psychological factors and their relation to recovery from stroke. The research question is identified here.

Chapter 3: A literature review which acknowledges the findings from Study One and develops the theoretical framework for Study Two (the experiment constructed), with explanations of the variables selected for the current study. The hypotheses are outlined here.

Chapter 4: Outlines the longitudinal methodology, the measures used and the procedure followed for Study Two.

Chapter 5: Presents an introduction to the quantitative analysis for Study Two.

Chapter 6: This chapter examines the statistical analysis for the Physical Recovery Model.

Chapter 6: This chapter examines the statistical analysis for the Psychological Recovery Model.

Chapter 8: Overall discussion of this research project.

Chapter 2

Study One: Do Psychological Factors Affect Stroke Risk And Recovery? A Systematic Review.

2.1 Summary

This chapter will firstly provide a rationale for conducting a systematic review, followed by the aims and methodology (inclusion criteria, exclusion criteria, search strategy, information on the data extraction sheet, search terms and the text selection process). The main body of the Chapter focuses on reviews papers published between 1990-2009. A summary table of the review papers are detailed along with tables that aid in a methodological assessment of these papers. The results detail country of investigation, setting of the research, consecutive vs. non-consecutive patients, sample size, power calculation, number of measurements, length of follow up, demographic information, stroke diagnosis, type and severity, psychological and outcome measures, method of analysis and attrition rates. These are further interpreted in the Discussion and implications of the findings are also discussed. An update of the review was additionally conducted for published studies between 2009- April 2013. To end the Chapter, aims and development of the current study are disclosed.

2.2 Rationale for Conducting a Systematic Review

Systematic reviews have gained importance in Health Psychology. Reviews which are not systematic introduce bias as the authors choose what they focus on, which can also mislead and contradict research that has actually been conducted (Atkins et al., 2004). A systematic review limits bias, is critical in nature and synthesises relevant literature together (Wright, Brand, Dunn, & Spindler, 2007), which reduces overall bias and results in stronger conclusions because of its analytical attributes.

Depression is the main psychological variable associated with stroke in the published literature, as an independent direct predictor. Many studies report depression leads to an increased risk in stroke (Jonas & Mussolino, 2000; Surtees et

al., 2008; Pan et al., 2011), has an effect on stroke recovery (Harwood, Gompertz, Pound, & Ebrahim, 1997; Paolucci et al., 1999; Pohjasvaara, Vataja, Leppavuori, Kaste, & Erkinjuntti, 2001; Wilz, 2007), can lead to a risk of recurrent stroke (Yuan et al., 2012) and also has been associated with death from stroke (Morris, Robinson, Andrzejewski, Samuels, & Price, 1993; House, Knapp, Bamford & Vail, 2001; May et al., 2002).

However it is less clear what other psychological variables are also important in the landscape of stroke and thus, the rationale behind this systematic review is to investigate which other areas of psychology have been researched in regards to stroke risk and recovery. A search of the literature has shown that no systematic review has been published with the same search terms that will be used in this review. Therefore, there is a strong rationale for the inclusion of a systematic review to help inform the direction of the current research.

This review will examine psychological variables and their effect on risk and recovery from stroke. As mentioned in section 1.6.3 of Chapter 1, medicine acknowledges five psychosocial areas that can affect vascular disease: depression, stress, social support, personality style and coping style. These factors will be entered into the search strategy in order to aid searching if simple search terms are too broad.

The following section will outline the aims of the systematic review.

2.3 Aims of the Systematic Review

This systematic review aims to:

(i) Investigate the current literature on psychology, risk of stroke and effect on physical recovery from stroke.

(ii) To decipher any gaps in the literature.

(iii) To form a research question which amalgamates points (i) and (ii). That is, to formulate a research question which incorporates previous psychological research which has contributed to the field of stroke recovery but to also incorporate new psychological factors not yet studied in the realm of stroke recovery. Also cognitive factors will be added to the research question at a later stage (see Chapter 3). Cognitive variables will not be included in this systematic review as including these factors

will result in an insufficient number of papers for a systematic review. From performing an exploratory search, zero papers met the inclusion criteria.

The next stage of this process will focus on the methodology of the review, which has 5 stages: Inclusion criteria, exclusion criteria, search strategy, the data extraction sheet and the search terms used.

2.4 Methodology

2.4.1 Stage 1: Inclusion Criteria

- Research must be written in English.
- Studies searched will be from 1990 – current (April 2013).[1]
- Published literature.
- Study methodology should be quantitative.
- Study design should be longitudinal in nature.
- Time points of the research must be clear.
- Any setting of the research will be accepted (hospitals, clinics, hospices etc.).
- Participants should be in the adult age range (18 years old +).
- Any stroke type will be accepted.
- Any stroke severity (minor – major) will be accepted.
- Psychological variables should be measured at more than one time point (therefore the psychological component of the studies should be longitudinal).

[1] There are no standard time limits for search strategies in a systematic review. Some studies have chosen 10 years as a cut-off point to concentrate on current literature (Querstret & Cropley, 2013), whilst others have chosen longer time frames (Cropley, Theadom, Pravettoni, & Webb, 2008).

Many systematic reviews have used 1990 as the start of their search strategy which allows for approximately 20 years of research to be scrutinized. For example, in reviews investigating advanced practice nurse outcomes (Newhouse et al., 2011), job stress (Lamontagne, Keegal, Louie, Ostry, & Lanbergis, 2007), severe periodontitis (Kassebaum, Bernabé, Dahiya, Bhandari, Murray, Marcenes, 2014), Malaria in China (Lu, Zhou, Horstick, Wang, Liu, & Muller, 2014), diabetes (Zabetian , Sanchez, Venkat Narayan, Hwang, & Ali, 2014), opioid related mortality (King, Fraser, Boikos, Richardson, & Harper), Alzheimer's disease (Chan et al., 2013), Pharmaceutical care services (Roughead, Semple & Vitry, 2005), immunization of Australian children (Lister, McIntyre, Burgess, O'Brien 1999) and PTSD in female veterans (Middleton & Craig, 2012).

- Participant report, not proxy.
- There should be at least one measure of physical outcome.
- Psychological variables must be analysed with recovery as the outcome variable, to determine the effect of psychological factors on risk or recovery.
- If more than one disease is studied within the same research study, stroke must be analysed separately in order to investigate its role in the study.

2.4.2 Stage 2: Exclusion Criteria

- Other study designs will not be included.
- Carers / spouses / healthcare professionals and other proxy measures will not be included.
- Transient Ischemic Attacks (TIA's).
- If there is not a physical outcome measure, the studies will not be included.

2.4.3 Stage 3: Search Strategy

The investigations incorporated into this review were located by hand searching references and mainly using electronic databases. For papers where details were unclear the authors of those studies were contacted for clarification. From 1990 – August 2009, Ingenta Connect, Embase, Psych Info, PubMed and Web of Science were searched. The systematic review was updated from September 2009 – April 2013 using Medline and Summon, more details of which are described in section 2.9.

2.4.4 Stage 4: Data Extraction Sheet

A data extraction sheet was constructed in order to facilitate the extraction of relevant information. These data included aims of the research papers, study design, population, sample size, if patients were recruited consecutively or not, setting for data gathering, demographics (age, gender, ethnicity), any interventions, stroke diagnosis, stroke type, stroke severity, psychological measures, measures for recovery from stroke, statistical analysis employed, results obtained, attrition and conclusions. See Appendix A for details.

2.4.5 Stage 5: Search Terms

"Stroke" and "Psychology" as search terms were too broad. This lead to more specific search terms being used bearing in mind the previous five areas of interest in vascular disease and psychology (depression, stress, social support, personality and coping) (see section 1.6.3).

2.4.5.1 Search Terms for 1990 – August 2009

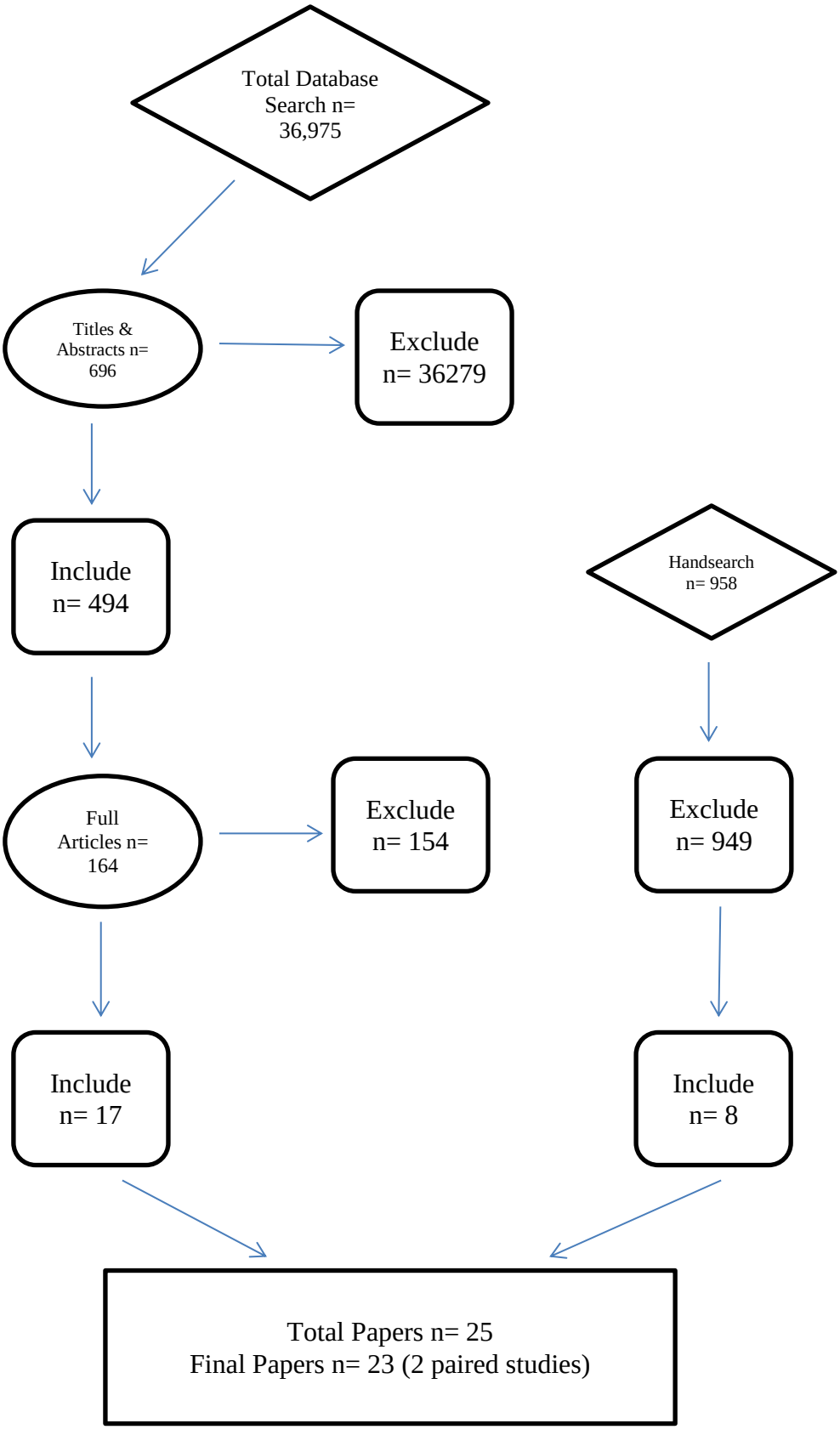
See Table 2.1 and Figure 2.1 for information derived from the search for 1990 – August 2009.

Table 2.1:

Search terms used for 1990 – August 2009 search

Search Terms	PsychInfo	Ingenta Connect	Embase	PubMed	Web of Science
Stroke AND Psychological Stress	68	15	190	363	128
Stroke AND Stress	479	539	4,070	4,448	3,175
Stroke AND Coping	169	35	225	632	0
Stroke AND Depression	1,364	392	3,621	3,218	6
Stroke AND Stress AND Coping	36	8	59	71	0
Stroke AND Personality	210	20	1,573	656	3
Stroke AND Comparative Optimism	0	0	2	4	0
Stroke AND Social Support	177	56	540	354	0
Stroke AND Quality of Life	434	266	2,859	1,736	0
Stroke AND Predictor AND Recovery	67	0	123	0	173
Stroke AND Stress NOT Physiological	362	0	4,029	0	0
Stroke AND Predictor AND Recovery AND Longitudinal	8	0	4	0	8

Figure 2.1: Text Selection Process 1990 – August 2009.



From these papers 25 relevant papers were identified. From these 25, 23 final studies were determined (two sets of studies used the same population and consequently are counted as one study each). These studies are:

1. Schubert D.S.P., Burns R., Paras W., & Siosen E. (1992a)
   &
   Schubert D.S.P., Burns R., Paras W., & Siosen E. (1992ab).

2. Johnston M., Morrison V., MacWalter R., & Partridge C. (1999)
   &
   Johnston M., Pollard B., Morrison V., & MacWalter R. (2004).

Although these papers are paired they do report different information. Consequently, where appropriate they will be treated separately. In the following section the full list of papers are detailed.

2.5. Review References From 1990 – August 2009

1. House, A., Dennis, M., Mogridge, L., Hawton, K., & Warlow, C. (1990). Stressful life events and difficulties preceding stroke. *Journal of Neurology, Neurosurgery, and Psychiatry, 53,* 1024-1028.

2. Morris, P.L.P., Robinson, R.G., & Raphael, B. (1990). Prevalence and course of depressive disorders in hospitalized stroke patients. *International Journal of Psychiatry in Medicine, 20* (4), 349-364.

3. Parikh, R.M., Robinson, R.G., Lipsey, J.R., Starkstein, S.E., Fedoroff, J.P., & Price, T.R. (1990). The impact of post stroke depression on recovery in ADL over a 2 year follow up. *Archives of Neurology, 47,* 785-789.

4. Morris, P.L.P., Raphael, B., & Robinson, R.G. (1992). Clinical depression is associated with impaired recovery from stroke. *The Medical Journal of Australia, 157,* 239-242.

5a. Schubert, D.S.P., Burns, R., Paras, W., & Siosen, E. (1992a). Increase of medical hospital length of stay by depression in stroke and amputation patients: A pilot study. *Psychotherapy and Psychosomatics, 57,* 61-66.

5b. Schubert, D.S.P., Burns, R., Paras, W., & Siosen, E. (1992b). Decrease of depression during stroke and amputation rehabilitation. *General Hospital Psychiatry, 14,* 135-141.

6. Schubert, D.S.P., Taylor, C., Lee, S., Mentari, A., & Tamaklo, W. (1992c). Physical consequences of depression in the stroke patient. *General Hospital Psychiatry, 14,* 69-76.

7. Morris, P.L.P., Robinson, R.G., & Samuels, J. (1993). Depression, introversion and mortality following stroke. *Australian & New Zealand Journal of Psychiatry, 27,* 443-449.

8. Loong, C.K., Ng, K.C.K., & Straughan, T.P (1995). Post-stroke depression: Outcome following rehabilitation. *Australian & New Zealand Journal of Psychiatry, 29,* 609-614.

9. Simonsick, E.M., Wallace, R.B., Blazer, D.G., & Berkman, L.F. (1995). Depressive symptomatology and hypertension-associated morbidity & mortality in older adults. *Psychosomatic Medicine, 57,* 427-435.

10. Elmstahl, S., Somner, M., & Hagberg, B. (1996). A 3 year follow-up of stroke patients: Relationships between activities of daily living and personality characteristics. *Archives of Gerontology & Geriatrics, 22,* 233-244.

11. Chang, A.M., & MacKenzie, A.E. (1998). State self esteem following stroke. *Stroke, 29*, 2325-2328.

12. Herrmann, N., Black, S.E., Lawrence, J., Szekely, C., & Szalai, J.P., (1998). The Sunnybrook Stroke Study: A prospective study of depressive symptoms and functional outcome. *Stroke, 29*, 618-624.

13a. Johnston, M., Morrison, V., MacWalter, R., & Partridge, C. (1999). Perceived control, coping and recovery from disability following stroke. *Psychology & Health*, *14*, 181-192.

13b. Johnston, M., Pollard, B., Morrison, V., & MacWalter, R. (2004) Functional limitations and survival following stroke: Psychological and clinical predictors of 3 year outcome. *International Journal of Behavioural Medicine*, *11* (4), 187-196.

14. van de Weg, F.B., Kuik, D.J., & Lankhorst, G.L. (1999). Post-stroke depression & functional outcome: A cohort study investigating the influence of depression on functional recovery. *Clinical Rehabilitation, 13*, 268-272.

15. Chemerinski, E., Robinson, R.G., & Kosier, J.T. (2001). Improved recovery in activities of daily living associated with remission of post stroke depression. *Stroke, 32*, 113-117.

16. Lai, S., Duncan, P., Keighley, J., & Johnston, D. (2002). Depressive symptoms and independence in BADL and IADL. *Journal of Rehabilitation Research and Development*, *39* (5), 589-596.

17. Cassidy, E.M., O'Connor, R., & O'Keane, V. (2004). Prevalence of post-stroke depression in an Irish & Its relationship with disability outcome following inpatient rehabilitation. *Disability & Rehabilitation, 26* (2), 71-77.

18. Nannetti, L., Paci, M., Pasquin, J., Lombardi, B., & Taiti, P.G. (2005). Motor and functional recovery in patients with post-stroke depression. *Disability & Rehabilitation*, *27* (4), 170-175.

19. Saxena, S.K., Ng, T-P., Koh, G., Yong, D., & Fong, N.P. (2007). Is improvement in impaired cognition and depressive symptoms in post-stroke patients associated with recovery in activities of daily living? *Acta Neurologica Scandinavica*, *115*, 339-346.

20. Bilge, C., Kocer, A., & Turk Boru, U. (2008). Depression and functional outcome after stroke: The effect of anti depressant therapy on functional recovery. *European Journal of Physical & Rehabilitative Medicine*, *44* (1), 13-18.

21. Bos, M.J., Linden, T., Koudstaad, P.J., Hofman, A., Skoog, I., Breteler, M.M.B., & Tiemeier, H. (2008). Depressive symptoms and risk of stroke: Rotterdam Study. *Journal Of Neurology, Neurosurgery & Psychiatry*, *79*, 997-1001.

22a. Ostir, G.V., Berges, I-M., Ottenbacher, M.E., Clow, A., & Ottenbacher, K.J. (2008). Associations between positive emotion and recovery of functional status following stroke. *Psychosomatic Medicine*, 70 (4), 404-409.

23. Hamzat, T.K., & Peters, G.O. (2009). Motor function recovery and quality of life among stroke survivors in Ibadan, Nigeria. A 6-month follow-up study. *European Journal of Physical Rehabilitation Medicine*, *45*, 179-83.

See Table 2.2 for a summary of the study characteristics. This will be followed by a section on the methodological quality assessment.

Table 2.2:

Summary table, showing details of the 30 review studies

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
1. House et al. (1990) UK.	128 stroke participants and 141 participants in a control group (gender not specified). Type of stroke not specified.	Case-control longitudinal design. Data collected at baseline, diagnosis of stroke only (T1), 113 participants in total for T2, 84 of which were seen 1 month post stroke, and 29 of which were seen at 6 months post stroke. There is a T3 follow up but the time of which is unclear.	Rehabilitation.	1. Bedford College Life Events & Difficulties Schedule (LEDS) 2.Mortality records.	Severely threatening life events are associated with an increased risk of stroke.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
2. Morris et al. (1990) Australia.	99 participants at T1 recruited 51 males, 48 females. T2, recruited 34 males and 22 females. 73 infarcts, 16 hem, 45 right hemisphere, 46 left hemisphere lesion, 8 brainstem strokes.	Longitudinal. Data collected at 2 months post stroke (T1) and 17 months post stroke (T2).	Rehabilitation.	1. Demographics & Medical info 2. Hollingshead Social Class 3. Composite International Diagnostic Interview (CIDI) – psychiatric exam with DSM III criteria 4. Montgomery & Asberg Depression Rating Scale (MADRS) 5. Abbreviated Barthel Index (BI)	Non depressed patients report less physical and cognitive impairment. But differences on BI and MMSE with depressed patients were small and not significant.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
				6. MMSE 7. Family psychiatric history	

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
3. Parikh et al. (1990) USA.	63 participants (42% male in depressed group and 66% male in non depressed group). 51 acute thromboembolic and 12 haemorrhagic stroke.	Longitudinal. Data collected at 2 weeks post stroke (T1) and 2 years post stroke (T2).	Rehabilitation.	1. MMSE 2. Hamilton Depression Rating Scale 3. Zung Self Rating Depression Scale 4. Present State Examination (PSE with DSM-III criteria 5. Social Functioning Examination – Quality of social relationships 6. Social Ties Checklist – Number of social	Major and minor depression is associated with a decrease in functional recovery. Depression may have a negative effect on motor and language recovery. Major depression remitted by 2 year follow up. Some non depressed patients develop depression. Recovery in ADL was slower in depressed patients than in non depressed patients.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
				connections 7. John Hopkins Functioning Inventory (JHFI)	

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
4. Morris et al. (1992) USA & Australia	49 participants (33 male, 16 female). 39 infarct, 5 haemorrhagic, 25 right hemisphere, 24 left hemisphere.	Longitudinal. Data collected at 2 months post stroke (T1), and 16 months post stroke (T2).	Rehabilitation.	1. Composite International Diagnostic Interview (CIDI) – psychiatric exam with DSM III criteria. 2. General Health Questionnaire 3. Karnofsky Performance Rating Scale – Functional status 4. Abbreviated Barthel Index 5.Mental Status Questionnaire (MSQ)	Clinical depression 2 months after stroke is associated with impaired recovery 16 months after stroke. Depression has a negative effect on functional status and cognitive performance.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
5a. Schubert et al. (1992a) * USA.	14 stroke participants (7 male, 7 female). (Also includes 17 amputee patients in separate analysis). Type of stroke not specified.	Longitudinal. Data collected within a week of admission (T1), and at discharge (T2).	Rehabilitation, physical and occupational.	1. Geriatric Depression Scale (GDS) 2. Modified Barthel Index (BI) 3. Length of Stay	Depression is associated with an increased length of stay.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
5b. Schubert et al. (1992b) * USA.	14 stroke participants (7 male, 7 female). (Also includes 17 amputee patients in separate analysis). Stroke type not specified, however location of strokes were: 8 parietal cortex, 4 frontal lobe, 3 occipital 3, 2 temporal lobe, 1 periventricular and 1 lacunar stroke.	Longitudinal. Data collected 11-28 days post stroke (T1) and 14-40 days post stroke (T2).	Rehabilitation.	1. Geriatric Depression Scale (GDS) 2. Modified Barthel Index (BI)	No correlation between depression and functional ability change in the stroke sample.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
6. Schubert et al. (1992c) USA.	21 participants (10 male, 11 female). 6 left hemisphere, 6 right hemisphere, 3 bilateral, 2 left brainstem and 1 subarachnoid haemorrhage.	Longitudinal. Data collected within a week of admission (T1), and at discharge (T2), approximately 4 weeks between T1 and T2.	Rehabilitation.	1. DSM III diagnosis 2. Beck Depression Inventory (BDI) 3. MMSE 4. Modified Barthel Index (BI)	Self reported levels of depression are associated with decreased physical functioning.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
7. Morris et al. (1993) Australia & USA.	94 participants (45 male, 39 female). 61 infarct, 13 haemorrhagic strokes. 40 right hemisphere, 37 left hemisphere, 7 brainstem/bilateral location.	Longitudinal. Data collected at 2 months post stroke (T1) and 15 months post stroke (T2)	Rehabilitation.	1. Demographics, medical info, social classes, comorbidities. 2. Karnofsky Scale – functional performance status 3. Abbreviated Barthel Index 4.MMSE 5. Composite International Diagnostic Interview (CIDI) 6. DSM-III	Depression is associated with increased mortality. And pre-stroke trait introversion is associated with increased mortality.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
8. Loong et al (1995) Singapore.	52 participants (29 male, 23 female). 33 infarcts, 15 haemorrhagic strokes. 31 right hemisphere and 12 left hemisphere strokes.	Longitudinal. Data collected at 1 week post stroke (T1) and at discharge (T2) – however, length of time between T1 & T2 is not specified and could vary for each participant.	Rehabilitation.	1. MMSE 2. Clinical Psychiatric Interview for major depression using DSM-III-R 3. Hamilton Rating for Depression 4. Barthel Index (BI)	Depression might not have a clear negative impact on rehabilitation. Depression on admission and improved mood at the end of rehab, was a good predictor of physical impairment outcome.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
9. Simonsick et al. (1995) USA.	Patient number not given. Gender not specified. Type of stroke not specified.	Longitudinal. Baseline (T1), 3 years (T2), 6 years (T3).	Rehabilitation.	1. Centre For Epidemiologic Studies Depression Scale (CES D) 2. Blood Pressure 3.London School of Hygiene Questionnaire (to find the presence of angina) 4. Lifestyle questions (smoking, drinking, physical activity, BMI) 5. Mortality records.	There was an increased risk of stroke in those with diagnosed hypertension and high levels of depression, especially in women. Those with poor BP control had a higher rate of stroke after 3 years.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
10. Elmstahl et al. (1996) Sweden.	66 participants (25 male, 41 female). 32 right hemisphere, 28 left hemisphere and 16 right cerebral lesions, 21 left lesions and 9 bilateral lesions.	Longitudinal. Data collected at baseline (T1), 1 year post stroke (T2), 3 years (T3).	Rehabilitation.	1. Katz Index of ADL – functional capacity 2. Activity Index – mental capacity, ADL functions & motor activity. 3. Eysenck Personality Inventory 4. Comprehensive Psychopathological Rating Scale (CPRS) – aggressiveness & depressed mood.	Active coping is associated with better ADL function at 1 & 3 year follow up. And active coping is associated with increased Activity Index in Multiple Regression Analysis. Extrovert personality is associated with increased Activity Index at 1 year follow up.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
				5. LGC – life satisfaction & life quality 6. Locus of Control 7. Coping strategies	

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
11. Chang & Mackenzie (1998) China.	152 participants (85 male, 67 female). 70 right sided CVA, 51 left sided CVA, 3 brain stem, 1 cerebellar stroke, 20 unclassified, 124 ischemic, 18 haemorrhagic.	Longitudinal. Data collected at baseline (T1), 2 weeks post stroke (T3), 3 months post stroke (T3).	Rehabilitation.	1. Demographic variables (age, marital status, education, religion, comorbidity, length of stay). 2. State Self-Esteem Scale 3. Rosenberg Self-Esteem Scale – Trait self esteem. 4. Social Support Questionnaire. 5. Modified Barthel Index (BI)	State self esteem is associated with functional ability after 3 months.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
12. Herrmann et al.(1998) Canada.	436 participants (51% men, 51% women reported). 388 infarct, 62 haemorrhage, 219 left hemisphere, 221 right hemisphere and 10 bilateral.	Longitudinal. Data collected at 3 months post stroke (T1) and 12 months post stroke (T2).	Rehabilitation.	1. Demographics & Medical history & tests 2. Neuropsychological battery, inc. MMSE HSS – cognitive assessment 3. Montgomery & Asberg Depression Rating Scale (MADRS) 4. SDS – Depression scale 5. Functional Independence Measure (FIM)	Depression is correlated with outcome.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
				6. Modified Rankin Scale (mRS)	

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
13a. Johnston et al. (1999) ^ Scotland.	71 participants (36 male, 35 female). Type of stroke not specified.	Longitudinal predictive study. Data collected within 3 weeks of stroke (T1), 1 month post stroke (T2), 6 months post stroke (T3)	Rehabilitation.	1. Recovery Locus of Control Scale (RLOC) – Perceived control 2. Exercise coping. 3. Hospital Anxiety & Depression Scale (HADS) 4. Barthel Index (BI) 5. Observer Assessed Disability 6. Clifton Assessment Procedures For The Elderly (CAPE) –	Depression & anxiety were not predictors. Perceived control at 1 month predicted Observer Assessed Measure & BI at 6 months.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
				Information & Orientation section for cognitive impairment. 7. Mental Status Questionnaire (MSQ) 8. Neurological Index – Neurological impairment.	

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
13b. Johnston et al. (2004) ^ Scotland.	101 participants (52 male, 49 female). 41 Left lesions.	Longitudinal predictive study. Data collected 10-20 days post stroke (T1), 1 month post stroke (T2), 6 months post stroke (T3), 1 year post stroke (T4), 3 years post stroke (T5).	Rehabilitation.	1. Demographic factors 2. Clinical measures. 3. Orgogozo Index – Neurological impairment 4. Barthel Index (BI) 5. Clifton Assessment Procedures For The Elderly (CAPE) – Information & Orientation section for cognitive impairment. 6. Mental Status Questionnaire (MSQ)	Depression & anxiety did not predict recovery. 6 month perceived control predicts independence at 3 years.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
				7. Recovery Locus of Control Scale (RLOC) – Perceived control 8. Exercise coping. 9. Hospital Anxiety & Depression Scale (HADS) 10. Engagement in Exercise 11. Satisfaction with Treatment and Advice 12. Confidence in Recovery	

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
14. van de Weg et al. (1999) Netherlands.	85 participants (42 male and 43 female). Stroke type not specified however, 50 left hemispheres reported.	Multicentre cohort study. Depressed group compared to non depressed group. Data collected at 3-6 weeks post stroke (T1) and 6 months (T2).	Rehabilitation.	1. Stroke diagnosed by a neurologist. 2. Geriatric Depression Scale (GDS) 3. DSM III R Criteria for Depression 4. Functional Independence Measure (FIM) 5. Rehabilitation Activities Profile (RAP)	Improvement in functional outcome, not related to presence of partner, sex, side of hemiparesis. RAP & FIM scores lower for depressed patients. No relationship between age / sex / presence of partner & depression. Patients with depression have significantly lower functional scores at onset & after 6 months.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
15. Chemerinski et al. (2001) USA.	171 participants. 2 groups were recruited from 2 hospitals, with 43% men from Maryland Hospital, and 63% men from Methodist Hospital. Ischemic & haemorrhage stroke included, but numbers not specified.	Longitudinal. Depression measured at baseline (T1) and depression & ADL measured at *either* 3 or 6 months (T2).	Rehabilitation.	1. Present State Examination (PSE) – modified semi-structured interview, used with DSM-IV to come to a conclusion about major or minor depression. 2.Hamilton Depression Scale 3. John Hopkins Functional Inventory (JHFI) – Functional physical impairment 4. MMSE – cognitive functioning	Patients with lower depression scores had better ADL scores.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
16. Lai et al (2002) USA.	459 participants (214 male, 245 female). 430 ischemic, 29 haemorrhagic.	Longitudinal. Data collected at 1 month post stroke (T1), 3 months post stroke (T2), and 6 months post stroke (T3).	Rehabilitation.	1. Demographics 2. Geriatric Depression Scale 3. SF36 (Physical Functioning Index) 4. Orpington Prognostic Scale – stroke severity 5. Barthel Index (BI) 6. Lawson IADL – Instrumental Activities of Daily Living 7. Charlson Comorbidity	Depression is associated with slower achievement of BADL & IADL.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
				Index	
17. Cassidy et al. (2004) Ireland.	50 participants (29 male, 21 female). 23 right hemisphere stroke left hemiparesis, 27 left hemisphere stroke right hemiparesis, 33 aphasic participants.	Longitudinal, prospective. Data collected at Baseline (T1) and at 2 months (T2).	Rehabilitation.	1. DSM IV Criteria for Depression 2. Hamilton Depression Rating Scale 3. Centre For Epidemiologic Studies Depression Scale (CES D) 4. Rankin Scale (RS) 5. Barthel Index (BI) 6. Mini-Mental State Examination (MMSE)	Major depression is common after stroke. Females have a higher risk of depression. Depression (in this sample) not related to functional disability following stroke or early functional outcome following rehabilitation. Post stroke disability (before rehabilitation) is predictive of functional outcome 2 months of rehabilitation.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
				7. Length of Stay 8.Effectiveness of Rehab	

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
18. Nannetti et al. (2005) Italy.	121 participants (post stroke depression group: 48.9% male, and non post stroke depression group 47% male). Stroke type not specified.	Longitudinal. Data collected 2 weeks post stroke (T1), 3-4 weeks post stroke (T2) and 3 months post stroke (T3).	Intensive rehabilitation and those with depression were treated with oral antidepressants.	1. Pfeiffer Test – Serious cognitive impairment 2. Goodglass & Kaplan Scale – Aphasia & speech problems 3. Cumulative Illness Rating Scale (CIRS) – Comorbidity 4. Geriatric Depression Scale 5. DSM-IV Criteria 6. Cornell Scale – Patients behaviour	Post stroke depression does not influence functional and motor recovery in the first 3 months.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
				7. Barthel Index (BI) 8. Fugl-Meyer Assessment Scale – Motor recovery	

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
19. Saxena et al. (2007) Singapore.	200 participants (55% male). 122 infarct, 19 hem, 69 left hemisphere lesion, 64 right hemisphere stroke.	Longitudinal. Data collected at admission (T1) and 6 months post stroke (T2).	Rehabilitation	1. Abbreviated Mental Test (AMT) – cognitive impairment. 2. Geriatric Depression Scale (GDS) 3. Barthel Index (BI) 4. National Institute of Health Stroke Scale (NIHSS) 5. Sociodemographic factors (age, gender, ethnicity, marital status & educational level).	Depression at T1 was associated with functional dependence. Depression at 6 months was not associated with functional dependence. Level of physical functioning at 6 months post stroke was associated with baseline level of cognitive impairment & cognitive improvement over 6 months.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
20. Bilge et al. (2008) Turkey.	40 (27 male, 13 female). 31 ischemic stroke and 9 haemorrhagic stroke.	Longitudinal. Data collected at 2 weeks post stroke (T1), 1 month post stroke (T2), 3 months post stroke (T3), 6 months post stroke (T4).	Rehabilitation & antidepressant use (Citalopram).	1. DSM-IV criteria for depression 2. Hamilton Depression Scale 3. MMSE 4. Barthel Index (BI) 5. Scandinavian Stroke Scale (SSS) 6. Rankin Scale	A decrease in post stroke depression is associated with an increase in functional recovery.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
21. Bos et al. (2008) Sweden.	4394 participants (40% men, 60% women). 190 ischemic strokes, 31 haemorrhagic strokes, 70 unspecified stroke.	Population based cohort study. Data collected at baseline 1997-1999 (T1), and follow up completed by 1 Jan 2005 (T2).	Rehabilitation.	1. Centre For Epidemiologic Studies Depression Scale (CES D) 2. Present State Examination – psychiatric interview, DSM-IV criteria used. 3. Blood pressure. 4. MMSE – Cognitive performance 5. Comorbidities 6.Reported strokes	Depressive symptoms are a strong risk factor for stroke in men, but not in women.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
22a. Ostir et al. (2008) UK & USA.	823 participants (51.5% female). Ischemic stroke (74.6%), haemorrhagic stroke (15.4%), other stroke (10%), with left hemiparesis (42.5%), right hemiparesis (39.5%), bilaterial paresis (2.9%) or no paresis (15.1%).	Longitudinal. Data collected within 72 hours of discharge (T1) and 3 months post stroke (T2).	Rehabilitation.	1. Socio-demographic variables 2. Health related measures 3. Four positive questions from the Centre For Epidemiologic Studies Depression Scale (CES D) – Positive emotion 4. Inpatient Rehabilitation Facilities-Patient Assessment Instrument (IRF-PAI) (the functional status	Discharge positive emotion is significantly associated with follow up Total FIM score, 3 months later.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
				items are from the Functional Independence Measure (FIM)	
23. Hamzet & Peters (2009) Nigeria.	20 participants (9 male, 7 female). 14 haemorrhagic, 2 ischemic, 2 left hemiplegia, the remaining 14 not commented on.	Longitudinal. Data collected at Baseline (T1), 1 month post stroke (T2), 2 months post stroke (T3), 3 months post stroke (T4), 4 months post stroke (T5), 5 months post stroke (T6) & 6 months post stroke (T7).	Anti hypertensive drugs. In patient rehabilitation not mentioned.	1. Demographics (age, gender, stroke type). 2. Modified Motor Assessment Scale (MMAS) – motor function. 3. World Health Organizations QoLBREF (WHOQoL BREF)	Only psychological and environmental domains of the WhoQoLBREF measure, showed a correlation with motor function at 1 month.

Studies that share the same population: * Schubert et al. (1992a & 1992b); ^ Johnston et al. (1999 & 2004)

2.6 Preliminary Methodological Quality Assessment

A preliminary methodological quality assessment of all the review papers was carried out to assess the quality of the review papers. This was achieved by creating a system to score the quality of each of the review papers. This assessment consisted of 10 items for the review of the effect of psychological factors on the risk of and recovery from stroke (Sample characteristics which includes age, gender, ethnicity; Stroke diagnosis; Stroke types; Length of study; Time points; Measurement of psychological variables; Measurement of physical recovery variables; Method of analysis; Missing data and Sample size & power). Please see Table 2.3 for more details on the items considered for the methodological assessment and the scoring criteria.

2.6.1 Scoring

Studies were given a rating of 'Good' (2 points), 'Intermediate' (1 point) and 'Poor' (0 points), for each item. This system was devised by the Researcher. Scores were summed to produce an overall score. Studies that scored 20-15 points were graded as 'Good', those that scored 14-8 were graded as 'Intermediate' and studies that were graded 7-0 were graded as 'Poor'. 10 studies were considered to be 'Good', 15 were considered to be 'Intermediate' and none of the studies were deemed to be 'Poor', therefore there were no further exclusions at this stage. Please see Table 2.4 for the methodological quality assessment scores.

After these papers have passed the methodological assessment they are discussed at length in the Results section (Section 2.7).

Table 2.3:
Criteria for Assessment of Methodological Quality of Review Papers

METHODOLOGICAL COMPONENT	RATING		
	Good	**Intermediate**	**Poor**
Sample Characteristics (Age, Gender, Ethnicity)	Clear information on age, gender and ethnicity.	Partial information on age, gender and ethnicity.	No information on age, gender and ethnicity.
Stroke Diagnosis	Clear definitions of diagnosis, using WHO, ICD codes, CT scans or confirmation from a Neurologist.	Other definitions of stroke diagnosis, or stroke diagnosis confirmed but not divulged.	Definition not given.
Stroke Types	Clear reporting of stroke types, e.g., Ischemic and Haemorrhagic and how many recorded.	Stroke types mentioned but number of types not reported or partial information on stroke.	Stroke types not mentioned.
Length of Study	Length of study over 1 year.	Length of study under 1 year.	N/A
Time Points	Over 2 time points reported.	2 time points reported.	N/A. The Exclusion Criteria states studies must be longitudinal and clear time points must be reported.
Measurement of Psychological Variables	Clear reporting of Psychological measures used.	Some aspects of Psychological measures used, were unclear.	N/A. The Inclusion Criteria states studies must include Psychological measures.
Measurement of Physical Recovery Variables	Clear reporting of physical recovery measures used.	Some aspects of the physical recovery measures were unclear.	N/A. The Inclusion Criteria states studies must include physical recovery measures.
Method of Analysis	Clear and specific statistical analysis.	Satisfactory basic analysis, or somewhat incomplete analysis.	Unclear analysis or absent analysis.
Missing Data	The studies recruited consecutive participants and report no missing data or all missing data is explained.	The studies recruited non-consecutive participants or missing data was not explained.	The studies did not recruit consecutive participants and missing data was not explained.
Sample Size & Power	Sample size was clear and/or power for analysis was reported.	Sample size was potentially adequate but no power calculation was reported.	Sample size is small coupled with no power calculation.

Table 2.4:
Methodological Assessment of the Review Papers

STUDY	Sample Characteristics (Age, Gender, Ethnicity)	Stroke Diagnosis	Stroke Types	Length of Study	Time Points	Measurement of Psychological Variables	Measurement of Outcome Variables	Method of Analysis	Missing Data	Sample Size & Power	Overall Methodological Rating
					GOOD						
1. Parikh et al. (1990)	Good	Good	Good	Good	Inter	Good	Good	Good	Inter	Inter	17
2. Elmstahl et al. (1996)	Inter	Good	Good	Good	Good	Good	Good	Good	Inter	Inter	17
3. Herrmann et al. (1998)	Good	Good	Good	Inter	Inter	Good	Good	Good	Good	Inter	17
4. Lai et al. (2002)	Good	Good	Good	Inter	Good	Good	Good	Good	Inter	Inter	17
5. Saxana et al. (2007)	Good	Good	Good	Inter	Inter	Good	Good	Good	Good	Inter	17
6. Morris et al. (1990)	Inter	Good	Good	Good	Inter	Good	Good	Good	Inter	Inter	16
7. Bos et al. (2008)	Inter	Good	Good	Good	Inter	Good	Good	Good	Poor	Inter	15
8. Morris et al. (1992)	Good	Good	Good	Inter	Inter	Good	Good	Inter	Good	Poor	15
9. Morris et al. (1993)	Inter	Poor	Good	Good	Inter	Good	Good	Good	Good	Inter	15
10. Nannetti et al. (2005)	Inter	Good	Poor	Inter	Good	Good	Good	Good	Good	Inter	15

STUDY	Sample Characteristics (Age, Gender, Ethnicity)	Stroke Diagnosis	Stroke Types	Length of Study	Time Points	Measurement of Psychological Variables	Measurement of Outcome Variables	Method of Analysis	Missing Data	Sample Size & Power	Overall Methodological Rating
INTERMEDIATE											
11. Chemerinski et al. (2001)	Good	Good	Inter	Inter	Inter	Good	Good	Good	Poor	Inter	14
12. Cassidy et al. (2004)	Inter	Good	Good	Inter	Inter	Good	Good	Good	Inter	Poor	14
13. Johnston et al. (2004) ^	Inter	Poor	Inter	Good	Good	Good	Good	Good	Inter	Inter	14
14. Ostir et al. (2008)	Good	Good	Good	Inter	Inter	Inter	Inter	Good	Inter	Inter	14
15. Hamzet et al. (2009)	Good	Poor	Good	Inter	Good	Good	Good	Good	Inter	Poor	14
16. Bilge et al. (2008)	Inter	Poor	Good	Inter	Good	Good	Good	Inter	Good	Poor	13
17. House et al. (1990)	Poor	Good	Poor	Good	Good	Good	Good	Poor	Inter	Inter	12
18. Chang et al. (1998)	Inter	Poor	Good	Inter	Inter	Good	Good	Good	Poor	Inter	12
19. Johnston et al. (1999) ^	Inter	Poor	Poor	Inter	Good	Good	Good	Good	Inter	Inter	12

STUDY	Sample Characteristics (Age, Gender, Ethnicity)	Stroke Diagnosis	Stroke Types	Length of Study	Time Points	Measurement of Psychological Variables	Measurement of Outcome Variables	Method of Analysis	Missing Data	Sample Size & Power	Overall Methodological Rating
20. Van de Weg et al. (1999)	Inter	Good	Poor	Inter	Inter	Good	Good	Inter	Inter	Inter	12
21. Schubert et al. (1992b) *	Good	Poor	Good	Inter	Inter	Good	Good	Inter	Poor	Poor	11
22. Loong et al. (1995)	Good	Poor	Good	Inter	Inter	Good	Good	Inter	Poor	Poor	11
23. Schubert et al. (1992c)	Inter	Good	Inter	Inter	Inter	Good	Good	Inter	Poor	Poor	11
24. Simonsick et al. (1995)	Inter	Poor	Poor	Good	Good	Good	Good	Inter	Poor	Poor	10
25. Schubert et al (1992a) *	Good	Poor	Poor	Inter	Inter	Good	Good	Inter	Poor	Poor	9
POOR											
N/A											

Studies that share the same population: * Schubert et al. (1992a & 1992b); ^ Johnston et al. (1999 & 2004)

2.7 Results

The results detail country of investigation, setting of the research, consecutive vs. non-consecutive patients, sample size, power calculation, number of measurements, length of follow up, demographic information, stroke diagnosis, type and severity, psychological and outcome measures, method of analysis and attrition rates.

2.7.1 Country of Investigation

The studies conducted in this systematic review are geographically global, with research originating from the UK (House et al., 1990; Johnston et al., 1999; Johnston et al., 2004; Cassidy et al., 2004 & Ostir et al., 2008), Sweden (Elmstahl et al., 1996 & Bos et al., 2008), Netherlands (van de Weg et al., 1999), Italy (Nannetti et al., 2005), the USA (Parikh et al., 1990; Morris et al., 1992; Schubert et al., 1992a; 1992b; 1992c; Simonsick et al., 1995; Chemerinski et al., 2001 & Lai et al., 2002), Canada (Herrmann et al., 1998), Australia (Morris et al., 1990 & Morris et al., 1993), Singapore (Loong et al., 1995 & Saxena et al., 2007), China (Chang et al., 1998), Turkey (Bilge et al., 2008) and Nigeria (Hamzet et al., 2009).

2.7.2 Setting

The settings of the studies are important to note as this can have an impact on the quality of the data collected. All studies first time point measurement was taken in the hospital (however, the Bos et al., 2008 study has not clearly specified where their setting for the first time point measurement was). Three studies only measured time spent in hospital as the time span of the studies were from admission to discharge (Schubert et al., 1992a, 1992b, 1992c & Loong et al., 1995), three studies also detailed the inclusion of measuring data in nursing homes (Morris et al., 1992; Herrmann et al., 1998 & Saxena et al., 2007) and other settings for data collection included interview by telephone follow up (Parikh et al., 1990 & Ostir et al., 2008) and neurology outpatient clinic (Parikh et al., 1990). However most studies did not specify the setting of follow up appointments (House et al., 1990; Morris et al., 1993; Simonsick et al., 1995; Chang et al., 1998; Johnston et al., 1999; van de Weg et al., 1999; Chemerinski et al., 2001; Lai et al., 2002; Cassidy et al., 2004; Johnston et al., 2004; Nannetti et al., 2005; Bos et al., 2008; Bilge et al., 2008 & Hamzet et al., 2009).

2.7.3 Consecutive vs. Non-Consecutive Patients

Consecutive patients were recruited by 11 of the review studies (Parikh et al., 1990; Morris et al., 1990; Morris et al., 1992; Morris et al., 1993; Herrmann et al., 1998; van de Weg et al., 1999; Cassidy et al., 2004; Nannetti et al., 2005; Saxena et al., 2007; Bilget et al., 2008 & Hamzet et al., 2009), whilst 12 studies recruited non-consecutive patients (House et al., 1990; Schubert et al., 1992a; 1992b; 1992c; Loong et al., 1995; Simonsick et al., 1995; Elmstahl et al., 1996; Chang et al., 1998; Johnston et al., 1999; Chemerinski et al., 2001; Lai et al., 2002; Johnston et al., 2004; Bos et al., 2008 & Ostir et al., 2008).

2.7.4 Sample Size

Sample sizes ranged from not reported to large. No sample size was reported by 1 study (Simonsick et al., 1995). The remaining papers all reported the sample size recruited: 14 participants (Schubert et al., 1992a; 1992b); 20 participants (Hamzet et al., 2009); 21 participants (Schubert et al., 1992c); 40 participants (Bilge et al., 2008); 49 participants (Morris et al., 1992); 50 participants (Cassidy et al., 2004); 52 participants (Loong et al., 1995); 63 participants (Parikh et al.,1990); 66 participants (Elmstahl et al., 1996); 71 participants (Johnston et al., 1999); 85 participants (van de Weg et al., 1999); 94 participants (Morris et al., 1993); 101 participants (Johnston et al., 2004); 104 participants (Morris et al., 1990); 121 participants (Nannetti et al., 2005); 128 participants (House et al., 1990); 152 participants (Chang et al., 1998); 171 participants (Chemerinski et al., 2001); 200 participants (Saxena et al., 2007); 436 participants (Herrmann et al., 1998); 459 participants (Lai et al., 2002); 823 participants (Ostir et al., 2008) and 4394 participants (Bos et al., 2008).

2.7.5 Power Calculation

No power calculations were reported from any of the review papers.

2.7.6 Number of Measurements

Longitudinal study designs have more than one measurement throughout the data collection period. The minimum requirement is therefore to have two time points recording psychological data, which the majority of the studies have satiated (Parikh et al., 1990; Morris et al., 1990; Morris et al., 1992; Schubert et al 1992a; 1992b; 1992c; Morris et al., 1993; Loong et al., 1995; Herrmann et al., 1998; Chang et al., 1998; van

de Weg et al., 1999; Chemerinski et al., 2001; Cassidy et al., 2004; Saxana et al., 2007; Bos et al., 2008 and Ostir et al., 2008). Review papers that went above the minimum of two time points included those with 3 time points (Simonsick et al., 1995; Chang et al., 1998; Elmstahl et al., 1996; Johnston et al., 1999; Lai et al., 2002 & Nannetti et al., 2005), studies with 4 time points (House et al., 1990 & Bilge et al., 2008), studies with 5 time points (Johnston et al., 2004) and 7 time points (Hamzet et al., 2009).

2.7.7 Length of Follow Up

As all of these studies are longitudinal in nature it is important to report on the length of follow up of the review studies. The timeframes for length of follow up varies across all of the studies. Some studies do not report clearly the length of the follow up, which is important if stroke recovery is to be assessed properly. House et al., (1990) report unclear time frames. They conducted 4 time points, with Time 3 combining 1 month and 6 months post stroke measurements and the Time 4 timeframe is not specified. Three other studies have reported unclear timeframes, with the only information being admission and discharge (Schubert et al., 1992a; 1992c; & Loong et al., 1995). Bos et al., 2008 have also reported time differences between time points in an unclear manner, with Time 1 at baseline (1997-1999) and Time 2 completed by 1 Jan 2005. From this is it unclear if the measurements for participants are uniformed or not. Hamzet et al., (2009) have not specified when their Time 1 baseline measurements was taken but have detailed the rest of their study timeframe (Time 2 taken at 1 month post stroke, Time 3 was taken at 2 months post stroke, Time 4 was taken at 3 months post stroke, Time 5 was taken at 4 months post stroke, Time 6 was taken at 5 months post stroke and Time 7 was taken at 6 months post stroke). Chemerinski et al., (2001) also failed to report when their Time 1 baseline measurement was from but provided information on the later time points (Time 2 was measured at either 3 months or 6 months). Elmstahl et al., (1996) have failed to report when their Time 1 baseline measurement was taken but provides information on the other time points (Time 2 was taken at 1 year post stroke and Time 3 was taken at 3 years post stroke). Simonsick et al., (1995) did not specify when their Time 1 baseline measurement was but provided information on the rest of the timeframe (Time 2 was taken at 3 years post stroke and Time 3 was taken at 6 years post stroke).

The remaining studies all have different lengths of reported duration; Time1: within 72 hours of discharge and Time2: 3 months post stroke (Ostir et al., 2008);

Time 1: at admission and Time 2: 6 months post stroke (Saxana et al., 2007); Time 1: baseline between 10-20 days post stroke, Time 2: 1 month post stroke, Time 3: 6 months post stroke, Time 4: 1 year post stroke and Time 5: 3 years post stroke (Johnston et al., 2004); Time 1: between 11-28 days post stroke and Time 2: between 25-68 days post stroke (Schubert et al., 1992b); Time 1: 2 weeks post stroke, Time 2: 3-4 weeks post stroke and Time 3: 3 months post stroke (Nannetti et al., 2005); Time 1: 2 weeks post stroke and Time 2: 2 months post stroke (Cassidy et al., 2004); Time 1: 2 weeks post stroke and Time 2: 3 months post stroke (Chang et al., 1998); Time 1: 2 weeks post stroke, Time 2: 1 month post stroke, Time 3: 3 months post stroke and Time 4: 6 months post stroke (Bilge et al., 2008); Time 1: 2 weeks post stroke and Time 2: 2 years post stroke (Parikh et al., 1990); Time 1: within 3 weeks of stroke, Time 2: 1 month post stroke and Time 3: 6 months post stroke (Johnston et al., 1999); Time 1: 3-6 weeks post stroke and Time 2: 6 months post stroke (van de Weg et al., 1999); Time 1: 1 month post stroke; Time 2: 3 months post stroke and Time 3: 6 months post stroke (Lai et al., 2002); Time 1: 2 months post stroke; Time 2: 16 months post stroke (Morris et al., 1992) and Time 1: 3 months post stroke and Time 2: 12 months post stroke (Herrmann et al., 1998).

2.7.8 Demographic Information

Demographic factors are important to report in order to make an assessment of a representative sample. Age, gender and ethnicity have been reported by 9 of the review papers (Parikh et al., 1990; Schubert et al., 1992a; 1992b; Loong et al., 1995; Herrmann et al., 1998; Chemerinski et al et al., 2001; Lai et al., 2002; Saxena et al., 2007; Ostir et al., 2008 & Hamzet et al., 2009). Age and gender have been reported by 8 of the review papers (Morris et al., 1990; Schubert et al., 1992c; Elmstahl et al., 1996; Cassidy et al., 2004; Chang et al., 1998; Johnston et al., 1999; van de Weg., 1999; Johnston et al., 2004 & Bos et al., 2008). Gender was only reported by 3 papers (Morris et al., 1992; Morris et al. 1993 & Bilge et al., 2008), whilst age was only reported by 2 papers (Simonsick et al., 1995 & Nannetti et al., 2005). However, House et al., 1990, reported no sample characteristics.

Demographic information on age varied. The youngest age included was 39 years of age and the oldest age included was 96 years of age. Age ranges were: 39-90 years old (Morris et al., 1990), 40-79 years old (Schubert et al., 1992a), 40-96 years old (Saxena et al., 2007), 47-72 years old (Schubert et al., 1992c), mean age of 51.4

years of age +/- 1.2 years (Cassidy et al., 2004), 55-74 years old (Ostir et al., 2008), late 50's to early 60's (Parikh et al., 1990), mean age of 61.5 years of age +/- 13.2 years (Chemerinski et al., 2001), mean age of 59.75 years of age (Loong et al., 1995), mean age of 60.68 years of age +/- 9.78 years (Hamzet et al., 2009), mean age of 61.4 years of age (van de Weg et al., 1999) 65+ years (Simonsick et al., 1995), mean age of 68.92 years of age in male participants and mean age of 73.80 years of age in female participants (Johnston et al., 2004), mean age of 69.44 years of age (Chang et al., 1998), mean age of 69.4 years of age (Johnston et al., 1999), mean age of 70 years of age +/- 11.4 years (Lai et al., 2002), modal age of 70-74 years old (Schubert et al., 1992b), mean age of 71.6 years old in a depressed group and 72.4 years of age in a non-depressed group (Nannetti et al., 2005), median age of 71.9 years old (Bos et al., 2008), mean age of 74.9 years of age +/- 11.6 years (Herrmann et al., 1998) and mean age of 75.6 years of age +/- 7.4 years for male participants and 81.1 years of age +/- 8.3 years of age for female participants (Elmstahl et al., 1996).

Ethnicity of the reported studies included Caucasian (n= *9*, Schubert et al., 1992a; 199b; n=*360*, Lai et al., 2002; *93%*, Herrmann et al., 1998; *96%*, Chemerinski et al., 2001 & *100%*, Morris et al., 1992), Chinese (*88.5%*, Loong et al., 1995 & *89%*, Saxena et al., 2007), non-Hispanic White (*79.2%*, Ostir et al., 2008), African American (*"slightly over half" of the 63 patients*, Parikh et al., 1990; n=*5*, Schubert et al., 1992a; 1992b; n= *78*, Lai et al., 2002 & *66%*, Chemerinski et al., 2001), African Nigerian (n= *16*, Hamzet et al., 2009), Malay (*3.8%*, Loong et al., 1995 & *8%*, Saxena et al., 2007), Indian (*3%*, Saxena et al., 2007 & *5.8%*, Loong et al., 1995) and "Other" (*1.9%*, Loong et al., 1995).

Gender reported was male (n= *7*, Schubert et al., 1992a; n= *9*, Hamzet et al., 2009; n= *10*, Schubert et al., 1992c; n= *21*, Cassidy et al., 2004; n= *25*, Elmstahl et al., 1996; n= *27*, Bilge et al., 2008; n= *29*, Loong et al., 1995; n= *33*, Morris et al., 1992; n= *34*, Morris et al., 1990; n= *36*, Johnston et al., 1999; n= *42*, van de Weg et al., 1999; n= *45*, Morris et al., 1993; n= *52*, Johnston et al., 2004; n= *85*, Chang et al., 1998; *40%*, Bos et al., 2008; *43%*, Chemerinski et al., 2001 & *51%*, Herrmann et al., 1998) and female (n= *7*, Schubert et al., 1992a; n= *7*, Hamzet et al., 2009; n= *11*, Schubert et al., 1992c; n= *13*, Bilge et al., 2008; n= *16*, Morris et al., 1992; n= *21*, Cassidy et al., 2004; n= *22*, Morris et al., 1990, n= *23*, Loong et al., 1995; n= *35*, Johnston et al., 1999; n= *39*, Morris et al., 1993; n= *41*, Elmstahl et al., 1996; n= *43*, van de Weg et al., 1999; n= *49*, Johnston et al., 2004; n= *67*, Chang et al., 1998; n=

245, Lai et al., 2002; *51%*, Herrmann et al., 1998; *51.5%*, Ostir et al., 2008; *55%*, Saxena et al., 2007 & *60%*, Bos et al., 2008). However, Simonsick et al., (1995) failed to report on gender.

2.7.9 Stroke Diagnosis

The stroke diagnosis is crucial to stroke studies because of the possibility of misdiagnosis with mimics (where the patient presents as a stroke but it is a different condition e.g., epilepsy or another condition) or TIA's (which are treated the same as a stroke and are sometimes included in analysis). Clear definitions of diagnosis using WHO, ICD codes, or confirmation from a Neurologist were utilised in the majority of the studies with 15 papers reporting a clear diagnosis **(**Parikh et al., 1990; Morris et al., 1990; Morris et al., 1992; Schubert et al.,1992c; Elmstahl et al., 1996; Herrmann et al., 1998; van de Weg et al., 1999; Chemerinski et al., 2001; Lai et al., 2002; Cassidy et al., 2004; Nannetti et al., 2005; Saxana et al., 2007; Ostir et al., 2008; Bos et al., 2008 & House et al., 1990). Definitions of stroke diagnosis not addressed by the research papers were evident in 8 of the studies (Schubert et al., 1992a; 1992b; Morris et al., 1993; Loong et al., 1995; Simonsick et al., 1995; Chang et al., 1998; Johnston et al., 1999; Johnston et al., 2004; Bilge et al., 2008 & Hamzet et al., 2009).

2.7.10 Stroke Type

As these studies are investigating stroke recovery it is important to assess how stroke type has been recorded. There is variability in how stroke has been recorded in the review studies from reporting on both ischemic and haemorrhagic strokes (Parikh et al., 1990; Chemerinski et al., 2001; Lai et al., 2002; Bilge et al., 2008 & Bos et al., 2008), only reporting on hemisphere or location of stroke (Schubert et al., 1992b; 1992c; Elmstahl et al., 1996; van de Weg et al., 1999; Johnston et al., 2004 & Cassidy et al., 2004), reporting both stroke type with location (Morris et al., 1990; Morris et al., 1992; Morris et al., 1993; Loong et al., 1995; Chang et al., 1998; Herrmann et al., 1998; Ostir et al., 2008 & Hamzet et al., 2009) and not specifying any stroke information at all (House et al., 1990; Schubert et al., 1992a; Simonsick et al., 1995; Johnston et al., 1999; Nannetti et al., 2005 & Saxena et al., 2007).

2.7.11 Stroke Severity

Stroke can cause difficulties in data collection so it is important to consider the stroke severity of the review studies which will also impact on the heterogeneity or homogeneity of the sample. Most studies however have not reported on stroke severity at all (Parikh et al., 1990; Morris et al 1990; House et al., 1990; Morris et al., 1992; Schubert et al., 1992a; 1992b; 1992c; Morris et al 1993.,Loong et al., 1995; Simonsick et al., 1995; Emstahl et al 1996; Herrmann et al., 1998; Chang et al., 1998; Johnston et al 1999; van de Weg et al., 1999; Chemerinski et al., 2001; Cassidy et al., 2004; Johnston 2004; Nannetti et al., 2005; Bos et al., 2008; Ostir et al., 2008; Bilge et al., 2008 & Hamzet et al., 2009). Only 1 study has acknowledged stroke severity as being classified as mild, moderate and severe (Lai et al., 2002). An additional study classified stroke as mild, moderate and severe, where mild and moderate were mixed together (Saxena et al., 2007).

2.7.12 Psychological Measures

Different psychological measures were used to assess psychological factors. These were Bedford College Life Events & Difficulties Schedule (LEDS) (House et al., 1990), Composite International Diagnostic Interview (CIDI) (psychiatric exam with DSM III criteria), (Morris et al., 1990, Morris et al., 1992 & Morris et al., 1993), Present State Examination (PSE) with DSM-III criteria (Parikh et al., 1990), with DSM-IV criteria (Chemerinski et al. 2001 & Bos et al. 2008), the DSM III diagnosis (Schubert et al., 1992c; Loong et al 1995 & van de Weg., 1999), the DSM IV diagnosis (Cassidy et al. 2004; Nannetti et al. 2005 & Bilge et al. 2008), Montgomery & Asberg Depression Rating Scale (MADRS) (Morris et al., 1990 & Herrmann et al., 1998), Hamilton Depression Rating Scale (Parikh et al 1990; Loong et al 1995; Chemerinski et al. 2001; Cassidy et al. 2004 & Bilge et al. 2008), Beck Depression Inventory (BDI) (Schubert et al., 1992c), Geriatric Depression Scale (Schubert et al., 1992a; 1992b; van de Weg 1999; Lai et al., 2002; Nannetti et al. 2005 & Saxena et al. 2007), Centre For Epidemiologic Studies Depression Scale (CES D) (Simonsick et al., 1995; Cassidy et al. 2004 & Bos et al. 2008), 4 positive questions from the Centre For Epidemiologic Studies Depression Scale (CES D) (Ostir et al., 2008), Hospital Anxiety & Depression Scale (HADS) (Johnston et al., 1999; 2004), Zung Self Rating Depression Scale (Parikh et al 1990; Herrmann et al., 1998), Social Functioning Examination (which measures quality of social relationships), Social Ties Checklist

(which measures number of social connections) (Parikh et al 1990), General Health Questionnaire (Morris et al., 1992), Eysenck Personality Inventory, Comprehensive Psychopathological Rating Scale (CPRS) (which measures aggressiveness & depressed mood), Life Quality Gerontological Centre scale (LGC) (which measures life satisfaction & life quality), Locus of Control, Coping strategies (Elmstahl et al., 1996), State Self-Esteem Scale, Rosenberg Self-Esteem Scale (Trait self esteem), Social Support Questionnaire (Chang et al., 1998), Recovery Locus of Control Scale (RLOC) (which measures perceived control) (Johnston et al 1999; 2004), Satisfaction with Treatment and Advice, and Confidence in recovery (Johnston et al., 2004) and the World Health Organizations Quality of Life Bref (WHOQoL BREF) (Hamzet et al., 2009).

2.7.13 Outcome Measures

The physical outcome measures varied from mortality statistics to disability measures to length of stay in hospitals. The physical outcome measures used were: Mortality records (House et al., 1990 & Simonsick et al. 1995), reported strokes (Bos et al. 2008), Abbreviated Barthel Index (BI) (Morris et al., 1990; Morris et al., 1992 & Morris et al., 1993), Modified Barthel Index (BI) (Schubert et al., 1992a; 1992b; 1992c & Chang et al., 1998), Barthel Index (Loong et al., 1995; Johnston et al., 1999; Lai et al., 2002; Johnston et al., 2004; Cassidy et al., 2004; Nannetti et al., 2005; Saxena et al., 2007 & Bilge et al., 2008), John Hopkins Functioning Inventory (JHFI) (Parikh et al., 1990 & Chemerinski et al., 2001), Karnofsky Performance Rating Scale (which measures functional status) (Morris et al., 1992 & Morris et al., 1993), Katz Index of ADL (which measures functional capacity), Activity Index (which measures mental capacity, activities of daily living functions & motor activity) (Elmstahl et al. 1996), Functional Independence Measure (FIM) (Herrmann et al., 1998 & van de Weg et al., 1999), Inpatient Rehabilitation Facilities-Patient Assessment Instrument (IRF-PAI) (which uses the functional status items from the Functional Independence Measure (FIM) (Ostir et al., 2008), Fugl-Meyer Assessment Scale (which measures motor recovery) (Nannetti et al., 2005), Modified Motor Assessment Scale (MMAS) (which measures motor function) (Hamzet et al., 2009), Orpington Prognostic Score (which measures stroke severity) (Lai et al., 2002), Modified Rankin Scale (mRS) (Herrmann et al., 1998), Rankin Scale (RS) (Cassidy et al., 2004 & Bilge et al., 2008), Observer Assessed Disability (Johnston et al., 1999), Rehabilitation Activities Profile (RAP)

(van de Weg., 1999), SF36 (Physical Functioning Index), Lawson IADL (Instrumental Activities of Daily Living) (Lai et al., 2002), National Institute of Health Stroke Scale (NIHSS) (Saxena et al., 2007), Scandinavian Stroke Scale (SSS) (Bilge et al., 2008) and length of stay in hospital (Schubert et al., 1992a & Cassidy et al., 2004).

2.7.14 Method of Analysis

The review studies have utilised different methods of analysis. These were, percentages (Simonsick et al., 1995), t-test and chi square (Morris et al., 1992 & Bilge et al., 2008), chi square (Schubert et al., 1992c), chi square and Spearman's correlation (Schubert et al., 1992a; 1992b), chi square and ANOVA (Herrmann et al., 1998), unpaired t-test, chi square and cross-tabulation (Loong et al., 1995), t-test, ANOVA and chi-squared (Morris et al., 1990 & Chemerinski et al., 2001), t-test and Fishers exact test (van de Weg et al., 1999), Spearman's correlation, Friedman's ANOVA and Wilcoxon Signed Rank Test (Hamzet et al 2009), t-tests, repeated measures ANOVA, chi square, factor analysis and multiple regression (Parikh et al., 1990), Mann-Whitney U Test, Spearman's correlation and stepwise multiple regression (Elmstahl et al., 1996), ANOVA and logistic regression (Morris et al., 1993), chi square, t-test, ANOVA and logistic multiple regression (Nannetti et al., 2005), Pearsons correlation and hierarchical multiple regression (Johnston et al., 2004), linear stepwise multiple regression (Chang et al., 1998), linear multiple regression (Johnston et al., 1999; Cassidy et al., 2004 & Ostir et al., 2008), logistic regression and multiple linear regression (Saxena et al., 2007), Cox proportional hazards model (Bos et al., 2008), time dependent Cox proportional hazards model (Lai et al 2002) and no statistical analysis was mentioned by House et al., 1990.

2.7.15 Attrition

Six studies failed to report on attrition at all (Parikh et al., 1990; Schubert et al., 1992a; 1992b; 1992c; Simonsick et al., 1995; Chang et al., 1998 & Chemerinski et al., 2001). One study reported detailed attrition from the sequential time points (Johnston et al., 1999; 2004). At Time 2 of the 1999 sample, attrition was reported for 13 deaths, 4 patients had cognitive impairment and 6 refused follow up; At Time 3 attrition reported was 2 deaths, 2 patients had cognitive impairment and 3 refused follow up (Johnston et al., 1999). Attrition for the 2004 study were Time 2 attrition reported 11 deaths, Time 3 attrition reported 16 deaths, Time 4 attrition reported 20 deaths, Time 5

attrition reported 42 deaths and a further 12 participants were lost due to being unable to participate because of poor health. One was cognitively impaired, 4 declined and 2 were lost to follow up. However it is not stated at which time points these further losses had occurred (Johnston et al., 2004).

Eight studies reported on loss of patients just once even though they had more than one time point recorded. Reasons for loss of attrition for this group were refusals (n= *1*, Hamzet et al., 2009; n= *3*, Nannetti et al., 2005; n= *4*, Elmstahl et al., 1996; n= *37* & Lai et al., 2002), unable to participate due to poor health (n= *1*, Nannetti et al., 2005 & n= *2*, Elmstahl et al., 1996), geographical relocations (n= *1*, Elmstahl et al., 1996; n= *1*, Hamzet et al., 2009 & n= *13*, Lai et al., 2002), deteriorated cognitive function (n= *1*, Hamzet et al., 2009), dementia (n= *2*, House et al., 1990), lost to follow up (n= *4*, Bilge et al., 2008) and mortality (n= *1* Hamzet et al., 2009; n= *2*, House et al., 1990; n= *2*, Bilge et al., 2008 & n= *32*, Lai et al., 2002).

The remaining 11 studies all had two time point data measurements. The reported reasons for attrition of these studies were because of refusals (n= *15*, Saxena et al., 2007; n= *24*, Morris et al., 1990; n= *44*, Ostir et al., 2008; n= *1045*, Bos et al., 2008 & *14%*, Herrmann et al., 1998), unable to participate due to poor health (n= *140*, Bos et al., 2008 & *15%*, Herrmann et al., 1998), geographical relocation (n= *2*, Morris et al., 1993; n= *3*, Morris et al., 1990 & *12%*, Herrmann et al., 1998), cognitive impairment (n= *3*, Morris et al., 1992 & *7%*, Herrmann et al., 1998), too aphasic (*5%*, Herrmann et al., 1998), excluded because of major depression (n= *1*, Cassidy et al., 2004), lost to follow up (n= *1*, Loong et al., 1995; n= *1*, van de Weg 1999; n= *2*, Morris et al., 1992; n= *8*, Morris et al., 1990; n= *8*, Morris et al., 1993; n= *26*, Ostir et al., 2008 & n= *35*, Saxena et al., 2007), missing data in questionnaires (n= *3*, Loong et al., 1995), recurrent stroke (n= *7*, Morris et al., 1992), death (n= *1*, van de Weg 1999; n= *8*, Morris et al., 1990; n= *9*, Saxena et al., 2007; n= *29*, Ostir et al., 2008 & n= *140*, Herrmann et al., 1998) and excluded but no explanation given as to why (n= *3*, Cassidy et al., 2004).

2.7.16 Findings

The conclusions these review papers came to about the relationship between psychology and recovery have mainly shown that post-stroke depression has a negative impact on functional recovery after a stroke, as reported by 12 studies (Parikh et al., 1990; Morris et al., 1992; Schubert et al., 1992a; Schubert et al., 1992c; Loong et

al., 1995; Simonsick et al., 1995; Herrmann et al., 1998; van de Weg., 1999; Chemerinski et al., 2001; Lai et al., 2002; Saxena et al., 2007 & Bilge et al., 2008), and post-stroke depression and pre-stroke trait introversion was associated with increased mortality (Morris et al., 1993).

However, 5 studies have disagreed and have reported there is no association between post-stroke depression and functional status (Morris et al., 1990; Schubert et al., 1992b; Johnston et al., 1999; 2004; Cassidy et al., 2004 & Nannetti et al., 2005) along with anxiety not being significant (Johnston et al., 1999; 2004). Pre-stroke depression (Bos et al., 2008) and pre-stroke severely life threatening events have also been reported as risk factors (House et al., 1990).

Positive emotion was measured by selecting 4 questions from the CES-D and was found to be significantly associated with functional status (Ostir et al. 2008), along with active coping, extrovert personality (Elmstahl et al., 1996), state self esteem (Chang et al., 1998), perceived control (Johnston et al., 1999; 2004) and the psychological and environmental domains of the WhoQoLBREF measure (Hamzet et al., 2009).

The results of this review will be further interpreted in the next section.

2.8 Discussion

This systematic review had specific exclusion criteria. Carers, spouses, health professionals and any other proxy measures used within the data gathering phase were excluded. This is because proxy measures are used extensively in stroke research (Pohjasvaara et al., 2001; Pohjasvaara et al., 2002; Desrosiers et al., 2002; Desrosiers et al., 2006; Wilz, 2007) because sufferers of stroke can experience problems with dysphasia (language impairment), dysphagia (swallowing problems) and dysarthria (problems with the muscles that help one to speak resulting in slurred speech) (Barnett, Mohr, Stein, & Yatsu, 1998) which can make communication difficult. These communicative problems in the stroke aftermath can lead researchers to search for proxy measurements however, these measurements may be inaccurate due to biasing from the proxy respondent. This may lead to inaccurate research results and brings into question the quality of the research, as proxy respondents will inevitably answer with their own opinion. Proxy ratings may be used to prevent exclusion of this data (Sneeuw et al., 1997) but research into stroke and psychology should not rely heavily on proxy ratings.

It may be more encouraging to the viability of stroke research to investigate different methods in extracting responses from participants with communicative impairment for example, devising touch screen technology and with participants with physical impairment, using Dragon voice activated software which would minimise the researcher/proxy-participant interaction and reduce bias.

TIA's were also excluded as TIA's differ from strokes as the disturbance of blood flow to the brain is temporary and therefore does not result in a lesion to the brain. With strokes permanent lesions occur in the brain causing brain death (Barnett, Mohr, Stein, & Yatsu, 1998). As the recovery effects of TIA's differ from stroke, all TIA studies which are included in stroke analysis are excluded in order to maintain the integrity of stroke data analysed.

A longitudinal study design was chosen because research of this nature can track changes in the same population over time (Bryman, 2008) and therefore the role of psychological variables on physical stroke risk and recovery is better analysed with longitudinal rather than cross-sectional study designs.

The review papers are geographically global with research from five of the seven continents from around the world – Europe, North America, Australia, Asia and Africa. Although research has a standardized procedure there are no global measures in place to ensure that all research is executed at the same standard and different countries may have different standards. Nevertheless, the spread of stroke research globally is a testament to the importance of this illness.

All the studies included were quantitative in method and analysis. This allows for comparisons between studies to take place. It is interesting to note that during the searching phase of this review not many qualitative papers were discovered, which may lead to a viable avenue to investigate.

It is salient to discuss the issue of bias in research to be able to acknowledge the weaknesses that are present in research designs. Reliability is important to consider because it is concerned with the repeatability of the study and the consistency of the test used to measure a concept or the consistency of different observer ratings. Internal reliability measures items on a scale to see if they are consistent. This is normally measured with a Cronbach's alpha statistic. Inter-observer consistency is tested when observer rated measures are tested because of the possible lack of consistency in different opinions. This is normally measured with a Kappa statistic (Bryman, 2008).

Biases are important to acknowledge as biases can affect the quality of research studies. Selection bias can be present with the identification and recruitment of the study population. Recruitment can cause bias if the recruitment procedure is not uniformed. Additionally bias is less likely to occur if the outcome is unknown at the time as in prospective studies compared to retrospective studies, where the outcome is already known. Interviewer bias is also important to acknowledge. This is concerned with differences in how information is interpreted. Bias can be present as the researcher knows which disease is being investigated and is therefore more attuned to information that fits in with risk factors and related variables. Recall bias may also play a role (Pannuci & Wilkens, 2010) as a participant has been diagnosed with an illness their recall about the events leading up to the diagnosis may be altered e.g., once diagnosed with a stroke a participant may report higher levels of stress or depression when asked how they have been feeling before the stroke occurred. Confounding factors are important to acknowledge in any research study. This is where a factor which is not measured affects the outcome. The best way to handle the effects of unknown factors producing confounding affects is to have true randomisation in a large sample (Pannuci & Wilkens, 2010). Social desirability bias can also occur and result in distorted data from participants due to them giving socially desirable answers to the researcher, which may be more common in face-to-face interaction rather than questionnaire completion or telephone interviewing (Bowling, 1997). Also a modified White Coat Effect (WCE) can occur. The WCE is when blood pressure readings are taken by a doctor or a nurse and by the very nature of taking the reading the patient can have an increase in blood pressure in reaction to the test (Saladini, Benetti, Malipiero, Casiglia, & Palatini, 2012; Garcia-Donaire et al., 2012). Consequently if a participant is in a hospital and being questioned on negative affect and so on, they may respond more to these questions due to being in hospital. Attrition bias is also important to acknowledge as this is concerned with the drop-out rate from the study which can lead to a biased outcome regarding the topic under investigation (Jüni & Egger, 2005) for example, healthy people may remain in the study thereby biasing the results causing a cohort bias.

It is difficult to know to what extent these biases are in the review papers as most of these biases take place in the data collection period. A preliminary methodological quality assessment was undertaken for the review papers. Some aspects of the study may score well and some aspects may score poorly however, the

final rating would combine these to score an 'Intermediate' rating. No studies were classed as 'Poor'.

The settings of the studies included hospitals, the home environment, nursing homes and telephone interviews. All studies first time point measurement was taken in the hospital (however, the Bos et al., 2008 study has not clearly specified where their setting for the first time point measurement was). In hospitals there may be some of the White Coat Effect and the fear of treatment bias (where participants may feel if they do not take part in the research it may affect their treatment). In face to face interviews at home and telephone interviews participants may demonstrate social desirability bias, they may change their answers to be more positive if family members are present or conversely they may be more honest in their responses as they are comfortable in their familiar environment. Some follow up settings were not divulged (House et al., 1990; Morris et al., 1993; Simonsick et al., 1995; Chang et al., 1998; Johnston et al., 1999; van de Weg et al., 1999; Chemerinski et al., 2001; Lai et al., 2002; Cassidy et al., 2004; Johnston et al., 2004; Nannetti et al., 2005; Bos et al., 2008; Bilge et al., 2008 & Hamzet et al., 2009). This information is important to disclose because without knowing this information the study is not repeatable and therefore not following the scientific procedure of the research process and also the reader may assume bias is present.

Consecutive patients are favoured over non-consecutive patients because consecutive patients lead to less biasing in the recruitment phase as participants are recruited in the order they are admitted to hospital. For non-consecutive patients the researcher has chosen them which will lead to a selection bias. Therefore 12 of the studies could be open to bias (House et al., 1990; Schubert et al., 1992a; 1992b; 1992c; Loong et al., 1995; Simonsick et al., 1995; Elmstahl et al., 1996; Chang et al., 1998; Johnston et al., 1999; Chemerinski et al., 2001; Lai et al., 2002; Johnston et al., 2004; Bos et al., 2008 & Ostir et al., 2008).

None of the review papers stipulated any power calculations therefore it is difficult to conclude the statistical viability of the research as insufficient power may lead to Type II errors. Power calculations determine the minimum sample size needed to reach statistical power. If n is less than 30, then we cannot assume a normal distribution (Levine, Stephan, Krehbiel, & Berenson, 2011) however, three studies have recruited less than 30 participants: 14 participants (Schubert et al., 1992a; 1992b); 20 participants (Hamzet et al., 2009); 21 participants (Schubert et al., 1992c), but they

have all used non-parametric tests which is correct, as these tests are for analysis with a non-normal distribution. Four studies are just over the threshold of $n > 30$: 40 participants (Bilge et al., 2008); 49 participants (Morris et al., 1992); 50 participants (Cassidy et al., 2004); 52 participants (Loong et al., 1995). These studies use both parametric and non-parametric tests which may cause some statistical problems as these studies are just over the threshold of $n > 30$, therefore the parametric tests may not be generalizable.

Five studies recruited a reasonable number of participants: 63 participants (Parikh et al.,1990), 66 participants (Elmstahl et al., 1996), 71 participants (Johnston et al., 1999), 85 participants (van de Weg et al., 1999), 94 participants (Morris et al., 1993), but a power calculation would be needed to verify if enough participants have been recruited to achieve statistical power. Six studies recruited a respectable number of participants: 101 participants (Johnston et al., 2004); 104 participants (Morris et al., 1990); 121 participants (Nannetti et al., 2005); 128 participants (House et al., 1990); 152 participants (Chang et al., 1998); 171 participants (Chemerinski et al., 2001) and four studies have recruited a valuable number of participants: 200 participants (Saxena et al., 2007); 436 participants (Herrmann et al., 1998); 459 participants (Lai et al., 2002); 823 participants (Ostir et al., 2008) and 4394 participants (Bos et al., 2008). This latter group should have reached statistical power. No sample size was reported by 1 study (Simonsick et al., 1995) this is atypical and should not be duplicated by future researchers.

The study design chosen was the longitudinal study design therefore at least 2 time point measurements are needed which 15 of the studies adhered to (Parikh et al., 1990; Morris et al., 1990; Morris et al., 1992; Schubert et al 1992a; 1992b; 1992c; Morris et al., 1993; Loong et al., 1995; Herrmann et al., 1998; Chang et al., 1998; van de Weg et al., 1999; Chemerinski et al., 2001; Cassidy et al., 2004; Saxana et al., 2007; Bos et al., 2008 & Ostir et al., 2008). Review papers that went above the minimum of two time points included those with 3 time points (Simonsick et al., 1995; Chang et al., 1998; Elmstahl et al., 1996; Johnston et al., 1999; Lai et al., 2002 & Nannetti et al., 2005), studies with 4 time points (House et al., 1990 & Bilge et al., 2008), studies with 5 time points (Johnston et al., 2004) and 7 time points (Hamzet et al., 2009). Exceeding two time point measurements are valuable for research as it is useful in tracking recovery and it also offers more statistical interpretation options.

Stroke recovery can change over time. This is better acknowledged with repeated measures over time to determine any changes in psychological and physical factors. The length of follow up is important as stroke presentation combined with stroke severity will determine stroke recovery. A longer follow up period is more beneficial to concluding any related factors compared with a shorter follow up period. This is useful as the effects of stroke at different time points can be assessed but also because of this, direct comparisons between studies cannot be done. In 9 of these studies there is not enough information on the *length* of timeframes (House et al., 1990; Schubert et al., 1992a; 1992c; & Loong et al., 1995; Simonsick et al., 1995; Elmstahl et al., 1996; Chemerinski et al., 2001; Bos et al., 2008; Hamzet et al., 2009). Consequently recovery really cannot be assessed effectively. House et al., (1990) in their study combined 1 month and 6 month post stroke measurements into their Time 3 measurement. This is a major flaw in the design of the study as 1 month post stroke recovery is still in the acute phase of illness and should not be amalgamated with 6 month post stroke recovery. This infers House et al., (1990) conducted a study with loose guidelines which also will impact on the statistical analysis and conclusions of the study. Their Time 4 measurement is not expressed, which is a concern as the focus of this study is about recovery from stroke and it additionally means this study is not repeatable. The Time 4 measurement may again be mixed due to the Time 3 measurement being mixed but no information is given on this.

Three studies have only disclosed admission and discharge from hospital as the timeframes (Schubert et al., 1992a; 1992c; & Loong et al., 1995), however the length of admission and discharge for patients can vary depending on the severity of stroke and in all these cases stroke severity is not specified, so again it is difficult to trust the conclusions of these studies.

Bos et al., (2008) have also reported time differences between time points in an unclear manner with Time 1 at baseline (1997-1999) and Time 2 completed by 1 Jan 2005. From the reporting from these Authors it is impossible to know without investigating the raw data the length of follow up for participants and therefore if they should be grouped together in time related analysis or not. Again stroke severity is not specified in this study so it is difficult to conclude the rate of stroke recovery and the changes in psychological responses.

Five papers failed to specify when the Time 1 baseline measurement was taken but provided information on the remaining time points (Simonsick et al., 1995;

Elmstahl et al., 1996; Chemerinski et al., 2001 & Hamzet et al., 2009). Stroke severity was again not specified so it is impossible to know if recruitment was able to be administered swiftly or if a time delay had to be adhered to due to recovery from stroke severity. Ostir et al., (2008) stated their baseline measurement as "within 72 hours of discharge" (p. 3), however the length of hospital stay was not divulged, consequently the reader does not know how long the patient stayed in hospital. Again stroke severity was not reported and therefore no assumptions can be made. It is difficult to ascertain how long the patients were admitted to hospital without investigating the raw data.

Demographic factors are important to report in order to make an assessment of a representative sample. Age, gender and ethnicity have been reported by 9 of the review papers (Parikh et al., 1990; Schubert et al., 1992a; 1992b; Loong et al., 1995; Herrmann et al., 1998; Chemerinski et al et al., 2001; Lai et al., 2002; Saxena et al., 2007; Ostir et al., 2008 & Hamzet et al., 2009). Age and gender have also been reported by 8 of the review papers (Morris et al., 1990; Schubert et al., 1992c; Elmstahl et al., 1996; Cassidy et al., 2004; Chang et al., 1998; Johnston et al., 1999; van de Weg., 1999; Johnston et al., 2004 & Bos et al., 2008). Gender was only reported by 3 papers (Morris et al., 1992; Morris et al. 1993 & Bilge et al., 2008), whilst age was only reported by 2 papers (Simonsick et al., 1995 & Nannetti et al., 2005). However House et al., 1990, reported no sample characteristics. This lack of information means this study cannot be generalizable and it is impossible to identify demographic themes from these studies as they are not offered. For the papers only reporting one or two demographic components it is difficult to assess whether the sample is generalizable.

The biggest spread of ages, were from 39-96 years of age, with the most common ages seeming to be in the 60's and 70's. One study did not give precise details of the ages but instead reported "late 50's to early 60's" (Parikh et al., 1990, p. 786) which should not be acceptable in research literature. Parikh et al., (1990) also reported that "slightly over half were black" (p. 786) in terms of reporting ethnicity. Again this is not giving the reader precise information on numbers which also raises the possibility of not trusting this research to report adequately other information, e.g., statistical data. There is a good spread of ethnic backgrounds when reporting ethnicity when all the studies are considered together with Caucasian (Schubert et al., 1992a; 199b; Lai et al., 2002; Herrmann et al., 1998; Chemerinski et al., 2001 & Morris et al.,

1992), Chinese (Loong et al., 1995 & Saxena et al., 2007), non-Hispanic Whites (Ostir et al., 2008), African Americans (Parikh et al., 1990; Schubert et al., 1992a; 1992b; Lai et al., 2002 & Chemerinski et al., 2001), African Nigerians (Hamzet et al., 2009), Malaysians (Loong et al., 1995 & Saxena et al., 2007) and Indians (Saxena et al., 2007 & Loong et al., 1995) being included. In one study there was an "Other" category (Loong et al., 1995) however the details of this category were not disclosed.

According to the Stroke Association (2013) the age group most vulnerable to stroke in the UK is 75 years old plus, followed by 65-74 years of age, and the rate of stroke decreases by each ten year age group and 66% of strokes in 2009 were experienced by people over the age of 65 in the U.S (Hall, Levant, & DeFrances, 2012).

McGruder, Malarcher, Antoine, Greenlund, & Croft, (2004) found a trend in racial/ethnic differences in stroke prevalence in the US, the rates of which were almost 1.5 times higher in African Americans compared to Whites or Hispanics. However, no data were available on the timing and type of stroke raising concerns about possible selection bias (Feigin & Rodgers, 2004) although it has been reported by the DoH (2005) that African Caribbean people are twice as likely to have a stroke compared to Caucasian people.

Gender was reported by all the review papers except for House et al., (1990), with nearly an equal spread between males and females. The Stroke Association (2013) report that in 2010 in the UK more women suffered a stroke compared to men (30,079 in women compared with 19,287 in men), however a 2008 Canadian study by Reid et al., concluded that the majority of gender differences in stroke were explained by confounding and more research should be conducted in this area.

Research studies in stroke should make certain the diagnosis for stroke they are using is reported as stroke can be misdiagnosed as TIA's, mimics and other misdiagnoses. Fifteen of the review papers reported good stroke diagnosis including, WHO definitions, ICD codes, confirmation from a Neurologist and CT scans (Parikh et al., 1990; Morris et al., 1990; Morris et al., 1992; Schubert et al.,1992c; Elmstahl et al., 1996; Herrmann et al., 1998; van de Weg et al., 1999; Chemerinski et al., 2001; Lai et al., 2002; Cassidy et al., 2004; Nannetti et al., 2005; Saxana et al., 2007; Ostir et al., 2008; Bos et al., 2008 & House et al., 1990). Eleven of the studies failed to report how stroke was diagnosed. Some of these papers were older in which case research standards may have been different (Schubert et al., 1992a; 1992b; Morris et al., 1993;

Loong et al., 1995; Simonsick et al., 1995; Chang et al., 1998; Johnston et al., 1999; Johnston et al., 2004; Bilge et al.,) but 2 of these papers were published in recent years (Bilge et al., 2008 & Hamzet et al. 2009), which is a concern as to why basic information is not deemed important to collect and divulge.

Stroke type is important to record because of the differences in the stroke itself, it can gives clues as to the causes of the stroke and it can be related to stroke severity. Ischemic strokes are more common than haemorrhagic strokes but haemorrhagic strokes are more often fatal (Stroke Association, 2013). Ischemic strokes are caused by a blockage in an artery that leads to the brain which can be the result of an unhealthy lifestyle such as poor diet, smoking, lack of exercise and drinking alcohol. Haemorrhagic strokes are caused by a ruptured vessel or artery in the brain from high blood pressure which causes pressure on the vessel walls. The risk of this type of stroke is often difficult to determine as opposed to the ischemic stroke which has more measureable risk factors (Barnett, Mohr, Stein, & Yatsu, 1998). It is useful to report the type of stroke as this can be compared with other factors such as demographic factors, ethnicity, age, risk factors and psychosocial variables, such as stress. There has not been full reporting of stroke type in the review studies for example, 6 studies have not reported on stroke type at all (House et al., 1990; Schubert et al., 1992a; Simonsick et al., 1995; Johnston et al., 1999; Nannetti et al., 2005 & Saxena et al., 2007), which should be highlighted as studies that claim to investigate stroke should include which strokes actually occurred.

Six studies have ignored stroke type and only reported on hemisphere or location of lesion (Schubert et al., 1992b; 1992c; Elmstahl et al., 1996; van de Weg et al., 1999; Johnston et al., 2004 & Cassidy et al., 2004). The remaining studies all reported on stroke type, with ischemic stroke indeed outnumbering haemorrhagic stroke (refer to Table 2.2 for details).

In research studies that are investigating recovery from stroke it is imperative to record stroke severity. Only including mild strokes will not yield fruitful data on the landscape of stroke and will produce a homogenous sample. Moderate and severe strokes should be recorded in order to create a heterogeneous sample which can facilitate obtaining a generalizable sample. Also stroke severity will undoubtedly have an effect on stroke recovery due to hemiplegia (paralysis of one side of the body) or hemiparesis (weakening of one side of the body), which will impact on stroke recovery. Stroke severity may also impact upon psychological wellbeing. As stroke is

an illness which can kill and cause long term disability it is imperative this information is available when considering studies that claim to investigate stroke recovery. Stroke also affects cognitive functioning and can cause dysphasia (language impairment) and aphasia (total loss of language). Due to these effects researchers often exclude this group which causes a cohort bias.

Most of these review papers have not commented on stroke severity (Parikh et al., 1990; Morris et al 1990; House et al., 1990; Morris et al., 1992; Schubert et al., 1992a; 1992b; 1992c; Morris et al 1993.,Loong et al., 1995; Simonsick et al., 1995; Emstahl et al 1996; Herrmann et al., 1998; Chang et al., 1998; Johnston et al 1999; van de Weg et al., 1999; Chemerinski et al., 2001; Cassidy et al., 2004; Johnston 2004; Nannetti et al., 2005; Bos et al., 2008; Ostir et al., 2008; Bilge et al., 2008 & Hamzet et al., 2009) which results in these researchers being restricted in being able to conclude on physical recovery from stroke. Physical outcome measures may be of some help in this regard but this has not been commented on in the review papers. The severity of the strokes of these investigations were not disclosed so it is difficult to conclude how big a role psychological variables can play in influencing recovery from stroke as it is not known how vast the gap between damage and recovery is. For example, a patient with mild cognitive, psychological or physical impairment, may encourage positive results in experiments but realistically the gap between damage and recovery may have been small. Conversely, a patient with severe cognitive, psychological and physical problems may make a noticeable change in these areas but on a grand scale they would appear not to have made a big change. And so in order for any conclusions to be drawn about the impact of psychological and cognitive factors in stroke recovery firstly, stroke severity must be reported on. The patients included must have some level of cognitive abilities in order to take part in the study in the first place and because of this, this may explain why they may recover due to this selective recruitment. Patients that have lower cognitive abilities may not recover well from stroke but they are unable to take part in the studies. This is an unfortunate gap in the literature but due to the nature of stroke studies this is a normal consequence of these types of experiments.

Saxena et al., (2007) amalgamated mild and moderate strokes together however, this is a major flaw in the research design as moderate strokes are obviously more physically and cognitively advanced compared to mild strokes and should not be added together in any statistical analysis. Only 1 study have acknowledged stroke severity as being classified as mild, moderate and severe (Lai et al., 2002).

There is no one standardised measure for psychological constructs. It is acceptable to have different measures but they should demonstrate good psychometric properties. Also measures can be used simply because they are popular but that does not mean they are necessarily good measures to employ. It is important to be critical when choosing measures.

A host of different measures were utilised and the measures may have been employed at different time points in the participant's rehabilitation. It is important to note the reliability of the measures used which can aid in assessing the contribution of the studies.

To measure depression 11 scales were utilised all of which have been reported to have good reliability: The Composite International Diagnostic Interview (CIDI) (which is a psychiatric exam with DSM III criteria), (used by Morris et al., 1990, Morris et al., 1992 & Morris et al., 1993) includes three measures – the Generalised Anxiety Disorder (GAD), Panic Disorder and Major Depression measurements. They used Kappa values to test inter-rater reliability (k > 0:94), test-retest reliability (k > 0.41 – k > 0.84) and validity (k > 0.77), which shows good psychometric properties (Andrews & Peters 1998) although the range shown for test-retest reliability includes a low range. The Geriatric Depression Scale (used by Schubert et al., 1992a; 1992b; van de Weg 1999; Lai et al., 2002; Nannetti et al. 2005 & Saxena et al. 2007) has a reported Cronbach's alpha coefficient of 0.92 (Ertan, Ertan, Kızıltan, & Uygucgil, 2005). The Centre For Epidemiologic Studies Depression Scale (CES D) (used by Simonsick et al., 1995; Cassidy et al. 2004 & Bos et al. 2008) has a reported Cronbach's alpha of 0.88 (Thombs, Hudson, Schieir, Taillefer, & Baron, 2008). The Hamilton Depression Rating Scale (used by Parikh et al 1990; Loong et al 1995; Chemerinski et al. 2001; Cassidy et al. 2004 & Bilge et al. 2008) has been reported to have an Cronbach's alpha value of 0.81 (Trajković et al., 2011). The Diagnostic and Statistical Manual of Mental Disorders (DSM) provides a standard classification for the assessment of mental disorders. The stipulated review papers used the DSM to assess depression. The DSM is an internationally respected criteria to use. The DSM III (used by Schubert et al., 1992c; Loong et al 1995 & van de Weg., 1999) has been reported to have Kappa values ranging between 0.40 – 0.86 (Segal, Hersen, & Van Hasselt, 1994). The DSM IV diagnosis (used by Cassidy et al., 2004; Nannetti et al., 2005 & Bilge et al., 2008) has been reported to have Kappa values of 0.29 – 0.81 (Mahoney, 1998). The Present State Examination (with DSM-III criteria) (used by

Parikh et al., 1990) and with DSM-IV criteria (used by Chemerinski et al., 2001 & Bos et al., 2008) is a semi-standardised interview, the name of which has been changed to Schedules for Clinical Assessment in Neuropsychiatry (SCAN) (Wing et al., 1990). The Kappa values reported for reliability for this measure has been reported as 0.48 to 1.0 (McGuffin, Katz, & Aldrich, 1986). The Beck Depression Inventory (BDI) (used by Schubert et al., 1992c) has a reported Cronbach's alpha of 0.88 for internal consistency (Visser, Leentjens, Marinus, Stiggelbout, & van Hilten, 2006). The Hospital Anxiety & Depression Scale (HADS) (used by Johnston et al., 1999; 2004) has been reported to have high internal consistency with a Cronbach's alpha coefficient of 0.884 (0.829 for anxiety and 0.840 for depression) (Michopoulos et al., 2008). The Zung Self Rating Depression Scale (used by Parikh et al., 1990 & Herrmann et al., 1998) has a reported Cronbach's alpha of 0.79 (Knight, Waal-Manning, & Spears, 1983). The Montgomery & Asberg Depression Rating Scale (MADRS) (used by Morris et al., 1990 & Herrmann et al., 1998) has had a reported Cronbach's alpha of 0.61 (Hammond, 1998).

Positive emotion was measured by 4 positive questions from the Centre For Epidemiologic Studies Depression Scale (CES D) (used by Ostir et al., 2008) however, information on the psychometric properties of only 4 questions from the CESD were difficult to obtain. Aggressiveness & depressed mood was measured by the Comprehensive Psychopathological Rating Scale (CPRS). This measure had an overall reported Kappa coefficient of 0.72 (mean taken from K0, 0.67, K1, 0.79 & 0.70) (van der Laan, Schimmel, & Heeren, 2005) (used by Elmstahl et al., 1996). Personality was measured by the Eysenck Personality Inventory and had reported Cronbach's alphas of 0.83 (Extrovert), 0.82 (Neuroticism), 0.73 (Lie) and 0.51 (Psychotic) (Goh, King & King 1982) (used by Elmstahl et al., 1996). Self esteem was measured using the State Self-Esteem Scale which has a reported Cronbach's alpha of 0.92 (Heatherton & Polivey, 1991) and the Rosenberg Self-Esteem Scale (Trait self esteem) which has a reported Cronbach's alpha of 0.77 to 0.88 (Rosenberg, 1965) (used by Chang et al., 1998). Stress was measured once and that was with the Bedford College Life Events & Difficulties Schedule (LEDS) (used by House et al., 1990). This is a semi structured interview for assessing life events and long term difficulties in adults. It has high inter rater reliability reported (κ=0.86) (Malkoff-Schwartz et al., 1998). Mental health was measured by using the General Health Questionnaire (used by Morris et al., 1992) and has a reported internal consistency of 0.85 (Chan & Chan, 1983). Quality of Life was

measured by using the World Health Organizations Quality of Life Bref (WHOQoL BREF) which has a reported internal consistency Cronbach's alpha of 0.68 – 0.82 across 4 domains of the measure (Skevington, Lotfy, & O'Connell, 2004) (used by Hamzet et al., 2009). Social Support was measured by the Social Support Questionnaire with a reported Cronbach's alpha for internal reliability as 0.97 (Sarason, Levine, Basham, & Sarason, 1983) (used by Chang et al., 1998). Social support was also measured by using the Social Functioning Examination (to measure the quality of social relationships) and the Social Ties Checklist (to measure the number of social connections) (used by Parikh et al., 1990). The Social Functioning Examination has been reported to have high inter-rater and test-retest reliability, however the values of these tests could not be obtained (Starr, Robinson, & Price, 1983). No information on psychometric properties could be obtained for the Social Ties Checklist.

No psychometric properties were obtained also for the Satisfaction with Treatment and Advice, and Confidence in Recovery measures (Johnston et al., 2004), Life Quality Gerontological Centre scale (LGC) (which measures life satisfaction & life quality), coping strategies (as the precise measure was not fully explained) and Locus of Control. Locus of Control (LoC) was difficult to find data on the psychometric properties because the precise measure of LoC was not stipulated (used by Elmstahl et al., 1996) and psychometric information on Recovery Locus of Control Scale (RLOC) (which measures perceived control) could also not be obtained (Johnston et al 1999; 2004).

Eight of the 28 measures had unobtainable reliability statistics and the remaining measures scored from moderate to high on reliability. It is difficult to conclude if these 8 measures are acceptable in capturing the desired data (Parikh et al 1990; Elmstahl et al., 1996; Johnston et al., 1999; 2004).

The physical outcome measures varied from mortality statistics to disability measures to length of stay in hospitals. These all measure something different about physical recovery. Some measures are not privy to the reliability statistic such as mortality records (used by House et al., 1990 & Simonsick et al. 1995), reported strokes (used by Bos et al. 2008) and length of stay in hospital (used by Schubert et al., 1992a & Cassidy et al., 2004).

One of the most frequent measures used to test physical function is the Barthel Index (BI). The Modified Barthel Index has reported Kappa coefficients of 0.52 – 0.68

(Fricke & Unsworth, 1996) (used by Schubert et al., 1992a; 1992b; 1992c & Chang et al., 1998) whilst the Barthel Index has reported Kappa coefficients of 0.57 – 0.81 (Fricke & Unsworth, 1996) (used by Loong et al., 1995; Johnston et al., 1999; Lai et al., 2002; Johnston et al., 2004; Cassidy et al., 2004; Nannetti et al., 2005; Saxena et al., 2007 & Bilge et al., 2008). Three studies used an Abbreviated Barthel Index but no psychometric data was available on this measure (Morris et al., 1990; Morris et al., 1992 & Morris et al., 1993) so it is unclear if the authors abbreviated the BI from their own volition or if there was another reason for using this measure.

A further thirteen measures have demonstrated good reliability. The Karnofsky Performance Rating Scale which measures functional status has been criticised for its reliability (Hutchinson, Boyd, & Feinstein, 1979; Schag, Heinrich, & Janz, 1984; Orr & Aisner, 1986) but has also been reported to have a Cronbach's alpha of 0.97 (Mor, Laliberte, Morris, & Wieman, 1984) (used by Morris et al., 1992 & Morris et al., 1993). The Katz Index of Activities of Daily Living (ADL) measures functional capacity and has a reported Cronbach's alpha of 0.87 (Ciesla, Shi, Stoskopf, & Samuels, 1993) and 0.94 (Hamrin & Lindmark, 1988) and the Activity Index (which measures mental capacity, ADL functions & motor activity) has a reported Cronbach's alpha of 0.94 (Hamrin & Wohlin, 1982) (used by Elmstahl et al. 1996). The Inpatient Rehabilitation Facilities-Patient Assessment Instrument (IRF-PAI) uses the functional status items from the Functional Independence Measure (FIM) (used by Ostir et al., 2008). The FIM has a reported Cronbach's alpha of 0.88 – 0.91 (Hsueh et al., 2002) (used by Herrmann et al., 1998 & van de Weg et al., 1999). The Fugl-Meyer Assessment Scale assesses motor recovery and has a reported Cronbach's alpha of 0.94 to 0.98 (Lin, Hsueh, Sheu, & Hsieh, 2004) (used by Nannetti et al., 2005). The Orpington Prognostic Score measures stroke severity and has reported Kappa scores of 0.53 – 0.84 for the 4 domains (Weir, Counsell, McDowall, Gunkel, & Dennis, 2003) (used by Lai et al., 2002). The Modified Rankin Scale (mRS) has a reported weighted Kappa of 0.78 – 0.93 (Wilson et al., 2002) (used by Herrmann et al., 1998) and the Rankin Scale (RS) has a weighted Kappa of 0.90 (Quinn, Dawson, Walters, & Lees, 2009) (used by Cassidy et al., 2004 & Bilge et al., 2008). The Rehabilitation Activities Profile (RAP) (used by van de Weg., 1999) has a reported Cronbach's alpha of 0.78 (Verhoef, Toussaint, Putter, Zwetsloot-Schonk, & Vliet Vlieland, 2008). The SF36 (Physical Functioning Index) has a Cronbach's alpha of 0.93 (Ten Klooster, Oude Voshaar, Taal, & van de Laar, 2011) (used by Lai et al., 2002). The National Institute

of Health Stroke Scale (NIHSS) (used by Saxena et al., 2007) has a reported intraclass correlation coefficient of 0.82 (Kasner et al., 1999) and the Scandinavian Stroke Scale (SSS) (used by Bilge et al., 2008) has reported weighted Kappa values ranging from 0.53 – 0.83 (Barber, Fail, Shields, Stott, & Langhorne, 2004).

It was difficult to obtain reliability data on the remaining 3 measures of the John Hopkins Functioning Inventory (JHFI) (used by Parikh et al., 1990 & Chemerinski et al., 2001), the Observer Assessed Disability (used by Johnston et al., 1999) and the Lawson IADL (used by Lai et al., 2002). Therefore these 5 studies may have weaker outcome measures.

2.8.1 Method of Analysis

The review studies have utilised different methods of analysis. These are important to acknowledge and to decipher if the appropriate tests were used. Parametric tests assume the underlying distribution of the sample is normal and have more statistical power. If the data are very skewed or ranked then non-parametric tests should be used but these are not as powerful as parametric tests. Semi-parametric tests encompass both parametric and non-parametric components.

Parametric tests that were used within the review studies were t-test (used by Morris et al., 1990; Parikh et al., 1990; Morris et al., 1992; Loong et al., 1995; van de Weg et al., 1999; Chemerinski et al., 2001; Nannetti et al., 2005; Bilge et al., 2008) which compares the mean scores of participants in the same group or in different groups. ANOVA was used (used by Morris et al., 1990; Parikh et al., 1990; Morris et al., 1993; Herrmann et al., 1998; Chemerinski et al., 2001; Nannetti et al., 2005) which compares the mean scores of more than two groups. Pearsons correlation (used by Johnston et al., 2004) correlates variables together. Multiple regressions are parametric tests but different kinds of regressions have been used. Linear multiple regression (used by Parikh et al., 1990; Johnston et al., 1999; Cassidy et al., 2004; Saxena et al., 2007 & Ostir et al., 2008) is when variables are entered at the same time in the regression model. Hierarchical multiple regression (used by Johnston et al., 2004) are where variables are entered in the order the researchers choose which can follow a theoretical sequence of their choosing. Stepwise multiple regression (used by Elmstahl et al., 1996; Chang et al., 1998) lets statistical software packages choose the order of the variables entered and logistic regression (used by Morris et al., 1993; Nannetti et al., 2005 & Saxena et al., 2007) uses a categorical variable as the dependent variable.

Small samples should not be used with multiple regressions because of the issue of generalizability which is problematic for the Cassidy et al., (2004) study which recruited 50 participants, a low number.

Non-parametric tests assume a non-normal distribution. The non-parametric tests included in the review papers are chi square (Parikh et al., 1990; Morris et al., 1992; Schubert et al., 1992a; 1992b &1992c; Loong et al., 1995; Herrmann et al., 1998; Nannetti et al., 2005 & Bilge et al., 2008) which places participants in categories of a variable or tests whether two categorical variables are related. Cross tabulation (used by Loong et al., 1995 & van de Weg et al., 1999) which assesses the relationship between at least two categorical variables. Spearman's correlation (Schubert et al., 1992a; 1992b; Elmstahl et al., 1996 & Hamzet et al., 2009) correlates variables together. The Friedman's ANOVA (used by Hamzet et al., 2009) measures variables at three different time points or under three different conditions. Fishers exact test (used by van de Weg et al., 1999) can be used when sample sizes are small as it is a test of statistical significance. Mann-Whitney U test (used by Elmstahl et al., 1996) compares the median values between two independent groups on a continuous measure and the Wilcoxon's Signed Rank Test (used by Hamzet et al., 2009) which measures participants twice and categorical variables are compared at Time 1 and Time 2.

Semi-parametric tests are when components of parametric and non-parametric tests are combined and are used in survival analysis. Cox proportional hazards model (used by Bos et al., 2008) is interested in how a risk (hazard) changes over time until an event occurs. Survival analysis is used for 'time to event' data e.g., time to disease recovery and the time dependent Cox proportional hazards model (used by Lai et al., 2002) is interested in survival time (time to death) as the dependent variable. The model can be built up in the usual way as you would for any regression with predictor/explanatory variables. The output is in the form of hazard ratios - which are like odds ratios from logistic regression models. They compare levels of a predictor variable in terms of 'hazards' (risk of dying over time) e.g. active treatment compared to placebo. A normal Cox model has predictor variables that are fixed in time e.g. sex, ethnicity etc. A time dependent model contains variables that change over time.

Other measures used were factor analysis (used by Parikh et al., 1990) which is not a measure to test hypotheses but is a data reduction technique by identifying groups among inter-correlations in a subscale. Basic percentages were used by one

study (Simonsick et al., 1995) and no statistical analysis was mentioned by House et al., 1990.

As far as the quality of statistical methods performed only 1 of the 17 tests performed give some concern. Simonsick et al., (1995) only used percentages as a form of data analysis which is very basic and does not control for chance or errors.

2.8.2 Attrition

It is important to report attrition data to acknowledge if attrition was due to death, cognitive impairments, comorbidities and any other reasons. Refusal to allow follow up visits may be linked to depression, progression of disease or even because of positive recovery (i.e., the participant may feel they have recovered well and have no further need to be a part of a research study). Therefore attrition bias may occur because healthy people may remain in the study thereby biasing the results.

Six studies failed to report on attrition at all (Parikh et al., 1990; Schubert et al., 1992a; 1992b; 1992c; Simonsick et al., 1995; Chang et al., 1998 & Chemerinski et al., 2001) therefore these studies may be at risk of attrition bias. Six studies reported on loss of patients just once, even though they had more than one time point recorded (House et al., 1990; Elmstahl et al., 1996; Lai et al., 2002; Nannetti et al., 2005; Bilge et al., 2008 & Hamzet et al., 2009) which shows these Authors have not fully disclosed their attrition rate at each time point. One study did report detailed attrition from the sequential time points (Johnston et al., 1999; 2004) from attrition caused by mortality, however caused confusion when describing other reasons for attrition (cognitive impairment, refusals and lost to follow up) by disclosing these participants at the end but not explaining where in time they were lost. The remaining 10 studies reported good attrition data (Morris et al., 1990; Morris et al., 1992; Morris et al., 1993; Loong et al., 1995; Herrmann et al., 1998; van de Weg 1999; Cassidy et al., 2004; Saxena et al., 2007; Ostir et al., 2008 & Bos et al., 2008), although Herrmann et al., (1998) switches between actual *n* and percentages. This is unconventional and can distort the reporting of attrition and lead to being unable to see direct comparisons.

Attrition has not been reported fully across all of the review papers, however from summing the scores (without including the percentages from Herrmann et al., 1998) the main reason for attrition is refusal of follow up (1314 participants), followed by death (371 participants), lost to follow up (159), unable to participate due to poor health (152), geographical relocations (22 participants), missing data (22 participants),

cognitive impairment (11 participants), recurrent stroke (7 participants), protocol violations (7 participants), no explanation given why participants were excluded (3 participants), dementia (2 participants) and due to major depression (1 participant). These are crude assumptions however, they do give an idea to the reasons for loss of data with refusal being the main reason. Limited research investigating why participants refuse to take part in studies have been conducted. Elskamp, Hartholt, Patka, van Beeck, & van der Cammen, (2012) conducted a qualitative study to investigate why older people refuse to be a part of follow up in falls prevention trials. They found reasons included participants felt they are too healthy if they were mobile, many participants reported their mobility impairment as the main reason, some reported they spend enough time in hospital and transport problems and cost were also explanations. Participants also reported they knew the reason for their fall and could not see the benefit of being a part of research. It would be beneficial to literature to have more research conducted on this area however, it is realised that this loss to follow up group will be difficult to investigate.

Attrition can lead to a cohort bias leaving patients who are more able to comply with the investigations inclusion criteria which in turn produces significant results. Acknowledging this consequence of stroke research should give researchers greater impetus to describe attrition rates and what effect this has had on their findings and conclusions.

2.8.3 Findings

The results gathered for this review suggest that depression (Parikh et al., 1990; Morris et al., 1992; Schubert et al., 1992a; Schubert et al., 1992c; Morris et al., 1993; Loong et al., 1995; Simonsick et al., 1995; Herrmann et al., 1998; van de Weg., 1999; Chemerinski et al., 2001; Lai et al., 2002; Saxena et al., 2007 & Bilge et al., 2008) and pre-stroke trait introversion (Morris et al. 1993) can negatively influence recovery from stroke. Depression has also been reported as a risk factor (Bos et al., 2008) along with severely life threatening events (House et al., 1990).

Whilst higher scores on positive emotion (Ostir et al. 2008) along with active coping, extrovert personality (Elmstahl et al., 1996), state self esteem (Chang et al., 1998), perceived control (Johnston et al., 1999; 2004) and the psychological and environmental domains of the WhoQoLBREF measure (Hamzet et al., 2009) are associated with less effects on physical recovery.

However 5 studies have disagreed and have reported there is no association between depression and functional status (Morris et al., 1990; Schubert et al., 1992b; Johnston et al., 1999; 2004; Cassidy et al., 2004 & Nannetti et al., 2005) and there is no association between anxiety and functional status (Johnston et al., 1999; 2004).

Some concerns about the review papers have been noted which will affect the strength of their conclusions. Ostir et al., (2008) assessed the effect of positive emotion on functional recovery. They use 4 items of positive emotion from the depression scale, the CESD. There is no Cronbach alpha information on these 4 items and no Cronbach alpha value was offered by the Authors. Four items to use as a predictor variable in regression analysis does seem like a weak measure.

In the statistics section the Authors mention depression, but depression was not listed as a variable in the Method section. If they are using the negative items on the CESD, it is not clear. And the outcome measure is the IRF-PAI, which incorporates items from the FIM measure. But in the results section they do not refer to the outcome as IRF-PAI, but as "Total FIM", which can lead to confusion. Other items of the IRF-PAI are not mentioned. There seems to be many discrepancies in this study.

Hamzet et al., (2009) recruited a low number of participants (16), which is too low to be able to draw any conclusion from and only 2 sub domains of the WhoQoLBREF measure correlated with motor function. The Authors state in their Discussion there was an ischemic dominance in the study, however in the Results section they say that 14 of the strokes were haemorrhagic and this is the majority stroke type. They also state side of stroke is not consistent but 14 out of the 16 strokes were not commented on, only 2 were reported with left hemiplegia. The reporting of strokes in this study seem to be in disarray and cast doubt on the conclusions these Authors make.

Chemerinski et al., (2001) are unclear with their methodology. These Authors state the second follow up is either at 3 OR 6 months post stroke, they do not explain which patients had 3 or 6 months follow up or why there is a difference and how this time difference can affect physical functioning and psychology and why they put this in the same analysis. It appears to the Reader, data collection was conducted loosely and participants at different stages were haphazardly placed in the same time point collection which will reduce the quality of the data gathered.

Cassidy et al., (2004) have committed a major flaw and it is a surprise this study was able to be published. These Authors did not request Ethics Committee

approval not to treat depressed post stroke patients, the Authors too admit this is unethical. This behaviour should not be replicated by future research. One other discrepancy has been noticed: they included a Barthel Index score of >14 prior to stroke in their inclusion criteria. However the patients were admitted after their first stroke and so it is unclear how a Barthel Index score was obtained prior to stroke onset and no information on retrospective data collection is mentioned.

House et al., (1990) have no statistical tests mentioned at all therefore it is difficult to conclude any findings from this study. The time points are unclear and not repeatable and therefore have less scientific viability. The measure for stress they utilised was the Bedford College Life Events & Difficulties Schedule (LEDS) which rates the death of a sibling as not severe. It makes the Reader question if this is a reasonable measure to use as research has shown sibling death to be related to risk of myocardial infarction (Rostila, Saarela, & Kawachi, 2013) and affects socioeconomic outcomes in their surviving sibling (Fletcher, Mailick, & Song, 2013). The time points of their data collection are also unclear with the Time 2 measurement containing 84 participants which were seen after 1 month and 29 participants which were seen after 6 months poststroke. One month poststroke is still in the acute stroke recovery phase and should not be mixed with 6 months post stroke where a range of recovery may have taken place.

Morris et al., (1990) report there may be selection bias in their study as patients are in hospital so they may be more disabled than people who are not in hospital but not as much as those who are severely disabled. This is true of all the research studies. They also acknowledge that diagnosing depression in stroke patients is imprecise although including psychiatric interviews could produce some reliability. They also state major depression is associated with higher mortality, however this may be due to the patients being older and having greater stroke severity. However, there is gross reporting of hemispheric lesions so conclusions cannot be made about lesion location and depression.

Schubert et al., (1992b) did not list the Barthel Index in their Method section, however it was included in the 1992a paper. Lai et al., (2002) have expressed a methodological concern over the heterogeneity of the stroke group, however one would think that homogeneity of the sample group would be more of an issue as Nannetti et al., (2005) express, as these studies all exclude patients with severe aphasia therefore potentially homogenising the sample groups. Van de Weg et al., (1999)

cannot generalise their findings because the type of stroke was not specified and aphasic patients were not included.

The articles that were accumulated in this systematic review were the only ones available. This can be interpreted as publication and citation bias, as published work can be indicative of selective reporting and not publishing unfavourable results. Unpublished and grey literature were not incorporated into this review as they were unobtainable. Additionally unpublished literature has not gone through peer review and so the quality of the research cannot be guaranteed (Pannucci & Wilkins, 2010).

Significant variables are of course reliant on which variables the Authors have deemed relevant enough to include in their studies. The main recurrent predictor from these 25 studies is depression (the higher the depression score the less recovery is achieved) and there are methodological flaws in the review papers, therefore there is scope for further research in regard to psychological predictors of recovery from stroke.

2.8.4 Conclusion

The implications for this review show that there is scope for further research in the area of psychological factors and their influence on stroke recovery. It is difficult to comment on the applicability of these findings because of the differences in the psychological and clinical measures used, the differences in the measures of recovery, length of the study duration, lack of demographic data, differences and lack of stroke definition and differences in statistical analyses.

However, the review papers do provide good research ideas and do give insight into the area of psychology and stroke recovery. In the following section, the implications of these findings are discussed.

2.9 Implications

It is the responsibility of the modern Health Psychologist to know anatomy, biology, cognitive neuropsychology and the initiation and progression of disease within the human body if we are to claim research in any health field. In the field of stroke we must understand the physical, cognitive, neurological and emotional aspects of stroke in order to produce good quality research. In Psychological studies there seems to be a train of thought that Psychologists do not have to learn the disease they are researching, which is evidenced with the lack of information on stroke severity and cognitive impairment. This gives Psychology a weaker footing in the research arena and should be addressed so future Psychological research exceeds the expectations we set for ourselves. It is the aim of this thesis to take a step in this direction.

Also it is important for future research to try and include the excluded groups, i.e., those with language impairment and physical impairment. This group of stroke survivors are an important group to study within the stroke realm especially in terms of psychological wellbeing. With the aid of technology the inclusion of this group may be managed without the use of proxy ratings.

2.10 Update to the systematic review September 2009 – April 2013

The systematic review was updated recently, with a search being conducted for articles between September 2009 – April 2013 to provide a complete review. Embase is no longer provided by Brunel University. Ingenta and PubMed do not allow filters by year and so Medline was searched in accordance from advice taken from the Psychology Subject Librarian at Brunel University, as Medline is a sister site to PubMed. Summon was recommended by the Psychology Subject Librarian as a search tool as it incorporates all the University databases (including Psychology) into one, and so the search of Ingenta and PubMed are included through Summon. Summon searches all databases that can be accessed by Brunel University except Statistics, Law and Finance. In regards to Psychology databases Summon searches, Ingenta, PubMed Central, Academic Search Complete, Cambridge Journals Online, Emerald e-books, Nature Publishing Group Journals, Oxford English Dictionary, PsycArticles, PsycInfo, Sage, ScienceDirect, Scopus, Taylor & Francis Journals, Web of Knowledge and Wiley Online Library.

The first part of the systematic review from 1990-2009 was conducted to serve as the foundation of this thesis. The outcome of the review informed the direction of the Literature Review and consequently the research questions and research design. Before the thesis was submitted it was pertinent to conduct an update (from 2009-2013) in order to investigate if other research had been published during the span of the current study. Otherwise the review would be out dated and questions regarding up-to-date research would not be able to be answered.

Hence, there is the main review and an update. If these were incorporated into one review, the examiners would not be able to clearly see which papers informed the research design. Even though the update from 2009-2013 did not add any new information it did yield 3 new papers. These 3 papers did not help to inform the research design therefore should be treated separately.

Additionally from 2009, active efforts were being made to satisfy the NHS ethical procedure thus ending the period for the systematic review as this information was put forward to the NHS Ethics Committee as forming the justification for the research. Chosen variables were decided upon at this stage. The update to the systematic review was added in order to offer a complete systematic review at time of submission.

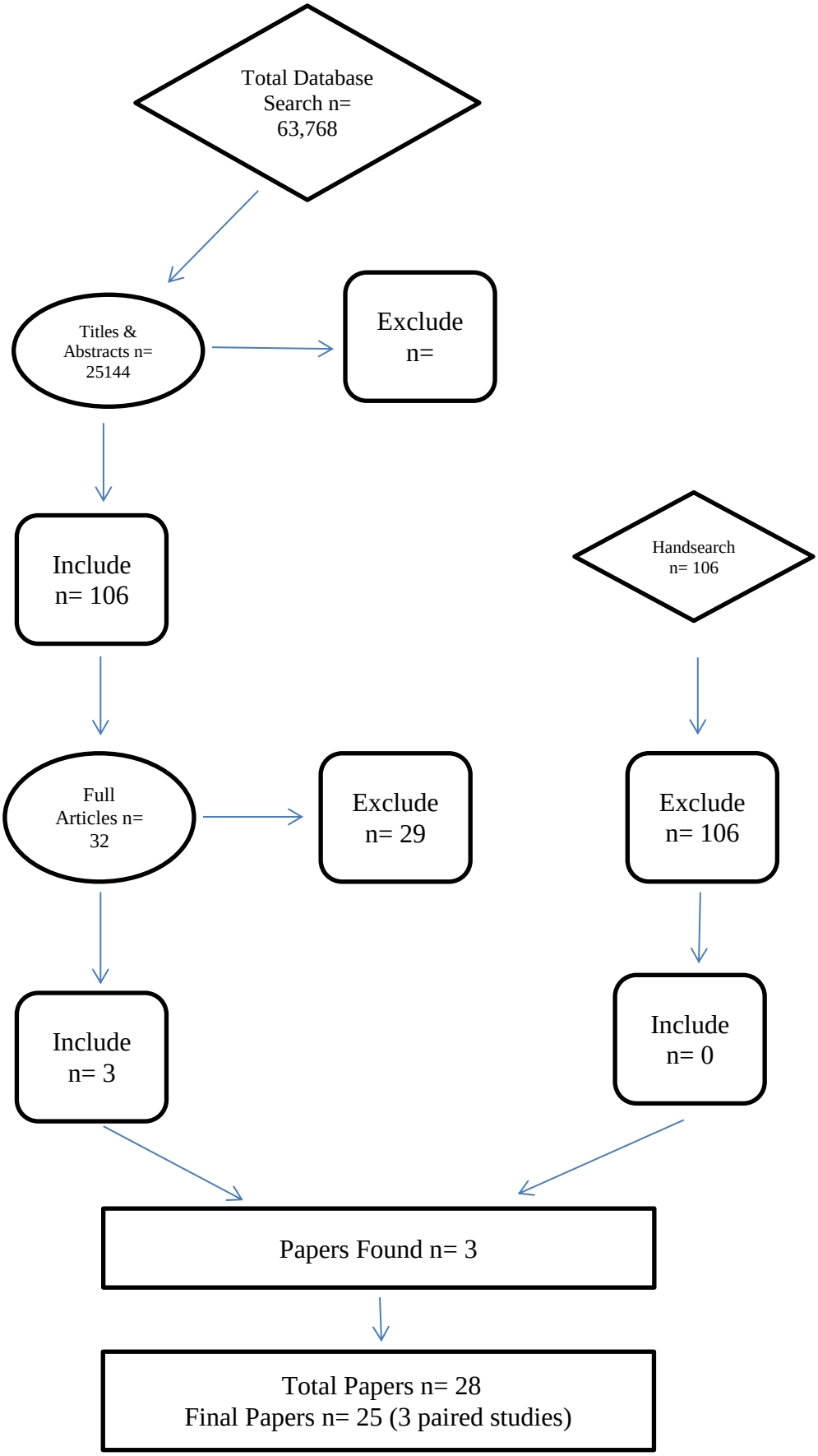
Please see Table 2.5 for information derived from the search for September 2009 – April 2013 and Figure 2.2 for the text selection process.

Table 2.5:

Search Terms Used for September 2009 – April 2013.

Search Terms	Summons	Medline
Stroke AND Psychological Stress	1,030	65
Stroke AND Stress	18,712	1,410
Stroke AND Coping	1,027	50
Stroke AND Depression	8,514	924
Stroke AND Stress AND Coping	307	12
Stroke AND Personality	1,364	44
Stroke AND Comparative Optimism	13	0
Stroke AND Social Support	4,741	151
Stroke AND Quality of Life	9,774	1,026
Stroke AND Predictor AND Recovery	1,144	109
Stroke AND Stress NOT Physiological	11,992	1,260
Stroke AND Predictor AND Recovery AND Longitudinal	94	5

Figure 2.2: Text Selection Process Sep 2009 – April 2013.



There were 3 relevant papers found in this search, however one study was found to share the same population as a study in the previous 1990 – August 2009 search. Consequently these papers are counted as one study, which constitutes the third pairing in this systematic review. This pairing is as follows:

3. Ostir G.V., Berges I-M., Ottenbacher M.E., Clow A.,Ottenbacher K.J. (2008)

&

Seale G.S., Berges I-M., Ottenbacher K.J., & Ostir G.V. (2010).

Therefore the final number of review papers included is 25. The complete references for these papers are detailed in the next section.

2.10.1 Review References Sep 2009 – April 2013

24. Donnellan, C., Hickey, A., Hevey, D., & O'Neill, D (2010) Effect of mood symptoms on recovery one year after stroke. *International Journal of Geriatric Psychiatry*, *25*, 1288-1295.

22b. Seale, G.S., Berges, I-M., Ottenbacher, K.J., & Ostir, G.V. (2010). Change in positive emotion and recovery of functional status following stroke. *Rehabilitation Psychology, 55* (1), 33-39.

25. West, R., Hill, K., Hewison, J., Knapp, P., & House, A. (2010). Psychological disorders after stroke are an important influence on functional outcomes: A prospective cohort study. *Stroke, 41*, 1723-1727.

These three papers did not add anything new to the findings of this review. Please see Appendix T for the table of characteristics and Appendix U for the methodological assessment table for these papers.

2.11 Why a Systematic Review was conducted rather than a Meta Analysis

Results from research using randomised controlled trials (RCT's) are used in meta analyses (Hunter & Schmidt, 2004). As the studies used in this systematic review were not RCT's they were not appropriate for a meta analysis.

Systematic reviews are frequent in health psychological reviews as is seen in Health Psychology Review. Some of these include reviews on adolescent alcohol use (Leung, Toumbourou & Hemphill, 2011), health belief model and adherence (Jones, Smith & Llewellyn, 2013), cancer (Merz, Fox & Malcarne 2014), illness beliefs (Urquhart Law, Tolgyesi & Horward, 2012), coping & pregnancy (Guardino & Schetter, 2013), obesity (Dombrowski et al., 2010), skin cancer (Dodd & Forshaw, 2010), partners of cardiac patients (Randall, Molloy, & Steptoe, 2009), carers of cancer patients (Ussher, Perz, Hawkins & Brack, 2009) and type 2 diabetes, (Thoolen, De Ritter, Bensing, Gorter, & Rutten, 2008). Therefore, a systematic review was conducted without meta analysis. It should also be noted that only one study gave any information on effect size from all of the review papers (Schubert et al. (1992a). Therefore there was insufficient information to conduct a meta analysis.

As the systematic review is complete, the next section will detail the aims and development of the current study and the psychological variables chosen for inclusion.

2.12 Aims & Development of the Current Study

The aims of this systematic review are as follows:

(i) Investigate the current literature on psychology, risk of stroke and effect on physical recovery from a stroke.

The results gathered for this review have found the following variables as being significant in regards to stroke recovery:

- Depression
- Positive emotion (taken from the CESD depression measure)
- State self esteem
- Severely threatening life events

- Pre-stroke trait introversion
- Extrovert personality
- active coping
- Perceived control,
- The psychological and environmental domains of the WhoQoLBREF measure.

This will facilitate in the forming of a research question for a quantitative study.

(a) Depression.

Depression and positive emotion are measured on the same scale (CES-D), therefore this can be treated as one variable. Depression has been shown to be very important in recovery from stroke.

Low self esteem is strongly related to depression risk whilst positive self esteem is related to less risk of depression (Sowislow & Orth, 2013). These factors can be treated as measuring similar attributes therefore, they will be treated as part of the same variable.

(b) Stress.

Severely life threatening events are stress factors. The role of stress and stroke is important as stress is a risk factor for stroke (Surtees et al., 2008). Consequently, stress will be treated as both an acute and chronic factor.

(ii) To decipher any gaps in the literature.

(c) Type D Personality.

Introversion and extroversion are personality dispositional traits which were briefly highlighted in this review (Morris et al. 1993 and Elmstahl et al. 1996). The Type D Personality (distressed personality) is a dispositional trait variable that has been used in heart disease research but not in stroke research so far. Heart disease and stroke are both vascular diseases and have many of the same symptoms. Type D personality is when individuals experience negative emotions and inhibit the expression of these emotions in social situations. Denollet, Pedersen, Vrints, & Conraads, (2006) conducted a study to find the relationship between Type D and cardiac events. They

found that participants with a Type D personality style had an increased risk of death or myocardial infarction after 5 years, compared with non-Type D personality types.

(d) Social Support.

Previous studies have shown psychosocial variables exhibit a strong association with the probability of suffering from heart disease (Kristofferzon, Löfmark, & Carlsson, 2005; Miller & Blackwell, 2006; Surtees et al., 2008). Importantly these factors have also been found to be associated with poorer outcomes following cardiac events. For example, Pedersen, Van Domburg, & Larsen, (2004) have shown that lower baseline levels of social support are associated with a 10% increased risk of further cardiac events and social support has been concluded to be important with other illnesses and has a close relationship with stress. Therefore social support will be considered as a variable even though no associations were found in the review papers that included social support (used by Parikh et al., 1990 & Chang & Mackenzie, 1998).

(e) Repressive Coping

Active coping was found to have a significant effect on ADL function (Elmstahl et al., 1996) therefore coping style is an important factor in considering recovery from stroke. However, it is also important to acknowledge maladaptive coping styles such as repressive coping. Repressive coping and stroke has not yielded any publications and therefore is a gap in the current literature. People with a repressive coping style are identified by showing high defensiveness and low trait anxiety. Repressors report low levels of distress whilst showing high physiological signs of stress. This indicates repressors may appear psychologically healthy but are prone to suffer from physical health problems (Myers et al., 2008). There seems to be a link between having a repressive coping style and being prone to coronary heart disease; in a longitudinal study spanning up to 10 years repressive coping was identified as being associated with long-term mortality in people with coronary heart disease (Denollet, 1999).

(f) Sense of Coherence (SoC).

Perceived control was used by 1 review study (Johnston et al., 1999;2004) measured by Recovery Locus of Control (LoC) Scale. However, the principles of LoC can be substituted by using Sense of Coherence. SoC measures coping with adverse experiences and measures comprehensibility, (“Do you usually feel that the things that

happen to you in your daily life are hard to understand?"), manageability ("Do you usually see a solution to problems and difficulties that other people find hopeless?") and meaningfulness ("Do you usually feel your daily life is a source of personal satisfaction?"). SoC scales have been reported to be more favourable than LoC scales (Flannery, Perry, Penk, & Flannery, 1994). Patients with a strong sense of coherence have demonstrated better recovery from stroke (Surtees et al., 2006).

(iii) To form a research question, which amalgamates points (i) and (ii). with added cognitive factors.

The Systematic Review has specifically investigated the association of psychological variables on stroke outcome at designated time points. This will facilitate the formation of the research question for the current study as Systematic Reviews are used to generate new hypotheses in future research as the function of the Systematic Review is to, in detail, examine a specific research topic (Khunti, 1999; Webb & Roe, 2007; Deb et al., 2013; Cheng et al., 2013; Campbell et al., 2014; Patel, Laffan, Waheed, & Brett, 2014). In the proposed project the relationship between psychosocial variables (depression, stress, social support, repressive coping, Type D personality and SoC) and cognitive factors from the 3 of the 4 cognitive domains (visuo-spatial impairment, memory and executive function) and their relation to recovery following stroke (quality of life & physical outcome) will be investigated at 3 fixed time points. Demographic information and risk factors will also be collected. These psychological and cognitive factors will be discussed in more detail in Chapter 3.

Chapter 3

Literature Review

3.1 Rationale

The rationale behind this chapter is to expand upon the findings of the systematic review in Chapter 2. Therefore depression, stress, social support, Type D personality, repressive coping and sense of coherence will be investigated. This is followed by a section on cognitive factors from 4 main cognitive domains and how they are related to depression, stress, social support, Type D personality, repressive coping and sense of coherence in relation to stroke recovery. This is to identify gaps in the literature and to identify cognitive variables for inclusion in the research design. The culmination of this Chapter ends with two designed theoretical models and a series of testable hypotheses.

3.2 Summary

The chapter is structured as follows. A brief introduction to some biological systems, are presented below in section 3.3. Each variable is then defined and its link with various illnesses is briefly discussed. This is followed by a discussion of cardiovascular and cerebrovascular disorders and the variable. Possible interventions are then outlined and finally, there is a discussion of how these variables can be measured. In each case, short self-report measures are favoured, in order to reduce participant fatigue. Reliability of the chosen measures is discussed in the Methodology section (4.7.4). This is followed by a section on cognitive factors from 4 main cognitive domains and how they are related to depression, stress, social support, Type D personality, repressive coping and sense of coherence in relation to stroke recovery. This is followed by sections on physical recovery and psychological recovery (quality of life). The Chapter ends with two theoretical models and hypotheses derived from the reviews.

3.3 Biological Markers

The immune system is a protective system against harmful organisms (Bennett Herbert, & Cohen, 1993). Psychological reactions can have an effect on

immune system functioning. Briefly, the hypothalamic pituitary adrenocortical (HPA) system, the sympathetic adrenomedullary system (SAM) and pro inflammatory cytokines is outlined to facilitate understanding of the link between psychology and the body, in the following sections.

3.3.1 Hypothalamic Pituitary Adrenocortical (HPA) System

The hypothalamic pituitary adrenocortical (HPA) system secretes glucocorticoids (such as cortisol), which is slowly released into the blood. Negative emotions such as anxiety and fear can stimulate the release of cortisol (Lundberg, 2005). Cortisol may be secreted in surplus in response to repeated stressful events and repeated secretions are a risk factor for vascular diseases (Kupper & Denollet, 2007). Cortisol impairs immune responses as it interferes with the communication between T-lymphocyte cells and cytokines. T-lymphocyte cells and cytokines are needed to fight infections (Taylor, 1995; Janeway, Travers, Walport, & Shlomchick, 2001).

3.3.2 Sympathetic Adrenomedullary (SAM) System

The sympathetic adrenomedullary (SAM) system secretes epinephrine (adrenaline), which prepares the muscles for action and norepinephrine (which is responsible for promoting increased attention and concentration) quickly into the blood, which readies the body for fight or flight against a threat. This is more in relation to physical demands rather than emotional. Over activation of the SAM system can cause narrowing of the blood vessels and thickening of the arteries, which can promote diseases of the vascular system (Lundberg, 2005).

3.3.3 Proinflammatory Cytokines

Cytokines are cells which aid the immune system and they are modulated by glucocorticoids (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002b). Negative emotions are associated with elevated pro-inflammatory cytokines (Kiecolt-Glaser, McGuire, Robles, & Glaser 2002a). There are two main types: Pro-inflammatory cytokines and anti-inflammatory cytokines. Pro-inflammatory cytokines worsen the disease by increasing inflammation and infection and reducing immune responses. Anti-inflammatory cytokines facilitate healing (Dinarello, 2000). HPA dysregulation

causes increased cortisol release and is associated with an increase of pro-inflammatory cytokines and hypertension (Girod & Brotman, 2004) and has been associated with ischemic heart failure (Deswal et al., 2001).

The following sections will review the psychological variables (depression, stress, social support, Type D personality, repressive coping and Sense of coherence).

3.4 Depression

In this section the definition of depression, how biology can relate to depression, theories of depression, depression and the relationship with disease, interventions for depression and how depression is measured in research studies will be reviewed.

3.4.1 Definition of Depression

Precise definitions of depression vary. There are major depressive disorder, atypical depression, bipolar depression, refractory and chronic depression (Carod-Artal, 2007). The Diagnostic and Statistical Manual of Mental Disorders (DSM-V, 2013) criteria, is the most universally used definition for clinical depression, which includes depressed mood, weight loss, loss of interest in pleasure, insomnia, fatigue, feelings of worthlessness, inability to concentrate and thoughts of death of at least 2 weeks duration.

3.4.2 Depression and Biology

Depression has been associated with activation of the HPA system which secretes cortisol in response to negative emotions (Bennett Herbert, & Cohen, 1993; Catalan, Gallart, Castellanos, & Galard, 1998; Pruessner, Hellhammer, Pruessner, & Lupien, 2003). In regard to risk factors for cerebrovascular disease, depression may increase hypertension (Davidson, Jonas, Dixon, & Markovitz, 2000). High releases of cortisol may be due to an impaired HPA system (Sher, 2005). Glucocorticoids (such as cortisol) inhibit potassium channel proteins, which are important for vascular tone regulation (Brem, Bina, Mehta, & Marshall, 1999) and affects the robustness of the immune system. This process would weaken the immune system thereby allowing disease progression to occur (e.g., risk factors for vascular disease

or any other disease where risk factors are present) (Janeway, Travers, Walport, & Shlomchick, 2001). This does not exclusively mean that depression will cause stroke. It means that a weakened immune system cannot fight the existing biological risk factors. Depression may cause hypertension as cortisol will weaken the immune system therefore being unable to fight the effects of pre-existing risk factors. Hypertension would not cause depression as hypertension is increased blood pressure and is a mechanical factor which does not stimulate the emotional release of cortisol. Chronic activation of the HPA system from depression or stress is indicative of an increase in pro-inflammatory cytokines which can lead to atherosclerosis. Therefore depressive systems could contribute to hypertension (Maes, Bosmans, Meltzer, Scharpe & Suy 1993; Steptoe & Brydon 2006).

Additionally a reduction in norepinephrine in the brain is associated with major depression. Norepinephrine is released by the SAM system as a response to a threat. Norepinephrine is responsible for promoting increased attention and concentration, a reduction of which would leave a person less able to utilise the fight or flight defensive strategies (Southwick, Vythilingam & Charney 2005).

3.4.3 Theories of Depression

Below is a brief overview of the main theories of depression: Seligman's learned helplessness theory and Beck's theory of depression. Seligman & Campbell (1965) developed the theory of learned helplessness. In this experiment based on classical conditioning following the works of Pavlov in 1902 (cited in Pavlov, 1941), dogs were placed in a box with a barrier which divided it into two parts. One side had electrodes on the floor, whereas the other side had no electrodes. One group of dogs were given electric shocks when the barrier was raised, therefore not being able to escape anywhere. When the barrier was lowered and the dogs were able to escape the shocks, they surprisingly did not. They stayed on the side with the electrodes and continued to be shocked behaving in a helpless manner. Conversely, dogs which had not been trapped previously would jump over the fence when experiencing shocks from the electrodes. Seligman believed we therefore learn depression from previous experiences and we develop beliefs that we cannot escape the situation, thereby learning to be helpless.

This theory has been revised for humans with the attributional style theory (Abrahamson, Seligman, & Teasdale, 1978). This theory postulates that when failure has occurred the person will try to attribute the failure to a reason. These reasons are determined by the persons beliefs, if they are attributed to personal reasons (internal) or environmental reasons (external), if the explanation for the failure is global or specific and if they are stable (e.g., something is unfair), or unstable (e.g., due to bad luck). Attributing reasons for failure to internal characteristics can lead to negative consequences on self-esteem. Attributing failure to global reasons can generalise thoughts of failure to other situations and attributing failure to stable characteristics will increase the duration of these thoughts. Depressed people tend to explain events in their life as personal, stable and global. The development of this theory has also included hopelessness (Abramson, Metalsky & Alloy 1988), much like Seligman's helplessness theory. Therefore, negative events interact with the personal, global and stable attributions to cause one to feel hopeless about a situation. Seligman believes the way to combat learned helplessness is to change our explanatory styles. That is to change the way we explain negative events to ourselves and to others (Gillham, Shatte, Reivich, & Seligman, 2001).

Beck (1967) developed a theory about depression and cognitive vulnerability. In this theory, Beck asserted that those that have learned maladaptive schemas and beliefs are more prone to developing depression. These are persistent negative ways of thinking which are about the self, the world and the future (the cognitive triad). This way of thinking skews information so it is processed negatively. These ways of thinking promote pessimism, low self esteem and unrealistic points of view. Beck believed the way to counter these maladaptive thought processes were through cognitive behavioural therapy which helps to train the depressed person in thinking differently and challenging automatic thoughts (Burns, & Beck, 1978).

In both of these landmark theories, believing unrealistic thoughts and being unable to find a solution to the problem (even if the solution is obvious), are indicative of depression. This could have an effect on adherence to treatment and efforts in the rehabilitative stage of stroke recovery (Cruess et al., 2010). Also the way to break the habitual cycle of negative thinking would entail a great effort to change thinking patterns.

Other theorists (Brown & Harris, 1989) believe that depression occurs in reaction to severe life events that have an emotional loss attached to it only. This theory does not identify other levels of life events as enough to produce depression, therefore does not acknowledge chronic everyday situations. These theories on depression have not made the same long term impact as the ones put forward by Seligman (1965) and Beck (1967).

3.4.4 Depression and Disease

Depression can be triggered by stressful events (Kessler, 1997) and this depressed mood can affect the course of diseases (Evans et al., 2005), for example, HIV/AIDS (Zimpel & Fleck, 2014), multiple sclerosis (Stepleman, Decker, Rollock, Casillas, & Brands, 2014) and Parkinson's disease (Allain, Schuck, & Maudui, 2000). However, in end stage renal disease there have been no strong relationships with depression (Devins et al., 1990; Christensen, Wiebe, Smith, & Turner, 1994), and also with cancer (haematological malignancies and rectal cancer) (Richardson, Zarnegar, Bisno, & Levine, 1990; Cody et al., 1994).

In a study of nearly 500 stroke, myocardial infarction, spinal cord injury and traumatic brain injury patients, 20% had suicidal thoughts between 3 months to 2 years post illness. When depression improved, suicidal thoughts lessened. When depression did not improve, suicidal thoughts persisted (Kishi, Robinson, & Kosier, 2001).

3.4.5 Depression and Cardiovascular Disease Recovery

As early as 1921 there have been reports that atherosclerosis and depression have a relationship (Kraeplin, 1921). Support for the relationship between depression, hypertension and vascular problems persists (de Castro et al., 2008).

In a study of hypertensive men at risk of cardiovascular disease those that experienced higher levels of discontentment were at 3 year follow up significantly more likely to have problems with carotid artery disease (Agewell, Wikstrand, Dahlof, & Fagerberg, 1996).

Depression and anxiety have been found to be predictive of hypertension development in a 16 year longitudinal study in normotensive participants (Jonas, Franks, & Ingram, 1997), therefore promoting the view that depression is a risk

factor for coronary heart disease. Similarly in a 15 year longitudinal study of nearly 3000 participants (Brown, Stewart, & Stump, 2011) depression was found to be predictive of coronary heart disease. Importantly, in the Multiple Risk Factor Intervention Trial with a follow up of 18 years those participants with increased depression scores had a higher risk of stroke mortality (Gump, Matthews, Eberly, & Chang, 2005).

3.4.6 Depression and Stroke Recovery

Depression is the most common psychiatric affliction suffered by stroke patients (Chemerinski & Robinson, 2000). In regard to risk factors for cerebrovascular disease, depression may increase hypertension (Davidson, Jonas, Dixon, & Markovitz, 2000). It has been reported in a study of nearly 500 participants that having biological risk factors for stroke and feelings of depression increases the risk of stroke, especially in men (Emmelin et al., 2003). The risk of depression on stroke patients have been echoed by many other studies (Colantonio, Kasi, & Ostfeld, 1992; Larson, Owens, Ford, & Eaton, 2001; Ohira et al., 2001; Lawrence, & Grasby, 2001; Nilsson & Kessing, 2004; Krishnan, Mast, Ficker, Lawhorne, & Lichtenberg, 2005; Salaycik et al., 2007).

Depression has been reported to improve over time. In a sample of 128 depressed patients compared to control participants, stroke patients had higher levels of depressive symptoms than controls, however at 12 month follow up depression levels were similar between the two groups (House et al., 1991). Nevertheless, this is not always the case and depression has also been reported to not decrease in a 12 month follow up study (Kotila, Numminen, Waltimo, & Kaste, 1983; Burvill et al., 1984).

Carod-Artal, Egido, Gonzalez, & Varela de Seijas, (2000) argue that post stroke depression affects health care use, functional recovery, cognitive function and quality of life. Depression affects physical recovery from a stroke (Robinson, 1997; Clarke, Black, Badley, Lawrence, & Williams, 1999; Desrosiers et al., 2007; Goodwin & Devanand, 2008), as depression has been found to impede physical recovery even 2 years post stroke (Parikh et al., 1990). However, in a study of over 70 stroke patients there was no association between depressive symptoms and functional impairment (Diamond, Holroyd, Macciocchi, & Felsenthal, 1995).

Parikh, Robinson, & Lipsey, (1990) reported depressed patients are more impaired at 2 years follow up compared with non-depressed patients in physical activities and language functions. However Morris, Raphael, & Robinson, (1992) suggested that 2 months after stroke depressed and non-depressed patients demonstrated an equal improvement in daily living skills.

Post stroke patients may become depressed because of disability and dependency on others, and having to accommodate loss of functions (Aben & Verhey, 2006). Lack of exercise and physical disability predicted depression in a 3 year follow up study in 101 stroke patients (Morrison, Pollard, Johnston, & MacWalter, 2005). Patients with depression after an acute stroke showed lower activities of daily living (ADL) 2 years post stroke, compared with non-depressed patients (Robinson, 1998). Good ADL at 1 month predicts depression at 3 months post stroke. This may be due to having to adjust to post stroke life quicker than patients still in the hospital (Singh et al., 2000). Depression at 3 months post stroke was associated with functional impairment at 1 year follow up and functional impairment at 3 months was associated with depression at 1 year follow up (Kotila, Numminen, Waltimo, & Kaste, 1999). However, the minority of published studies report no association (Chang, Ng, & Paulin, 1995).

Stroke mortality studies have reported mortality at 12 and 24 months post stroke, which was predicted by 1 month post stroke depressive symptoms in a U.K. study (House, Knapp, Bamford, & Vail, 2001) and distress has predicted fatal ischaemic stroke in a study of over 2000 men from the Caerphilly study (May et al., 2002). Additionally, in a sample of over 6 1/2 thousand stroke free participants those that displayed more depressive symptoms had a higher stroke mortality rate (Everson, Roberts, Goldberg, & Kaplan, 1998).

3.4.7 Interventions for Depression in Stroke Patients

Recovery from stroke can improve if depression is treated (Aben et al., 2001). There have been studies conducted on depression and stroke interventions mainly on antidepressant treatments such as selective serotonin reuptake inhibitors. These are considered effective in treating post stroke depression (Andersen, Vestergaard, & Lauritzen, 1994a; Arseniou, Arvaniti, & Samakouri, 2011). In a study combining antidepressant therapy with a short course of psychosocial-behavioural therapy a

reported 47% decrease in depression ratings were recorded compared with stroke patients who were treated with only antidepressant medication (Mitchell et al., 2009). Psychological therapies such as cognitive behavioural therapy (CBT) have been reported as being effective (Lincoln & Flannaghan, 2003) and a current multi-centre randomised controlled trial on augmented CBT (including occupational therapy and movement therapy) as an intervention for post stroke depression is under investigation (Kootker, Fasotti, Rasquin, van Heugten, & Geurts, 2012).

Depression after stroke is often untreated in patients as physical impairments of stroke may disguise depression (Lee, Tang, Yu, & Cheung, 2007). It is important to recognise depression in stroke patients as it is a treatable condition (Linden, Blomstrand, & Skoog, 2007). In a systematic review on the frequency of depression after stroke, Hackett, Yapa, Parag, & Anderson, (2005) conclude there is a lack of effective treatment of depression using psychological therapies and/or antidepressants.

3.4.8 Measures of Depression

Below are some of the most frequently used self-rating depression scales in the literature. These have been briefly critiqued in order to choose an appropriate measure.

a) **The Geriatric Depression Scale (Sheikh & Yesavage, 1986).** This is a 15 item measure which records activities, interests, mood, isolation, memory impairment. This measure is often used in patients who additionally have mild to moderate dementia. As dementia is an exclusion criteria (see section 4.6.2), this measure was not considered any further.

b) **The Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1963)** consists of 21 questions and records mood, guilt, disappointment, suicidal thoughts, concentration, sleep impairment, tiredness, appetite loss, weight loss, sex drive and health status. As these responses include a number of physical health measures, which can also be part of the illness (e.g., changes in appetite, fatigue, changes in sleeping pattern and loss

of sexual interest) and because it is a fairly long questionnaire this measure was not considered any further.

c) **The Hamilton Depression Rating Scale (Hamilton, 1960)** is a 17 item measure which assesses depressed mood, feelings of guilt, suicide, insomnia, work and activities, psychomotor retardation, agitation, anxiety, somatic symptoms, sexual symptoms, hypochondriasis, insight and loss of weight. The items are weighted differently for each section. Because of this it is difficult to ascertain what the final scores may mean. In a systematic review by Bagby, Ryder, Schuller, & Marshall, (2004) of 71 studies, the conclusion was that the Hamilton Depression Rating Scale is not a measure researchers should continue to use. For example, two participants may have the same score but they may have scored highly on one section and scored low on another or vice versa. The items are scored and are summed at the end. This leads to unclear conclusions. Additionally the items are inadequately designed. This scale is measuring concepts of depression which are out dated and based on older versions of the DSM. The authors of this review conclude "It is time to retire the Hamilton depression scale" (Bagby, Ryder, Schuller, & Marshall, 2004, p. 2175) and suggest the usage of a modern scale. Therefore this measure was not considered any further.

d) **The Zung Self Rating Depression Scale (Zung, 1965)** is a 20 item measure recording mood, disturbed sleep, appetite, sex drive, weight, physiological processes, tiredness, restlessness and suicidal thoughts. This scale has not been updated since 1965 and it, much like the Hamilton Depression Scale may be measuring out dated concepts of depression. Also factor analysis of this measure has concluded a final summed score should not be used and instead 3 subscales should be considered separately (cognitive, affective and somatic symptoms) (Kitamura, Hirano, Chen, & Hirata, 2004). For these reasons this measure was not considered any further.

e) **The Centre for Epidemiologic Studies Depression Scale (CESD) (Radloff, 1977)** is a 20 item measure and is designed for large scale studies investigating the epidemiology of depressive symptomatology. This measure records negative affect, sleep impairment and concentration loss experienced in the previous week. The CESD, has been updated and shortened to a 10 item version (CESD-10) (Andresen, Malmgren, Carter, & Patrick, 1994). Bearing in mind respondent fatigue (Anastasi, 1976) this measure was considered good for use in large scale studies as it is both short and has good internal consistency (see 4.7.1). Unlike the BDI there is only one physical question about sleep impairment. Consequently, this measure was used in the current study. More details are described in Chapter 4 Section 4.8.4.

The next section will discuss stress.

3.5 Stress

In this section the definition of stress, how biology can relate to stress, theories of stress, stress and the relationship with disease, interventions for stress and how stress is measured in research studies will be reviewed.

3.5.1 Definition and Theories of Stress

Stress has been defined in a number of ways to encompass biological and psychological stress. These are made evident in the leading theories of stress. Selye (1976) developed the General Adaption Syndrome to explain how the body responds to stressors. This process discharges glucocorticoids, which quickens the cardiovascular beat however, this process suppresses the immune system. If this continues the body is put under strain which exposes it to disease.

Stress can also vary by how it is perceived which can influence its frequency, intensity and length. Lazarus & Folkman (1984) stated there are three health outcomes in response to a stressor: somatic health, functioning in work and social living and morale or life satisfaction. The mediator between the person and their environment is appraisal. This is where the person determines what effect the stressor will have on them and if a threat is possible. Reappraisal is considering the situation as it progresses to adapt to changes that may come. Stress is managed by coping. These authors suggest there are two types of coping: problem focused coping and

emotion focused coping. Problem focused coping includes reappraisal of a situation and acknowledging strengths. Emotion focused coping reduces emotional distress by blaming and avoiding or pursuing social support.

Due to the manifestations of stress and the effect on the immune system, stress may accelerate the course of disease progression. Lazarus and Folkman, (1984) believe stress is influenced by the environment, appraisal of the environment, coping and personality factors and therefore measuring stress is very complicated. Reasons for stress have been cited as death of loved ones, illnesses of family or friends and financial problems (Chiriboga, Black, Aranda, & Markides, 2002).

3.5.2 Stress and Biology

In this section, research which has investigated stress and the HPA system, the SAM system, hypertension, atherosclerosis, cardiovascular disease and pro-inflammatory cytokines will be explored.

Stroke itself is a stressor because activation of the HPA and the SAM system occurs during a stroke event (Johannson, Olsson, Carlberg, Karlsson & Fungerlund 1997; Ahmed, de la Torre, & Wahlgren 2004). Cortisol is the main glucocorticoid which is released in response to stress (McEwen 2000; Habra, Linden, Anderson, & Weinberg 2003) or when anticipating stress (Smyth et al., 1998) which can elevate blood pressure (Levy, Hullman, Strond & White 1944).

Stress can cause increases in cardiac (blood pressure and cardiac output) or vascular responses (elevation in vascular peripheral resistance) (Manuck 1994). Increased HPA activation can cause an increase in platelet aggregation (Stratakis & Chrousos 1995) which can lead to hypertension (Goble & Le Grande 2008) and elevated blood pressure after a stroke (Bedi, Varshney & Babbar 2000; Ahmed, de la Torre, & Wahlgren 2004). Extended stress arousal can lead to stress related diseases (Nielsen, Kristensen, Schnohr, & Gronbaek, 2008). Cortisol can exacerbate vascular disease as it can decrease the growth hormone which is related to an elevated risk of cardiovascular disease (Matthews, Woodall, & Allen, 1993; Hew, O'Neal, Kamarudin, Alford, & Best, 1998) and develop atherosclerosis (Karmarck et al., 1997; Barnett, Spencer, Manuck & Jennings 1997; Kunst, del Rios, Groenhof & Mackenbach 1998). High cortisol levels after a stroke has been associated with increased risk or mortality and morbidity (Davalos et al., 1996).

Pro-inflammatory cytokines have been associated with acute stress (Maes et al., 1998). Acute stress raises blood pressure which can lead to brain haemorrhaging. Stroke is usually the result of progressive damage to the arteries of the brain over years, however the effect stress has on the arteries is unknown although it is generally believed that it does increase the risk of stroke and heart attack. Consequently the relationship between chronic stress and stroke is undetermined (Stroke Association 2007).

This would be consistent with Lazarus and Folkman's (1984) theory. People who appraise events or the stroke itself as threatening will exhibit a physical stress response. This will activate the HPA and SAM system. Over-activation of these systems can result in increases in vascular responses, which may lead to vascular illnesses such as stroke or heart disease.

3.5.3 Stress and Disease

Extended stress arousal is associated with stress-related diseases (Nielsen, Kristensen, Schnohr, & Gronbaek, 2008) such as: neurological disorders (Parkinson's disease and multiple sclerosis) (al' Absi & Wittmers, 1999), gastric ulcers (Hamilton, 1950), asthma (Wright, Rodriguez, & Cohen, 1998) and all cause mortality (stroke, heart disease, diabetes and cancer) (Nielsen, Kristensen, Schnohr, & Gronbaek, 2008). Severe psychological stress has been associated with increased blood pressure (Kadojic, Demarin, Kadojic, Mihaljevic, & Barac, 1999; al' Absi & Wittmers, 1999; Matthews et al., 2004) and smoking (Harmsen, Rosengren, Tsipogianni, & Wilhelmsen, 1990).

3.5.4 Stress and Cardiovascular Disease

Results of the relationship between stress and vascular disease are not clear-cut. For example the Whitehall II study of over 7000 men and women concluded psychological stressors do not predict high blood pressure (Carroll, Smith, Sheffield, Shipley, & Marmot, 1995). Nonetheless, psychological stress has been associated with increased intima-media thickness of the carotid artery (the two inner layers of the arterial wall) (Everson, Lynch, & Chesney, 1997).

Hypertension is a risk factor for coronary heart disease, which links stress with coronary heart disease (Denollet, 1997; Kop, 1999; Nielsen, Kristensen,

Schnohr, & Gronbaek, 2008; Hamer, Molloy, & Stamatakis, 2008). Stress has been associated with a higher risk of coronary heart disease in the Whitehall II study. Those that reported higher levels of stress had a higher risk of suffering a myocardial infarction (Nabi et al., 2013) however, in the Whitehall II cohort study there was not an association between stress and inflammatory markers (Steptoe, 2007), therefore there is some dispute in the literature.

Stress has also been associated with a higher risk of mortality from coronary heart disease (Frasure-Smith, 1991; Denellot, Pedersen, Vrints, & Conraads, 2006) and decreases in HRQoL in coronary heart disease patients (Staniute, Brozaitiene, & Bunevicius, 2013).

3.5.5 Stress and Stroke

The relationship between psychological stress and stroke is not clear. Studies have concluded a relationship between stress and stroke does exist (Harmsen, Rosengren, Tsipogianni, & Wilhelmsen, 1990) with stress decreasing stroke recovery (SoRelle, 2001), but other studies have found no significant relationship (Eckar, 1954; Macko et al., 1996; Nielsen et al., 2005).

Increased stress-related blood pressure led to a risk of ischaemic stroke in a study of 2682 Finnish men between the ages of 42 – 60. Those with high responses to stress were associated with a higher chance of having a stroke in this 11 year longitudinal study (Everson et al., 2001). However a study of 151 patients found no association between stressful incidents and cerebrovascular disease (Peris, Martin-Gonzalez, Valiente, Ruiz, & Vioque, 1997).

3.5.6 Interventions for Stress Reduction for Stroke Patients

Interventions that are aimed to reduce stress on stroke patients and to aid their recovery have been investigated on yoga and mindfulness techniques which have been found to be beneficial in the reduction of stress in stroke patients in studies in the U.S. (Lawrence, Booth, Mercer, & Crawford, 2013; Lazaridou, Philbrook, & Tzika, 2013). However, most intervention studies in the stroke field have focused on relieving stress on stroke caregivers (Servaes, Draper, Conroy, & Bowring, 1999; Hartke & King, 2003; Legg et al., 2011; King et al., 2012).

3.5.7 Measures of Stress

Below are some of the most frequently used stress measures in the literature. These have been briefly critiqued in order to choose an appropriate measure.

a) The systematic review in Chapter 2 identified the **Bedford College Life Events & Difficulties Schedule (LEDS) (Brown & Harris, 1978)** as a predictor related to stroke recovery. However, this measure is a semi structured interview therefore was not be eligible for inclusion in the current research.

b) **The Stress Appraisal Measure (SAM) (Peacock & Wong, 1990)** is a 28 item measure which assesses three perceptions of a stressor: threat, challenge and centrality (perceived importance for wellbeing). To find good quality studies using this measure were scarce even though it has recently been translated into Turkish (Durak & Senol-Durak, 2013). Taking into account the length and ambiguity of the quality of this measure, it was not considered to be included in the current research.

c) **The Life Events Checklist (Cohen, Tyrrell, & Smith, 1991)** is a 69 item measure investigating sources of stress (e.g., death of a loved one) and how much it has affected the participant. The scoring of this questionnaire is unevenly weighted with different questions being scored differently. Because of this the final score is ambiguous and not comparable across samples. This type of questionnaire is dependent on the participant having experienced a specific set of stressors. It is also too long to be given in an acute stroke setting, therefore this questionnaire was disregarded.

d) **The Perceived Stress Scale (PSS) (Cohen, Karmack, & Mermelstein, 1983)** is the most widely used measure of perceived stress (Andreou et al., 2011). This 14 item measure focuses on how the participant has handled general stress in the past month. This questionnaire is well used and applicable to use in an acute stroke sample, therefore this measure was used

in the current study. More details of this measure are described in Chapter 4, Section 4.8.4.

The next section will discuss social support.

3.6 Social Support

In this section the definition of social support, how biology can relate to social support, theories of social support, social support and it's relationship with disease, interventions for social support and how social support is measured in research studies will be reviewed.

3.6.1 Definition and Theories of Social Support

There is not a universal definition of social support (Glass, Matcher, Belyea, & Feussner, 1993; Beckley, 2007). Some definitions regard actual support received and some focus on perceived support (Knapp & Hewison, 1998; Beckley, 2006), with perceived support being associated with better health compared to actual received support (Uchino, 2004). Social networks provide stability, predictability, integration and rewards (Cohen & Wills, 1985).

Social support has been defined by Sarason, Sarason, Shearin, & Pierce, (1983) as the number of friends that supply social support and also the level of satisfaction of this support. There are two main theories which seek to account for the role of social support. Firstly the Main Effect Hypothesis suggests the absence of social support is stressful and the presence of it is beneficial to health. It is said to mediate the stress-illness link and can have a direct effect on health. Emotional support can reduce emotional arousal in a stressed individual as it can inhibit physiological mechanisms and reduce physical damage to the heart and arteries therefore protecting the immune system (Cooper, 1984).

The second theory is the Stress Buffering Hypothesis. Again this theory suggests social support mediates the stress-illness link but by buffering the individual from the stressor. The individual's appraisal of a potential stressor is influenced by social support. No direct effects of support on health or stressors are assumed but the relationship between them is in some way altered and social support only influences health under conditions of stress (Sarason, Sarason, Shearin, & Pierce, 1983; Cohen & Syme, 1985; Knapp & Hewison, 1998).

However a study of 17,047 participants of the general Dutch population from the Morbidity and Interventions in General Practice study reported support does not buffer against the effects of stress on health. This could be due to recruiting participants who may not have a severe illness thereby not testing this claim thoroughly (Tijhuis, Flap, Foets, & Groenewegan, 1995).

Payne & Jones (1987) argue the socio-psychological mechanism by which the buffering hypothesis works are not defined. In addition "social support" can be categorised into sub sections: instrumental support (physical help), informational support (advice), social companionship (support through activities), esteem support, emotional support and appraisal support (Wills, 1985; House, Umberton, & Landis, 1988; Friedland & McColl, 1989; Sherbourne & Stewart, 1991; Cimarolli & Boerner, 2005). Other definitions of social support include size of social networks and the quantity of relationships & resources from others (House & Khan 1985).

The main theories do not take into account this differentiation suggesting it could be considered in future research particularly since different 'types' may vary in importance depending on age. What is to be considered at present is whether social support exerts an influence on health status and whether this varies according to stress level.

Social support can be both positive and negative. Negative social support is defined as social conflict, social undermining and insensitivity (Cimarolli & Boerner, 2005). The most distressing form of social interaction is unpleasant communication with members of a shared social network (Bolger, DeLongis, Kessler, & Schilling, 1989). Negative social support has detrimental influences on health which eclipses the effects of positive social support. The Stress exacerbation model explains when there is more than one stressor, higher demands are placed on the persons coping strategies, which make the stressors harder to address compared with if there is one stressor to address (Rook, 1998). If an individual has poor quality social support physical health can be damaged for example, longitudinal epidemiological research has provided evidence that social isolation can increase risk of morbidity and mortality from all causes (Seeman et al., 1993).

3.6.2 Social Support and Biology

In this section research which has investigated social support with cortisol and cytokines will be explored.

The HPA system has been reported to decrease in people with positive social support, this has been interpreted that social support may inhibit cortisol activation in response to a stressful situation (Legros, Chiodera, Geenan, & von Frenckell, 1987; Kirschbaum, Klauer, Filipp, & Hellhammer, 1995). In other research this was expanded by explaining physical contact promotes the release of oxytocin, commonly referred to as the "bonding hormone". Oxytocin suppresses cortisol and reduces blood pressure and heart rate (Uvnas-Moberg 1998; Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003; Southwick, Vythilingam, & Charney, 2005). Low social support was associated with enhanced SAM system activation (Hughes, Sherwood, Blumenthal, Suarez, & Hinderliter, 2003), whilst perceived social support was associated with brain serotonin transporter availability, in a study of Chinese healthy volunteers (Huang et al., 2013).

Cytokines are important in repair from disease and they are modulated by glucocorticoids, which can be affected by stress. When couples are in conflict they recover slower from this and produce lower levels of cytokines. When couples have positive social support they release higher levels of cytokines (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002b).

In a study of T cells, patients that had higher social support had higher T-cell recovery compared to those with lower social support (Mohr & Genain, 2004) and higher natural killer cells (Uchino, Uno, & Holt-Lunstad, 1999). Additionally, loneliness has been associated with elevated systolic blood pressure (Hawkley, Masi, Berry, & Cacioppo, 2006).

3.6.3 Social Support and Disease

Social isolation is predictive of disease progression and death from disease from all cause mortality (Berkman & Syme, 1979; House, Landis, & Umberson, 1988; Morris, Robinson, Raphael, & Bishop, 1991; Hawkley & Cacioppo, 2003; Lyyra & Heikkinen, 2006; Ikeda et al., 2008) and higher rates of HIV (Lee & Rotheram-Borus, 2001), cancer (Hibbard & Pope, 1993) and negative health behaviours such as smoking, unhealthy diets and low exercise participation (Reblin

& Uchino, 2008). Poor social support is linked with promoting suicidal thoughts (Kishi, Robinson, & Kosier, 2001), lower psychological wellbeing and higher distress (Mellor, Stokes, Firth, Hayashi, & Cummins, 2008).

Higher social ties are related to reduced morbidity and mortality (Olsen, 1993). Social support has been associated with decreased cancer rates (type of cancer was not stipulated) and social support may enhance recovery only, but not prevent disease (Vogt, Mullooly, Ernst, Pope, & Hollis, 1992). Social support may influence disease severity, progression, recovery and incidence (Cohen, 1988) and reduce mortality in diseases such as vascular diseases and cancers (type of cancer not stipulated) (DeVries, Craft, Glasper, Neigh, & Alexander, 2007), tuberculosis (Holmes, 1956) and reduces depression in elderly people with unipolar depression. The depressive effects on the neuroendocrine system may be protected by social support (Hays, Steffen, Flint, Bosworth, & George, 2001) increasing treatment adherence in Type 2 diabetes patients (Osborn & Egede, 2012), increasing HRQoL in men with prostrate cancer (Paterson, Jones, Rattray, & Lauder, 2013) and having a positive effect on breast cancer survivors (Cheng et al., 2013). Social support is associated with better recovery from hip operations (Cummings et al., 1988), cancer recovery (type of cancer not stipulated) (Wortman, 1984) and body image issues such as paralysis (Labi, Philips, & Gresham, 1980).

Cohen, Doyle, Skoner, Rabin, & Gwaltney, (1997) devised an experiment in which consenting participants were exposed to the common cold virus. They found that participants with varied social networks were less likely to be infected compared to those with less social support. In a study of over 2500 adults from the general population, support in work and marriage has been found to be protective against all-cause morbidity and mortality (Hibbard & Pope, 1993).

3.6.4 Social Support and Cardiovascular Disease

Social support may have an effect on hypertension as perceived loneliness in the general Hispanic population of over 60 years of age predicted hypertension, stroke and heart disease (Tomaka, Thompson, & Palacios, 2006). Low social support and social isolation has been found to be associated with cardiovascular disease (Rosengren, Wilhelmsen, & Orth-Gomer, 2004). Social isolation is physiologically stressful to the cardiovascular system and low social integration and low perceived

social support has been found to be related to cardiovascular disease. This was concluded from the results of a 6-year longitudinal study (Watson, Shively, Kaplan, & Line, 1998; Orth-Gomer, Rosengren, & Wilhelsen, 1993). Also, research has shown social support to be beneficial as it can increase length of life and lower levels of cardiovascular reactivity during stressful situations (Knox and Uvnas-Moberg, 1998), thus maintaining cardiovascular health (Reed, McGee, Yano, & Feinleib, 1983).

Those who are more socially isolated have a 1.5 increased risk of suffering a myocardial infarction (Ali, Merlo, Rosvall, Lithman, & Lindström, 2006) and present poorer recovery (Denellot et al., 1996). Cardiac patients with poor marital support and depression are at risk of a poorer prognosis (Compare et al., 2013) whilst low social support decreases HRQoL in coronary heart disease patients (Staniute, Brozaitiene & Bunevicius 2013).

Social support has also been associated with mortality from cardiovascular disease. Five hundred and three women with suspected coronary heart disease who reported social isolation were more at risk of mortality at 2 year follow up (Rutledge et al., 2004). In a 15 year longitudinal study of 2603 participants from the general population from Portland, Oregon it was found that social network scope (number of contacts in different domains), was predictive of mortality from ischaemic heart disease (Vogt, Mullooly, Ernst, Pope, & Hollis, 1992), whilst highly stressed socially secluded men had a higher risk of death following a myocardial infarction (Ruberman, Weinblatt, Goldberg, & Chaudhary, 1984).

3.6.5 Social Support and Stroke

Social support also affects cerebrovascular disease. After stroke social networks decrease (Knapp & Hewison, 1998; Hilari & Northcott, 2006). In general it is asserted that people with a partner recover better from a stroke (Jorgensen et al., 2008). In an Australian sample of 76 stroke patients those with poorer perceived social support had higher depression and a longer depressive period compared to those with higher perceived social support (Morris, Robinson, Raphael, & Bishop, 1991). Married men reported more benefit from their marriage in regards to recovery at home after a stroke compared with unmarried men. However, married women

reported less benefits compared with unmarried women (Clarke, Black, Badley, Lawrence, & Williams, 1999).

Social support is associated with lower levels of post stroke depression (Brugha, Bebbington, Stretch, MacCarthy, & Wykes, 1997) with lack of positive social support leading to depression (Astrom, Adolfsson, & Asplund, 1993). Social isolation (knowing less than 3 people you can rely on) was associated with higher events post-stroke (Boden-Albala, Litwak, Elkind, Rundek, & Sacco, 2005), whilst being a member of multiple social groups before stroke is beneficial as after stroke, some of these groups are likely to be maintained (Haslam et al., 2008). The more social support a patient had before the stroke showed the less need to be in a nursing home (Colantonio, Kasl, Ostfeld, & Berkman, 1993).

Stroke patients' level of social isolation or social support determines feelings of their recovery during the recuperation period (Haun, Rittman, & Sberna, 2008). Attainment of social needs results in positive adjustment to impairments from stroke (Evans & Northwood, 1981). Quantity of social support was found to be predictive of community participation after stroke more so than quality of social support, in a study of 95 stroke patients at 3 and 6 month follow up (Beckley, 2007).

In the Japan Public Health Centre-based Prospective Study Cohort II study, low levels of social support was related to increased stroke risk in men (Ikeda et al., 2008). Social support was also found to be a protective factor against stroke risk in a sample of Chinese patients (Tang et al., 2005).

Improved functioning in stroke patients has been related to having good social support four to six weeks post stroke, whilst poor social support was predictive of reduced functional improvement (Glass, Matcher, Belyea, & Feussner, 1993). Stroke patients with severe impairment but with high social support made better gains over time in recovery compared to those with low social support (Glass & Maddox, 1992). Patients with low social support and mild stroke had reductions in functional improvement 3 and 6 months post stroke. This could be due to milder strokes being deemed as less deserving of ongoing support (Glass, Matcher, Belyea, & Feussner, 1993).

However, there can also be negative physical effects of social support. Too much social support can lead to lower levels of motivation (Watzlawick & Coyne, 1980) and instrumental support can have a negative effect on physical recovery

(McLeroy, DeVellis, DeVellis, Kaplan, & Toole, 1984). Mulley (1985) concluded that too much instrumental support can have a negative effect as this hinders the patient in learning to do matters for themselves.

3.6.6 Social Support Interventions with Stroke Patients

Out of 10 studies in a systematic review investigating the effect of social support interventions on outcome after stroke discharge (Salter, Foley, & Teasell, 2009), only 2 studies reported a significant relationship. Care management at home (Dennis, O'Rourke, Slattery, Staniforth, & Warlow, 1997) and care coordination had a significant impact on depression (instead of usual care) (Claiborne 2006). However, the overwhelming amount of research shows social support interventions do not have a positive impact post stroke (Friedland & McColl, 1992; Mant, Carter, Wide, & Winner, 2000; Clark, Rubenach, & Winsor, 2003; Lincoln, Francis, Lilley, Sharma, & Summerfield, 2003; Corr, Phillips, & Walker, 2004; Boter & HESTIA Study Group, 2004; Tilling, 2005; Burton & Gibbon, 2005).

3.6.7 Measures of Social Support

Below are some of the most frequently used social support measures in the literature. These have been briefly critiqued in order to choose an appropriate measure.

a) **The Social Functioning Examination (Starr, Robinson, & Price, 1983)** was found to be a significant predictor in the systematic review. However, this measure is a semi structured interview so will not be considered for inclusion in the current research.

b) **The Social Ties Checklist (Starr, Robinson, & Price, 1983)** was also reported in the systematic review from Chapter 2. This is a 10 item measure which quantifies the number of social ties the participant has. As can be seen in the prior review of the literature, perceived support is reported to be more valuable than simply counting contacts. For this reason this measure was not considered for inclusion in the current study.

c) **The Social Support Questionnaire (Sarason, Levine, Basham, & Sarason, 1983)** was also identified by the systematic review. This is a 27 item questionnaire which asks the participant to name the number of contacts that provide support and to rate how satisfied they are with this support. As this is a 27 item questionnaire it was considered too long for the current study due to the potential of participant fatigue (Anastasi, 1976).

d) **The Social Network Index (Cohen, Doyle, Skoner, Rabin, & Gwaltney, 1997)** is a 23 item measure. It records which social groups the participant is involved in (e.g., parents, in-laws and children). These questions may be inappropriate to ask elderly participants as relatives and parents may be deceased. For this reason and because of the length this questionnaire will not be considered for inclusion in this research.

e) **The Multidimensional Scale of Perceived Social Support (MSPPS) (Zimet, Dahlem, Zimet, & Farley, 1988)** is a 12 item scale which measures perceived social support, with three subscales (family, friends and significant other). This measure is short and user friendly therefore was used in the current research. More details of this measure are described in Chapter 4, Section 4.8.4.

The next section will discuss Type D Personality.

3.7 Type D Personality

In this section the definition of Type D personality, how biology can relate to Type D personality, theories of Type D personality, Type D personality and it's relationship with disease, interventions for Type D personality and how Type D personality is measured in research studies will be reviewed.

3.7.1 Definition and Theory of Type D Personality

Type D is the "distressed personality" style and is considered a measure of suppression of negative emotions (Denellot et al., 1996). This construct consists of two factors: negative affectivity (experiencing negative emotions regardless of

situation) and social inhibition (in social situations, inhibiting self-expression to avoid social disapproval) (Denellot, 2005). Negative affect includes depressed affect, anxiety, hostility and anger. Social inhibition includes social unacceptance and social disapproval (Habra, Linden, Anderson, & Weinberg, 2003). Type D people experience disability, anger, distress, low social support, depression and pessimism (Denollet, 2000). Type D may also indirectly influence health behaviours by not adhering to treatment or not asking doctors questions and advice (Denellot et al., 1996). In a healthy British sample, Type D was associated with decreased levels of social support and lower health behaviours (Williams et al., 2008).

3.7.2 Type D Personality and Biology

In this section research which has investigated Type D personality with pro-inflammatory cytokines, cortisol, intima media thickness of carotid arteries, and autonomic cardiac control, will be explored.

A pro-inflammatory cytokine called tumor necrosis factor alpha, is believed to increase blood pressure and cause vasoconstriction of arteries and can cause blood platelets to clump together which can result in a thrombosis. Type D has been found to be an independent predictor of tumor necrosis factor alpha (Denellot et al., 2003).

Participants with Type D personality have in response to acute stress higher cortisol levels and cardiovascular reactivity in an undergraduate sample, in a healthy sample (Habra, Linden, Anderson, & Weinberg 2003) and in an acute coronary syndrome sample (Whitehead, Perkins-Porras, Strike, Magid & Steptoe 2007).

Type D personality has been associated with changes in autonomic cardiac control compared with non- Type D participants in an active mental stressor task, however no relationship has been found with passive mental stressors (Kupper, Denollet, Widdershoven, & Kop, 2013). Additionally, the intima media thickness of carotid arteries in 40-60 year old patients in Iran had mixed results for it's association with Type D personality, compared to normal carotid arteries. When using t-tests the results were not significant, however when using chi square, the results were significant. Therefore the investigators of this study conclude there is a relationship between Type D personality and intima media thickness of carotid arteries. (Khorvash, Rahimi, & Bagherian-Saraoudi, 2013). However, as parametric testing is more stringent than non parametric testing, the findings of this study should

be treated with caution. Consequently more research should be devised on the biological mechanisms of Type D (Kupper & Denollet, 2007).

3.7.3 Type D Personality and Disease

Type D personality has had an effect on multiple disease conditions. For example, in a study of adherence with a mandibular advancement device to help with sleep disordered breathing (Dieltjens, Vanderveken, & Van den Bosch, 2013), satisfaction with information about cancer survival in cancer patients (Husson, Denollet, Oerlemans, & Mols, 2013), Parkinson's disease, multiple sclerosis (Dubayova, Krokavcova, & Nagyova, 2013), peripheral artery disease (Aquarius, Denollet, de Vries, & Hamming, 2007) and a study has been reported of patients that went to the hospital with non cardiac chest pain, where Type D personality patients were more likely to have panic disorder and higher anxiety and depression scores (Kuijpers, Denellot, Wellens, Crijns, & Honig, 2007).

3.7.4 Type D Personality and Cardiovascular Disease

The vast majority of studies including Type D have been conducted on hypertension and cardiac problems. Type D is associated with hyper responsivity (which is a precursor to hypertension) (Gerin et al., 2000), hypertension (Svansdottir et al., 2013; Kupper, Pelle, & Denollet, 2013) and coronary heart disease (Denellot, 2005).

Emotional inhibition has been reported to increase cardiovascular reactivity and decrease recovery after coronary heart disease (Denollet, 2000) and increasing cardiac events (Denollet, Vaes, & Brutaert, 2000). Type D and coronary heart disease is associated with stress, depression (Pedersen et al., 2006), dissatisfaction with life (Denellot, 1998a), low self rated wellbeing, anger, tension (Denollet & De Potter, 1992), social alienation (Denellot, Sys, & Brutsaert, 1995), anxiety (Schiffer et al., 2005; Schiffer, Pedersen, Broers, Widdershoven, & Denollet, 2008), depression, low quality of life (Schiffer, Pedersen, Widdershoven, & Denollet, 2008), fatigue (Smith et al., 2007), sense of coherence (Karlsson et al., 2007) and posttraumatic stress disorder in cardiovascular patients (Pedersen & Denollet, 2004).

In Denellot's studies, he controlled for cardiac markers that could affect disease progression (such as left ventricular function and coronary obstructive

disease), however the Type D personality style still influenced adverse recovery from cardiovascular disease (Denellot et al., 1996). Type D also predicted health related quality of life even when medical markers did not (Pedersen, Theuns, Muskens-Heemskerk, Erdman, & Jordaens, 2007) and after cardiac rehabilitation (Pelle et al., 2008).

Type D cardiac patients also believe there are less advantages of medical interventions (Pedersen & Denollet, 2003). However, they report more cardiac symptoms, but are less likely to inform a cardiologist or nurse (Schiffer, Denollet, Widdershoven, Hendricks, & Smith, 2007) and they expend less energy on exercise. For example, on a 6 minute walking test after coronary heart bypass surgery, Type D patients walk less compared to non Type D patients when there is no difference in heart rate (Attila, Istvan, Istvan, & Gabor, 2007). There have also been long lasting effects of Type D on cardiovascular recovery. After a 5 year follow up study in coronary heart disease patients, Type D was still associated with coronary problems (Denellot et al., 1996).

A minority of research studies have reported no association between Type D and coronary heart disease. In a recent study, of disease free participants, Type D was investigated in regard to incident risk of coronary heart disease in a 10 year follow up study. However, this research yielded no significant results (Larson, Barger, & Sydeman, 2013).

Type D personality has also been associated with mortality form cardiovascular disease (Schiffer, Smith, Pedersen, Widdershoven, & Denollet, 2010). In 2 longitudinal studies with 105 and 268 patients, Type D was found to predict higher mortality rates from coronary heart disease (Erdman, Duivenvoorden, Verhage, Kazemier, & Hugenholtz, 1986) and Type D patients suffered higher rates of myocardial infarction and death after being fitted with stents (Pedersen et al., 2004).

3.7.5 Type D Personality and Stroke

To date there are no published studies on Type D personality and stroke recovery.

Type D personality is unlikely to be a *cause* of stroke, as the cause of stroke is a disturbance of blood flow to the brain and care must be taken not to make

statements such as these without strong supporting biological/neurological evidence. In order to address the question of whether Type D personality causes a stroke would require neurological data. Neurological data is information on the brain itself, for example, size and location of the stroke lesion (Ganesan, Ng, Chongc, Kirkhama, Connelly, 1999). This is not the focus of the PhD.

To date, no studies on stroke and Type D have been published and therefore, can Type D personality be a cause of stroke has not been broached. In Type D studies, Type D personality is normally treated as an independent predictor (e.g. Aquarius, Denollet, de Vries, & Hamming, 2007). Therefore, in the theoretical models in this thesis that have been constructed based on the previous literature, Type D personality was treated mainly as an independent variable predicting physical recovery and quality of life. However, Type D personality has been included as a mediator between stress and physical recovery based on the suggestion by Lazarus & Folkman (1984) that personality may be a mediator. As Type D was the only personality measure taken this was then hypothesized to be a potential mediator.

3.7.6 Interventions for Type D Personality and Stroke

There are no specific interventions for Type D personality however, interventions can be suggested. For example, improving health related behaviours, interpersonal functioning and mood status (Sher, 2005; Tulloch & Pelletier, 2008). Pelle, van den Broek, & Denollet, (2012) suggest CBT, mindfulness techniques, relaxation techniques and pharmacotherapy.

3.7.7 Measures of Type D Personality

Below are the main measures used to assess Type D personality. These have been briefly critiqued in order to justify its use as an appropriate measure.

a) **The standard assessment of negative affectivity, social inhibition and Type D personality (DS 16) (Denollet, 1998a)** is made up of two subscales: negative affectivity and social inhibition. This measure was created in order to produce a short scale without the need of presenting to participants two long scales.

b) In 2005, Denollet updated the scale **(DS 14)** reducing it by two items to improve the reliability. This scale is now used widely to measure Type D personality and was used in this study. More details of this measure are described in Chapter 4, Section 4.8.4.

The next section will discuss the repressive coping style.

3.8 Repressive Coping Style

In this section the definition and theories of repressive coping, how biology can relate to repressive coping, it's relationship with disease, interventions for repressive coping and how repressive coping is measured in research studies will be reviewed.

3.8.1 Definition and Theories of Repressive Coping

Repressive coping (repressors) is the disposition to repress or avoid negative affect (Myers, 2000; 2010; Rutledge & Linden, 2003). One of the defining and robust findings is that in potentially stressful situations repressors report lower levels of distress but are physiologically reactive (Asendorpf & Scherer, 1983; Barger, Kircher, & Croyle, 1997; Benjamins, Schuurs, & Hoogtraten, 1994; Derakshan & Eysenck, 1997, 2001a, 2001b; Gudjonsson, 1981; Jamner & Schwartz, 1986; Lambie & Baker, 2003; Newton & Contrada, 1992; Pauls & Stemmler, 2003; Weinberger, Schwartz, & Davidson, 1979).

Repressors are operationally defined as scoring low on trait anxiety scales (measured by various trait anxiety scales), and scoring highly on defensiveness (often measured with the Marlowe-Crowne Social Desirability Scale (MC) (Crowne & Marlowe, 1964) (Weinberger, Schwartz, & Davidson 1979). Repressors, as defined by Weinberger et al., are always operationalised by self-report measures (see Myers, 2000; 2010 for reviews). Many studies indicate that individuals with a repressive coping style avoid negative affect, especially to self-relevant threat stimuli and do not have conscious experience of anxiety (Myers, 2000, 2010). This avoidance may be preceded by a rapid vigilance stage, which may involve automatic and non-conscious processes (Derakshan, Eysenck, & Myers, 2007).

Apart from the repressor group, three control groups are usually identified using the same typology: a further low trait anxiety group who are low on defensiveness *(low-anxious)* and two high trait anxiety groups, one of which is low on defensiveness *(high-anxious)* and one of which is high on defensiveness *(defensive high-anxious).* Repressors are either compared with the individual control groups or a composite of control groups (see Myers, 2010 for a review).

3.8.2 Repressive Coping and Biology

In this section research which has investigated the repressive coping style with the HPA system, the SAM system, natural killer cells, cholesterol and blood pressure will be explored.

Repressors, although avoiding negative affect demonstrate increases in the SAM system (Levine et al., 1987; King, Taylor, Albright, & Haskell, 1990) and the HPA system (Giese-Davis, Sephton, Abercrombie, Duran, & Spiegal, 2004).

In a sample of healthy college male students, repressors (compared with nonrepressors), had a larger pattern of natural killer cells, lower circulating CD4 cells (T-helper cells), increased fasting insulin levels (which illustrates metabolic dysfunction), lower high-density lipoprotein (HDL) (also known as "bad cholesterol") and a higher total/ HDL cholesterol ratio (which increases risk of vascular disease) (Barger, Marsland, Bachan, & Manuck, 2000). Additionally, repressors have been reported as exhibiting increased blood pressure (King, Taylor, Albright, & Haskell, 1990; Grossman, Watkins, Risticcia, & Wilhelm, 1997; Gleiberman 2007).

3.8.3 Repressive Coping and Disease

There is considerable evidence which indicates that the repressive coping style as defined by Weinberger et al., (1979) may be associated with adverse physical health. This is potentially serious as repressors comprise a significant percentage of various populations, accounting for between 10 and 20% of non-clinical populations (e.g., Myers & Reynolds, 2000; Myers & Vetere, 1997; Phipps & Srivastava, 1997), between 30 and 50% of patients with various chronic illnesses (e.g., Cooke, Myers, & Derakshan, 2003; Myers, Davies, Evans, & Stygall, 2005a), and up to 50% of

elderly groups (Brown et al., reported in O'Leary, 1990; Erskine, Kvavilashvili, Conway, & Myers 2007).

There is a body of evidence linking repressive coping with poor physical health. For example, melanoma patients were significantly more repressed than cardiovascular patients and controls (Kneier & Temoshok, 1984) and a high percentage of repressors have been identified in women after taking a breast biopsy test (Kreitler et al., 1993). Poorer prognosis in repressive breast cancer patients compared to non repressors has been reported in 2 prospective studies (Jensen, 1980; Giese-Davis et al., 2004; 2006).

3.8.4 Repressive Coping and Cardiovascular Disease

The most robust, longitudinal findings linking repressive coping and poor physical health have been in the area of cardiovascular disease. Early studies from the 1980s indicated that repressors with cardiovascular disease retained low levels of information when given information about heart disease. For example, in hospitalized patients who were recovering from a myocardial infarction, repressors gained less information about cardiac risk factors. Six months later it was found that repressors who had gained high risk information reported more complications (e.g. arrhythmias and fluid retention) and poorer functioning (sleep disturbance and depression) compared with non repressors (Shaw et al., 1985).

The Montreal Heart Attack Readjustment Trial was a randomized control trial of psychosocial interventions for post myocardial infarction patients (N = 1376). The intervention involved screening and treating nonspecific psychological distress and was based on evidence that increases in stress may lead to poor prognosis after a myocardial infarction. At five years follow-up repressors and two control groups, low-anxious and high-anxious were identified. The programme was associated with significantly worse survival in both male and female repressors (Frasure-Smith et al., 2002).

Additionally 731 patients with coronary heart disease from two prospective studies were followed up at 5 and 10 years with a mean follow-up time of 6.6 years. Twenty two percent of patients were classified as repressors who were at increased risk for death/myocardial infarction (Denellot, Martens, Nyklicek, Conraads, & de Gelder, 2008).

3.8.5 Repressive Coping and Stroke

To date there are no published studies on repressive coping and stroke recovery.

3.8.6 Interventions for Repressive Coping

As discussed in 3.8.1 repressors do not have conscious awareness of anxiety, making it difficult to develop standard interventions for repressors. Such interventions have yet to be developed.

3.8.7 Measures of Repressive Coping

Repressive coping is characterised by measuring two constructs: defensiveness and trait anxiety.

a) Defensiveness

The Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe, 1960) is the most frequently used measure for defensiveness in repressive coping studies (Brosschot & Janssen, 1998; Myers & Derakshan, 2004). This measure assesses response bias (i.e., the degree to which individuals attempt to present themselves in a favourable light) and has been classically used as the defensiveness component to assess the repressive coping style. This measure has 33 items.

The M-C SDS was shortened by Reynolds in 1982 to a 12 item scale and is termed the M-C SDS Form B. This was used in the current study as it is user friendly, short and easy to administer. More details of this measure are described in Chapter 4, Section 4.8.4.

b) Trait Anxiety

Trait anxiety can be measured with different scales. The Manifest Anxiety Scale (Taylor 1953) is often used. This measure is a 59 item questionnaire. However, as this measure is long it was not considered for the current study.

Another popular measure is the Spielberger Trait Anxiety Scale (STAI) (1970) which measures state and trait anxiety with 40 items. The

STAI measures state anxiety (how one feels in the moment) and trait anxiety (how one normally feels). In 1992 Marteau & Bekker, created the 6 item short-form of the state scale of the Spielberger State-Trait Anxiety Inventory (STAI). This is a short questionnaire which condenses the most highly correlated anxiety-present and anxiety-absent items from the full-form of the STAI (20 items) into six items. As this measure is short, user friendly, and easy to administer it was included in the current study. More details of this measure are described in Chapter 4, Section 4.8.4.

The next section will discuss sense of coherence (SoC).

3.9 Sense of Coherence (SoC)

In this section the definition and theories of SoC, how biology can relate to SoC, the relationship with disease, interventions for SoC and how SoC is measured in research studies will be reviewed.

3.9.1 Definition and Theory of SoC

Antonovsky coined the term Sense of Coherence in 1971. He researched a group of Israeli women in concentration camps in the Second World War and their adjustment to menopause. He believed SoC is a stress adaptive strategy and has three components: comprehensibility (cognitive), manageability (instrumental/ behavioural) and meaningfulness (motivational). Comprehensibility refers to the ability to predict problems that will be encountered in the future. Manageability refers to the ability to use resources to solve a problem and Meaningfulness refers to ability to perceive challenges as a necessary obstacle to endeavour to survive in the future. Antonovsky focused on the positive effects of psychology on health. To focus on the positive origins of health is called a salutenogenic perspective, to focus on a negative origins of health is called a pathogenic perspective (Antonovsky, 1979).

Antonovsky (1987) states people must cope with stressful situations to avoid negative stress. SoC is a stress adaptive strategy; however Antonovsky asserts it is not a coping style or a personality trait but a dispositional orientation (Antonovsky, 1993). A strong SoC illustrates the ability to manage, understand and find the meaningfulness in challenging situations. A weak SoC demonstrates an inability to

manage, understand and find meaningfulness in challenging situations. Strong SoC has been described as being a buffer against social stress (Richardson & Ratner, 2005), much like social support.

3.9.2 SoC and Biology

In this section research which has investigated SoC with cholesterol and blood pressure will be explored.

Svartvik et al., (2000 & 2002) discovered lower lipid levels, with low levels of high-density lipoproteins (HDLs) and high levels of triglycerides in women reporting a weak SoC, compared to women with a stronger SoC. Additionally, women with a higher SoC reported less symptoms of ill health. These studies embark on researching the relationship between SoC, health behaviours and physiological processes.

In a study of healthy non smoking premenopausal middle-aged women, those with a strong SoC had significantly lower systolic blood pressure and total cholesterol compared with women with a weak SoC. This may show that women with a strong SoC can manage stress better than women with a weak SoC, therefore this is demonstrable in these biomedical markers (Lindfors, Lundberg & Lundberg 2005). However, more studies linking SoC with health should be encouraged.

3.9.3 SoC and Disease

SoC has been associated with the reduction of pain in a sample of 387 older patients with chronic illnesses (Wiesmann, Dezutter, & Hannich, 2014), atopic disease (Takaki & Ishii, 2013), postmenopausal women with recently diagnosed primary or recurrent breast cancer (Kenne Sarenmalm, Browell, Persson, Fall-Dickson, & Gaston-Johansson, 2013), cancer and heart disease (Surtees, Wainwright, Luben, Khaw, & Day, 2003), stomach problems, dyspepsia, diabetes, heart disease and stroke (Nilsson, Holmgren, & Westman, 2000). During follow up of the EPIC-Norfolk study those with a strong SoC, had 20% lower risk of all-cause mortality compared with those with a weak SoC (Wainwright et al., 2008).

Twenty one thousand participants were recruited in the European Prospective Investigation into Cancer (EPIC-Norfolk) study through GP registers. SoC was associated with a higher mortality rate (Surtees, Wainwright, Luben, Khaw, & Day,

2003). Also, those with a strong SoC demonstrated quicker adaption to adverse experiences compared to those with a weak SoC (Surtees, Wainwright, & Khaw, 2006).

SoC was also associated with psychosocial factors of rehabilitation but not physical factors. Antonovsky's theory purports, that SoC would have an influence on both physical and psychological aspects of disease (Benz, Angst, Lehmann, & Aeschlimann, 2013). Therefore there is some debate in the literature.

SoC affects health behaviours. People with a high SoC are more likely to eat healthily and not smoke (Wainwright et al., 2007), have lower risk of alcohol problems (Midanik, Soghikan, Ransom, & Polen, 1992) and less coronary heart disease (Poppius, Tenkanen, Hakama, & Heinsalmi, 1999) which would ultimately affect physical health.

3.9.4 SoC and Cardiovascular Disease

SoC has been reported to affect coronary heart disease patients. In a longitudinal study of HRQoL in coronary heart disease patients, measured at baseline, and followed up between 1-2 years follow up, SoC predicted HRQoL (Silarova et al., 2012).

3.9.5 SoC and Stroke

There have been limited studies on stroke patients and SoC with the largest reported study being the EPIC-Norfolk study, with nearly twenty two thousand participants. In this study stroke risk was assessed from participants recruited from GP registers. Surtees et al., (2006) tested the use of SoC (using a 3 item measure) for stroke risk. The findings revealed a weak SoC was independently related to stroke risk after controlling for risk factors for stroke.

However, most studies focus on SoC and caregiver burden not on stroke patients (Van Puymbroeck, Hinojosa, & Rittman, 2008; Chumbler, Rittman, & Wu, 2008; Forsberg-Warleby, Moller, & Blomstrand, 2002).

3.9.6 Interventions for SoC

There are at the present no intervention strategies for SoC (Brainin & Tuomilehto, 2007).

3.9.7 Measures of SoC

SoC was originally a 29 item scale which was also shortened to a 13 item scale by its original creator Antonovsky (Antonovsky 1979; 1987). For this research study the 3-item measure (Lundberg & Nystrom Peck, 1995) was used because it has been previously used in stroke research (Surtees et al., 2006) and it is easy to administer. However, this is a short scale with a restricted range of responses. More details for this measure are described in Chapter 4, Section 4.8.4, p.224.

In the next section cognitive factors will be reviewed with the view to incorporating specific measures into the research design.

3.10 Cognitive factors

Typically in Health Psychology research cognitive factors are not incorporated therefore there are limited studies which can be reported. Cognitive factors are important in recovery from stroke and therefore will be acknowledged in this thesis and incorporated into the design of Study Two. Accordingly, cognitive factors will be discussed in this section.

Cognitive impairment after stroke can affect half the stroke population (Hochstenbach, den Otter, & Mulder, 2003) and therefore is a persistent culprit which influences the experience and recovery from stroke. However, neuropsychological consequences of stroke can often be overlooked (Dennis, O'Rourke, Lewis, Sharpe, & Warlow, 2000).

Many studies use the Mini Mental State Examination (MMSE) (Folstein, Folstein, & McHugh, 1975) which is a short test for dementia (Nys et al., 2005) but does not measure any other cognitive impairment (Jaillard, Naegele, Trabucco-Miguel, LeBas, & Hommel, 2009). However, it is used as a general measure for cognitive impairment which is inadequate (Fatoye et al., 2007). Using a cognitive battery can expose more cognitive impairment rather than relying on the MMSE (Jaillard, Naegele, Trabucco-Miguel, LeBas, & Hommel, 2009). Therefore, the MMSE will not be used in this study.

Cognitive impairment can predict functional outcome (Paolucci et al., 1996; Zinn, Dudley, Bosworth, Hoenig, Duncan, & Horner, 2004; Oksala, Jokinen, & Melkas, 2009) and is associated with dependent living (Tatemichi et al., 1994;

Pohjasvaara, Erkinjuntti, Vataja, & Kaste, 1998), depression and reduced quality of life (Nys et al., 2006).

There are 4 main cognitive domains: Language, memory, visuo-spatial disturbances and executive function (Kolb & Wishaw 2009; Baars & Gage, 2010). These will be briefly discussed in the following sections.

3.10.1 Language

Language impairments predominately occur with stroke lesions in the left hemisphere of the brain in the parietal lobe (Purves et al., 2008; Kolb & Wishaw 2009). This can result in dysphasia (language impairment) or aphasia (total loss of language). Dysphasia is the common language cognitive impairment which occurs after stroke. There are three main types; Expressive dysphasia, receptive dysphasia and a combination of both. Expressive dysphasia is when a person can fully understand what is being said to them but they have difficulties when trying to express a reply. Receptive dysphasia is when the person has trouble understanding and receiving information being said to them and the third type is a combination of both (Warlow, 2008). As language impairments can cause difficulties with research designs and gaining informed consent many stroke patients with language impairments are excluded. In a systematic review investigating depression and inclusion and exclusion of aphasic patients after stroke from a total of 129 studies, 13 studies acknowledged aphasia. From the remaining 116 studies, 92 studies excluded aphasic patients (Townend, Brady, & McLaughlan, 2007).

However, excluding these patients reduces the generalizability of results (Townend, Brady & McLaughlan 2007). In order to retain these patients proxy measures may be used (Hilari & Northcott 2006). Proxy measures are when a third party (e.g., a family member or health professional) answers the questions on behalf of the participants. However proxy responses are subjective and may not be reflective of the participant's true responses (Sneeuw et al., 1997).

In the sections below language impairment (dysphasia) studies with the psychological factors identified previously in regards to stroke recovery are outlined.

3.10.1.1 Dysphasia, Depression & Stroke

In a study of 61 participants Lim & Ebrahim (1983) concluded that dysphasic patients are less likely to have their depressive symptoms acknowledged and dealt with as these patients have difficulty in communicating their symptoms to medical staff. It is little wonder that dysphasia can result in depression.

Depression has been associated with dysphasia in a study of 106 participants with first time stroke with 3 and 12 month follow up (Kauhanen et al., 1999) and in a 3 year longitudinal study with 80 participants (Astrom, Adolfsson, & Asplund, 1993). In this study 25% of patients had major depression at onset. This increased to 31% at 3 months, declined to 16% at 12 months and increased to 29% at 3 years post stroke. These two studies were the only ones reported in a systematic review on psychosocial risk factors and dysphasia (Ouimet, Primeau, & Cole 2001). Communication impairment was a strong predictor of depression at 6 months post stroke follow up in a study of 123 stroke patients in the UK (Thomas & Lincoln, 2006). This can impact on stress levels and ability to cope (Laures-Gore, Hamilton, & Matheny, 2007).

3.10.1.2 Dysphasia, Stress & Stroke

No studies found were found in a search of dysphasia, psychological stress and stroke recovery.

3.10.1.3 Dysphasia, Social Support & Stroke

Language impairments can adversely affect social support as isolation can arise (Sarno, 1997). Independence can also be affected with relationships with family increasing, whilst relationships with friends decrease (Hilari & Northcott, 2006). In a 3 year longitudinal study with 80 stroke participants, major depression was associated with social isolation and dysphasia (Astrom, Adolfsson, & Asplund, 1993).

3.10.1.4 Dysphasia, Type D Personality & Stroke

No studies were found on dysphasia, Type D personality and stroke recovery.

3.10.1.5 Dysphasia, Repressive Coping & Stroke

No studies were found on dysphasia, repressive coping and stroke recovery.

3.10.1.6 Dysphasia, Sense of Coherence & Stroke

No studies were found on dysphasia, SoC and stroke recovery.

Language impairments are important to acknowledge as it is one of the cognitive domains, however this domain is not a main focus of the thesis.

In the next section verbal and visual short term memory will be discussed.

3.10.2 Memory

Stroke lesions can cause deficits in memory functions of survivors. In particular short term memory is of an interest and is commonly affected after a stroke (Stroke Association, 2015). The dorsolateral prefrontal cortex is important for working memory which manages short term verbal memory and short term visual memory (Banich 2004). This section will briefly discuss the importance of short term verbal memory and short term visual memory with depression, stress, social support, Type D personality, repressive coping and sense of coherence in relation to stroke recovery.

3.10.2.1 Verbal Short Term Memory

Short term memory is information currently maintained by the brain for a limited time and capacity (Ward 2010). Working memory allows for the short term recall of items, normally about 7 items for 10 seconds (Banich 2004). Short term memory problems after stroke increase the need for length of stay in hospital and the need for therapies post discharge. Memory problems can hinder rehabilitation due to not remembering when and how to do exercises or not adhering to treatment medications (Galski, Bruno, Zorowitz, & Walker, 1993).

In the sections below verbal memory studies with the psychological factors identified previously in regards to stroke recovery are outlined.

3.10.2.1.1 Verbal Short Term Memory, Depression & Stroke

Memory and depression were reported as important cognitive and emotional consequences of stroke in a study of 111 participants 3 months post stroke (Passier, Visser-Meily, & van Zandvoot, 2010). Memory impairment can contribute to the continuation of depression in stroke patients (Kauhanen et al., 1999). Memory, language and visuoperception impairments have been associated with moderate to severe depression in stroke patients (Nys et al., 2005).

3.10.2.1.2 Verbal Short Term Memory, Stress & Stroke

No studies were found on verbal short term memory, stress and stroke recovery.

3.10.2.1.3 Verbal Short Term Memory, Social Support & Stroke

No studies were found on verbal short term memory, social support and stroke recovery.

3.10.2.1.4 Verbal Short Term Memory, Type D Personality & Stroke

No studies were found on verbal short term memory, Type D personality and stroke recovery.

3.10.2.1.5 Verbal Short Term Memory, Repressive Coping & Stroke

No studies were found on verbal short term memory, repressive coping and stroke recovery.

3.10.2.16 Verbal Short Term Memory, Sense of Coherence & Stroke

No studies were found on verbal short term memory, SoC and stroke recovery.

As there are prominent gaps in the literature in these areas it would be advantageous for research to address these.

3.10.2.17 Measures of Verbal Short Term Memory

Short term memory is normally tested using span tasks. The mechanisms involved in this could include a phonological store and rehearsal of the information

subvocally (Baddeley & Hitch, 1974; Baddeley, Lewis, & Vallar, 1984). The most frequently used span task is the Forward Digit Span (Wechsler 1945). The Forward Digit Span is a test of verbal working memory capacity and attention, where the participant is asked to repeat a sequence of numbers in the same order. This test is further explained in Chapter 4, Section 4.8.5.

In the next section, visual memory is discussed.

3.10.2.18 Visual Short Term Memory

For visual short term memory the visuospatial sketchpad is used as a visual store and is controlled by the central executive. Much like the rehearsal that is entailed with verbal short term memory, visual short term memory also entails the rehearsal of visual images (Baddeley & Hitch, 1974; Baddeley, Lewis, & Vallar, 1984).

Reductions in visual memory have been associated with disability and lower quality of life in a study of 307 participants 5 years post stroke (Barker-Collo, Feigin, Parag, Lawes, & Senior, 2010) and low visual memory has been related to poorer physical activity one year after stroke (Pahlman, Savborg, & Tarkowski, 2012). However, other studies in this area are lacking.

In the sections below visual short term memory studies with the psychological factors identified previously in regards to stroke recovery are outlined.

3.10.2.19 Visual Short Term Memory, Depression & Stroke

Depressed stroke patients have more chance of also having cognitive impairment (Downhill & Robinson 1994) such as short term memory and visual memory impairments although this area is in need of further research (Barker-Collo 2007).

3.10.2.20 Visual Short Term Memory, Stress & Stroke

No studies were found on visual short term memory, psychological stress and stroke recovery.

3.10.2.21 Visual Short Term Memory, Social Support & Stroke

No studies were found on visual short term memory, social support and stroke recovery.

3.10.2.22 Visual Short Term Memory, Type D Personality & Stroke

No studies were found on visual short term memory, Type D personality and stroke recovery.

3.10.2.23 Visual Short Term Memory, Repressive Coping & Stroke

No studies were found on visual short term memory, repressive coping and stroke recovery.

3.10.2.24 Visual Short Term Memory, Sense of Coherence & Stroke

No studies were found on visual short term memory, SoC and stroke recovery.

3.10.2.25 Measures for Visual Short Term Memory

A frequently used measure for visual memory is the Rivermead Behavioural Memory Test (Wilson, Cockburn, & Baddeley, 1985). This test is a battery of tests for visual short term recognition memory. Of particular interest is the object recognition test. This test will be further explained in Chapter 4, Section 4.8.5.

In the next section visuo-spatial impairment is discussed.

3.10.3 Visuo-spatial impairment

A frequent visuo-spatial impairment suffered by stroke survivors is hemispatial neglect (or visual neglect). This is when there is intact sensory and motor function but one side of visual space is neglected. This occurs after a lesion primarily in the right parietal lobe as the parietal lobe processes spatial information (Ward, 2010). Hemispatial neglect is the decreased awareness of stimuli on the patients contralesional side (on the opposite side of vision, from the side of stroke lesion), but acknowledge the side of vision on their ipsilesional side (same side of vision as the stroke lesion) (Parton, Malhotra, & Husain, 2004).

Even in a darkened room neglect patients still only look at the non-neglected side of space which strongly suggests this is not a problem with visual processing but a problem of impaired attention as only specific information is selected for processing (Banich 2004). Practising visual scanning can help with visual neglect (Cicerone et al., 2000) and using an eye patch encourages the patient to be more conscious turning their head and looking around **(**Jutai et al., 2003). Visual neglect can improve over time, with up to 70% of patients recovering at 3 months post stroke (Jutai et al., 2003), however the effects of neglect may not completely disappear (Banich 2004).

The role of visual neglect on stroke recovery has mixed findings. Some studies suggest there is a relationship, whilst others do not. Visual neglect and older age have been reported as predictors of poor functional outcome at 3, 6 and 12 month follow up in a study of 57 stroke patients (Jehkonen et al., 2000). Visual neglect may hinder the effects of functional recovery (Sunderland, Wade, Langton, & Hewer, 1987; Bailey, Riddoch, & Crome, 2002) and rehabilitation (Barrett & Muzaffar, 2014). Rehabilitation could include exercises for neglect which will aid traditional rehabilitation. Improvements in visual neglect would help patients to functionally recover by performing physical tasks better (Jones & Shinton 2006; van Wyk, Eksteen, & Rheeder, 2014). In a study of 113 patients from Hong Kong, neglect has been concluded to adversely affect recovery as visual problems may cause injuries as the patient is unaware of their surroundings. (Siong, Woo, & Chan, 2014).

In a Cochrane systematic review of visual neglect training and effects on activities of daily living, no conclusions were drawn (Pollock et al., 2011), suggesting visual neglect has not been researched fully with functional recovery and attempts at repairing neglect often do not incorporate also improving physical recovery (Vossell, Kukolja, & Fink, 2010). It is apparent that more research in this area is needed.

In the sections below visual neglect studies with the psychological factors identified previously in regards to stroke recovery are outlined.

3.10.3.1 Visual Neglect, Depression & Stroke

Visuospatial tasks can be ill performed by depressed patients (Elliott et al., 1996) and can affect depression in stroke patients (Tsai et al., 2003). One hundred

and forty three patients were assessed 3 weeks post stroke. Acute neglect was a strong predictor of depression in a first ever study on this subject (Nys et al., 2006). Further studies exploring visual neglect, depression and stroke are lacking.

3.10.3.2 Visual Neglect, Stress & Stroke

No studies were found on visual neglect, psychological stress and stroke recovery.

3.10.3.3 Visual Neglect, Social Support & Stroke

No studies were found on visual neglect, social support and stroke recovery.

3.10.3.4 Visual Neglect, Type D Personality & Stroke

No studies were found on visual neglect, Type D personality and stroke recovery.

3.10.3.5 Visual Neglect, Repressive Coping & Stroke

No studies were found on visual neglect, repressive coping and stroke recovery.

3.10.3.6 Visual Neglect, Sense of Coherence & Stroke

No studies were found on visual neglect, SoC and stroke recovery.

3.10.3.7 Measures of Visual Neglect

These disturbances are best identified with more than one validated task of two of the most sensitive tasks: a line bi-section and a cancellation task (Azouvi et al., 1996; Ferber & Karnath, 2001). A detailed explanation of these measures will be given in Chapter 4, Section 4.8.5.

In the next section executive function is discussed.

3.10.4 Executive Function

Executive function is a meta-cognitive function and controls which functions are utilised and which functions are not. Therefore it governs different areas such as language, perception, memory and so on (Goldberg, 2001). Executive dysfunction is the lack of flexibility in processing information where automatic responses are resisting controlled responses (Banich 2004). Controlled behaviour would elicit executive function (Ward 2010). Executive function can be affected by high stress and depression (Lawrence & Grasby, 2001). Some processing demands controlled attention (less practised behaviours), whilst other processing is automatic (practised behaviours) (Shiffrin & Schneider, 1977; Banich, 2004). The anterior cingulate cortex is activated when there is response conflict (Bench et al., 1993; Banich, 2004) and processes cognitively demanding information and response selection (Gruber, Rogowska, Soraci, & Yurgelun-Todd, 2002).

Executive function has been found to be related to a decrease in activities of daily living in stroke patients (Chung, Pollock, Campbell, Durward, Hagen, 2013; Middleton, Lam, & Fahmi, 2014). However, direct effects in stroke recovery are lacking.

In the sections below executive function studies with the psychological factors identified previously in regards to stroke recovery are outlined.

3.10.4.1 Executive Function, Depression & Stroke

There is uncertainty if depression causes cognitive impairment or if cognitive impairment causes depression (Spalletta, Guida, & Caltagirone, 2003). Symptoms of depression may cause cognitive impairment, although the impairment effects may not last (Nussbaum 1994).

Burt et al., (1995) concluded people with depression reported remembering negative information rather than positive information. This supports other findings which assert depressed people do not process all available information that could help them in problem solving (Conway and Giannopoulous 1993), which demonstrates a bias in attention.

Executive function and depression has been identified as being present at the same time in 22% of stroke patients. Patients with both do have more problems with activities of daily living but this was not statistically significant. Symptoms of

executive dysfunction and depression remained for 2 years (Bour, Rasquin, Limburg, & Verhey, 2011). Studies focusing on executive function and depression in relation to stroke recovery are lacking.

3.10.4.2 Executive Function, Stress & Stroke

No studies were found on executive function, psychological stress and stroke recovery.

3.10.4.3 Executive Function, Social Support & Stroke

No studies were found on executive function, social support and stroke recovery.

3.10.4.4 Executive Function, Type D Personality & Stroke

No studies were found on executive function, Type D personality and stroke recovery.

3.10.4.5 Executive Function, Repressive Coping & Stroke

No studies were found on executive function, repressive coping and stroke recovery.

3.10.4.6 Executive Function, Sense of Coherence & Stroke

No studies were found on executive function, SoC and stroke recovery.

3.10.4.7 Measures for Executive Function

The most classic test for executive function is the colour word Stroop test (Stroop 1935). This test presents a list of words in different colours. The participant has to ignore the colour ink the word is written in, but read the word it spells (e.g., the word “blue” will be written in the colour ink red). This test will be used in the current study. More detailed explanation will be given in Chapter 4, Section 4.8.5.

3.11 Conclusion for the Inclusion of Cognitive Factors

Cognitive factors occur frequently with stroke. As can be seen from the literature review there are many gaps in the literature where cognition has not been researched fully with factors such as depression, stress, repressive coping, Type D personality and SoC in relation to recovery from stroke. This provides a compelling justification for the inclusion of cognitive measures, in an attempt to address these gaps in research.

In the next section physical recovery will be addressed.

3.12 Physical Recovery

From the previous literature discussed thus far psychological variables have been reported to affect physical recovery from stroke. The two main measures for recording physical recovery are discussed below.

3.12.1 Measures for Physical Recovery

The two main measures that are used for rating clinical physical functioning in stroke are the Barthel Index (BI) (Mahoney & Barthel, 1965) and the modified Rankin Scale (mRS) (Bonita & Beaglehole, 1988) (Roberts & Counsell, 1998; Sulter, Steen, & De Keyser, 1999).

The Barthel Index (Mahoney & Barthel, 1965) is a 10 item observer rated scale for activities of daily living recording feeding, bathing, grooming, dressing, bowel function, bladder function, toilet use, mobility, transferring to bed and to chair and walking up the stairs.

The modified Rankin Scale (mRS) (Bonita & Beaglehole, 1988) is an observer rated scale and is used for measuring the degree of disability or dependence of people who have suffered a stroke. The scale is categorised from no symptoms to slight disability, to moderate disability, to severe disability to death. The mRS is more in keeping with the research question which focuses on physical recovery and less on activities of daily living, therefore the mRS was chosen for inclusion in the study. A detailed explanation of this measure will be given in Chapter 4 Section 4.8.7 (p. 218).

Physical recovery has been the main outcome measure associated with the systematic review in Chapter 2 and the review of studies in this chapter. The addition

of QoL will also be added as an outcome variable therefore covering physical and psychological recovery. In the following sections QoL will be discussed in relation to the existing variables and recovery from stroke, along with interventions and measures of QoL.

3.13 Quality of Life (QoL)

3.13.1 Definition of QoL

Quality of life is a subjective construct and there is no universal definition (Kim, Warren, Madill, & Hadley, 1999). However, the World Health Organisation (WHO) has defined QoL as an "individual's perceptions of his position in life, in the context of the culture and value system in which he lives and in relation to his goals, standards, and concerns" (WHOQoL Group, 1998, p. 551). There are many different quality of life definitions, however it is believed QoL should include psychological, physical, social, functional and general health aspects (Kauhanen et al., 2000). This lack of a clear definition of QoL makes it difficult to compare studies (de Haan et al., 1993).

3.13.2 QoL and Disease

QoL has been reported to have an effect on various health conditions such as Crohn's disease (Gazzard, 1987), gastro-oesophageal reflux disease (Irvine, 2004), multiple system atrophy (Krismer et al., 2013), hypertension (Agewell, Wikstrand, & Fagerberg, 1998) and heart disease (Hofer, Lim, Guyatt, & Oldridge, 2004).

QoL can be influenced by other psychosocial factors and additionally, there are conflicting findings in the literature regarding the effect of QoL on stroke recovery. Some studies report the increase of QoL when participants adjust to stroke, and some studies report a steady decrease in QoL. The following sections will discuss the relationship between QoL, depression, stress, social support, Type D personality, repressive coping, SoC and recovery from stroke.

3.13.3 QoL, Depression & Stroke

Depression may have an adverse effect on health related quality of life. Participants with more depressive symptoms and low social support report lower QoL. Depression has accounted for 32% of the variance and social support accounted for 9% of the variance in a multiple regression analysis on recovering stroke patients (Kim, Warren, Madill, & Hadley, 1999). Depression was predictive of lower QoL, whilst treated depression was associated with improved QoL (Naess, Waje-Andreassen, Thomassen, Nyland, & Mhyr, 2006). Four years post stroke depression and cognitive impairment predicted lower QoL (Haacke et al., 2006) and functional and cognitive impairment were associated with lower QoL, in a cross sectional study of first ever stroke patients (Gurcay, Bal, & Cakci, 2009). However, cognitive impairment was assessed using the Mini-Mental State Examination (MMSE) (a short critique of MMSE studies are given in Section 3.10).

QoL has frequently been reported to be influenced by depression and reduced physical independence in stroke patients (Lofgren, Gustafson, & Nyberg, 1999; Carod-Artal, Egido, Gonzalez, & de Seijas, 2000; Patel, McKevitt, Lawerence, Rudd, & Wolfe, 2007). Additionally in a study of stroke, myocardial ischemia and lower back pain patients, depressed patients reported lower QoL (Fruhwald, Loffler, Eher, Saletu, & Baumhackl, 2001). Depressed post stroke patients reported lower levels of QoL compared with non-depressed patients in a 6 month longitudinal study (Teoh, Sims, & Milgrom, 2009). Depression was also negatively associated with QoL in a sample of stroke patients in a prospective cohort of Chinese patients, however QoL increased with increasing physical improvements (Kwok et al., 2006). These studies suggest depression has a negative influence on QoL.

3.13.4 QoL, Stress & Stroke

Studies investigating QoL, stress and stroke mainly focus on caregiver burdens (Scholte op Reimer, de Haan, Rijinders, Limburg, & van den Bos, 1998; Gaugler, 2010; Jaracz, Grabowska-Fudala, & Kozubski, 2012; Kniepmann, 2012; Bhattacharjee, Vairale, Gawali, & Dala, 2012; Clay et al., 2013).

Limited studies have been conducted on QoL, psychological stress and stroke recovery however, Baune & Aljeesh (2006) conducted a study on patients in the Gaza Strip with hypertension and stroke and the relation between psychological

stress and QoL. They concluded psychological stress was related to one domain of the WHOQoL-BREF (the Global domain), whilst being insignificant in the remaining domains (physical, psychological, social and environmental domains). More studies in this area should be conducted.

3.13.5 QoL, Social Support & Stroke

Social support can protect against declining QoL in stroke patients (Tang et al., 2005) or enhance declining QoL with decreased social support (King, 1996). In a qualitative study on post stroke survivors and their caregivers the importance of social relationships was the main theme that arose in regard to factors which are salient to QoL. Survival of stroke has been described as a "social effort" by this team as stroke survivors invariably become dependent on others (Lynch et al., 2008, p.522).

Communication impairments can have an impact on social support. Patients with communicative impairments that had more contact with their children and relatives after stroke rated their QoL as lower compared with patients with communication impairments who had the same amount of contact. However, the low scoring group could be more afflicted by physical impairments (therefore needing more social support) and the higher scoring group could be benefitting from a stronger sense of control (Chow, 1997). Patients with communication impairments who had more contact with their friends reported higher QoL. This could be due to having a social life outside their home (Hilari & Northcott, 2006). In a Polish study participants that lived with family scored higher on QoL, whilst depression and physical disability impacted on QoL in this sample (Jaracz & Kozubski, 2003). Emotional support and marital status were predictive of good QoL in Polish stroke patients (Jaracz, Jaracz, Kozubski, & Rybakowski, 2002). Additionally, in a study of 100 patients whom were discharged after 1 year there was a relationship between social support and QoL (Gottlieb, Golander, & Bar-Tel, 2001). In another study, the environment and social interaction components of QoL were lower at 12 months post stroke compared with 3 months post stroke, despite physical functioning remaining unchanged. This demonstrates how lack of social support can affect recovery (Kwok et al., 2006).

3.13.6 QoL, Type D Personality & Stroke

No studies were found on QoL, Type D personality and stroke recovery.

3.13.7 QoL, Repressive Coping & Stroke

No studies were found on QoL, repressive coping and stroke recovery.

3.13.8 QoL, Sense of Coherence & Stroke

No studies were found on QoL, SoC and stroke recovery.

3.13.9 QoL, Verbal & Visual Short Term Memory & Stroke

QoL and verbal memory have been reported to have no relationship in aneurysmal subarachnoid hemorrhages (Al-Khindi, MacDonald & Schweizer, 2010). However, studies in this area are lacking. No studies were found on QoL and short term visual memory.

3.13.10 QoL, Visual Neglect & Stroke

In a study of 143 patients acute neglect was a strong predictor of QoL 6 months post stroke in a first ever study on this subject (Nys et al., 2006) and visual neglect at discharge has been associated with lower QoL in 528 Italian patients (Franceschini, La Porta, & Agosti, 2010). However, no other studies were found on QoL and visual neglect in stroke patients.

3.13.11 QoL, Executive Function & Stroke

In a study of 45 stroke patients executive function was found to have a direct effect on QoL in a regression analysis (Brookes et al., 2014). However QoL and executive function were analysed in a study of 81 post stroke patients, where no conclusions were drawn (D'Aniello et al., 2014) with similar results being echoed by Al-Khindi, MacDonald, & Schweizer, 2010. However, more studies should be conducted in this area.

3.13.12 QoL, Physical Recovery & Stroke

There seem to be mixed findings regarding the relationship between QoL and physical recovery from stroke. The relationship between stroke and QoL has been

reported to be strong with stroke patients consistently scoring low on QoL (Wyller et al., 2006), whilst in an Australian stroke sample patients had similar QoL compared to the general public even though their physical functioning was poorer (Hackett, Duncan, Anderson, Broad, & Bonita, 2000). Six years after stroke there seems to be good adjustments in QoL which was evident in a sample of 1761 patients, with 639 patients survived at 6 years (Hackett, Duncan, Anderson, Broad, & Bonita, 2000). Therefore participants with disabilities can maintain a good QoL (Albrecht & Devlieger, 1999) as severe stroke patients have reported higher QoL compared with those with moderate strokes (Engs, Yu, & Luistro, 2001). These changes may be due to response shifting.

QoL as measured using the Sickness Impact Profile improved over 3 months post stroke in a sample of patients from Hong Kong with functional ability being the strongest predictor of QoL (MacKenzie & Chang, 2002). This is disputed by Pan, Song, Lee, & Kwok, (2008), who assert mood is more important than functional status in QoL as functional gains did not change one year after stroke in their study, however depression did and this was related to QoL.

Studies which report consistent low QoL with stroke include a Canadian study in which physical improvements post stroke do not necessarily translate into improvements in QoL. This study did not take into account depression and cognitive impairment therefore they cannot conclude which factors may affect this relationship (Madden, Hopman, Bagg, Verver, & O'Callaghan, 2006).

In a German sample of elderly stroke patients after 1 year follow up patients had increased physical functioning but a reduction in QoL. Significant others became more important and patients viewed themselves as not significant to other people (Lalu, 2003). It seems that a drop in self-esteem has resulted in a decreased QoL.

Between 1 month and 6 month post stroke there was no significant change in QoL scores in first ever ischemic Chinese stroke patients, however length of stay in hospital did predict QoL (Lee, Tang, Tsoi, Fong, & Yu, 2009). These authors used the Rankin Scale to measure QoL with a single item measure. Consequently, these results should be treated with caution as the Rankin scale records physical disability and is not a measure of QoL.

In a longitudinal study measuring 3, 6 and 12 months post stroke QoL, QoL decreased over the 12 months despite there being stable neurologic and physical functioning (Suenkeler et al., 2002).

Disability has a negative effect on QoL in stroke (Jonnson, Lindgren, Hallstrom, Norrving, & Lindgren, 2005; Nichols-Larsen, Clark, Zeringue, Greenspan, & Blanton, 2005). In a study of first ever stroke patients QoL was found to be not significant in physical recovery after stroke. Levels of distress did not improve as physical recovery improved (Horgan, O'Regan, Cunningham, & Finn, 2009) this may be due to age and functional dependence (Kwa, Limburg, & de Haan, 1996).

There does seem to be a trend indicating that after a stroke QoL decreases due to loss of independence (Gallien et al., 2005) but despite significant ongoing physical disability survivors of stroke can appear to adjust well psychologically to their illness within time (Hackett et al., 2000). In a study from Auckland, New Zealand, stroke participants were followed up for 2 decades. QoL improves over time despite living with disability (Anderson et al., 2004). In a study of stroke patients with 4 year follow up patients did not achieve pre-QoL status despite making good recovery and independence in living situations (Niemi, Laaksonen, Kotila, & Waltimo, 1988).

However, it is important to acknowledge the stroke severity of participants, as without this information these findings cannot be placed into context and many of these studies do not report on this. It is usual for health psychological studies to not report on stroke characteristics which ultimately causes difficulties when comparing across studies.

3.13.13 QoL Intervention

There are no specific interventions for QoL however, as depression and social support can affect QoL interventions in those areas will have a repercussion on QoL.

3.13.14 Measures of QoL

Below are some of the most frequently used QoL measures in the literature. These have been briefly critiqued in order to choose an appropriate measure.

a) **The Stroke Impact Scale (Duncan, Wallace, Lai, Johnson, Embretson, Laster, 1999)** is a 59 item measure recording physical functioning, memory, attention, mood, communication, activities of daily living, mobility, hand use and community participation. An additional question asks the participant to rate their recovery as a percentage.

This measure may produce multicollinearity because of the repetition in factors being measured for example, physical functioning, memory, attention, mood and mobility. Additionally this measure is too long to use in an acute stroke setting, therefore this measure was not considered for use in the current study.

b) **Stroke Specific Quality of Life Scale (Williams, Weinberger, Harris, Clark, & Biller, 1999)** is a 50 item measure with each item weighted differently. This measure records energy, family roles, language, mobility, mood, personality, self care, social roles, thinking, upper extremity function, vision and work productivity. Many of the items measured here are repeated in the main set of variables chosen for analysis and so there will be the issue of multicollinearity if this questionnaire is used. Also, the length of the questionnaire is too long to be used in conjunction with the other measures. For this reason this questionnaire was not considered any further.

c) **Burden of Stroke Scale (Doyle, McNeil, Mikolic, Prieto, Hula, Lustig, Ross, Wambaugh, Gonzalez-Rothi, & Elman, 2004)** is a 64 item measure recording physical limitations (mobility, self-care and swallowing), psychological distress (mood, satisfaction, restriction, energy & sleep), and cognitive limitations (communication, cognition and social relations). This measure overlaps with variables chosen to be in the study such as mood, physical limitations and cognitive limitations. This can produce multicollinearity. Also as this measure is long it is not viable to use alongside the other study variables, and therefore this measure was disregarded.

d) The Medical Outcomes Study 36-item short-form health survey (SF-36) (Ware Jr. & Sherbourne, 1992) is one of the most frequently used measures for QoL (Kalantar-Zadeh, Kopple, Block, & Humphreys, 2001). This is a 36 item scale measuring general health in participants which is comprised of two components measuring physical (physical functioning, role physical, bodily pain and general health) and mental (vitality, social functioning, role emotional and mental health) outcomes. Therefore there are both objective (physical functioning) and subjective (feelings and experiences) (George & Bearon, 1980) components.

The SF 36 is a general measure of health related quality of life and not a specific stroke measure (Anderson, Laubscher, & Burns, 1996). However stroke measures tend to be too long and are difficult to administer in an acute stroke setting. In a study by Carod-Artal, Egido, Gonzalez, & Varela de Seijas (2000) QoL was measured in 118 stroke patients using the SF-36 and the Sickness Impact Profile. The results demonstrated that both measures were interrelated. Because of this Suenkeler et al., (2002) justified using the SF-36 in their longitudinal study of stroke and QoL because the SF-36 has been well validated in studies with other illnesses and therefore enables comparisons and it is more participant friendly to answer. For these reasons the SF 36 was included in the current study. More details of this measure are described in Chapter 4, Section 4.8.6 (p.217).

3.14 Conclusion and Current Study

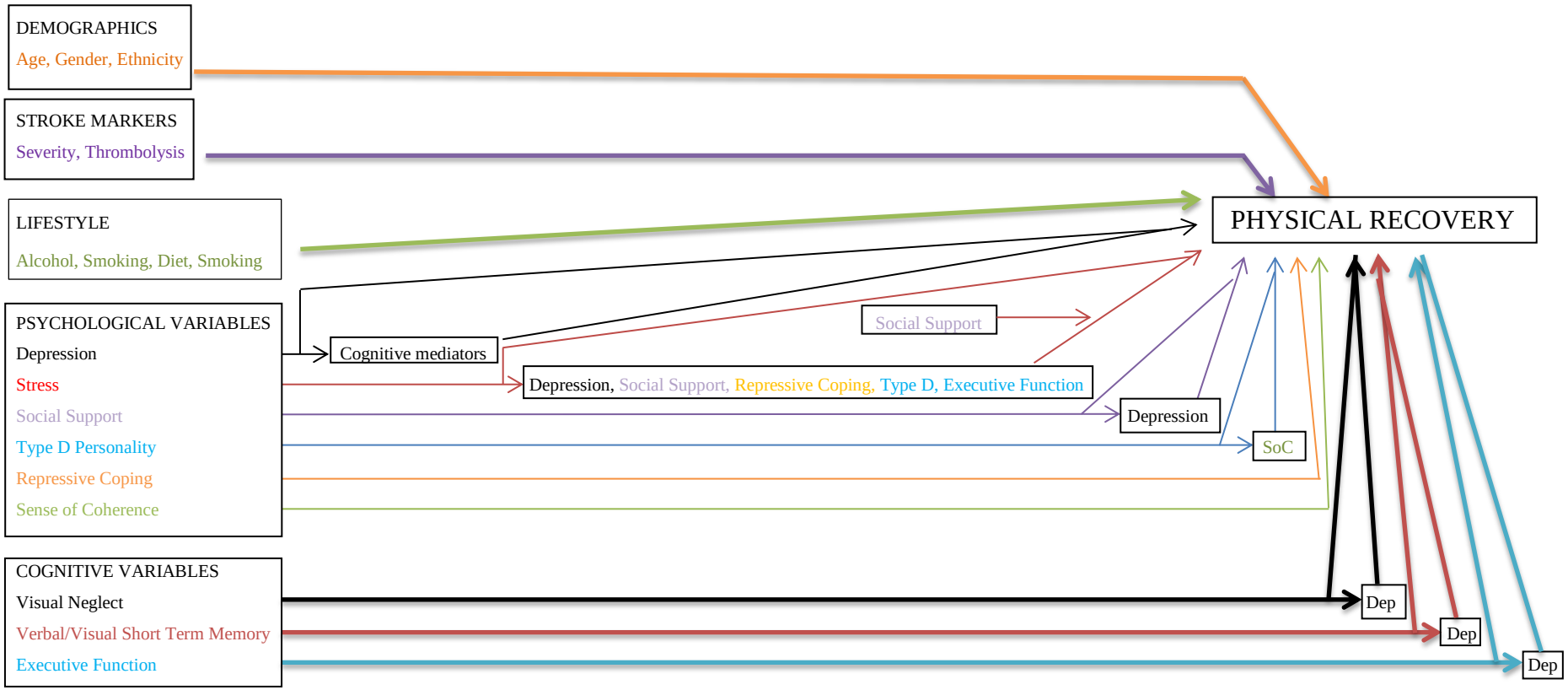
From the review of the literature depression, stress, social support, Type D personality, repressive coping, SoC and cognitive factors (verbal short term memory, visual short term memory, visual neglect and executive function) will be investigated in regard to their influence on psychological (QoL) and physical recovery from stroke.

In Sections 3.16 and 3.17, two theoretical models based on the literature are presented, one for physical recovery as the outcome and one for QoL as the outcome, predicted by psychological and cognitive factors. These models are the same for each time point. In Section 3.18, the research hypotheses to be tested are outlined and an explanation of how they were constructed is explained.

3.15 Theoretical Model for Physical Recovery

Figure 3.1

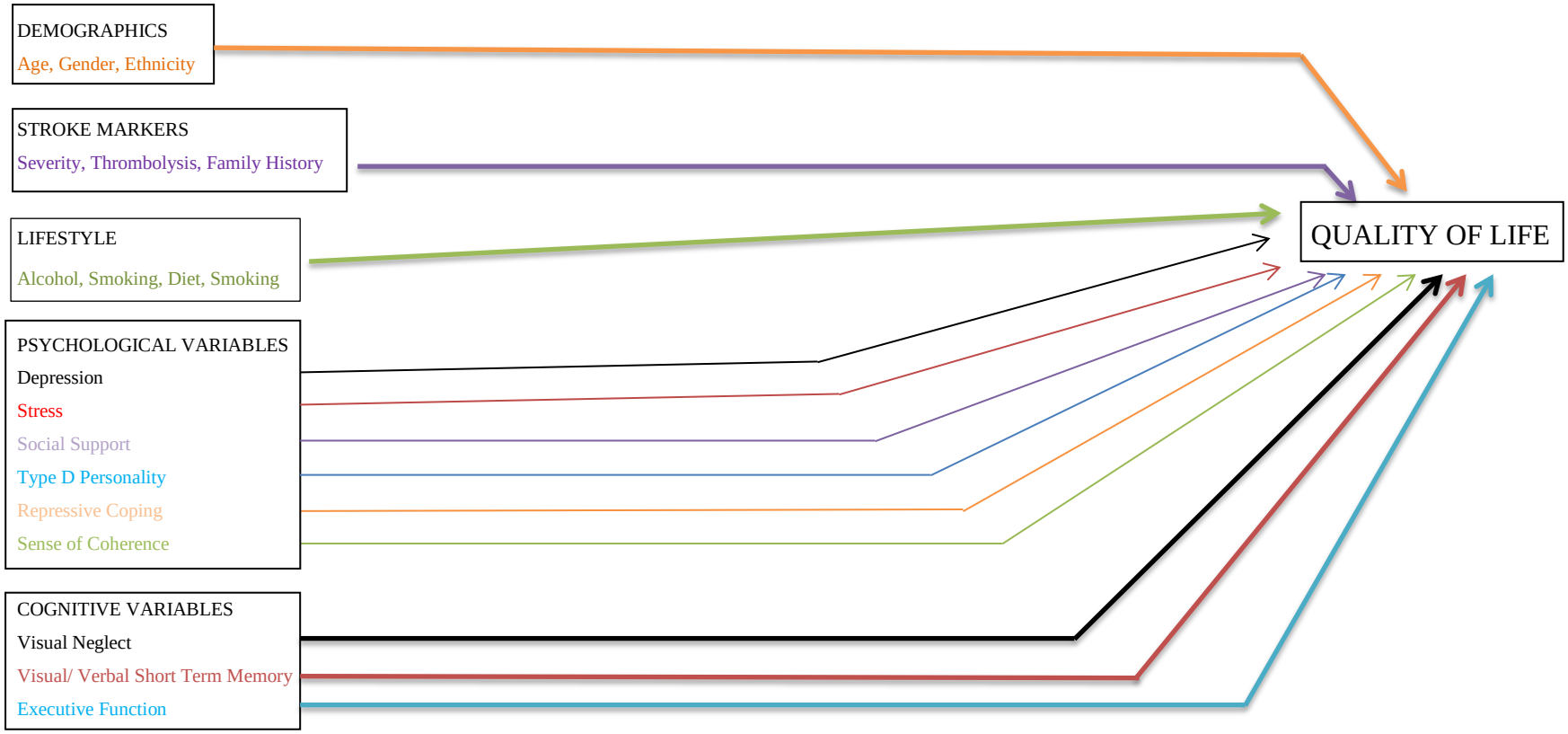
Theoretical Model for Physical Recovery



3.16 Theoretical Model for Quality of Life Recovery

Figure 3.2

Theoretical Model for Psychological Recovery



3.17 Hypotheses Generation

The following hypotheses are based on the groundwork of this thesis from the Systematic Review in Chapter 2 and the Literature Review in Chapter 3. As can be seen by the theoretical models the aim for this thesis was to investigate if psychological and cognitive variables are associated with physical or psychological recovery from stroke. In an extensive systematic review searching 100,743 research studies, 23 studies (2 paired studies) were identified which fulfilled the specific inclusion criteria of assessing if psychological variables have an association with stroke outcome, measured over more than one time point and excluding all proxy responses. These studies assessed the association between psychological variables and stroke outcome at fixed different time points**,** for example, active coping at baseline was associated with increased ADL function at 1 & 3 years follow up (Elmstahl et al., 1996), perceived control at 6 months predicted independence 3 years post stroke (Johnston et al., 2004), patients with depression at baseline had significantly lower functional scores at onset & after 6 months (van de Weg., 1999) and positive emotion (low depression) at hospital discharge was significantly associated with follow up Total FIM scores, 3 months later (Ostir et al., 2008).

The hypotheses in the following section are in line with the guidance of the Systematic Review, testing associations of psychological (and additionally, cognitive) variables with stroke outcome over 3 fixed time points, with data collected at Time 1: 0-6 weeks post stroke, Time 2: 3 months post stroke and Time 3: 6 months post stroke.

3.17.1 Hypotheses for the Physical Recovery Model.

This section is a written version of the Physical Recovery Model. There are 3 main hypotheses for this model, each with sub hypotheses.

***H_1*: Time 1 variables predict Time 1 Physical recovery.**

Main Hypothesis: It is predicted that after demographic, stroke markers and lifestyle variables are controlled for, psychological and cognitive variables at the fixed Time 1 point (T1) predicts physical recovery at the fixed T1 point. This hypothesis can be divided into the following sub sections:

a) High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 0-6 weeks post stroke (Time 1) will predict poorer physical recovery at a fixed time point of 0-6 weeks post stroke (Time 1).

b) High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at a fixed time point of 0-6 weeks post stroke (T1) will predict poorer physical recovery at a fixed time point of 0-6 weeks post stroke (T1).

c) T1 visual neglect will mediate T1 depression and T1 physical recovery.

d) T1 visual memory will mediate T1 depression and T1 physical recovery.

e) T1 verbal memory will mediate T1 depression and T1 physical recovery.

f) T1 executive function will mediate T1 depression and T1 physical recovery.

g) T1 depression will mediate T1 stress and T1 physical recovery.

h) T1 social support will mediate T1 stress and T1 physical recovery.

i) T1 repressive coping will mediate T1 stress and T1 physical recovery.

j) T1 Type D personality will mediate T1 stress and T1 physical recovery.

k) T1 executive function will mediate T1 stress and T1 physical recovery.

l) T1 social support will moderate T1 stress and T1 physical recovery.

m) T1 depression will mediate T1 social support and T1 physical recovery.

n) T1 SoC will mediate T1 Type D personality and T1 physical recovery.

o) T1 depression will mediate T1 visual neglect and T1 physical recovery.

p) T1 depression will mediate T1 visual short term memory and T1 physical recovery.

q) T1 depression will mediate T1 verbal short term memory and T1 physical recovery.

r) T1 depression will mediate T1 executive function and T1 physical recovery.

H_2: Time 1 and 2 variables predict Time 2 Physical recovery.

Main Hypothesis: It is predicted that after demographic, stroke markers, lifestyle variables and previous significant main variables are controlled for, psychological and cognitive variables at the fixed Time 1 and 2 points will predict physical recovery at the fixed Time 2 point. This hypothesis can be divided into the following sub sections:

a) High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 0-6 weeks post stroke (Time 1) will predict poorer physical recovery at a fixed time point of 3 months post stroke (Time 2).

b) High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 3 months post stroke (Time 2) will predict poorer physical recovery at a fixed time point of 3 months post stroke (Time 2).

c) High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at a fixed time point of 0-6 weeks post stroke (T1) will predict poorer physical recovery at a fixed time point of 3 months post stroke (T2).

d) High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at a fixed time point of 3 months post stroke (T2) will predict poorer physical recovery at a fixed time point of 3 months post stroke (T2).

e) T2 visual neglect will mediate T2 depression and T2 physical recovery.

f) T2 visual memory will mediate T2 depression and T2 physical recovery.

g) T2 verbal memory will mediate T2 depression and T2 physical recovery.

h) T2 executive function will mediate T2 depression and T2 physical recovery.

i) T2 depression will mediate T2 stress and T2 physical recovery.

j) T2 social support will mediate T2 stress and T2 physical recovery.

k) T2 repressive coping will mediate T2 stress and T2 physical recovery.

l) T2 Type D personality will mediate T2 stress and T2 physical recovery.

m) T2 executive function will mediate T2 stress and T2 physical recovery.

n) T2 social support will moderate T2 stress and T2 physical recovery.

o) T2 depression will mediate T2 social support and T2 physical recovery.

p) T2 SoC will mediate T2 Type D personality and T2 physical recovery.

q) T2 depression will mediate T2 visual neglect and T1 physical recovery.

r) T2 depression will mediate T2 visual short term memory and T2 physical recovery.

s) T2 depression will mediate T2 verbal short term memory and T2 physical recovery.

t) T2 depression will mediate T2 executive function and T2 physical recovery.

H_3: Time 1, 2 and 3 variables predict Time 3 Physical recovery.

Main Hypothesis: It is predicted that after demographic, stroke markers, lifestyle variables and previous significant main variables are controlled for, psychological and cognitive variables at the fixed Time 1, 2 and 3 points will predict physical recovery at the fixed Time 3 point. This hypothesis can be divided into the following sub sections:

a) High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 0-6 weeks post stroke (Time 1) will predict poorer physical recovery at a fixed time point of 6 months post stroke (Time 3).

b) High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 3 months post stroke (Time 2) will

predict poorer physical recovery at a fixed time point of 6 months post stroke (Time 3).

c) High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 6 months post stroke (Time 3) will predict poorer physical recovery at a fixed time point of 6 months post stroke (Time 3).

d) High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at a fixed time point of 0-6 weeks post stroke (T1) will predict poorer physical recovery at a fixed time point of 6 months post stroke (T3).

e) High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at a fixed time point of 3 months post stroke (T2) will predict poorer physical recovery at a fixed time point of 6 months post stroke (T3).

f) High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at a fixed time point of 6 months post stroke (T3) will predict poorer physical recovery at a fixed time point of 6 months post stroke (T3).

g) T3 visual neglect will mediate T3 depression and T3 physical recovery.

h) T3 visual memory will mediate T3 depression and T3 physical recovery.

i) T3 verbal memory will mediate T3 depression and T3 physical recovery.

j) T3 executive function will mediate T3 depression and T3 physical recovery.

k) T3 depression will mediate T3 stress and T3 physical recovery.

l) T3 social support will mediate T3 stress and T3 physical recovery.

m) T3 repressive coping will mediate T3 stress and T3 physical recovery.

n) T3 Type D personality will mediate T3 stress and T3 physical recovery.

o) T3 executive function will mediate T3 stress and T3 physical recovery.

p) T3 social support will moderate T3 stress and T3 physical recovery.

q) T3 depression will mediate T3 social support and T3 physical recovery.

r) T3 SoC will mediate T3 Type D personality and T3 physical recovery.

s) T3 depression will mediate T3 visual neglect and T1 physical recovery.

t) T3 depression will mediate T3 visual short term memory and T3 physical recovery.

u) T3 depression will mediate T3 verbal short term memory and T3 physical recovery.

v) T3 depression will mediate T3 executive function and T3 physical recovery.

3.17.2 Hypotheses for the Psychological (QoL) Recovery Model.

This section is a written version of the Psychological Recovery Model. There are 3 main hypotheses for this model, each with sub hypotheses.

***H_4*: Time 1 variables predict Time 2 QoL.**

Main Hypothesis: It is predicted after demographic, stroke markers and lifestyle variables are controlled for psychological and cognitive variables at the fixed T1 point will predict psychological recovery (QoL) at the fixed T2 point. This hypothesis can be divided into the following sub sections:

a) High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 0-6 weeks post stroke (Time 1) will predict poorer QoL at a fixed time point of 3 months post stroke (Time 2).

b) High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at a fixed time point of 0-6 weeks post stroke (T1) will predict poorer QoL at a fixed time point of 3 months post stroke (T2).

***H_5*: Time 2 variables predict Time 2 QoL.**

Main Hypothesis: It is predicted that after demographic, stroke markers, lifestyle variables and previous significant main variables are controlled for, psychological and cognitive variables at the fixed T2 point will predict psychological recovery (QoL) at the fixed T2 point. This hypothesis can be divided into the following sub sections:

a) High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 3 months post stroke (Time 2) will predict poorer QoL at a fixed time point of 3 months post stroke (Time 2).

b) High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at a fixed time point of 3 months post stroke (T2) will predict poorer QoL at a fixed time point of 3 months post stroke (T2).

H_6: Time 1, 2 and 3 variables predict Time 3 QoL.

Main Hypothesis: It is predicted that after demographic, stroke markers, lifestyle variables and previous significant main variables are controlled for, psychological and cognitive variables at the fixed Time 1, 2 and 3 points will predict psychological recovery (QoL) at the fixed Time 3 point. This hypothesis can be divided into the following sub sections:

a) High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 0-6 weeks post stroke (Time 1) will predict poorer QoL at a fixed time point of 6 months post stroke (Time 3).

b) High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 3 months post stroke (Time 2) will predict poorer QoL at a fixed time point of 6 months post stroke (Time 3).

c) High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 6 months post stroke (Time 3) will predict poorer QoL at a fixed time point of 6 months post stroke (Time 3).

d) High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at a fixed time point of 0-6 weeks post stroke (T1) will predict poorer QoL at a fixed time point of 6 months post stroke (T3).

e) High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at a fixed time point of 3 months post stroke (T2) will predict poorer QoL at a fixed time point of 6 months post stroke (T3).

f) High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at a fixed time point of 6 months post stroke (T3) will predict poorer QoL at a fixed time point of 6 months post stroke (T3).

The Physical Recovery Model is tested in Chapter 6 and the Psychological Recovery Model is tested in Chapter 7. The Methodology of this study is explained in Chapter 4.

Chapter 4

Methodology

4.1 Rationale and Summary

The rationale for this chapter is to outline a design for a longitudinal study investigating the effects of psychological and cognitive variables on the psychological and physical recovery from stroke. This is to ensure the study is repeatable and therefore could be replicated or modified in the future.

The Chapter begins with the aims, study design, ethical approval and ethical issues. This is followed by information on the power calculation, sample size, participant recruitment, inclusion criteria, exclusion criteria, measures used, procedure and proposed statistical analysis.

4.2 Aim

The aim of this study is to develop a quantitative conceptual framework to predict the extent of recovery after a stroke using psychological and cognitive variables as predictors. The next section explains the study design.

4.3 Study Design

This research project has a longitudinal study design. Three separate time point measurements were used: Time 1 (baseline 0-6 weeks post stroke), Time 2 (3 months post stroke) and Time 3 (6 months post stroke). Baseline measures were taken between March 2010 – Jan 2011 and full follow up assessment was completed by July 2011.

Independent variables included questionnaire measures and cognitive tests. The questionnaire measures used were depression, stress, social support, Type D personality, repressive coping and sense of coherence. The cognitive tests measured

were the line bi-section, the bells cancellation task, the Rivermead Behavioural Memory Test, the forward digit span and the Stroop colour-word task.

The dependent variables were measures of recovery. Psychological recovery was measured using the the Medical Outcomes Study 36-item short-form health survey (SF-36). Physical recovery was measured using the modified Rankin Scale (mRS).

Before the study can commence ethical approval is needed. The following two sections discuss ethical approval and ethical issues.

4.4 Ethical Approval

Ethical approval was given by Brunel University's Psychology Ethics Board (PsyRec) and from the National Health Service (NHS), The Hammersmith and Queen Charlotte's & Chelsea Research Ethics Committee using the Integrated Research Applications System (IRAS) (see Appendix B). Research & Development approval (R&D) was then obtained from The Hillingdon Hospital and Northwick Park Hospital. Once formal clearance was obtained, Stroke Consultants and Stroke Research Nurses gave permission for eligible participants to be approached (more on this in the Procedure Section 4.9, p.237). Risk assessment was developed by Brunel University to ensure safety practices were adhered to when data collection involved visiting participants in their homes (at Time 2 and Time 3).

4.5 Ethical Issues

Stroke affects mental capacity which can complicate gaining informed consent. Stroke Consultants and Stroke Research Nurses aided in selecting participants for recruitment based on their diagnoses. In accordance with the Mental Capacity Act 2005, Section 34.2, if a participant is withdrawn due to loss of capacity, no new data will be collected. Existing data that has been collected whilst the participant was able to consent will still be used in the study. During the course of the research, no participants withdrew in accordance with the Mental Capacity Act 2005.

Participants with communicative problems such as dysphasia were included in the recruitment phase however, only those with expressive dysphasia were included. Expressive dysphasia is when a participant understands what is being said to them, and they can understand the consent from process but they have difficulty in speaking. This can be combated in some part, by slowing down the process and allowing the participant to have time to respond. Participants with receptive dysphasia have to be excluded from the study. Receptive dysphasia is when participants cannot understand the information they are presented with, hence not being able to complete the informed consent process. The Stroke Consultants and Stroke Research Nurses aided with separating these groups of dysphasic patients.

The Hammersmith and Queen Charlotte's & Chelsea Research Ethics Committee held a meeting in regard to the application for the approval of this study. The main enquiries were levelled at the number and length of the proposed measures to be completed by the participants. As the first time point data was to be collected in the acute stroke phase, concerns regarding stress on the participants were raised. Therefore, short measures of variables were to be chosen in place of long measures to reduce the amount of questions and tests placed on participants' time in this acute recovery period.

The REC agreed to approve all questionnaire measures and the cognitive battery as they were all justifiable components of the research study. However, as the Time 1 data point was recorded in the acute stroke period, the REC was concerned regarding the inclusion of the QoL measure due to the length of the questionnaire (36 questions). Due to the issue of participant fatigue and duress that may be caused as a result of repetitive testing, measures at Time 1 had to be reduced. Some of these questions ask about how one feels about physical impairment. This was deemed inappropriate to ask in the acute stroke phase as participants' may have paralysis and weakening of limbs and discussing this immediately after stroke may cause distress. Also, as the study was proposed with a longitudinal design it was asserted that Time 1 QoL was not needed, as Time 1 independent variables would be predicting Time 2 and Time 3 outcome. Therefore, QoL was not measured at Time 1. The timeframes

of length of visit estimated for each participant were approximately 25 minutes at Time 1; 40 minutes at Time 2 and 40 minutes at Time 3.

It is important to note the basis for the proposal of this research was formed with the Systematic Review which investigated the association of psychological variables at fixed time points on recovery outcomes at fixed time points (see Chapter 2, p. 58). The Systematic Review did not examine any other topics, as is its purpose. This research is concerned with the predictive value of psychological and cognitive variables on outcome at *fixed* time points. For this reason, QoL was addressed at Times 2 and 3.

Once ethical approval was obtained from this lengthy process, a similar process of justification was needed to satisfy the R&D departments at Hillingdon Hospital and Northwick Park Hospital. After this approval was completed, the study could commence.

In order to calculate a benchmark for the number of participants needed to achieve statistical power, a power calculation must be determined. This is discussed in the next section.

4.6 Power Calculation & Sample Size

To calculate the number of participants needed to reach statistical power, a power analysis using G Power (http://www.psycho.uni-duesseldorf.de/abteilungen/aap/gpower3/ 2009) was conducted to estimate the required participant numbers for the 8 psychosocial measures using a medium effect size (0.5) and a power of 80% (0.8) and a type 2 error rate set at 20%. A minimum of 119 participants were required to reach statistical power.

Once the power calculation has been determined, participant recruitment has a goal. In the next section details of the participants are disclosed.

4.7 Participants

One hundred and forty three participants were recruited from a sample of 224 available participants. Eighty five male and 58 female participants were recruited

between the ages of 19 and 95 (mean age 67.7). Participants were categorised as White British (n = 99), White Other (n = 11), British Indian (n = 1), British Pakistani (0), Asian Other (12), British Black African (1), British Black Caribbean (7) and Black Other (13). For the purposes of statistical analysis, 3 main groups were formed: Caucasian (110), Asian (13) and African & Caribbean (20).

These participants were consecutively recruited from The Hillingdon Hospital (n = 48) and Northwick Park Hospital (n = 95) at Time 1. At Time 2, 108 participants were followed up and at Time 3, 101 participants were followed up. Details on loss to follow up are presented in table 4.1.

The setting of data collection was as follows: All Time 1 data collection (143) was collected in hospital. Time 2 data collection was collected in hospitals (11), the home (92) and care homes (5). Time 3 data collection was collected in hospitals (2), the home (93) and care homes (6).

Table 4.1

Loss to follow up at Time 2 and 3.

	TIME 2		TIME 3	
Loss To Follow Up Reason	N	%	N	%
Death	11	7.7%	1	0.7%
Unable To Participate	2	1.4%	1	0.7%
Another Stroke	0	0%	0	0%
Refused	15	10.5%	7	4.9%
Lost	7	4.9%	0	0%
TOTAL ATTRITION	35	24.5%	9	6.3%

4.7.1 Inclusion Criteria for Participant Recruitment

1) Lesion Location – Any lesion location.
2) Stroke severity – Any stroke severity.
3) Stroke Number – Any stroke experienced by patient (i.e., 1st, 2nd, 3rd).
4) Expressive Dysphasic and Aphasic patients are to be included.
5) Language – English must be understood.

4.7.2 Exclusion Criteria for Participant Recruitment

1) Receptive Aphasia and Dysphasia (due to inability to consent).
2) Existing diagnosis of the cognitive disorders, dementia and delirium.
3) Previous psychiatric disorders, schizophrenia and delusional disorders.
4) Neurodegenerative disorders, multiple sclerosis, Parkinson's disease and Alzheimer's disease.
5) Learning disabilities, Downs Syndrome, Asperger's and Autism.
6) Inability to comprehend the consent from (unable to give informed consent).
7) Transient Ischemic Attack (TIA), e.g., stroke symptoms had resolved within 24 hours.

In the following sections measures are disclosed. These include stroke markers, demographic variables, psycho-social and cognitive scales and outcome measures.

4.8 Measures

4.8.1 Stroke Markers

a) Oxford Community Stroke Project (OCSP, also referred to as the Bamford Stroke Classification) (Bamford, 1991).

This measure categorises four subtypes of cerebral infarct stroke based on clinical localisation of the infarct.

(i) **TAC** — Total Anterior Circulation Stroke

(ii) **PAC** — Partial Anterior Circulation Stroke

(iii) **LAC** — Lacunar Stroke

(iv) **POC** — Posterior Circulation Stroke

Additional letters of ***S, I*** *and* ***H*** *are added* ***after*** *the categories to add to the classifications:*

- *S: Syndrome* - Categorisation made before imaging when the stroke type is undetermined (ischemic or haemorrhagic).

- *I: Infarction* - Categorisation made after imaging when infarct without haemorrhage is shown.

- *H: Haemorrhage* - Categorisation made after imaging when haemorrhage is demonstrated.

This measure was completed by the Stroke Consultant.

b) Trial of ORG 10172 in Acute Stroke Treatment (TOAST) (Adams et al., 1993)

This measure classifies participants with ischaemic stroke into 5 etiologic groups.

(i) Large Artery Athersclerosis (Embolus / Thrombus)
(ii) Cardioembolism (High Risk / Medium Risk)
(iii) Small Vessel Occlusion (Lacune)
(iv) Stroke of other determined aetiology
(v) Stroke of undetermined aetiology

(vi) InterCerebral Haemorrhage (ICH)

This measure was completed by the Stroke Consultant. An extra category of ICH was added upon advice of the Stroke Consultant.

c) **Hemisphere of stroke**, was recorded as Left or Right, taken from the clinical notes.
d) **Stroke type recorded as Ischemic or Haemorrhagic**, taken from the clinical notes.
e) **Stroke number**, was recorded as the number of strokes experienced by the participant, 1st, 2nd, 3rd etc., taken from the clinical notes and the participant.
f) **Physical stroke severity**, was recorded as Mild / Moderate / Severe. This was an observer rated measure from the Researcher.
g) **Thrombolysis treatment**, was recorded as No / Yes / Not Applicable. This information was taken from the clinical notes and checked by the Stroke Consultant.

4.8.2 Demographic Factors

a) **Age**, in years. This was taken from the medical notes.

b) **Gender**, categorised as male and female.

c) **Education**, categorised as Less Than Secondary School / Secondary School / College / Undergraduate / Postgraduate.

d) **Marital Status**, categorised as Never Married / Co-Habiting / Married / Divorced / Widowed.

e) **Ethnicity**, was recorded as White British / White Other / Black British Caribbean / Black British African / Black British Other / British Asian Indian / British Asian Pakistani / British Asian Other. If numbers are low in each category, they will be collapsed into Caucasian / Asian / African & Caribbean.

f) **Occupation** was defined using the National Statistics Socio-Economic Classification (NS-SEC), which was based on the Social Class based on Occupation criteria (formerly the Registrar General's scale of Social Class and Socio-economic groups), which has been used frequently in Britain (Office For National Statistics 2013), which ranges from Professionals to unemployed status.

g) **Retired**, was measured as Yes or No.

4.8.3 Risk Factors

a) **Alcohol**. This was measured as No Never / No Now/ Yes Now. They were collapsed into Yes and No responses. They were also asked how much they have drunk in the past and for how long.

b) **Smoking**. Participants were asked about their current smoking status and this was measured as No Never / No Now/ Yes Now. They were collapsed into Yes and No responses. They were also asked about how much they have smoked in the past and for how long.

c) **Participant-reported blood pressure**, was classified as Low / Normal / High / Do Not Know.

d) **Participant-reported Cholesterol**, was classified as Low / Normal / High / Do Not Know.

e) **Family history of heart disease**, was classified as No / Yes / Do Not Know.

f) **Family history of stroke**, was classified as No / Yes / Do Not Know.

g) **Participant-reported Diet**, was classified as Unhealthy / Moderate / Healthy. These were collapsed into Healthy and Unhealthy responses.

h) **Participant-reported Exercise**, was classified as None / Mild / Moderate / A Lot. These were collapsed into regular exercise and non-regular exercise.

i) **Anti depressants**, were recorded as No or Yes.

4.8.4 Psycho-social scales

a) Centre for Epidemiologic Studies Short Depression Scale (CES-D 10) (Andresen, Malmgren, Carter, & Patrick, 1994).

This is a short self-assessment test that measures depressive feelings and behaviours during the past week and is derived from the 20-item CES-D. Examples of questions include measuring negative affect ("I felt depressed"), positive emotion ("I was happy"), physical effect ("My sleep was restless") and cognitive factors ("I had trouble keeping my mind on what I was doing"), which is scored on a Likert scale of Rarely or None of the Time / Some or a Little of the Time / Occasionally or a Moderate Amount of the Time and All of the Time. This measure is scored out of 30. If participants score above 10, they are considered to be demonstrating signs of depressive symptomatology.

Measures are assessed with the Cronbach's alpha coefficient which measures the internal reliability of a scale (Cronbach, 1951) or a Kappa coefficient which is used to test the reliability between raters to assess inter-rater reliability (Cohen 1960).

Andresen et al., (1994) tested this measure on Mexican immigrants in a mental health research study and reported a Kappa value of 0.97. Further

research includes HIV-positive patients who are enrolled in an antiretroviral therapy program in Canada (kappa coefficient = 0.82) (Zhang et al., 2012), depression in psychiatric patients (Cronbach's alpha = 0.80) (Nishiyama, Ozaki, & Iwata, 2009) and patients with spinal cord injury (Cronbach's alpha = 0.86) (Miller, Anton, & Townson, 2008).

b) <u>Perceived Stress Scale (PSS-14) (Cohen, Karmack, & Mermelstein, 1983).</u>

This measure records perceived stress in the previous month using a 14 item questionnaire with examples of questions in the scale including "In the last month, how often have you been upset because of something that happened unexpectedly?" and "In the last month, how often have you been able to control the irritations in your life?", recorded on a Likert scale (Never / Almost Never / Sometimes / Fairly Often / Very Often). There are no cut off points for this measure as comparisons are made between participants within the sample.

The reliability of the scale was tested by the constructers which yielded a Cronbach's alpha coefficient in two college samples (0.84 and 0.85) and in a smoking cessation sample (0.86) (Cohen, Karmack, & Mermelstein, 1983).

Further research using this measure has included stress experienced by participants whose family members or significant others committed suicide (Cronbach's alpha = 0.89) (Mitchell, Crane, & Kim, 2008), cardiac patients that smoke (Cronbach's alpha = 0.85) (Leung, Lam, & Chan, 2010) and workers recruited from hospitals, financial offices and universities (Cronbach's alpha = 0.82) (Andreou et al., 2011).

c) <u>Multidimensional Scale of Perceived Social Support (MSPPS) (Zimet, Dahlem, Zimet, & Farley, 1988).</u>

For this measure participants respond to 12 statements that assess their perception of the level of social support (support from relationships)

available to them in 3 areas (family, friends and significant others). Each of these areas had four questions each. For example: “My family really tries to help me” (Family), “I can count on my friends when things go wrong” (Friends) and “There is a special person who is around when I am in need” (Significant Other). Questions were scored on a 7 item Likert scale ranging from Very Strongly Disagree to Very Strongly Agree. This measure is scored by the total mean value of the scale.

The Cronbach’s alpha coefficient for the three subscales upon construction were 0.87 (Family), 0.85 (Friends) and 0.91 (Significant Other) in an undergraduate sample. The overall value for the whole scale was 0.88 (Zimet et al., 1988).

The MPPS has shown good internal consistency in other studies, in controls and pathological samples in Turkish adults (Cronbach’s alpha = 0.77 – 0.92) (Eker & Arker, 1995), in generalised anxiety disorder and controls in an elderly sample (Cronbach’s alpha = 0.87 – 0.94) (Stanley, Beck, & Zebb, 1998) and in women who attended postnatal clinics in Uganda, with a Cronbach’s alpha of 0.82 (Family), 0.80 (Friends), 0.79 (Significant Other) and with a total alpha of 0.83 (Nakigudde, Musisi, Ehnvall, Airaksinen, & Agren, 2009).

d) <u>Repressive coping</u>

Repressive coping is classified by two measures: a defensiveness measure and an anxiety measure. In this research study the Marlowe-Crowne Social Desirability Scale (M-C SDS Form B) was used to measure defensiveness and the Six-item short-form of the state scale of the Spielberger State-Trait Anxiety Inventory (STAI) was used to measure anxiety. Repressors score high on defensiveness and low on anxiety. For analysis, median splits are used where the upper median of the M-C SDS Form B and the lower median of the Six Item STAI are analysed. In this study repressors are identified as scoring above 9 on the M-C SDS Form B and below 11 on the six item STAI.

(i) Marlowe-Crowne Social Desirability Scale (M-C SDS Form B) (Reynolds, 1982).

The Marlowe-Crowne Social Desirability Scale (M-C SDS) (Crowne & Marlowe, 1960) assesses response bias (i.e., the degree to which individuals attempt to present themselves in a favourable light) and has been classically used as the defensiveness component to assess the repressive coping style.

The M-C SDS was shortened by Reynolds in 1982 to a 12 item scale and is termed the M-C SDS Form B. This measure includes questions such as "I sometimes try to get even rather than forgive and forget", "I sometimes feel resentful when I don't get my way" and "I am always willing to admit when I have made a mistake". These questions are scored as Yes or No. The maximum value for the Marlowe-Crowne Form B is 12.

Reynolds demonstrated the reliability (0.75) of the measure in a sample of undergraduate students using the Kuder-Richardson formula 20 reliability (Richardson & Kuder, 1939).

Further studies mainly using undergraduate samples have shown moderate to good reliability, e.g., Cronbach alphas of 0.88 (Fischer & Fick, 1993), 0.64 (Barger, 2002) and 0.61 (Loo & Thorpe, 2000).

(ii) Six-item short-form of the state scale of the Spielberger State-Trait Anxiety Inventory (STAI) (Marteau & Bekker, 1992).

This is a short questionnaire which condenses the most highly correlated anxiety-present and anxiety-absent items from the full-form of the STAI (20 items) into six items. The STAI measures state anxiety (how one feels in the moment) and trait anxiety (how one normally feels). Responses are scored as Not At All / Somewhat / Moderately / Very Much and includes questions such as, "I feel content", "I feel worried" and "I feel calm". The maximum value for the 6 item STAI is 24.

This shortened form of the questionnaire has shown good internal reliability in a sample of medical and nursing students, pregnant women with healthy scans and pregnant women with abnormal scans (Cronbach's alpha = 0.82) (Marteau & Bekker, 1992), parents with children with cystic fibrosis, congenital hypothyroidism and healthy infants (Cronbach's alpha = 0.81) (Tluczek, Henriques, & Brown, 2009) and before and after preconception counselling (Cronbach's alpha = 0.83) (van der Bij, de Weerd, Cikot, Steegers, & Braspenning, 2003).

Repressive coping is one of four groups in the theory put forth by Weinberger, Schwartz, & Davidson in 1979. The other three control groups are low anxious (low trait anxiety and low defensiveness) *high-anxious* (high trait anxiety and low defensiveness) and *defensive high-anxious* (high trait anxiety and high defensiveness). These groups are assigned according to specific combinations of the two subscales. The majority of research on repressive coping assigns repressors to a specific group which is primarily achieved through the use of median splits (Shaw et al., 1986; Jensen, 1987; Denollet, Martens, Nyklicek, Conraads, & de Gelder, 2008; Burns, 2000; Myers 2010). However, tertiary and quartile splits have also been used (Derakshan & Eysenck, 1997; Myers & Steed, 1999; Myers & Derakshan, 2004a, 2004b).

Weinberger (1990) asserted that using continuous scores of two measures in a multiple regression analysis to investigate the interaction term would not be able to identify repressors as ratings on the two measures would be divergent (i.e., low on anxiety and high on defensiveness) therefore using an interaction term would not be viable in identifying this group. The strength in utilising categorical variables is that asymmetrical groups can be identified.

Mendolia (2002) assessed repressors on one measure which was a composite scale of the Marlow-Crowne Social Desirability Scale (MC SDS) and the Manifest Trait Scale (MAS), called the Index of Self Regulation of Emotion (ISE), where MC SDS scores were subtracted from MAS scores. This study aimed to expand the concept and methodology of repressive

coping. Mendolia (2002) also compared the continuous scores to the traditional categorical scores and concluded they can be comparable if there is an appropriate sample size and statistical power for the continuous scores although there is an admission that a participant may have different MC SDS and MAS scores but ultimately have the same ISE score. This could cause errors in conclusions. The Mendolia (2002) paper has been cited 26 times since it's publication compared with Weinberger et al's (1978) publication which has been cited 748 times. Between these two methods of classifying repressors', Weinbergers' taxonomy has the most durability and stability.

e) <u>DS14: Standard Assessment of Negative Affectivity, Social Inhibition, and Type D Personality (Denollet, 2005).</u>

This is a 14 item scale containing 7 Negative Affectivity items (which covers dysphoria, worry and irritability) and 7 Social Inhibition items (which covers discomfort in social interactions, reticence and social poise). Examples of the negative affectivity questions are "I take a gloomy view of things" and "I often make a fuss about unimportant things" and examples of social inhibition questions are "I make contact easily when I meet people" and "I often feel inhibited in social interactions". Questions are recorded on a Likert scale of False / Rather False / Neutral / Rather True and True and have a maximum score of 28.

To determine a score of Type D personality a participant must score above 10 on both the negative affect scale and the social inhibition scale. From this a dichotomous variable of Yes or No Type D is produced. It is important to note that the Type D variable is a *combination* of negative affectivity and social inhibition. Negative affectivity and social inhibition will not be used separately in this study as predictors but jointly to refer to the Type D personality style, as Type D personality is the variable of interest.

Johan Denollet is the researcher responsible for providing the theory and method behind the data capture of Type D personality. Denollet (2005)

designed the DS14 which is the only main, up-to-date measure for Type D, which was preceded by the DS16 (Denollet, 1998).

A cut off of 10 on both subscales are used to classify Type D personality as this has been calculated by Denollet by using median splits in a coronary heart disease sample in the 2005 study and is now the benchmark cut off point. This variable is then converted into a categorical variable of Yes/No. Again, the subscales are not used separately but are joined together with a specific combination. To keep them as subscales and use them as continuous variables, is not to measure Type D. Therefore, this variable cannot be used in this way.

Many studies have used Type D in this way (Denellot, Sys, & Brutsaert, 1995; Denellot, 1998a ; Denollet, Vaes, & Brutaert, 2000; Denollet, 2000; Gerin et al., 2000; Schiffer et al., 2005; Pedersen et al., 2006; Aquarius, Denollet, de Vries, & Hamming, 2007; Kuijpers, Denellot, Wellens, Crijns, & Honig, 2007; Smith et al., 2007; Pedersen, Theuns, Muskens-Heemskerk, Erdman, & Jordaens, 2007; Attila, Istvan, Istvan, & Gabor, 2007; Schiffer, Pedersen, Broers, Widdershoven, & Denollet, 2008; Pelle et al., 2008; Schiffer, Pedersen, Widdershoven, & Denollet, 2008; Dieltjens, Vanderveken, & Van den Bosch, 2013; Svansdottir et al., 2013; Kupper, Pelle, & Denollet, 2013; Husson, Denollet, Oerlemans, & Mols, 2013; Dubayova et al., 2013; Larson, Barger, & Sydeman, 2013).

Additionally, research investigating use of the Type D variable dimensionally (NA x SI), have yielded no significant reports (Coyne et al.,2011; Grande et al., 2011) and studies which have compared the traditional Type D method to a newer continuous method have also been unsuccessful (Williams et al., 2012; Stevenson & Williams, 2014).

Reliability analysis by Denollet (2005) yielded good results with analysis being conducted on a coronary heart disease sample (Cronbach's alpha 0.88) and a hypertension sample (Cronbach's alpha 0.86). Further

research which assessed the reliability of the DS14 was conducted on cardiac patients with a reported Cronbach's alpha of 0.79 (negative affectivity) and 0.80 (social inhibition) (Vilchinsky et al., 2012), healthy participants with a reported Cronbach's alpha of 0.86 (negative affectivity) and 0.71 (social inhibition) (Pedersen ,Smith, Yagenska, Shpak, & Denollet, 2009) and cardiology patients, psychosomatic patients and healthy workers, with a reported Cronbach's alpha of 0.87 (negative affectivity) and 0.86 (social inhibition) (Grande et al., 2004). However, these latter studies do not report the Cronbach's alpha value for the combination of subscales which assesses the Type D personality style.

f) <u>3-Item Sense of Coherence scale (Lundberg, & Nystrom Peck, 1995)</u>

Sense of Coherence is a stress adaptive strategy and was originally a 29 item scale which was also shortened to a 13 item scale by its original creator Antonovsky (Antonovsky, 1979; 1987). For this research study the 3-item measure was used. It is a self-report measure designed to assess each of the component constructs: comprehensibility, ("Do you usually feel that the things that happen to you in your daily life are hard to understand?"), manageability ("Do you usually see a solution to problems and difficulties that other people find hopeless?") and meaningfulness ("Do you usually feel your daily life is a source of personal satisfaction?") and is measured on a Likert scale of Yes / Sometimes and No. The scores are summed from 0-6, with lower scores indicating a stronger SoC and higher scores indicating a weaker SoC.

Lundberg & Nystrom Peck (1995) used a representative sample of the Swedish population to test reliability which yielded a Kappa coefficient of 0.61. Other research demonstrated lower reliability (Cronbach's alpha = 0.35) from the EPIC-Norfolk United Kingdom study (Surtees et al., 2006) and in a general German population (Cronbach's alpha = 0.45) (Schumann et al., 2003). In these latter two examples the Cronbach's alpha coefficient is low,

however Lundberg & Nystrom Peck, (1995) reported a higher Kappa coefficient. In the current study, as participant fatigue is an important issue to consider, shorter versions of measures have been chosen in accordance with advice from the REC. Also, as the conclusions of the systematic review directed the use of a control measure (Johnston et al., 1999, 2004), therefore even though previous reliability of this measure was low, it was included in the design and the data collected.

4.8.5 Measures of Cognitive deficits in three major domains

Cognitive measures do not use reliability statistics such as Cronbach's alpha and Kappa coefficients as these statistics are normally used for questionnaire measures. Other reliability statistics are used with these measures.

a) Visuo-spatial disturbance was assessed with 2 brief measures. These disturbances are best identified with more than one validated task and therefore the current study utilises two of the most sensitive tasks, which have been shown to be doubly dissociable (Azouvi et al., 1996; Ferber & Karnath, 2001):

(i) The Bells Cancellation Test (Gauthier, Dehaut, & Joanette, 1989).

The Bells Cancellation test is a test of visual neglect. It requires participants to find and cancel (circle or stroke through with a pen) 34 target bells distributed on an A4-sized sheet of paper, which also contains 315 irrelevant distractor items. Although the items look randomised the page is divided into seven columns with 5 bells to each column.

In the original research study from Gauthier and his team (1989), the sample used was a stroke sample. In this sample half of the group made up to three omissions, which lead to the conclusion that 3 or more omissions on either side of the page would demonstrate

a deficit. Test-retest reliability at 2 week follow up yielded an intraclass correlation coefficient of 0.69.

The test is extremely sensitive to spatial bias that commonly follows stroke but impaired performance may also be exacerbated by other deficits (Husain & Rorden, 2003).

(ii) <u>The line bi-section (Binder, Marshall, Lazar, Benjamin & Mohr 1992).</u>

The line bi-section requires participants to mark the apparent midpoint of a long horizontal line printed on an A4 sheet. Participants with visuo-spatial disturbances will not mark the line at the mid-point but to the left or right (Binder, Marshall, Lazar, Benjamin, & Mohr, 1992). The cancellation task and line bi-section task may measure slightly different forms of visuo-spatial disturbance, e.g. the line bi-section has been shown to correlate with shifts in the perceived body-midline that do not necessarily predict neglect (Richard, Honoré, Bernati, & Rousseaux, 2004).

The intraclass correlation coefficient used to examine test-retest reliability for the line bi-section has been reported to be 0.47 (Machner, Mah, & Gorgoraptis, 2012).

b) Short term memory was assessed with two tests:

(i) <u>The Rivermead Behavioural Memory Test (Wilson, Cockburn, & Baddeley, 1985).</u>

The Rivermead Behavioural Test is a battery of tests for visual short term recognition memory. In this study the object recognition battery was used. This is comprised of a set of 10 picture cards (i.e., a book, a star etc.), which are presented to the participants who were asked to remember them. After an approximate 10 minute delay the participants are shown a different set of 20 cards, which include the

original 10 pictures and 10 distractor pictures. The participant is asked to identify which of them they remember from the previous set.

Inter rater agreement is reported to be 100% (Wilson, Cockburn, & Baddeley, 1985) and has been used in samples of stroke, Parkinson's disease, heart problems, dementia and healthy aging (Lezak, Howieson, & Loring, 2004).

(ii) <u>The Forward Digit Span (Wechsler, 1945).</u>

The Forward Digit span is a test of verbal working memory capacity and attention. The test starts with a sequence of two digits, the participant is asked to repeat the sequence in exactly the same order. This is then followed by a new sequence and on every other trial the sequence length is increased by one. There are 8 trials, with the first trial consisting of 2 digits and the longest trial (trial 8), consisting of 9 digits. The test ends when participants make errors on two successive sequences or when two trials of digit length 9 are completed.

The digit span results can be categorised as correct responses of 6 digits and above demonstrating a normal range of memory, 5 correct responses shows a marginal range, 4 correct responses shows a borderline range and 3 and below shows a deficit in memory span (Lezak, Howieson, & Loring, 2004).

Test-retest reliability has been reported as 0.89 (Snow, Tierney, Zorzitto, & Leal, 1989).

c) <u>Stroop Colour Naming Test (Stroop 1935).</u>

The Stroop Colour Naming Test is a classic test for executive function. This test has a congruent (control) trial and an incongruent trial. The congruent trial is a vertically displayed list of 24 neutral words (i.e., "when", "over" etc.) written in 4 different colours (blue, green, yellow and red). The participant will be asked to ignore the word but read out the colour it is in.

They were then given another 24 words vertically displayed, however these will have colour names written in a different colour (e.g., the word "blue" will be written in the colour ink red). The participant will be asked to ignore what the word says but to say the colour it is written in. These words are vertically displayed in order to reduce the impact of visual neglect on the Stroop task.

Reliability of the Stroop test was not recorded by Stroop (1935) and limited studies have been conducted on this, however the test retest reliability of the colour word Stroop has been reported in some studies e.g., the intraclass correlation coefficient of 0.86 in a female undergraduate sample (Siegrist, 1997) and 0.671, in a mixed undergraduate sample (Franzen, Tishelman, Sharp, & Friedman, 1987).

Error numbers and time taken to complete the task are recorded. The time is calculated by subtracting the incongruent trial from the congruent trial. This number should be a positive number because the Stroop effect shows interference in the incongruent trial, thus the incongruent trial should take longer to complete. Participants demonstrating a reversal in the standard Stroop effect (in which the incongruent trials were quicker than the congruent trials hence, not showing the Stroop effect) were removed from further analysis. Also, participants with errors on the congruent (control) task that exceeded 2 standard deviations from the mean were excluded. It could not be reasonably concluded that these participants were performing the task correctly.

4.8.6 Physical Recovery

a) Modified Rankin Scale (mRS) (Bonita & Beaglehole, 1988).

The Modified Rankin Scale is an observer rated scale and is used for measuring the degree of disability or dependence in people who have suffered a stroke. It is a 7 point scale which is categorised from no symptoms to slight disability, to moderate disability, to severe disability to death. Post stroke

Rankin scores at Time 1, Time 2 and Time 3 were provided by the researcher.

Bonita & Beaglehole (1988) did not report any reliability statistics however, the Modified Rankin Scale (mRS), has been reported to have a weighted Kappa of 0.78 – 0.93 (Wilson et al., 2002).

4.8.7 Psychological Recovery

a) The Medical Outcomes Study 36-item short-form health survey (SF-36) (Ware Jr., & Sherbourne 1992).

This is a 36 item quality of life (QoL) scale measuring general health in participants which is comprised of two components measuring physical (physical functioning, role physical, bodily pain and general health) and mental (vitality, social functioning, role emotional and mental health) outcomes. Responses are measured on various Likert scales from 3 to 6 options with questions such as "Does your health now limit you in these activities, vigorous activities, moderate activities etc." and "In the past 4 weeks have you felt full of life?". This measure has a final percentage total, with higher scores indicating a better QoL and lower scores indicating a weaker QoL.

However, Ware & Sherbourne (1992) do not report any Cronbach alpha, or a Kappa coefficient. Nevertheless, the measure has been shown to have good reliability in other research, such as acute stroke patients (Cronbach alpha = 0.70) (Almborg, & Berg, 2009), participants in Indian suburbs and villages (Cronbach alpha = 071 – 0.88 for subscales) (Sinha, van den Heuvel, & Arokiasamy, 2013) and with Chinese medical students (Cronbach alpha = 0.79) (Zhang, Bo, Lun, Guo, & Liu, 2012).

The procedure for the research study is disclosed in the following section.

4.9 Procedure

At the Hillingdon Hospital, the Researcher (PSKD) attended bi-weekly ward rounds in which eligible participants were identified by the consultant. The consultant introduced the researcher to the participant and if the participant allowed, the researcher would approach the participant and begin to explain what the study is regarding. At Northwick Park Hospital, the researcher was provided with a patient list and along with a Stroke Research Nurse (delegated by the consultant) eligible participants were identified and approached.

Participants were told this investigation was to discover if there is a relationship between psychological factors and recovery after a stroke. Participants were then asked to read an information sheet on the aims of the study (see Appendix C). If participants were unable to read due to vision problems, the researcher read out the participant information sheet and thoroughly explained all the content which included the participants right to withdraw, anonymity (their name would not be used but they would be instead given a code), confidentiality, how to complain and how abstaining from taking part in this research would not affect their medical treatment, before filling out any questionnaires.

If the participant was agreeable they were presented with an informed consent sheet to sign (see Appendix D). Each participant was invited to ask any questions they wished before and after completing the ethical procedure and testing.

As participants were in the acute phase of stroke recovery the researcher was flexible when and if breaks were needed due to incontinence, tiredness, lunch time, physiotherapy appointments, visitor appointments, brain scans and so on. Testing was completed on the same day for all participants.

The measures were given at three time points: Time 1 (baseline 2-4 weeks post-stroke), Time 2 (3 months post-stroke) and Time 3 (6 months post-stroke). All measures were given at all three times points except for the SF 36 which was given at Time 2 and Time 3 only. The measures took approximately 25 minutes at Time 1; 40 minutes at Time 2 and 40 minutes at Time 3. Time 1 measurements are all recorded in the hospital. Time 2 and 3 measurements are recorded mostly in the patients home or in nursing homes they have been allocated to.

At the end of the Time 3 assessment respondents were given a debriefing sheet (see Appendix E) which was also verbally explained to them, explaining which measures were used and what they were intending to record. Participants were again reminded they had the right to withdraw and contact details of the Stroke Association and Stroke Clubs were given if further information or support was desired. Participants were also invited to receive the results of the study after analysis was complete.

The final section of this Chapter will introduce and justify the statistical tests used.

4.10 Statistical Analysis

Data were analysed using the IBM Statistical Package for the Social Sciences (SPSS) Version 20. Moderation and the Bootstrap test for mediation were conducted using the PROCESS macro for SPSS (Hayes, 2012).

In the following chapter descriptive statistics and frequencies were analysed. Pearsons correlation were used to detect if multicollinearity was present. A preliminary repeated measures within-subject ANOVA was performed to investigate changes at the 3 time points for each variable. The main analysis used was hierarchical regressions. This analysis was chosen in order to investigate the predictive value of psychological and cognitive variables on the outcome variables. In Denellot's 1996 study he controlled for cardiac factors to be able to investigate the predictive value of Type D personality above that of cardiac markers. Surtees et al., (2006) also controlled for stroke markers to again be able to investigate the role of SoC on stroke risk above stroke markers. These are achieved through hierarchical multiple regressions. Therefore, demographic variables, stroke risk and lifestyle factors will be controlled in order to investigate the role of psychological and cognitive variables at specific time points in relation to psychological and physical recovery from stroke at specific time points. In addition mediation and moderation analysis will also be conducted for variables outlined in the theoretical models using the up to date PROCESS macro for SPSS (Hayes, 2012).

Chapter 5

Results: Introduction to Study Two

5.1 Rationale and Summary of Results Chapters

The rationale behind this chapter is to introduce the analysis for Study 2. Before testing the 2 theoretical models, the statistical data will be described. This chapter will cover information on data preparation, normality tests, histograms, normal Q-Q plots, outliers, skewness and kurtosis, multicollinearity, the use of categorical variables and reliability. Descriptive data will then be summarised including information on participants (respondents and non-respondents), setting of testing, information on demographic, stroke markers, risk and lifestyle factors.

A descriptive short analysis is conducted to assess if the main psychological and cognitive factors demonstrate significant effects of time using repeated measures ANOVA. This is followed by the description of a screening process to select variables for the main hierarchical regression analysis. This is because the amount of data collected will cause the regression analysis to be saturated. Ten participants per variable is the guideline used (Field, 2013) to avoid overloading the regression models. Therefore, the utmost care has been taken to reduce the number of variables that can be included for analysis. This ultimately means the data collected and described was not all included in the final analysis. Mediation and moderation will also be introduced in this Chapter to be tested in the final analysis. The final analysis is outlined in Chapter 6 (for the Physical Recovery Model) and Chapter 7 (for the Psychological Recovery Model).

The results presented in this chapter were extracted from the data collected in Study Two. Data were analysed using the IBM Statistical Package for the Social Sciences (SPSS) Version 20.

5.2 Data Preparation

Parametric assessment was evaluated on scale variables. There are different ways to assess normality for example viewing histograms, evaluating skewness and kurtosis or conducting a Kolmogorov-Smirnov (KS) test (Field, 2013).

Firstly, the data were checked for any irregularities by checking the frequencies of each continuous variable and investigating the descriptive data for categorical variables. Normality tests will be discussed in the next section.

5.2.1 Normality Tests

Normality can be tested by using the Kolomogorov-Smirnoff (KS) test, where a normal distribution is concluded from a non-significant result. However, this test has been described as a corrupt method (Carver, 1978) as it has been criticised for not being sensitive to parametric testing leading to false positive conclusions. Distorted findings can occur due to differences in variance, sample size, skewness or kurtosis, which can lead to the conclusion that parametric tests are not advisable when they could be (Carver, 1978; Thompson, 1992 & Aguirre, Zarahn, & D'Esposito, 1998). The KS test may also not acknowledge the effect sizes (therefore the repeatability of the sample is fragile) (Oakes 1986) and the sampling variation can be overestimated as the test itself is over sensitive (Tabachnick & Fidell, 2007).

There has been research which highlights there is a lack of understanding about the limitations of significance testing and researchers rely too heavily on them even though there are inaccuracies within the test itself (Oakes, 1986). For this reason the KS test was not used and instead an individual investigation of the variables was conducted (see sections 5.2.2, 5.2.3, 5.2.4, 5.2.5).

Histograms will be discussed in the next section.

5.2.2 Histograms

Firstly, normality was assessed by observing histograms with a normal distribution curve. Most of the histograms were acceptable for a normal distribution relationship. There were a minority of variables that deviated from the norm (T1

Social Support and T3 Social Support).

As these deviations were in the minority and parametric tests can withstand some deviation from the norm (Pallent, 2013), it was decided to include these with the other measures with good normal distribution (Figures 5.1, 5.2, 5.3 and 5.4 illustrate normal distributions and slight deviation from the norm).

Normal Q-Q plots will be discussed in the next section.

5.2.3 Normal Q-Q Plots

Normal Q-Q plots were also analysed. In these plots the variables scores were plotted against a line which represents the normal distribution. Upon observing these plots, scores were situated around the norm. In Figures 5.1 & 5.2 a normal distribution is given using the variable Time 2 QoL. In Figures 5.3 & 5.4 an example is given of a variable (Time 3 Social Support), which is slightly non-normally distributed. As can be seen from the Q-Q plot this deviation is minor.

Outliers will be discussed in the next section.

5.2.4 Outliers

The next stage of the data preparation was to investigate outliers. There were no random outliers as assessed by scatter graphs and box plots. Participants that scored high or low on variables scored this way across variables and across time points, therefore these were not treated as outliers as they were not random and were retained in the data.

Additionally the means and medians of each variable were assessed to check if they were close in value as this can illustrate if the central point is distorted. Also, the mean was compared to the trimmed mean of variables. The 5% trimmed mean removes the top and bottom 5% of variables and a new mean is calculated. From this the effect of extreme scores on variables can be seen. If the mean and the 5% trimmed mean are close it shows the effect of extreme scores do not dramatically affect the overall variable (Pallent, 2013). After analysing the mean and the 5%

Figure 5.1
Histogram of Time 2 QoL with a normal distribution curve

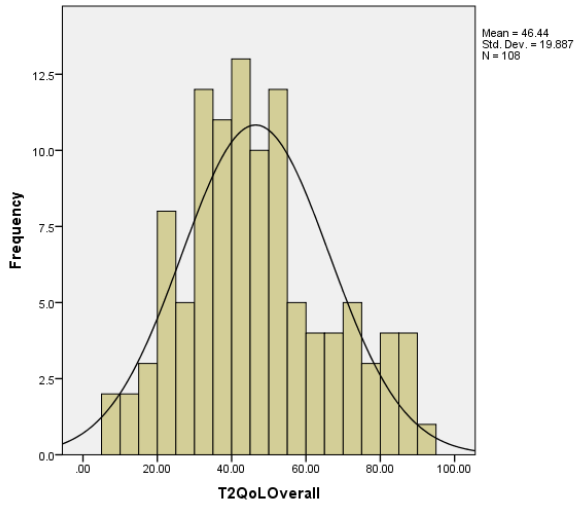


Figure 5.2
Normal Q-Q Plot of Time 2 QoL

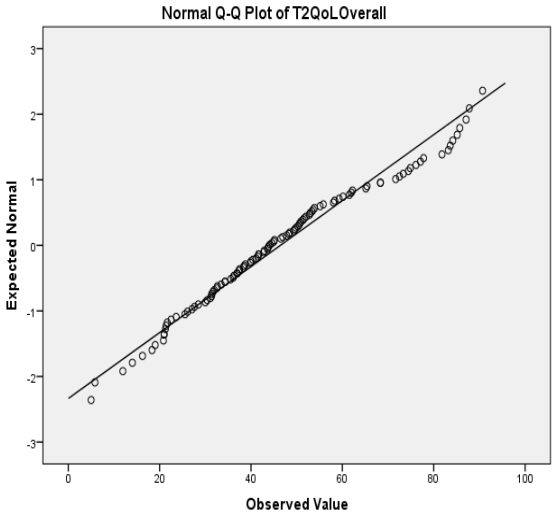


Figure 5.3
Histogram of Time 3 Social Support with a slight deviation from the norm

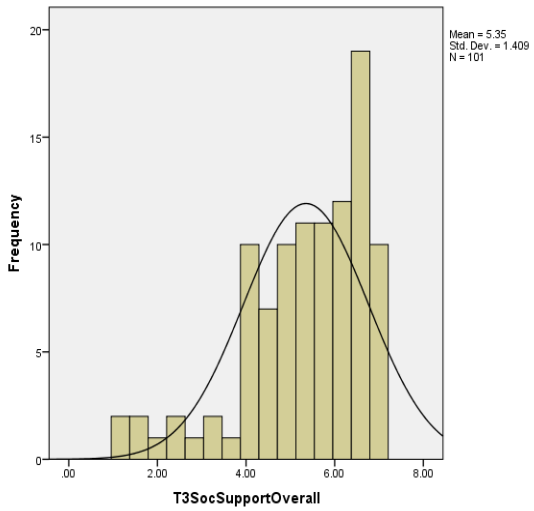
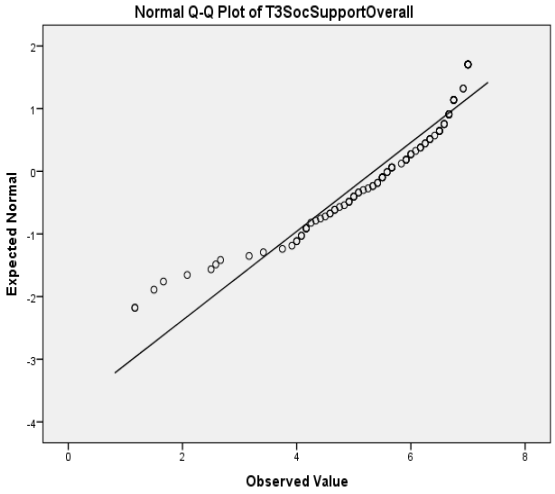


Figure 5.4
Normal Q-Q Plot of Time 3 Social Support



trimmed mean it was concluded that extreme scores did not have a strong impact on the mean, therefore extreme scores were kept.

Skewness and kurtosis will be discussed in the next section.

5.2.5 Skewness and Kurtosis

Normality can also be assessed with skewness and kurtosis (Pallent, 2013). Positive skewness shows there are more cases to the right hand side of the distribution and negative skewness means there are more cases to the left hand side of the distribution. Positive kurtosis indicates a peaked distribution and negative kurtosis indicates a flat distribution (Tabachnick & Fidell, 2007). Skewness and kurtosis was evaluated for the scale variables. Skewness decreases as the sample size increases and so the size of sample influences overall conclusions with larger sample sizes being more advantageous (Nunnally & Bernstein, 1994). Tabachnick & Fidell, (2007) state that skewness and kurtosis is reduced with a large sample (200+ cases).

Bulmer (1979) suggests skewness between -1 and +1 is acceptable and kurtosis between -2 and +2 is acceptable although there are no official cut off points for skewness and kurtosis in statistics literature. Skewness and kurtosis was satisfied in this sample.

Transforming variables will be discussed in the next section.

5.2.6 Transforming Variables

Transformation is practised when in the skewed distribution the mean is not a good indicator of the central tendency and so the median is used instead. The mean, median and 5% trimmed mean were assessed and as these were close in value transformation was not necessary.

Transformed variables are harder to interpret. If scales are widely used transformation can make results difficult to interpret and reduces comparability between studies. It is also a controversial topic as to transform means to alter the data to a way the researcher prefers which can introduce bias and lead to hesitant conclusions (Tabachnick & Fidell, 2001). Howell (1997) has suggested if the data is close to the normal distribution there is little value in transforming.

Multicollinearity will be discussed in the next section.

5.2.7 Multicollinearity

Multicollinearity is a correlation (*r*) above 0.9. This presents a difficulty as it illustrates two variables are measuring the same construct and therefore cannot be present in an analysis together because they increase the error rates.

When $r = 0.9$ and above in a regression analysis the accuracy of the analysis is diminished and therefore highly correlated variables should be kept separate or collapsed together (Tabachnick & Fidell, 2001).

Investigating Pearson's correlations, there were no variables with *r* above 0.9, which means multicollinearity is not present in the data. In regression analysis, *r* above 0.7 is the rule of thumb for multicollinearity (Pallent, 2013). This is monitored more details of which will be given in the main analysis in Chapter 6.

The correlation table is large. The utmost care was taken to produce concise tables for use as best as possible. Tables 5.1 – 5.9 present correlation matrices for the control, psychological and cognitive variables and physical and psychological outcomes at all time points. Pearson's Product Moment Correlation was used to calculate *r* for the majority of continuous with continuous variables. Point Biserial Correlation is a special case of the Pearson's Product Moment Correlation. This method was used to calculate *r* between continuous and discrete dichotomous variables (gender and thrombolysis). Discrete dichotomous variables are those variables which belong to either one group or another, for example, whether a participant has had thrombolysis treatment or not. Biserial Correlation was used to calculate *r* between continuous and artificially dichotomous variables (stroke recovery and repressive coping). Artificially dichotomous variables are variables which exist along a continuum but are separated into groups, for example, repressive coping scores. Point Biserial correlations are calculated in the same way as Pearson's *r*. Biserial correlations are not able to be computed in SPSS and have to be calculated by hand using an equation (see Equation 1):

$$r\mathrm{b} = \frac{r\mathrm{pb}\sqrt{(P_1\,P_2)}}{y} \quad (1)$$

Note. *r*b = biserial correlation. *r*pb = point biserial correlation. P_1 = proportion of cases that are coded 0. P_2 = proportion of cases that are coded 1. *y* = ordinate of the normal distribution.

The *r* statistic cannot be calculated for two discrete or artificial dichotomous variables (Field, 2013). Therefore *r* is unavailable for stroke severity with repressive coping and repressive coping with repressive coping (e.g., Time 1 repressive coping with Time 2 repressive coping).

The use of categorical variables will be discussed in the next section.

5.2.8 The use of Categorical Variables

Categorical variables may be used in correlations and multiple regression analyses. If a variable has more than two levels it should be converted into a dummy variable. There have been many surplus demographic, lifestyle and risk factor variables collected during the course of this research study, the inclusion of which would oversaturate the final regression models rendering them weak. Therefore a selection of the most pertinent variables to be controlled for, which best fit the hypotheses have been chosen. These are age, gender, stroke severity (2 dummy variables) and thrombolysis treatment (2 dummy variables). These variables occupy 6 spaces in the hierarchical regression analysis. The *n* for the smallest model (Time 3, QoL as an outcome) is 94, therefore leaving only 3 spaces for the main study variables. Therefore further inclusion of demographic, lifestyle and risk factor variables cannot be added.

The measure for stroke severity has 3 levels: mild, moderate and severe. Mild was used as the reference category therefore 2 dummy variables were calculated for stroke severity:

1. Mild stroke was considered the reference category and was coded as 0 and moderate stroke was coded as 1.

2. Mild stroke was considered the reference category and was coded as 0 and severe stroke was coded as 1.

In both of these categories the surplus group was also coded as 0.

Thrombolysis treatment also has 3 levels: no treatment, yes treatment and not applicable (N/A). No treatment was used as the reference category therefore 2 dummy variables were calculated for thrombolytic treatment:

1. No to treatment was considered the reference category and was coded as 0 and Yes to treatment was coded as 1.
2. No to treatment was considered the reference category and was coded as 0 and N/A was coded as 1.

In both of these categories the surplus group was also coded as 0.

Gender was coded as 0 for male and 1 for female. Repressive coping was coded as 0 for non-repressors and 1 for repressors. Type D personality and depression were measured on a scale however, to decipher groups they were divided into categories. For the Pearson's correlation these variables can be used in their continuous form. For the hierarchical multiple regressions these variables can be used in their categorical form to provide interpretations on which groups have or have not produced significant findings. Depression was coded as 0 for non-depressed (all responses below a total of 10) and 1 for depressed (all responses 10 and above) in accordance with the cut-off points selected by the creators of the scale (Andresen, Malmgren, Carter, & Patrick, 1994). Type D personality was coded 0 for non-Type D personality (scores of below 10 on both negative affectivity and social inhibition subscales) and 1 for Type D personality (scores of 10 and above on both negative affectivity and social inhibition subscales) in accordance with the guidelines set by the creator of this measurement (Denollet, 2005). Further details of these measurements are described in the Methodology Chapter (Chapter 4, Section 4.8.4, pages 224-232).

Table 5.1

Pearson correlation of control, Time 1 variables and all outcomes (QoL and mRS).

Scale	Gender	Stroke (Mild vs. Mod)	Stroke (Mild vs. Severe)	Thromb (No vs. Yes)	Thromb (No vs. N/A)
Age	.07	.05	.15	-.12	-.06
Gender	-	-.04	.16	-.09	-.10
Stroke (Mild vs. Moderate)		-	-.56**	-.04	.08
Stroke (Mild vs. Severe)			-	.03	.10
Thromb (No vs. Yes)				-	-.15
Thromb (No vs. N/A)					-
T1 D	-.06	.02	-.04	.05	-.03
T1 S	-.28**	.09	-.19	.05	-.75
T1 SS	-.05	.01	-.13	-.01	-.01
T1 tD	-.16	-.20	-.03	.06	.10
T1 RC	.06	-	-	-.15	-.02
T1 SoC	.03	.14	-.01	.02	-.05
T1 LB	-.05	.17	.01	.12	-.03
T1 B	-.22**	.05	.01	-.13	.12
T1 R	-.16	-.05	-.10	-.05	.03
T1 FDS	-.16	.10	-.13	.13	-.04
T1 St T	.05	.13	-.09	.15	-.08
T1 St E	.26**	-.10	.06	-.03	-.05
T1 mRS	.19*	.10	.51**	.43**	.18
T2 mRS	.25**	-.08	.10	.22*	-.06
T3 mRS	.40**	-.06	.19	.42**	-.08
T2 mRS	-.26**	.08	-.26	-.20*	.16
T3 QoL	-.33**	.03	-.07	-.32**	.29**

Note. D= Depression, S= Stress, SS= Social Support, tD= Type D personality, RC= Repressive Coping, SoC= Sense of Coherence, LB= Line Bisection, B= Bells Cancellation, R= Rivermead Behavioural Memory Test, FDS= Forward Digit Span, St T= Stroop Times, St E= Stroop Errors, QoL= Quality of Life, mRS = Modified Rankin Scale.

N's range from 77 to 143 due to missing data. ** $p < .01$ and * $p < .05$.

Table 5.2

Pearson correlation of control & Time 2 & Time 3 variables.

Scale	Age	Gender	Stroke (Mild vs. Mod)	Stroke (Mild vs. Severe)	Thromb (No vs. Yes)	Thromb (No vs. N/A)
T2 D	.03	.12	.14	.10	-.15	-.15
T2 S	.05	-.04	.09	.16	-.13	-.01
T2 SS	-.12	.03	-.13	.01	.07	.01
T2 tD	-.06	.03	.05	.06	-.05	-.06
T2 RC	.10	.03	-	-	-.04	.08
T2 SoC	-.07	.01	.14	-.18	-.12	.08
T2 LB	-.15	-.14	.06	-.41**	.08	.06
T2 B	-.31**	-.01	-.14	-.13	.17	-.09
T2 R	-.13	.01	-.13	-.13	.13	.01
T2 FDS	-.21*	.01	-.22	.16	.14	-.05
T2 St T	.12	.21	-.05	.09	-.05	-.13
T2 St E	.34**	.20	.14	.03	-.08	.02
T3 D	.11	-.01	-.06	.16	-.23*	.02
T3 S	.06	-.02	.01	.29*	-.25	.10
T3 SS	-.23*	.05	.01	-.03	.05	-.07
T3 tD	-.09	.02	-.17	.12	-.03	-.11
T3 RC	.38**	.11	-	-	.01	.04
T3 SoC	.07	.08	-.10	.25	-.05	-.05
T3 LB	-.05	.16	-.15	.12	.02	-.08
T3 B	-.31**	-.07	.05	-.40**	.18	-.11
T3 R	-.15	.18	.06	-.32*	.02	.04
T3 FDS	-.10	.07	-.14	.03	.05	-.02
T3 St T	.22*	.12	.06	.32*	-.20	.06
T3 St E	.27*	.12	.09	.01	-.21	.09

Note. D= Depression, S= Stress, SS= Social Support, tD= Type D personality, RC= Repressive Coping, SoC= Sense of Coherence, LB= Line Bisection, B= Bells Cancellation, R= Rivermead Behavioural Memory Test, FDS= Forward Digit Span, St T= Stroop Times, St E= Stroop Errors, QoL= Quality of Life, mRS = Modified Rankin Scale. *N*'s range from 77 to 143 due to missing data. ** $p < .01$ and * $p < .05$.

Table 5.3

Pearson correlation of Time 1 variables

Scale	T1 S	T1 SS	T1 tD	T1 RC	T1 SoC	T1 LB	T1 B	T1 R	T1 FDS	T1 St T	T1 St E
T1 D	.51*	-.19*	.33*	-.56**	.28*	.04	.16	.07	.09	-.03	-.04
T1 S	-	-.20*	.30**	-.47**	.34**	.16	.01	.09	.04	-.01	-.14
T1 SS		-	-.20*	-.02	-.20*	.06	-.07	-.09	.14	.09	.08
T1 tD			-	-.41**	.41**	-.13	.16	-.02	.01	-.08	.07
T1 RC				-	-.38**	.17	-.11	-.11	-.02	.18	-.17
T1 SoC					-	.06	.02	-.19*	-.07	.20*	.31**
T1 LB						-	-.11	-.05	.11	.02	-.17
T1 B							-	.46**	.01	.02	-.04
T1 R								-	.12	-.10	-.05
T1 FDS									-	-.27**	-.31**
T1 St T										-	.11
T1 St E											-

Note. D= Depression, S= Stress, SS= Social Support, tD= Type D personality, RC= Repressive Coping, SoC= Sense of Coherence, LB= Line Bisection, B= Bells Cancellation, R= Rivermead Behavioural Memory Test, FDS= Forward Digit Span, St T= Stroop Times, St E= Stroop Errors, QoL= Quality of Life, mRS = Modified Rankin Scale.

N's range from 106 to 143 due to missing data. ** $p < .01$ and * $p < .05$.

Table 5.4

Pearson correlation of Time 1 variables with all outcomes (QoL and mRS).

Scale	T1 mRS	T2 mRS	T2 mRS	T3 QoL	T3 QoL	
T1 D	.09	-.05	-.20*	-.02	-.14	
T1 S	-.10	-.01	-.01	-.03	-.14	
T1 SS	-.14	-.05	.28**	.05	.12	
T1 tD	.10	-.01	-.05	-.06	.07	
T1 RC	.02	-.02	.17	.21	-.03	
T1 SoC	.03	.20*	-.27**	.16	-.23*	
T1 LB	.06	.08	.01	.08	-.03	
T1 B	-.20*	-.16	.21*	-.26**	.31**	
T1 R	-.08	-.18	.07	-.34**	.08	
T1 FDS	-.04	-.10	.15	-.12	.01	
T1 St T	.09	.02	.02	.15	-.03	
T1 St E	.01	.24*	-.22*	.12	-.14	
T1 mRS	-	.34**	-.43**	.55**	-.36**	
T2 mRS		-	-.68**	.83**	-.58**	
T2 QoL			-	-.61**	.79**	
T3 mRS				-	-.63**	
T3 QoL						-

Note. D= Depression, S= Stress, SS= Social Support, tD= Type D personality, RC= Repressive Coping, SoC= Sense of Coherence, LB= Line Bisection, B= Bells Cancellation, R= Rivermead Behavioural Memory Test, FDS= Forward Digit Span, St T= Stroop Times, St E= Stroop Errors, QoL= Quality of Life, mRS = Modified Rankin Scale.

N's range from 77 to 119 due to missing data. ** $p < .01$ and * $p < .05$.

Table 5.5

Pearson correlation of Time 1 and 2 variables with outcome at all points (QoL and mRS).

Scale	T2 D	T2 S	T2 SS	T2 tD	T2 RC	T2 SoC	T2 LB	T2 B	T2 R	T2 FDS	T2 St T	T2 St E
T1 D	.36**	.26**	-.36**	.32**	-.30*	.40**	-.04	-.01	-.01	-.15	-.02	.14
T1 S	.26**	.02*	-.04	.24*	-.22	.09	-.01	-17	-.03	.08	-.24*	-.10
T1 SS	-.36**	-.28**	.79**	-.30**	.10	-.24*	.07	.15	.14	.17	-.09	-.18
T1 tD	.32**	.17	-.19*	.68**	-.29*	.29**	.04	-.01	-.07	.24*	-.03	-.03
T1 RC	-.49*	-.46*	-.09	-.41**	-	-.18	.12	-.29	.05	-.20	-.20	-.46**
T1 SoC	.40**	.32**	-.29**	.49**	-.38**	.43**	-.02	-.08	-.08	-.09	-.25*	.14
T1 LB	-.04	.01	.12	-.17	.14	.01	-.25**	-.12	-.15	.21*	-.07	-.21
T1 B	-.01	-.10	-.02	.22*	-.13	.04	.28**	.65**	.18	-.05	.21	-.19
T1 R	-.01	-.04	.06	.09	.01	.03	.19	.44**	.29**	.05	-.06	-.26*
T1 FDS	-.15	-.20*	.17	-.14	.09	-.18	.07	.31**	.21*	.64**	-.12	-.38**
T1 St T	-.02	-.04	.04	-.09	.01	-.02	-.01	-.03	.01	-.21	.51**	.30**
T1 St E	.26**	-.04	-.01	.08	.09	-.06	.31**	-.17	-.04	-.05	-.31**	.11

Scale	T2 D	T2 S	T2 SS	T2 tD	T2 RC	T2 SoC	T2 LB	T2 B	T2 R	T2 FDS	T2 St T	T2 St E
T1 mRS	.15	.09	-.14	.05	.18	-.02	-.17	-.23*	-.05	-.04	.06	.12
T2 mRS	.31**	.36**	-.07	.07	.17	.03	-.23*	-.26**	-.26**	-.12	.12	.19
T3 mRS	.19	.25*	.03	-.03	.11	-.05	-.22*	-.27**	-.25*	-.15	.13	.18
T2 QoL	-.63*	-.68**	.25**	-.31**	.19	-.25**	-.23*	.34**	.29**	.09	-.11	-.15
T3 QoL	-.37**	-.45	.10	-.13	.10	-.06	.21	.35**	.28**	.09	-.15	-.20

Note. D= Depression, S= Stress, SS= Social Support, tD= Type D personality, RC= Repressive Coping, SoC= Sense of Coherence, LB= Line Bisection, B= Bells Cancellation, R= Rivermead Behavioural Memory Test, FDS= Forward Digit Span, St T= Stroop Times, St E= Stroop Errors, QoL= Quality of Life, mRS = Modified Rankin Scale.

N's range from 77 to 143 due to missing data. ** $p < .01$ and * $p < .05$.

Table 5.6

Pearson correlation of Time 1 and 3 variables with outcome at all points (QoL and mRS).

Scale	T3 D	T3 S	T3 SS	T3 tD	T3 RC	T3 SoC	T3 LB	T3 B	T3 R	T3 FDS	T3 St T	T3 St E
T1 D	.36**	.28*	-.09	.31**	-.30*	.17	.05	-.06	-.15	.08	-.07	-.13
T1 S	.16	.17	-.12	.24*	.23	.07	.06	.07	.03	.20*	.09	-.14
T1 SS	-.11	-.02	.68**	-.21*	.16	-.12	-.07	-.06	.07	.07	-.06	-.02
T1 tD	-.01	.05	-.14	.60**	-.31*	.24*	-.07	.06	.09	-.04	.11	.27*
T1 RC	-.20	-.15	-.15	-.21	-	-.18	-.05	.02	.05	-.24	.27	.53**
T1 SoC	.34**	.35**	-.21*	.57**	-.43**	.46*	-.01	-.13	-.11	-.01	.21	.25*
T1 LB	.11	.11	.11	-.14	.13	-.01	.25*	-.13	-.04	.25*	-.05	.05
T1 B	-.19	-.24*	.09	.05	.19	-.03	.01	.65**	.24*	-.01	.07	-.13
T1 R	-.05	-.24*	.13	-.02	.18	-.03	.15	.27**	.20*	.09	-.22*	-.20
T1 FDS	-.06	-.05	.20	-.13	.05	.01	.10	.10	.15	.58**	-.05	-.18
T1 St T	.13	.11	.08	.07	-.11	.07	.02	-.13	-.09	-.21	.66**	.14
T1 St E	.01	-.05	-.09	.14	.05	.16	-.25*	-.23	-.20	-.33**	.14	.33**

Scale	T3 D	T3 S	T3 SS	T3 tD	T3 RC	T3 SoC	T3 LB	T3 B	T3 R	T3 FDS	T3 St T	T3 St E
T1 mRS	-.06	.06	.01	-.06	.19	.05	-.05	-.33**	-.14	-.12	.26*	.21
T2 mRS	.15	.34**	.08	.03	.04	.24	-.03	-.39	-.19	-.09	.30**	.37**
T3 mRS	.19	.37**	.07	.04	.03	.28**	-.04	-.41**	-.22*	-.18	.33**	.37**
T2 QoL	-.41**	-.42**	.20	-.16	.20	-.31**	.06	.38**	.14	.10	-.15	-.14
T3 QoL	-.57**	-.59**	.12	-.26	.26*	-.40**	.08	.45**	.24*	.10	-.19	-.17

Note. D= Depression, S= Stress, SS= Social Support, tD= Type D personality, RC= Repressive Coping SoC= Sense of Coherence, LB= Line Bisection, B= Bells Cancellation, R= Rivermead Behavioural Memory Test, FDS= Forward Digit Span, St T= Stroop Times, St E= Stroop Errors, QoL= Quality of Life, mRS = Modified Rankin Scale.

N's range from 77 to 143 due to missing data. ** $p < .01$ and * $p < .05$.

Table 5.7

Pearson correlation of Time 2 variables with outcome at all points (QoL and mRS).

Scale	T2 D	T2 S	T2 SS	T2 tD	T2 RC	T2 SoC	T2 LB	T2 B	T2 R	T2 FDS	T2 St T	T2 St E
T2 D	-	.80*	-.33**	.59**	-.61**	.49**	-.14	-.18	-.21*	-.10	.17	.01
T2 S		-	-.24*	.49*	-.53**	.42*	-.22*	-.28**	-.34**	-.16	.07	-.11
T2 SS			-	-.35**	.22	-.28**	-.07	-.23*	.07	.10	-.05	-.03
T2 tD				-	-.61**	.57**	.05	-.10	-.22*	-.11	.22*	.05
T2 RC						.43**	.14	.09	.23	-.09	-.06	-.15
T2 SoC						-	.19	-.11	-.01	-.08	.14	.21
T2 LB							-	.42**	.49**	.07	0.9	-.01
T2 B								-	.50**	.21*	-.01	-.21
T2 R									-	.21*	.01	-.05
T2 FDS										-	-.15	-.31**
T2 St T											-	.51**
T2 St E												-

Scale	T2 D	T2 S	T2 SS	T2 tD	T2 RC	T2 SoC	T2 LB	T2 B	T2 R	T2 FDS	T2 St T	T2 St E
T1 mRS	-.19*	.15	.01	-.14	.18	.03	-.02	-.17	-.23*	-.05	-.04	.06
T2 mRS	.25**	.31**	.37**	-.07	.11	.13	.03	-.23*	-.26**	-.26**	-.12	.12
T3 mRS	.40**	.20	.25*	-.03	.13	.05	-.05	-.22*	-.27**	-.25*	-.15	.13
T2 QoL	-.26**	-.63**	-.68**	.25**	.25*	-.39**	-.25**	.23*	.34**	.29**	.09	-.11
T3 QoL	-.33**	-.37**	.45**	.10	.14	-.23**	-.06	.21*	.35**	.28**	.09	-.15

Note. D= Depression, S= Stress, SS= Social Support, tD= Type D personality, MC= Marlowe-Crowne, STAI= Spielberger Trait-Strait Anxiety Inventory, SoC= Sense of Coherence, LB= Line Bisection, B= Bells Cancellation, R= Rivermead Behavioural Memory Test, FDS= Forward Digit Span, St T= Stroop Times, St E= Stroop Errors, QoL= Quality of Life, mRS = Modified Rankin Scale.

N's range from 77 to 108 due to missing data. ** $p < .01$ and * $p < .05$.

Table 5.8

Pearson correlation of Time 3 variables with outcome at all points (QoL and mRS).

Scale	T3 D	T3 S	T3 SS	T3 tD	T3 RC	T3 SoC	T3 LB	T3 B	T3 R	T3 FDS	T3 St T	T3 St E
T3 D	-	.81	-.22	.50	-.58**	.48	.04	-.28	-.34	-.03	-.03	-.09
T3 S		-	-.12	.54**	-.58*	.51**	-.03	-.34**	-.31**	-.02	-.10	-.05
T3 SS			-	-.25*	-.11	-.04	.04	.13	.10	.01	-.12	.01
T3 tD				-	-.69**	.53**	-.15	-.08	-.16	-.09	.17	.13
T3 RC						.37**	.14	.13	.31*	-.09	.15	.19
T3 SoC						-	.05	-.11	-.18	.04	.05	.10
T3 LB							-	.01	.15	.27**	-.13	-.07
T3 B								-	.36**	.14	-.13	-.19
T3 R									-	.18	.01	.01
T3 FDS										-	-.12	-.20
T3 St T											-	.36**
T3 St E												-

Scale	T3 D	T3 S	T3 SS	T3 tD	T3 RC	T3 SoC	T3 LB	T3 B	T3 R	T3 FDS	T3 St T	T3 St E
T1 mRS	-.06	.06	.01	-.06	.19	.05	-.05	-.33**	-.14	-.12	.26	.21
T2 mRS	.15	.34**	.08	.03	.04	.24*	-.03	-.39**	-.18	-.09	.30	.37**
T3 mRS	.19	.37**	.07	.04	.03	.28**	-.04	-.40**	-.22*	-.18	.33	.37**
T2 QoL	-.41**	-.42**	.20	-.16	.20	-.31**	.06	.38**	.14	.10	-.15	-.14
T3 QoL	-.57**	-.59**	.12	-.26**	.26*	-.40**	.08	.45**	.24*	.10	-.18	-.17

Note. D= Depression, S= Stress, SS= Social Support, tD= Type D personality, RC= Repressive Coping, SoC= Sense of Coherence, LB= Line Bisection, B= Bells Cancellation, R= Rivermead Behavioural Memory Test, FDS= Forward Digit Span, St T= Stroop Times, St E= Stroop Errors, QoL= Quality of Life, mRS = Modified Rankin Scale.

N's range from 77 to 101 due to missing data. ** $p < .01$ and * $p < .05$.

Table 5.9

Pearson correlation of Time 2 and 3 variables

Scale	T2 D	T2 S	T2 SS	T2 tD	T2 RC	T2 SoC	T2 LB	T2 B	T2 R	T2 FDS	T2 St T	T2 St E
T3 D	.42**	.44**	-.12	.24*	-.36**	.22*	-.14	-.28**	-.28*	-.05	.13	-.03
T3 S	.40*	.45**	-.10	.25*	-.22	.24*	-.13	-.28**	-.24*	-.10	.12	-.02
T3 SS	-.28**	-.23*	.77**	-.27*	.19	-.17	-.08	-.38*	.15	.18	.05	-.09
T3 tD	.44**	.33**	-.25*	.73**	-.41**	.38**	.12	-.09	-.05	-.13	.24*	.02
T3 RC	-.25	-.15	-.04	-.39**	-	-.31	-.06	.06	.15	-.28*	-.09	.21
T3 SoC	.32*	.03**	-.12	.40**	-.23*	.43**	-.05	-.11	-.07	.01	.15	.08
T3 LB	.03	.04	.01	-.09	-.01	-.04	-.32**	-.02	-.10	.11	-.13	-.09
T3 B	-.17	-.24*	-.01	.02	.08	-.01	.45**	.70**	.48**	.08	-.15	-.33**
T3 R	-.05	-.16	.06	-.04	.17	-.20	.04	.34**	.41**	.11	-.01	-.06
T3 FDS	-.10	-.14	.05	-.09	.04	-.23*	.04	.19	.10	.66**	-.20	-.28*
T3 St T	.11	.01	-.15	.10	.08	.07	.05	-.10	.02	-.17	.60**	.49**
T3 St E	.01	-.12	.01	.14	.25	.16	.03	-.31**	-.01	-.21	.40**	.60**

Note. D= Depression, S= Stress, SS= Social Support, tD= Type D personality, RC= Repressive Coping, SoC= Sense of Coherence, LB= Line Bisection, B= Bells Cancellation, R= Rivermead Behavioural Memory Test, FDS= Forward Digit Span, St T= Stroop Times, St E= Stroop Errors, QoL= Quality of Life, mRS = Modified Rankin Scale. *N*'s range from 77 to 107 due to missing data. ** $p < .01$ and * $p < .05$.

5.2.9 Reliability

Cronbach's Alpha measure for internal consistency assesses if items on a scale measure the same construct. The general rule of thumb is a coefficient of above 0.7 demonstrates a good internal consistency score (DeVellis, 2012).

Table 5.10 reports the Cronbach Alphas for the questionnaires used. "If item deleted" were reported only if it would increase the value of Alpha. However, as these increases were minimal no items were deleted. The only measure with values below 0.7 was the SoC measure. Therefore this measure was not included in further analysis.

The next section details descriptive data.

Table 5.10

Cronbach alpha coefficients for all scales

Variable	Time 1	Time 2	Time 3
PSS-14 (Stress)	0.81	0.82 (If Question 4 deleted, Cronbach's Alpha would be 0.87)	0.81 (If Question 4 deleted, Cronbach's Alpha would be 0.87)
Multidimensional Scale of Perceived Social Support	0.90	0.91	0.90
CES-D 10 (Depression)	0.75 (If Question 5 deleted, Cronbach's Alpha would be 0.76)	0.75 (If Question 7 deleted, Cronbach's Alpha is 0.76)	0.82
SoC 3 (Sense of Coherence)	0.31 (If Question 1 deleted, Cronbach's Alpha would be 0.33)	0.57	0.41 (If Question 2 deleted, Cronbach's Alpha would be 0.45)
Marlowe-Crowne Form B (Social Desirability)	0.68	0.69	0.73 (If Question 6 deleted, Cronbach's Alpha would be 0.73)
Six Item STAI (Anxiety)	0.82	0.89	0.82

Variable	Time 1	Time 2	Time 3
DS14 (Type D Personality)	0.81	0.85 (If Question 3 is deleted, Cronbach's Alpha would be 0.86)	0.80
SF 36 (Quality of Life)	N/A	0.93	0.92

5.3 Descriptive data

The following are summaries of descriptive data including information on participants (respondents and non-respondents), setting of testing, information on demographic, stroke markers, risk and lifestyle factors.

At Time 1, 143 participants were recruited from a sample of 202 available participants. At Time 2, 108 participants were followed up and at Time 3, 101 participants were followed up. Table 5.11 illustrates the loss to follow up at Time 2 and 3.

Table 5.11

Total loss of follow up in the overall study.

	Time 2		Time 3	
	N	%	N	%
Deceased	10	7.00%	1	0.70%
Unable To Participate	2	1.40%	1	0.70%
Another Stroke	0	0%	0	0%
Refused	16	11.20%	7	4.90%
Lost	7	4.90%	0	0%
Total Attrition	35	24.50%	9	6.30%

At Time 2 there was loss of data from 35 participants. However, the 2 participants that were unable to participate due to poor health were followed up and able to participate at Time 3, therefore they were included in Time 3. Therefore, the attrition rate was 29.37%.

The Time 1 recruitment numbers exceeded the power calculation which concludes 119 participants were needed to reach statistical power (see Chapter 4, Section 4.6, p. 214). At Time 2 and Time 3, participant numbers fell just below the 119 recommendation.

Participants were recruited at Time 1 solely from the hospital environment with Time 2 and 3 recruitment being collected mostly being in the home environment (see table 5.12).

Table 5.12

Setting of testing.

Test Setting		Time 1	Time 2	Time 3
Hospital	n	143	11	2
	%	100%	10.20%	2.00%
Home	n	0	92	93
	%	0%	85.20%	92.10%
Care Home	n	0	5	6
	%	0%	4.60%	5.90%

Table 5.13

Descriptive data for gender, ethnicity, education, occupation, retired status and marital status.

	Variable	*N*	%
Gender	Female	58	40.60%
	Male	85	59.40 %
Ethnicity	Caucasian	110	76.90%
	South Asian & East Asian	13	9.10%
	African & Caribbean	20	14.00%
Education	Less Than Secondary	22	15.60%
	Secondary	67	47.50%
	College	35	24.80%
	Undergraduate	9	6.40%
	Postgraduate	8	5.70%
Occupation	Professional	4	2.80%
	Managerial/ Technical	11	7.70%
	Skilled (Non Manual)	26	18.30%
	Skilled (Manual)	19	13.40%
	Partly Skilled	37	26.10%
	Unskilled	45	31.70%
Retired	Yes	90	62.90%
	No	53	37.10%

Variable		N	%
Marital Status	Never Married	21	14.70%
	Co-Habiting	9	6.30%
	Married	64	44.80%
	Divorced	15	10.50%
	Widowed	34	23.80%

The mean age for the study sample was 67.72 years (SD = 15.98), with a range from 19-96 years. There were more males (59.40%) then females, with Caucasians being the biggest ethnic group (76.90%). Just under half of all participants had secondary level schooling (47.50%) with postgraduate education accounting for the lowest amount of education achieved (5.70%). The highest percentage for occupation was in unskilled jobs (31.70%), with 62.90% being currently retired. Just under half of the participants were married (44.80%) with widowed participants accounting for 23.8% of the cohort. Co-habiting couples accounted for the lowest group (6.3%) followed by those that were divorced (10.50%).

Table 5.14

Descriptive data for number of stroke experienced at recruitment.

	Stroke Number				
	1	2	3	4	5
N	111	25	4	1	2
%	77.60%	17.50%	2.80%	0.70%	1.40%

Table 5.14 illustrates the frequency of stroke experienced by the participants with the majority experiencing a first stroke (77.60%) at recruitment. A second stroke was experienced by 17.50% at recruitment, a third stroke was by 2.80% at recruitment, a fourth stroke was experienced by 0.70% at recruitment and a fifth stroke was experienced by 1.40% at recruitment.

Table 5.15

Stroke Markers.

Variables		*N*	%
Stroke Type	Infarct	125	87.40%
	Hem	18	12.60%
Stroke Hemisphere	Left	64	45.40%
	Right	77	54.60%
Physical Stroke Severity	Mild	60	42.30%
	Moderate	58	40.10%
	Severity	25	17.60%
Thrombolysis	No	99	72.30%
	Yes	20	14.60%
	N/A	18	13.10%
TOAST[1]	Large Artery Atherosclerosis	20	18.30%
	Cardioembolism	30	45.90%
	Small Vessel Occlusion	44	86.20%
	Unknown	2	1.80%
	ICH	13	11.90%
Bamford[2]	TACS	1	1.00%
	PACS	48	49.50%
	LACS	29	29.90%
	POCS	19	19.60%

1 TOAST = Trial of ORG 10172 in Acute Stroke Treatment (TOAST)

2 Bamford Test = **TAC** — Total Anterior Circulation Stroke/ **PAC** — Partial Anterior Circulation Stroke/ **LAC** — Lacunar Stroke/ **POC** — Posterior Circulation Stroke.

Table 5.15 shows the majority of stroke recorded were infarctions with nearly an even distribution between the right (53.80%) and left (45.40%) hemispheres. The most frequent stroke severity recorded was of moderate level (40.10%), with the majority of participants experiencing small vessel occlusion (86.20%) and partial anterior circulation strokes (49.50%). The majority of the sample (72.30%) did not have thrombolysis treatment, with an additional 13.1% not being eligible and 14.60% receiving thrombolysis.

Table 5.16

Risk factors: Self reported family history of stroke and heart disease recorded at Time 1 visit.

Variables		*N*	%
Family History of Stroke	No	85	60.70%
	Yes	54	38.60%
	Do Not Know	1	0.70%
Family History of Heart Disease	No	8	61.90%
	Yes	51	38.10%

The majority of participants reported a family history of stroke (60.70%) and heart disease (61.90%).

Table 5.17

Self reported alcohol consumption status measured at 3 time points.

Alcohol Consumption		Time 1	Time 2	Time 3
No Never	*n*	16	14	14
	%	11.30%	13%	13.90%
No Now	*n*	33	54	44
	%	23.40%	50%	43.60%
Yes Now	*n*	92	40	43
	%	65.20%	37%	42.60%

Table 5.17 indicates that the majority of participants currently consuming alcohol (65.20%) at Time 1. However this decreases at Time 2 (37.00%), with nearly equal numbers currently consuming alcohol (43.60%) and currently consuming alcohol (43.60%) at Time 3.

Table 5.18

Self reported smoking status measured at 3 time points.

Smoking	Time 1	Time 2	Time 3
No Never	58	43	42
	41.10%	39.80%	41.60%
No Now	61	51	45
	43.30%	37.20%	44.60%
Yes Now	22	14	14
	15.60%	13.00%	13.90%

Table 5.18 indicates that the majority of participants were not currently smoking (No Never: 41.10% and No Now: 43.30%) at Time 1 and this trend continued into Time 2 and Time 3.

Table 5.19

Self reported diet status measured at 3 time points.

Diet	Time 1	Time 2	Time 3
Healthy	81	57	58
	58.30%	53.30%	57.40%
Moderate	37	43	32
	26.90%	40.20%	31.70%
Unhealthy	21	7	11
	15.10%	6.50%	10.90%

Table 5.19 depicts the majority of responses for self reported diet at Time 1 (58.30%); Time 2 (53.30%) and Time 3 (57.40%) were rated as healthy.

Table 5.20

Self reported exercise status measured at 3 time points.

Exercise	Time 1	Time 2	Time 3
None	55	34	31
	39.90%	31.80%	30.70%
Mild	72	6	65
	52.20%	61.70%	64.40%
Moderate	10	7	5
	7.20%	6.50%	5.00%
A Lot	1	0	0
	0.70%	0%	0%

Table 5.20 indicates that mild exercise status was the most frequent response at Time 1 (52.20%), Time 2 (61.70%) and Time 3 (64.40%).

Table 5.21

Self reported anti depressant medication status measured at 3 time points.

Antidepressant Medication	Time 1	Time 2	Time 3
No	132	93	86
	93.00%	65.00%	85.10%
Yes	10	15	15
	7.00%	10.50%	14.90%

Table 5.21 indicates a slight increase in anti depressant use from Time 1 (7.00%), Time 2 (10.50%) and Time 3 (14.90%). However, the majority of participants did not use anti depressant medication.

The following section reports on a brief preliminary analysis conducted with repeated measures within-subjects ANOVA.

5.4 Preliminary analysis

Repeated measures within-subjects ANOVA were conducted to investigate how scale variables changed at the 3 times points for the same participants. This analysis uses data that was complete from participants in all conditions (Field, 2013). This test investigates if there were any significant differences between participants at the 3 fixed time points. Table 5.22 records the means and standard deviations for all the variables and summarises the significant pairwise comparisons with Bonferroni correction.

Table 5.22

Summary of one-way repeated measures ANOVA characteristics.

Variable	*N*	Mean	*SD*	Pairwise Comparison
T1 Depression	99	1.15	0.67	T1 Depression/ T2 Depression **
T2 Depression	99	0.92	0.60	T1 Depression/ T3 Depression **
T3 Depression	99	0.90	0.67	
T1 Stress	99	1.38	0.70	
T2 Stress	99	1.59	0.74	
T3 Stress	99	1.54	0.71	
T1 Social Support	99	5.51	1.28	
T2 Social Support	99	5.38	1.45	
T3 Social Support	99	5.30	1.41	
T1 Type D	98	21.22	10.83	
T2 Type D	98	21.22	10.93	
T3 Type D	98	19.98	10.75	
T1 Line Bisection	93	0.22	1.73	
T2 Line Bisection	93	-0.21	1.10	
T3 Line Bisection	93	-0.10	1.27	
T1 Bells	92	27.32	8.45	Bells (T1)/ Bells (T2)*
T2 Bells	92	29.08	7.02	Bells (T1)/ Bells (T3)***
T3 Bells	92	29.96	6.63	
T1 RBMT	93	10.0	.86	
T2 RBMT	93	9.33	1.31	
T3 RBMT	93	9.24	1.50	

Variable	*N*	Mean	*SD*	
T1 Forward Digit Span	96	8.69	2.51	Forward Digit Span (T2)/
T2 Forward Digit Span	96	9.11	2.58	Forward Digit Span (T3) **
T3 Forward Digit Span	96	8.59	2.51	
T1 Stroop Reaction	64	24.67	28.28	Stroop Reaction Times (T1)/
T2 Stroop Reaction	64	17.55	13.45	Stroop Reaction Times (T3) **
T3 Stroop Reaction	64	16.80	17.83	
T1 Stroop Errors	64	5.03	5.90	
T2 Stroop Errors	64	3.64	4.87	
T3 Stroop Errors	64	3.69	4.74	
T1 mRS	100	2.89	1.21	mRS (T1)/ mRS (T2) **
T2 mRS	100	2.56	1.04	mRS (T1)/ mRS (T3) **
T3 mRS	100	2.52	1.00	
T2 QoL	99	46.94	19.09	
T3 QoL	99	48.91	19.17	

Note. *** $p < .001$ ** $p < .01$ and * $p < .05$. *N*'s range from 64 to 100 due to missing data.

Mauchly's Test of Sphericity should be above 0.05, which meets the assumption of equal variance. Wilks Lambda Multivariate testing demonstrates if there is a significant effect of time and to determine the effect size of the results partial eta squared (η_p^2) is reported (Pallent, 2013).

Significant changes over time were identified for depression, the Bells cancellation task, forward digit span, Stroop reaction times and physical recovery. For depression, Mauchly's Test of Sphericity was 4.98, with a significant effect of

time, Wilk's Lambda = 0.88, F (2, 97) = 6.65, p = 0.02, and multivariate η_p^2 = 0.12, which demonstrates a large effect size.

For the Bells cancellation task, Mauchly's Test of Sphericity was 0.05, with a significant effect of time, Wilk's Lambda = 8.63, F (2, 90) = 7.12, p = 0.001, and multivariate η_p^2 = 0.14, which demonstrates a large effect size.

For the forward digit span, Mauchly's Test of Sphericity was 0.47, with a significant effect of time, Wilk's Lambda = 0.93, F (2, 94) = 3.54, p = 0.03, and multivariate η_p^2 = 0.07, which demonstrates a small effect size.

For Stroop reaction times, Mauchly's Test of Sphericity was 0.01, with a significant effect of time, Wilk's Lambda = 0.89, F (2, 62) = 3.92, p = 0.03, and multivariate η_p^2 = 0.11, which demonstrates a moderate to large effect size.

For physical recovery (mRS), Mauchly's Test of Sphericity was 0.01, with a significant effect of time, Wilk's Lambda = 0.88, F (2, 98) = 6.54, p = 0.002, and multivariate η_p^2 = 0.12, which demonstrates a large effect size.

To determine significant changes between time points pairwise comparisons with Bonferroni corrections were calculated. For depression there were significant differences between Time 1 and Time 2, and Time 1 and Time 3. This indicates that depression significantly decreased at Time 2 and Time 3 compared with Time 1. This is the only psychological variable with significant differences across time ($p < .001$).

The other main variables that demonstrated significant changes over time were 3 cognitive variables; The Bells task, the forward digit span and Stroop reaction times. For the Bells task there were significant improvements between Time 1 and Time 2 ($p < .05$) and Time 1 and Time 3 ($p < .001$), with visual neglect improving over time. For the forward digit span there was a significant reduction between Time 2 and Time 3 ($p < .001$). This means that poorer verbal memory was reported at Time 3 indicating poor recovery of memory function. There was also a significant change between Time 1 and Time 3 Stroop reaction times ($p < .01$) with a significant improvement in Stoop reaction time recorded.

The modified Rankin Scale was the only outcome variable that had significant changes over time. Time 1 was significantly different from Time 2 ($p <$

.01) and Time 1 was significantly different from Time 3 ($p < .01$) with a reduction in mRS scores, which indicates an improvement in physical ability over time.

Stress, social support, Type D personality, the line bisection, the RBMT and Stroop errors did not exhibit significant effects at the 3 time points (see Table 5.24).

The next section details the screening procedure for the main analysis.

5.5 Screening Procedure

As there were many demographic data, potential risk factors and stroke markers that were measured during this research, a screening phase was instigated in order to choose the relevant variables to be included in the final regression model with 10 participants per independent variable in mind (Field, 2013). Adding too many variables into a regression model can cause problems such as collinearity effects, overfitting and Type I errors.

Collinearity occurs when variables are highly correlated. This may also occur if too many variables are entered into a regression model. Overfitting also occurs when a model is overloaded. In this situation discrete changes in the data can be over exaggerated, therefore possibly causing Type I errors to occur. A Type 1 error is when false positive conclusions are reported causing the true nature of the relationship between variables to become distorted and random error being detected instead (Everitt, 2002).

Stroop reaction times and Stroop errors were not included in the final analysis. This is because of the loss of viable data due to the reverse Stroop effect and the number of errors which exceeded 2 standard deviations from the mean on the congruent task. To include the Stroop task would reduce the final number for analysis to 65 cases, therefore there would be data loss across all variables in the regression. Sense of Coherence was removed from the final analysis due to the low Cronbach's alpha value that was calculated (see Section 5.2.8, Table 5.10, page 254).

As there were still too many variables a data reduction process was needed. As the data were longitudinal in design with many variables collected, multiple testing had to be conducted. Consequently it was important to reduce the number of

variables to be analysed to be able to fit the regression model and to include only those variables pertinent to the research hypotheses.

Hierarchical regression analysis was used to screen out non-significant variables. There were many variables collected in the study, which were not specific to the hypotheses, for example demographic, risk factor and lifestyle variables.

The variables chosen to be controlled for were age and gender for demographics, and stroke severity and thrombolysis treatment for stroke markers. Depression, stress, social support, Type D personality and repressive coping were used as the psychological predictor variables for T1, T2 and T3. The line bisection, the bells task, the forward digit span and the RBMT were used as cognitive predictors for T1, T2 and T3. The modified Rankin Scale was used as an outcome variable for T1, T2 and T3. Quality of life was used as the outcome variables for T2 and T3.

From the screening procedure the variables to be controlled for were entered in step 1 throughout the analysis. Gender and thrombolysis were removed as gender was not significant at all across analyses and thrombolysis was inconsistent from being significant across analyses. Age and stroke severity were retained to be used in the final analysis. Many psychological and cognitive variables were not significant in the screening procedure thus reducing the model size. All significant analyses are presented in Appendix U (p.533).

The next section will discuss multiple testing.

5.6 Multiple Testing

Multiple testing can produce false positive results (Type I error). This is when testing multiple hypotheses can produce significant results due to chance. There are different methods to address the Type I error, the most common of which is to use the Bonferroni correction. This correction can be calculated as $\alpha \div$ number of tests.

When dealing with a set of hypotheses firstly the family which it belongs to must be decided. Each hypothesis which contains sub hypotheses (e.g., $H_{1\ a,\ b,\ c,\ d}$, and $H_{2\ a,\ b,c,\ d}$) can be considered a family. In the case of the current study only those sub

hypotheses that have progressed past the screening phase would be tested, therefore not all sub hypotheses will be included. As an example for H_2 (as H_1 is not significant), the Bonferroni correction would be 0.05 ÷ 3 = .02. Therefore the significance level using a Bonferroni correction would be $p < .02$ for that particular family. For H_3, the Bonferroni correction would be 0.05 ÷ 4 = .01. Therefore the significance level using a Bonferroni correction would be $p < .01$ for H_3 (Westfall & Young 1993; Shaffer, 1995). It is important to remember that for each family of hypotheses different variables were investigated (e.g., Time 1 predicting Time 2 outcome and then Time 1 predicting Time 3 outcome), thus, the combination of variables change and do not remain static. Therefore repetitive testing of the same variables does not occur.

However the Bonferroni correction has been viewed as being too conservative and detrimental to analyses (Perneger, 1998) and may produce Type II errors (false negatives) at the cost of controlling for Type I errors and additionally the importance of a test depends upon the number of other analyses that have been tested (Shaffer, 1995). This debate still continues illustrated by this extract taken from a 2012 publication:

"The Bonferroni correction directly targets the Type 1 error problem, but it does so at the expense of Type 2 error. By changing the p value needed to reject the null (or equivalently widening the uncertainty intervals) the number of claims of rejected null hypotheses will indeed decrease on average. Although this reduces the number of false rejections, it also increases the number of instances that the null is not rejected when in fact it should have been. Thus, the Bonferroni correction can severely reduce our power to detect an important effect." (Gelman, Hill & Yajima, 2012, p. 192).

These authors have also declared that the Type I error may not be as important as reported as it is rare that the null hypothesis is always true and therefore multiple testing is not the problem at hand but the modelling of the analysis is more important.

In order to take a neutral stance and to accept both the effects of Type I and Type II errors the Bonferroni correction was calculated, however significant results

that exceeded the Bonferroni correction were also reported in order to guard against criticisms of committing a Type II error.

5.7 Conclusion

This Chapter has given the background of the quantitative study (Study Two). Data preparation, normality tests, histograms, normal Q-Q plots, outliers, skewness and kurtosis, transforming variables, multicollinearity, the use of categorical variables and reliability of measures have been acknowledged. Additionally, descriptive data and a preliminary analysis using repeated measures within-subjects ANOVA, was reported. This Chapter ended with sections on the screening procedure used for the final analysis and multiple testing.

In the following Chapter the Physical Recovery Model is tested.

Chapter 6

Results: Physical Recovery Model

6.1 Rationale and Summary

The rationale behind this chapter is to present the findings of analysis investigating the role of psychological and cognitive variables at 3 fixed time points (Time 1, Time 2 and Time 3) on physical recovery at 3 fixed time points (Time 1, Time 2, Time 3). The theoretical model was presented in Section 3.15, p. 204. This chapter analyses H_1, H_2 and H_3 which were initially outlined in Section 3.17.1, p. 207-211, using hierarchical multiple regression, the bootstrap test for mediation and moderation analyses. Psychological and cognitive predictors were analysed separately in order to provide a more rigorous analysis. The next section describes mediation and moderation analysis.

6.2 Mediation

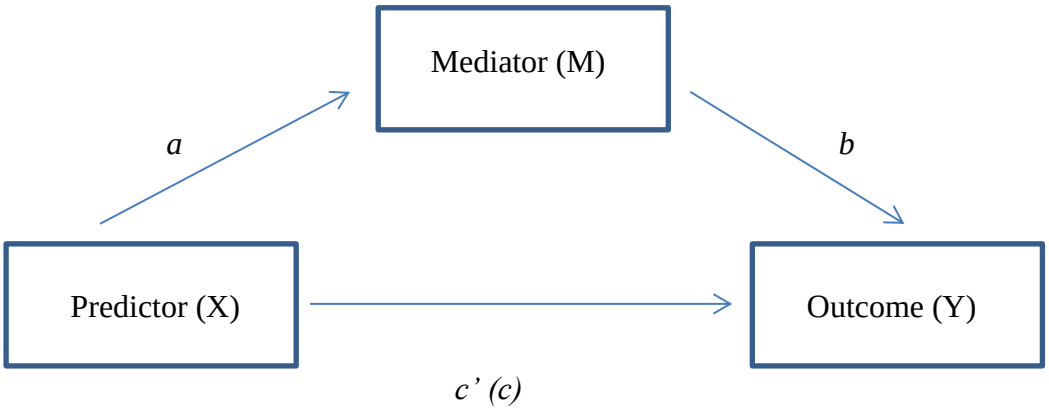
Mediation is when a third variable has a relationship between the predictor and the outcome variable (see Figure 6.1). The third variable is the mediator. As reported in Chapter 3, the Physical Recovery Model (Section 3.16, p. 200) predicted the presence of mediators between the predictor and outcome variables, e.g., depression was predicted to be a mediator between stress and physical recovery outcome (this relationship was hypothesized for all 3 fixed time points). Baron & Kenny (1986) propose a mediator must satisfy four criteria:

a) The predictor should be significantly correlated with the mediator.
b) The mediator should be significantly correlated with the outcome.
c) The predictor should be significantly correlated with the outcome.
d) The relationship between the predictor and the outcome is no longer significant (full mediation).

Partial mediation may also be detected of there is a reduction in the relationship between the predictor and the outcome.

Figure 6.1

Diagram of a mediation model.



Note. *a* = effect of X on M. *b* = effect of M on Y. *c* = direct effect of X on Y. *c'* = indirect effect of X on Y via M.

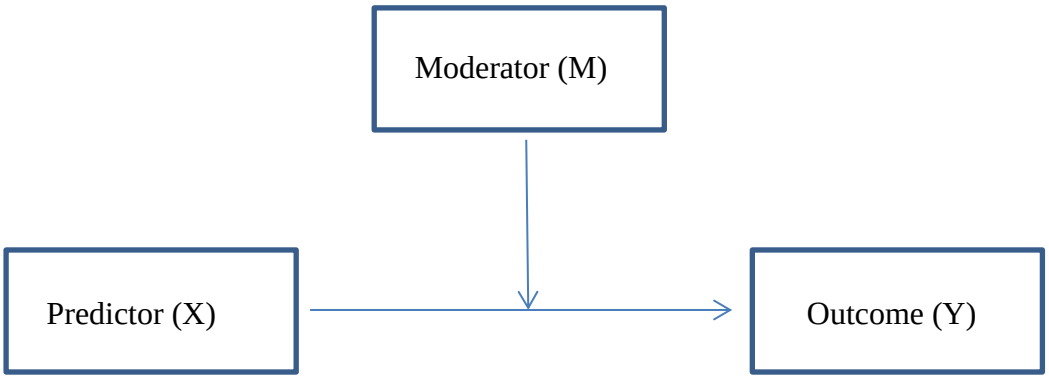
Significant mediating relationships are further discussed in Chapter 8. Moderation is discussed in the following section.

6.3 Moderation

Moderation is the effect of at least two predictor variables on an outcome variable (Baron & Kenny, 1986). The moderating variable affects the relationship between the predictor and the outcome (see Figure 6.2). As reported in Chapter 3, the Physical Recovery Model (Section 3.16, p. 200) predicts the presence of one moderator between the predictor and the outcome variable, e.g., social support was predicted to be a moderator between stress and physical recovery outcome (this relationship was hypothesized for all 3 fixed time points).

Figure 6.2

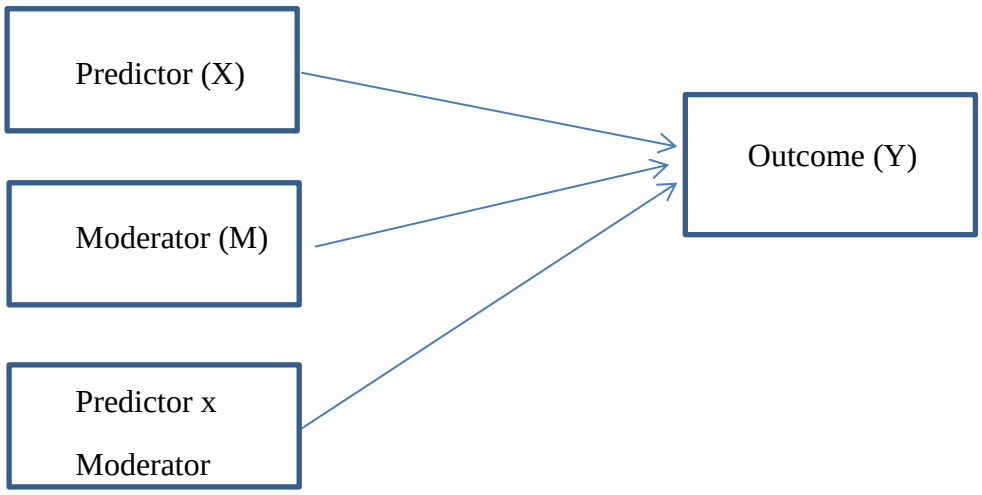
Diagram of a moderating model.



This is explored statistically by calculating the interaction effect between the predictor and the moderator on the outcome (see Figure 6.3).

Figure 6.3

Statistical moderation model.



The next section reports on the findings of H_1.

6.4 *H_1 a-r*: Time 1 variables predict Time 1 Physical recovery (at fixed time points).

From the screening procedure Time 1 (T1) psychological variables and T1 cognitive variables were not significant in predicting physical recovery. Investigating the correlation table there were no correlations between variables. This is further discussed in Chapter 8.

The next section reports on the findings of H_2.

6.5 *H_2*: Time 1 and 2 variables predict Time 2 Physical recovery (at fixed time points).

All analyses presented here are controlled by age and stroke severity based on the screening procedure.

H_2 *a, c, d, e, f, g, h, i, j, k, l, m, n, o, p, q, s* and *t* were not significant in the screening phase and therefore are not eligible to be discussed in this Chapter. H_2 *b, f,* and *r* were significant and are outlined in Sections 6.5.1, .6.5.2 and 6.5.3. The Bonferroni correction calculated for H_2 was $p = .02$.

6.5.1 *H_2 b*: High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 3 months post stroke (Time 2) will be associated with poorer physical recovery at a fixed time point of 3 months post stroke (Time 2).

In the screening procedure T2 stress and T2 repressive coping were significant and therefore were entered in the final analysis. A hierarchical multiple regression analysis was conducted to investigate if psychological variables at the fixed T2 point could predict physical recovery at the fixed T2 point when demographic and stroke markers were controlled for. Table 6.1 illustrates the outline for the model.

Table 6.1

Outline of H_2 b model.

Variables
Step 1
Demographic (age) & Stroke markers (stroke severity)
Step 2
T2 Psychological variables (T2 Stress & T2 Repressive coping)

Step 1 consisted of the control variables (age and stroke severity). This was to allow the investigation of T2 stress and T2 repressive coping (repressors vs. non repressors) as predictors of T2 mRS over and above the effects of the control variables at the fixed 3 month post stroke time (T2). The variables chosen to be entered in this sequence are based on H_2 *a*. Means and standard deviations for the model variables are shown in Table 6.2.

Table 6.2

Means and standard deviations for the model variables.

Variable	*M*	*SD*
T2 Rankin	2.53	1.05
Age	67.93	14.52
Stroke Severity (Mild vs.Mod)	.43	.50
Stroke Severity (Mild vs. Severe)	.21	.41
T2 Stress	1.59	.75
T2 Non-Repressors vs. Repressors	.39	.49

Multicollinearity was checked and there were no violations as the tolerance values were all above 0.10, the Variance Inflation Factor (VIF) values were all below

10 and there were no correlations above r = 0.7 (see Table 6.3 for the correlations table). Normality, homoscedasticity and linearity were also satisfied.

To assess if outliers were causing a distortion the critical chi-square value using an alpha value of .001 (Pallent, 2013) was investigated. This value was 20.52. The Mahalanobis distance was below this at 19.38. Cooks distance was below 1 (0.08), therefore there were no violations of outliers.

Table 6.3

Correlations among T2 mRS, Age, Stroke Severity, T2 Stress & T2 repressive coping.

Variable	2.	3.	4.	5.	6.
1. T2 mRS	.21*	.26**	.41***	.36***	.09
2. Age	-	.03	.14	.05	.08
3. Stroke (Mild vs. Mod)		-	-.44***	.07	-.01
4. Stroke (Mild vs. Severe)			-	.11	.02
5. T2 Stress				-	-.42***
6. T2 Non-Rep vs. Rep					-

Note. *** $p < .001$ ** $p < .01$ and * $p < .05$.

Table 6.3 illustrates the absence of mulicollinearity. The control variables were the most significant variables in the correlation matrix, with T2 stress exhibiting the most significant psychological effect associated with physical recovery (r = .36***).

A hierarchical multiple regression analysis was conducted to investigate the predictive value of T2 stress and T2 repressive coping on T2 physical recovery when demographic and stroke markers were controlled for at the fixed time point of 3 months post stroke. Age and stroke severity were entered in Step 1 explaining 41.8% of the variance. After T2 stress and T2 repressive coping were entered in Step 2, the total variance explained by the model was 49.7% (adjusted R^2) (R^2 = 52.1%), F (5,

101) = 21.95, $p < 0.001$, N= 107 (25.17% missing data), with each step being significant at $p < 0.001$.

Table 6.4

Regression coefficients for H_2 b.

Variable	*B*	*SE B*	β	Δ*R*
Step 1				.42
Age	.01	.01	.11	
Stroke (Mild vs. Mod)	1.13	.18	.54***	
Stroke (Mild vs. Sev)	1.62	.22	.63***	
Step 2				.10
Age	.01	.01	.08	
Stroke (Mild vs. Mod)	1.03	.16	.49***	
Stroke (Mild vs. Sev)	1.46	.20	.56***	
T2 Stress	.50	.11	.35***	
T2 Non-Rep vs. Rep	.46	.16	.22**	

Note. *** $p < .001$ ** $p < .01$ and * $p < .05$. Stroke severity (Mild vs. Mod) was dummy coded 0 and 1. Stroke severity (Mild vs. Sev) was dummy coded 0 and 1. Repressors vs. Non-Repressors was coded 0 and 1.

In the final model stroke severity had the highest contribution to T2 mRS (see Table 6.4) with moderate (β= .49, t (105) = 6.24, $p < .001$) and severe strokes (β= .56, t (105) = 7.14, $p < .001$) at stroke onset predicting a strong relationship with physical recovery at T2, with the higher severity at baseline predicting poorer recovery at T2. For example a one unit increase (from 0 to 1) in severe stroke severity scores predicted a .56 increase in poorer recovery scores from stroke and a one unit increase (from 0 to 1) in moderate stroke severity predicted a .49 increase in poorer recovery from stroke.

Additionally, the results from this analysis illustrate that once age and baseline severity were controlled for there were psychological factors which were

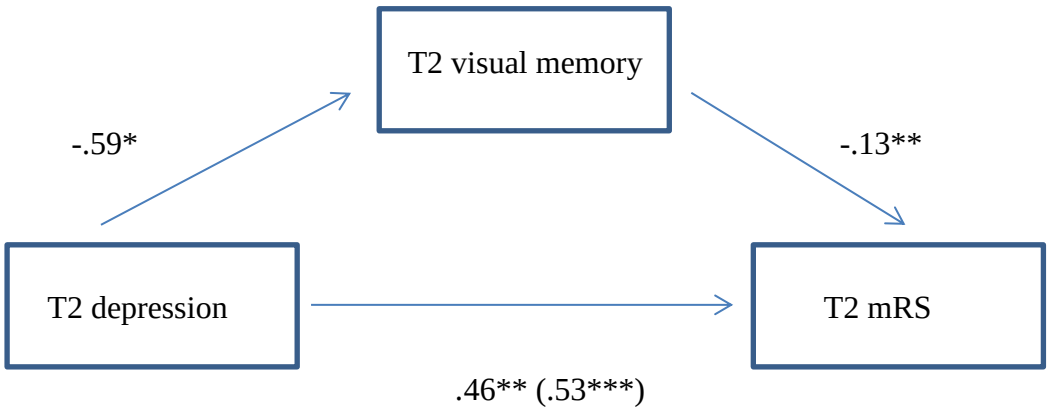
strong enough to make a contribution to the model such as psychological mood and coping style. At the fixed T2 point, higher stress scores predicted poorer T2 mRS scores (β= .35, *t* (105) = 4.57, *p* < .001). A one *SD* rise (.75) in stress scores at T2 predicted a .35 rise in poorer recovery scores. Additionally, repressive coping was significant once it was entered into the model (β= .22, *t* (105) = 2.82, *p* < .001). Repressors demonstrated poorer physical recovery compared to non-repressors at T2. A one *SD* increase in T2 repressive coping scores (.49) predicted a .22 increase in poorer recovery scores. However, T2 stress offered a unique psychological contribution to the model (β= .35, *p* < .001).

6.5.2 *H_2f*: T2 visual memory will mediate T2 Depression and T2 physical recovery.

The mediating model for *H_2f* is presented in Figure 6.4. The bootstrap test for mediation was conducted with the PROCESS macro for SPSS which conducts multiple regressions and mediation analyses (Hayes, 2012).

Figure 6.4

Mediation model of T2 visual memory mediating T2 depression andT2 mRS.



Note. *** *p* < .001 ** *p* < .01 and * *p* < .05.

The relationship between T2 depression and T2 mRS was mediated by T2 visual memory scores (from the RBMT measure). As Figure 6.4 illustrates the unstandardized regression coefficients of the *a* path between T2 depression and T2 visual memory was significant (*B*= -.59, *t* (102) = 2.18, *p* < .05) along with the *b* path between T2 visual memory and T2 mRS (*B*= -.13, *t* (102) = -2.19, *p* < .01) and the *c* path between T2 depression and T2 mRS (*B*= .53, *t* (102) = 3.19, *p* < .001). Unstandardized indirect effects were computed for each of the 5,000 bootstrapped samples (Preacher & Hayes, 2004) and the 95% confidence interval for the indirect effect was calculated. Mediation analyses confirmed the mediating role of T2 visual memory between T2 depression and T2 mRS (*B*= .08, CI .01, .18). The *c'* path between T2 depression and T2 mRS remained significant (*B*= .46, *t* (102) = 2.71, *p* < .001) when controlling for T2 visual memory, therefore illustrating partial mediation. An effect size of κ^2 = .05, 95% CI .01, .10 was calculated which represents a small effect size.

The negative relationship between T2 depression and T2 visual memory was not expected. Higher T2 depression scores predicted a decrease in T2 visual memory scores (*B*= -.59). Additionally higher T2 visual memory scores were predictive of lower T2 mRS scores, which was an expected result (*B* = -.13). The *c* and the *c'* path both illustrate a positive relationship between T2 depression and T2 mRS (*B*= .53 and *B*= .46), therefore higher T2 depression scores predicted poorer T2 mRS scores.

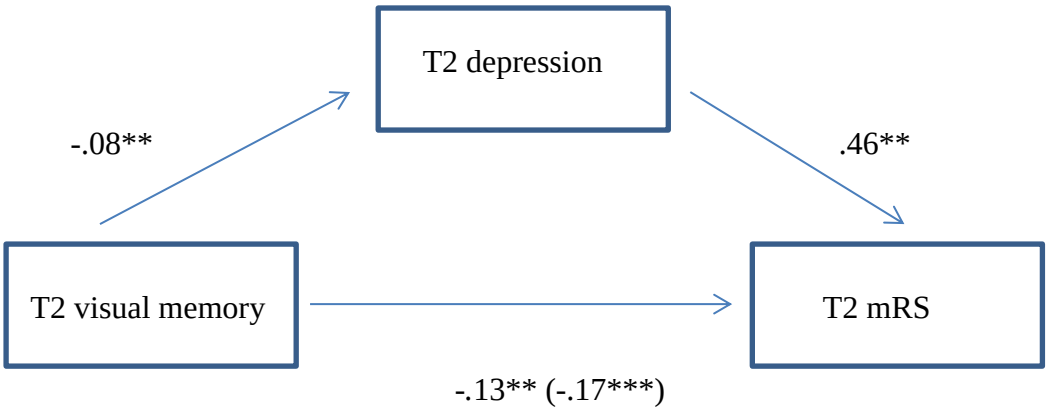
In this model the *a* path had a significance level of *p* = .03, whilst the Bonferroni correction set a threshold of *p* = .02. This difference between the Bonferroni correction and the p value is minor. At the risk of performing a Type II error, this result was reported.

6.5.3 *H_2r:* T2 depression will mediate T2 visual short term memory and T2 physical recovery.

The mediating model for *H_2p* is presented in Figure 6.5.

Figure 6.5

Mediation model of T2 visual memory mediating T2 depression andT2 mRS.



Note. *** $p < .001$ ** $p < .01$ and * $p < .05$.

The relationship between T2 visual memory (measured on the RBMT) and T2 mRS was mediated by T2 depression. As Figure 6.5 illustrates the unstandardized regression coefficients of the *a* path between T2 visual memory and T2 depression was significant (*B*= -.08, *t* (102) = -2.18, $p < .01$) along with the *b* path between T2 depression and T2 mRS (*B*= .46, *t* (102) = 2.71, $p < .01$) and the *c* path between T2 depression and T2 mRS (*B*= -.17, *t* (102) = -2.74, $p < .001$). Unstandardized indirect effects were computed for each of the 5,000 bootstrapped samples (Preacher & Hayes, 2004) and the 95% confidence interval for the indirect effect was calculated. Mediation analyses confirmed the mediating role of T2 depression between T2 visual neglect and T2 mRS (*B*= -.03, CI -.08, -.01). The *c'* path between T2 visual memory

and T2 mRS remained significant (B= -.13, t (102) = -2.19, p < .01) when controlling for T2 depression, therefore illustrating partial mediation. An effect size of κ^2 = .06, 95% CI .02, .11 was calculated which represents a medium effect size.

The negative relationship between T2 visual memory and T2 depression was expected illustrating higher scores in T2 visual memory predicted lower scores in T2 depression (B= -.08). Additionally higher scores in T2 depression predicted higher scores in T2 mRS, which was an expected result (B = .46). The *c* and the *c'* path both illustrate a negative relationship between T2 visual memory and T2 mRS (B= -.17 and B= -.13), therefore higher T2 visual memory scores predicted improvements in T2 mRS scores.

The next section reports on the findings of H_3.

6.6 H_3: Time 1, 2 and 3 variables predict Time 3 Physical recovery.

All analyses presented here are controlled by age and stroke severity based on the screening procedure.

H_3 *a, d, e, g, h, i, j, k, l, m, n, o, p, q, r, s, t, u* and *v* were not significant predictors and therefore are not eligible to be discussed in this Chapter. H_3 *b, c* and *f* were significant and are outlined in Sections 6.6.1, .6.6.2 and 6.6.3. The Bonferroni correction calculated for H_3 was $p = .02$.

6.6.1 H_3 *b*: High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 3 months post stroke (Time 2) will predict poorer physical recovery at a fixed time point of 6 months post stroke (Time 3).

and

H_3 *c*: High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 6 months post stroke (Time 3) will predict poorer physical recovery at a fixed time point of 6 months post stroke (Time 3).

This section will cover both H_3 *b* and *c*. In the screening procedure T3 stress and T3 social support were significant in predicting T3 mRS. A hierarchical multiple regression analysis was conducted to investigate if psychological variables at T2 (H_3 *b*) and T3 (H_3 *b*) could predict physical recovery at T3 when demographic and stroke markers were controlled for. Table 6.5 illustrates the outline for the model.

Table 6.5

Outline of H_3 b and c model.

Variables
Step 1
Demographic (age) & Stroke markers (stroke severity)
Step 2
T2 Psychological predictors (T2 stress
T2 repressive coping)
Step 3
T3 Psychological variables (T3 Stress
T3 social support)

In Step 1 the control variables (age and stroke severity) were entered. In Step 2 the previous T2 significant variables were entered (T2 stress and T2 repressive coping) to be assessed in their prediction of T3 mRS and also to be controlled for once T3 stress and T3 social support were entered in Step 3. The variables chosen to be entered in this sequence are based on $H_{3.}$ Means and standard deviations for the model variables are shown in Table 6.6.

Table 6.6

Means and standard deviations for the model variables.

Variable	*M*	*SD*
T3 Rankin	2.48	.94
Age	67.56	14.46
Stroke Severity (Mild vs. Mod)	.43	.50
Stroke Severity (Mild vs. Severe)	.21	.41
T2 Stress	1.59	.74
T2 Non-Repressors vs. Repressors	.40	.50
T3 Stress	1.54	.71
T3 Social Support	5.32	1.41

Multicollinearity was checked by assessing the tolerance and VIF values. There were no violations as the tolerance values were all above 0.10, the Variance Inflation Factor (VIF) values were all below 10, and there were no correlations above r = 0.7 (see Table 6.7 for the correlations table).

To assess if outliers were causing a distortion the critical chi-square value using an alpha value of .001 (Pallent, 2013) was investigated. This value was 26.13. The Mahalanobis distance was below this at 20.88. Cooks distance was below 1 (0.16), therefore there were no violations of outliers. Normality, homoscedasticity and linearity were also satisfied.

Table 6.7

Correlation for T3 mRS, age, stroke severity, T2 Stress, T2 Repressive coping, T3 Stress & T3 Social Support.

Variable	2.	3.	4.	5.	6.	7.	8.
1. T3 mRS	.39***	.18*	.47***	.25**	.10	.38***	.06
2. Age	-	.03	.20*	.06	.06	.04	-.21*
3. Stroke Severity (Mild vs. Mod)		-	-.46***	.02	-.02	-.02	.01
4. Stroke Severity (Mild vs. Severe)			-	.14	.03	.22**	-.04
5. T2 Stress				-	-.42***	.45***	-.23**
6. T2 Non-Rep vs. Rep					-	-.17*	.15
7. T3 Stress						-	-.12
8. T3 Social Support							-

Note. *** $p < .001$ ** $p < .01$ and * $p < .05$.

Table 6.7 illustrates the absence of mulicollinearity. The control variables were the most significant variables in the correlation matrix, with T3 stress exhibiting the most significant psychological effect associated with physical recovery (r = .38***), followed by T2 stress (r = .25**).

A hierarchical multiple regression analysis was conducted to investigate the predictive value of T3 Stress and T3 social support on T3 physical recovery when demographic, stroke markers and previous psychological variables were controlled for (T2 stress and T2 repressive coping). Age and stroke severity were entered in Step 1 accounting for 47% of the variance. T2 stress and T2 repressive coping were entered in Step 2 and explained an additional 4% of the variance. After T3 stress and T3 social support were entered into Step 3, the total variance explained by the model was 55.4% (adjusted R^2) (R^2= 58.6%), F (7, 91) = 18.37, p < 0.001, N= 99 (30.77% missing data), with each step being significant at p < 0.001.

Table 6.8

Regression coefficients for H_3

Variable	*B*	*SE B*	β	Δ*R*
Step 1				.47
Age	.02	.01	.25**	
Stroke Severity (Mild vs. Mod)	.86	.16	.45***	
Stroke Severity (Mild vs. Sev)	1.44	.20	.63***	
Step 2				.04
Age	.02	.01	.24**	
Stroke Severity (Mild vs. Mod)	.82	.16	.44***	
Stroke Severity (Mild vs. Sev)	1.34	.19	.59***	
T2 Stress	.27	.10	.22**	
T2 Non Rep vs. Rep	.32	.15	.17*	
Step 3				.07
Age	.02	.01	.28***	
Stroke Severity (Mild vs. Mod)	.78	.15	.41***	
Stroke Severity (Mild vs. Sev)	1.22	.19	.53***	
T2 Stress	.19	.11	.15	
T2 Non-Rep vs. Rep	.29	.14	.15*	
T3 Stress	.31	.10	.24**	
T3 Social Support	.12	.05	.18**	

Note. *** $p < .001$ ** $p < .01$ and * $p < .05$. Stroke severity (Mild vs. Mod) was dummy coded 0 and 1. Stroke severity (Mild vs. Sev) was dummy coded 0 and 1. Repressors vs. Non-Repressors was coded 0 and 1.

In the final model stroke severity was again the strongest predictor of stroke recovery in all Steps. Age also featured as a prominent predictor. In the final model, stroke severity explained the most significant contribution to T3 mRS (see Table 6.8) with moderate (β= .41, t (97) = 5.34, $p < .001$) and severe stroke (β= .53, t (99) = 6.56, $p < .001$) at stroke onset predicting a strong relationship with physical recovery at T3, with higher severity at Time 1 predicting poorer recovery at T3. For example a

one unit increase (from 0 to 1) in severe stroke severity predicted a .53 increase in poorer recovery from stroke and a one unit increase (from 0 to 1) in moderate stroke severity predicted a .41 increase in poorer recovery from stroke. This illustrates moderate and severe strokes were associated with poorer recovery scores.

Age in the final model demonstrated a positive predictive relationship with T3 mRS (β= .28, t (99) = 3.95, p < .001), with a one unit (*SD*) increase (14.46) predicting a .28 increase in T3 mRS scores. This illustrates older participants reported poorer recovery scores.

In Step 2 (H_3 *b*), T2 stress and T2 repressive coping significantly predicted T3 mRS. T2 stress (β= .22, t (99) = 2.66, p < .01) demonstrated a positive relationship with T3 mRS, where a one unit (*SD*) increase (.74) in stress scores predicted a .22 increase in poorer recovery scores. Additionally, T2 repressive coping (β= .18, t (99) = 2.12, p < .01) demonstrated that a one unit (*SD*) increase in repressor scores predicted a .17 increase in poorer recovery scores. Therefore, repressors are more likely than non-repressors to recover slower from stroke, although the β was small.

The Bonferroni correction was calculated as p = .02. The repressors vs. non-repressors variable exceeded this threshold but was significant at p = .04. At the expense of possibly committing a Type II error, repressive coping was retained in this analysis and reported.

In the final model (H_3 *c*) T3 stress and T3 social support significantly predicted T3 mRS. T3 stress (β= .24, t (99) = 3.04, p < .01) demonstrated a positive relationship with T3 mRS, where a one unit (*SD*) increase (.71) in stress scores predicted a .24 increase in poorer recovery scores. Additionally, T3 social support (β= .18, t (99) = 2.50, p < .01) demonstrated that a one unit (*SD*) increase (1.41) in social support scores predicted a .18 increase in poorer recovery scores. This illustrates the more social support this sample reported at T3, the worse their recovery was at T3.

In the final model, severe stroke severity made the biggest unique contribution (β= .3**) with T3 stress making the biggest psychological contribution (β= .24**). The results from this analysis indicate that T2 repressors, higher T2 and

T3 stress scores and higher T3 social support scores are related to poorer physical recovery at T3.

6.6.2 *H_3f*: High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at the fixed time of 6 months post stroke (Time 3) will predict poorer physical recovery at the fixed time of 6 months post stroke (Time 3).

In the screening procedure for cognitive variables T3 Bells cancellation task (for visual neglect) was the only significant cognitive variable in predicting T3 mRS. A hierarchical multiple regression analysis was conducted to investigate if the Bells cancellation task at the fixed 6 month post stroke period (T3) could predict physical recovery at T3 when demographic and stroke markers were controlled for. Table 6.9 illustrates the outline for the model.

Table 6.9

Outline of H_3f model.

Variables
Step 1
Demographic (age) & Stroke markers (stroke severity)
Step 2
T3 Cognitive variable (T3 Bells)

In Step 1 the control variables were placed (age and stroke severity). This was to allow the investigation of T3 Bells as a predictor of T3 mRS over and above the effects of the control variables at the fixed 6 month post stroke time (T3). The variables chosen to be entered in this sequence are based on *H_3f*. Means and standard deviations for the model variables are shown in Table 6.10.

Table 6.10

Means and standard deviations for the model variables.

Variable	*M*	*SD*
T3 Rankin	2.50	.93
Age	66.68	14.88
Stroke Severity (Mild vs. Mod)	.43	.50
Stroke Severity (Mild vs. Severe)	.23	.42
T3 Bells	29.86	6.66

Multicollinearity was checked by assessing the tolerance and VIF values. There were no violations as the tolerance values were all above 0.10, the Variance Inflation Factor (VIF) values were all below 10, and there were no correlations above r = 0.7 (see Table 6.11 for the correlations table).

To assess if outliers were causing a distortion, the critical chi-square value using an alpha value of .001 (Pallent, 2013) was investigated. This value was 18.47. The Mahalanobis distance was below this at 15.88. Cooks distance was below 1 (0.01), therefore there were no violations of outliers.

Table 6.11

Correlations among T3 mRS, Age, Stroke Severit & T3 Bells

Variable	2.	3.	4.	5.
1. T3 mRS	.38***	.17*	.48***	-.40***
2. Age	-	.06	.17*	-.31**
3. Stroke (Mild vs. Mod)		-	-.48***	.04
4. Stroke (Mild vs. Severe)			-	-.27**
5. T3 Bells				-

Note. *** $p < .001$ ** $p < .01$ and * $p < .05$.

Table 6.11 illustrates the absence of mulicollinearity. The control variables were the most significant variables in the correlation matrix. T3 Bells exhibits a significant correlation with physical recovery at T3 (r = -.40***).

A hierarchical multiple regression analysis was conducted to investigate the predictive value of T3 Bells cancellation (visual neglect) on T3 physical recovery when demographic and stroke markers were controlled for. Age and stroke severity were entered in Step 1 and explained 49% of the variance. After T3 Bells was entered into Step 2, the total variance explained by the model was 50.2% (adjusted R^2) (R^2 = 52.3%), F (4, 90) = 24.68, $p < 0.001$, N= 95 (33.57% missing data), with each step being significant at $p < 0.001$.

Table 6.12

Regression coefficients for H_3.

Variable	*B*	*SE B*	β	Δ*R*
Step 1				.49
Age	.02	.01	.24**	
Stroke Severity (Mod)	.88	.16	.47***	
Stroke Severity (Sev)	1.47	.19	.67***	
Step 2				.03
Age	.01	.01	.18*	
Stroke Severity (Mod)	.86	.16	.46***	
Stroke Severity (Sev)	1.35	.19	.62***	
T3 Bells	-.03	.01	-.20**	

Note. *** $p < .001$ ** $p < .01$ and * $p < .05$. Stroke severity (Mild vs. Mod) was dummy coded 0 and 1. Stroke severity (Mild vs. Sev) was dummy coded 0 and 1.

Stroke severity was again the strongest predictor of stroke recovery in all Steps. Age also featured as a prominent predictor. In the final model stroke severity had the highest explanatory contribution to T3 mRS (see Table 6.12) with moderate

(β= .46, *t* (93) = 5.45, *p* < .001) and severe stroke (β= .62, *t* (95) = 7.05, *p* < .001) at stroke onset predicting a strong relationship with physical recovery at T3, with the higher severity at baseline predicting poorer recovery at T3. For example a one unit increase (from 0 to 1) in severe stroke severity scores predicted a .63 increase in poorer recovery scores and a one unit increase (from 0 to 1) in moderate stroke severity scores predicted a .46 increase in poorer recovery scores.

Age in the final model demonstrates a positive predictive relationship with T3 mRS (β= .18, *t* (95) = 2.37, *p* < .05), with a one unit (*SD*) increase (14.88) predicting a .18 increase in poorer recovery scores. This illustrates older participants report worse recovery at 6 months post stroke.

In the final model T3 Bells significantly predicted T3 mRS. T3 Bells (β= -.20, *t* (95) = -2.55, *p* < .01) demonstrated a negative relationship with T3 mRS, where a one unit (*SD*) increase (6.66) in T3 Bells scores predicted a -.20 decrease in T3 mRS scores. This illustrates that as visual neglect at T3 improves, T3 recovery improves.

In the final model, severe stroke severity made the biggest unique contribution (β= .62***), while visual neglect at T3 was the only cognitive variable to make a contribution to physical recovery at T3 (β= -.20*).

In the following section the results are summarised.

6.7 Results Summary

In this section the results are summarised.

1. *H_1 a-r*: Time 1 variables predict Time 1 Physical recovery (at fixed time points).

No significant variables were established at Time 1.

2. *H_2 b*: High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 3 months post stroke (Time 2) will be associated with poorer physical recovery at a fixed time point of 3 months post stroke (Time 2).

In this analysis T2 stress and T2 repressive coping confirmed *H_2 b.* The control variables (age and stroke severity) accounted for 42% of the variance, with T2 stress and T2 repressive coping accounting for an additional 10% of the variance.

i. Moderate strokes at baseline predicted poorer recovery at T2.
ii. Severe strokes at baseline predicted poorer recovery at T2.
iii. High stress scores at T2 predicted poorer recovery at T2.
iv. Repressors at T2 experience poorer recovery at T2.

3. *H_2f:* T2 visual memory will mediate T2 Depression and T2 physical recovery.

In this analysis *H_2 f* was confirmed. The relationship between T2 depression and T2 mRS was mediated by T2 visual memory scores (from the RBMT measure).

i. The *a* path reported a negative relationship between T2 depression and T2 visual memory, with higher T2 depression scores predicting lower T2 visual memory scores. The Bonferroni correction for H_2 was calculated at $p = .02$. The *a* path exceeded this at $p = .03$.
ii. The *b* path reported higher T2 visual memory scores were predictive of lower T2 mRS scores at the fixed time points.
iii. The *c* and the *c'* path both reported a positive relationship between T2 depression and T2 mRS illustrating higher T2 depression scores predicted poorer T2 mRS scores.

4. *H_2r:* T2 depression will mediate T2 visual short term memory and T2 physical recovery.

In this analysis *H_2 r* was confirmed. The relationship between T2 visual memory (measured on the RBMT) and T2 mRS was mediated by T2 depression.

i. The *a* path reported a negative relationship between T2 visual memory and T2 depression illustrating higher scores in T2 visual memory predicted lower scores in T2 depression.
ii. The *b* path reported higher scores in T2 depression predicted higher scores in T2 mRS at the fixed time points.
iii. The *c* and the *c'* path both illustrated a negative relationship between T2 visual memory and T2 mRS therefore higher T2 visual memory scores predicted improvements in T2 mRS scores.

5. H_3 *b*: High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 3 months post stroke (Time 2) will predict poorer physical recovery at a fixed time point of 6 months post stroke (Time 3).

In this analysis T2 stress and T2 repressive coping confirmed H_3 *b*. The control variables (age and stroke severity) accounted for 47% of the variance, with T2 stress and T2 repressive coping accounting for an additional 4% of the variance.

i. Moderate strokes at baseline predicted poorer recovery at T3.
ii. Severe strokes at baseline predicted poorer recovery at T3.
iii. Older age was associated with poorer recovery at T3.
iv. Higher stress scores at T2 was predictive of poorer recovery at T3.
v. Repressors at T2 experience poorer recovery at T3. The Bonferroni correction calculated for H_3 was $p = .02$. T2 repressors vs non-repressors exceeded this value at $p = .04$.

6. H_3 *c*: High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 6 months post stroke (Time 3) will predict poorer physical recovery at a fixed time point of 6 months post stroke (Time 3).

In this analysis T3 stress and T3 social support confirmed *H_3 c*. The control variables (age and stroke severity) accounted for 47% of the variance, with T3 stress and T3 social support accounting for an additional 7% of the variance.

i. Moderate strokes at baseline predicted poorer recovery at T3.
ii. Severe strokes at baseline predicted poorer recovery at T3.
iii. Older age was associated with poorer recovery at T3.
iv. Higher stress scores at T3 predicted poorer recovery at T3.
v. Higher scores at T3 social support predicted poorer recovery at T3.

7. *H_3f*: High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at the fixed time of 6 months post stroke (Time 3) will predict poorer physical recovery at the fixed time of 6 months post stroke (Time 3).

In this analysis the T3 Bells cancellation task confirmed *H_3 f*. The control variables (age and stroke severity) accounted for 49% of the variance, with T3 Bells accounting for an additional 3% of the variance.

i. Moderate strokes at baseline predicted poorer recovery at T3.
ii. Severe strokes at baseline predicted poorer recovery at T3.
iii. Older age was associated with poorer recovery at T3.
iv. Higher T3 Bells scores predicted improvements in recovery at T3.

Chapter 7

Results: Psychological Recovery Model

7.1 Rationale and Summary

The rationale behind this chapter is to present the findings of analysis investigating the role of psychological and cognitive variables at 3 fixed time points (Time 1, Time 2 and Time 3) on psychological recovery (QoL) at 2 fixed time points (Time 2 and Time 3). The theoretical model is presented In Section 3.16, p. 205. This chapter will analyse H_4, H_5 and H_6 which were initially outlined in Section 3.17.2 p. 211-213. Psychological and cognitive predictors were analysed separately in order to provide a more rigorous analysis.

7.2 H_4: Time 1 variables predict Time 2 QoL.

All analyses presented here are controlled by age and stroke severity based on the screening procedure.

H_4 *b* was not significant in the screening phase and therefore is not eligible to be discussed in this Chapter. H_4 *a* was significant and is outlined in Section 7.2.1. The Bonferroni correction calculated for H_4 was $p = .05$.

7.2.1 H_4 *a*: High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 0-6 weeks post stroke (Time 1) will predict poorer QoL at a fixed time point of 3 months post stroke (Time 2).

In the screening procedure T1 social support was significant and therefore entered in the final analysis. A hierarchical multiple regression analysis was conducted to investigate if this psychological variable at T1 could predict QoL at the fixed T2 point when age and stroke severity were controlled for. Table 7.1 illustrates the outline for the model.

Table 7.1

Outline of H_4 a model.

Variable
Step 1
Demographic (age) & Stroke markers (stroke severity)
Step 2
T1 Psychological variables (T1 social support)

Step 1 consisted of the control variables (age and stroke severity). This was to allow the investigation of T1 social support as predictors of T2 QoL over and above the effects of the control variables at the fixed 3 month post stroke time (T2). The variables chosen to be entered in this sequence are based on *H_4 a*. Means and standard deviations for the model variables are shown in Table 7.2.

Table 7.2

Means and standard deviations for the model variables.

Variable	*M*	*SD*
T2 QoL	46.44	19.89
Age	68.18	14.67
Stroke Severity (Mild vs. Mod)	.43	.50
Stroke Severity (Mild vs. Severe)	.20	.40
T1 Social Support	5.52	1.24

Multicollinearity was checked and there were no violations as the tolerance values were all above 0.10, the Variance Inflation Factor (VIF) values were all below 10, and there were no correlations above r = 0.7 (see Table 7.3 for the correlations table). Normality, homoscedasticity and linearity were also satisfied.

To assess if outliers were causing a distortion, the critical chi-square value using an alpha value of .001 (Pallent, 2013) was investigated. This value was 18.47. The Mahalanobis distance was slightly above this at 20.86. This was due to only one case. In data sets there will be a few cases that will exceed this criteria however, if it is minimal it is acceptable to keep this data (Pallent, 2013). Cooks distance was below 1 (0.20), therefore there were no violations of outliers.

Table 7.3

Correlations among T2 QoL, Age, Stroke Severity & T1 Social Support.

Variable	2.	3.	4.	5.
1.T2 QoL	-.26**	-.18*	-.20*	.28**
2. Age	-	.01	.13	-.04
3. Stroke Sev (Mild vs. Mod)		-	-.44***	-.01
4. Stroke Sev (Mild vs. Sev)			-	.01
5. T1 Social Support				-

Note. *** $p < .001$ ** $p < .01$ and * $p < .05$.

Table 7.3 illustrates the absence of mulicollinearity. The control variables were the most significant variables in the correlation matrix, with T1 social support exhibiting a strong significant relationship with QoL (r = .28**).

A hierarchical multiple regression analysis was conducted to investigate the predictive value of T1 social support on T2 QoL when demographic and stroke markers were controlled for. Age and stroke severity were entered into Step 1 explaining 17.4% of the variance. After T1 social support was entered in Step 2, the total variance explained by the model was 22% (adjusted R^2) (R^2 =24.9%), F (4, 103) = 8.54, $p < 0.001$, N= 108 (24.48% missing data), with each step being significant at $p < 0.001$.

Table 7.4

Regression coefficients for H_4 a.

Variable	*B*	*SE B*	β	Δ*R*
Step 1				0.17
Age	-.30	.12	-.22*	
Stroke Severity (Mild vs. Mod)	-12.57	4.0	-.31**	
Stroke Severity (Mild vs. Sev)	-15.00	4.92	-.31**	
Step 2				.08
Age	-.28	.12	-.21*	
Stroke Severity (Mild vs. Mod)	-12.47	3.81	-.31**	
Stroke Severity (Mild vs. Sev)	-15.13	4.71	-.31**	
T1 Social Support	4.39	1.37	.27**	

Note. *** $p < .001$ ** $p < .01$ and * $p < .05$. Stroke severity (Mild vs. Mod) was dummy coded 0 and 1. Stroke severity (Mild vs. Sev) was dummy coded 0 and 1.

In the final model stroke severity explained the largest contribution to T2 QoL (see Table 7.4) with moderate (β= -.31, t (106) = -.3.27, ** $p < .01$) and severe strokes (β= -.31, t (106) = -.3.2, ** $p < .01$) at stroke onset predicting a strong relationship with QoL at T2, with higher severity at baseline predicting poorer QoL at T2. For example a one unit increase (from 0 to 1) in severe stroke severity scores predicted a -.31 decrease in T2 QoL scores and a one unit increase (from 0 to 1) in moderate stroke severity also predicted a -.31decrease in T2 Qol scores.

Age in the final model demonstrated a negative predictive relationship with T2 QoL (β= -.21, t (106) = -2.39, $p < .05$), with a one unit (*SD*) increase (19.89) predicting a -.21 decrease in T2 QoL scores. This illustrates older participants reported lower T2 QoL scores.

Additionally, the results from this analysis illustrate that once age and baseline severity were controlled for T1 social support was strong enough to make a contribution to the model. At the fixed T1 point, T1 social support was significant once it was entered into the model (β= .27, t (106) = 3.21, $p < .001$). This reveals a

one *SD* increase in T1 social support scores (1.24) predicted a .27 increase in T2 QoL scores.

The next section reports on the findings of H_5.

7.3 H_5: Time 1 and 2 variables predict Time 2 QoL.

All analyses presented here are controlled by age and stroke severity based on the screening procedure.

H_5 *b* was not significant in the screening procedure and therefore was not eligible to be discussed in this Chapter. H_5 *a* was significant and is outlined in Sections 7.3.1. . The Bonferroni correction calculated for H_2 was $p = .05$.

7.3.1 H_5 *a*: High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 3 months post stroke (Time 2) will predict poorer QoL at a fixed time point of 3 months post stroke (Time 2).

In the screening procedure T2 depression and T2 stress were significant in predicting T2 QoL. A hierarchical multiple regression analysis was conducted to investigate if psychological variables at T2 could predict psychological recovery at T2 when demographic, stroke markers and previous significant T1 social support was controlled for. Table 7.5 illustrates the outline for the model.

Table 7.5

Outline of H_5 a model.

Variable
Step 1
Demographic (age) & Stroke markers (stroke severity)
Step 2
T1 Psychological variables (T1 Social Support)
Step 3
T2 Psychological variables (T2 depression and T2 stress)

In Step 1 control variables (age and stroke severity) were entered. In Step 2 the previous T1 significant variable was entered (T1 social support) to be controlled for once T2 depression and T2 stress were entered in Step 3 as predictors of T2 QoL. The variables chosen to be entered in this sequence are based on H_5 *a*. Means and standard deviations for the model variables are shown in Table 7.6.

Table 7.6

Means and standard deviations for the model variables.

Variable	*M*	*SD*
T2 QoL	46.66	19.84
Age	67.93	14.52
Stroke Severity (Mild vs. Mod)	.43	.50
Stroke Severity (Mild vs. Severe)	.21	.41
T1 Social Support	5.52	1.25
T2 Depression	.41	.49
T2 Stress	1.59	.75

Multicollinearity was checked and there were no violations as the tolerance values were all above 0.10, the Variance Inflation Factor (VIF) values were all below

10, and there were no correlations above r = 0.7 (see Table 7.7 for the correlations table). Normality, homoscedasticity and linearity were also satisfied.

To assess if outliers were causing a distortion, the critical chi-square value using an alpha value of .001 (Pallent, 2013) was investigated. This value was 24.32. The Mahalanobis distance was below this at 24.22. Cooks distance was below 1 (0.06), therefore there were no violations of outliers.

Table 7.7

Correlations among T2 QoL, Age, Stroke Severity, T1 Social Support, T2 Depression & T2 Stress.

Variable	2.	3.	4.	5.	6.	7.
1. T2 QoL	-.25**	-.20*	-.20*	.29**	-.60***	-.68***
2. Age	-	.03	.14	-.04	.08	.05
4. Stroke Severity (Mild vs. Mod)		-	-.44***	-.01	.16*	.07
5. Stroke Severity (Mild vs. Sev)			-	.01	.09	.11
6. T1 Social Support				-	-.21**	-.28**
7. T2 Depression					-	.62***
8. T2 Stress						-

Note. *** $p < .001$ ** $p < .01$ and * $p < .05$.

Table 7.7 illustrates the absence of mulicollinearity. The control variables were significant variables in the correlation matrix, with T3 stress exhibiting the most significant psychological effect associated with physical recovery (r = -.68***), followed by T2 depression (r = -.60***).

A hierarchical multiple regression analysis was conducted to investigate the predictive value of T2 depression and T2 stress on T2 QoL when demographic, stroke markers and previous T1 social support were controlled for. Step 1 explained

17.8% of the variance in T2 QoL, whilst Step 2, explained an additional 0.8% of the variance. After T2 depression and T2 stress were entered in Step 2, the total variance explained by the model was 56.1% (adjusted R^2) (R^2 = 58.6%), F (6, 100) = 23.59, p < 0.001, N= 107 (25.17% missing data), with each step being significant at p < 0.001.

Table 7.8

Regression coefficients for H_5

Variable	*B*	*SE B*	β	Δ*R*
Step 1				0.18
Age	-.26	.12	-.19*	
Stroke Severity (Mild vs. Mod)	-13.31	3.99	-.33**	
Stroke Severity (Mild vs. Sev)	-15.83	4.94	-.32**	
Step 2				.08
Age	-.25	.12	-.18*	
Stroke Severity (Mild vs. Mod)	-13.26	3.82	-.33**	
Stroke Severity (Mild vs. Sev)	-16.05	4.72	-.33**	
T1 Social Support	4.45	1.36	-.28**	
Step 3				.33
Age	-.23	.09	-.17*	
Stroke Severity (Mild vs. Mod)	-8.86	2.95	-.22**	
Stroke Severity (Mild vs. Sev)	-10.18	3.62	-.21**	
T1 Social Support	1.68	1.07	.11	
T2 Depression	-6.95	3.37	-.17*	
T2 Stress	-13.02	2.24	-.49***	

Note. *** p < .001 ** p < .01 and * p < .05. Stroke severity (Mild vs. Mod) was dummy coded 0 and 1. Stroke severity (Mild vs. Sev) was dummy coded 0 and 1.

The control variables (age and stroke severity) were predictive in each step of the model. In the final model stroke severity and age explained significant

relationships to T2 QoL (see Table 7.8) with moderate (β= -.22, *t* (105) = -3.01, *p* < .01) and severe strokes (β= -.21, *t* (105) = -2.81, *p* < .01) at stroke onset predicting a strong relationship with QoL at T2, with higher severity at Time 1 predicting poorer QoL at T2. For example a one unit increase (from 0 to 1) in severe stroke severity predicted a -.21 decrease in poorer QoL at T2 and a one unit increase (from 0 to 1) in moderate stroke severity scores predicted a -.22 decrease in poorer T2 QoL scores. This illustrates moderate and severe strokes were associated with poorer T2 QoL scores.

Age in the final model demonstrated a negative predictive relationship with T2 QoL (β= -.17, *t* (105) = -2.53, *p* < .05), with a one unit (*SD*) increase (14.52) predicting a -.17 decrease in T2 QoL scores. This illustrates older participants reported lower T2 QoL scores.

In the final model after the previous significant variable had been controlled for (T1 social support), T2 depression and T2 stress significantly predicted T2 QoL. T2 depression (β= -.17, *t* (105) = -2.07, *p* < .05) demonstrated a positive relationship with T2 QoL, where a one unit (*SD*) increase (.49) in depression scores at T2 predicted a -.17 decrease in T2 QoL scores. Additionally, T2 stress (β= -.49, *t* (105) = -5.82, *p* < .001) demonstrated that a one unit (*SD*) increase (.75) in stress scores at T2 predicted a -.49 decrease in T2 QoL scores. This illustrates higher depression and stress scores at the fixed T2 point was predictive of lower T2 QoL scored in this sample.

In the final model, T2 stress made the biggest unique contribution (β= -.49***) surpassing stroke markers. The results from this analysis indicate that an increase in T2 depression and T2 stress was related to poorer T2 QoL.

The next section reports on the findings of H_6.

7.4 H_6: Time 1, 2 and 3 variables predict Time 3 QoL.

All analyses presented here are controlled by age and stroke severity based on the screening procedure.

In this section H_6 *a, b* and *c* were significant and are outlined in Section 7.4.1. H_6 *d* and *f* were significant and are outlined in Section 7.4.2. . The Bonferroni correction calculated for H_6 was $p = .03$.

7.4.1 H_6 *a:* High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 0-6 weeks post stroke (Time 1) will predict poorer QoL at a fixed time point of 6 months post stroke (Time 3).

and

H_6 b: **High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 3 months post stroke (Time 2) will predict poorer QoL at a fixed time point of 6 months post stroke (Time 3).**

and

H_6 c: **High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 6 months post stroke (Time 3) will predict poorer QoL at a fixed time point of 6 months post stroke (Time 3).**

This section covers H_6 *a, b* and *c*. In the screening procedure T1, T2 and T3 stress were the psychological variables that were significant in predicting T3 QoL. A hierarchical multiple regression analysis was conducted to investigate if psychological stress at T1 (H_6 *a*), T2 (H_6 *b*) and T3 (H_6 *c*) could predict psychological recovery at T3 when demographic and stroke markers were controlled for. Table 7.9 illustrates the outline for the model.

Table 7.9

Outline of H_6 a, b and c model.

Variable
Step 1
Demographic (age) & Stroke markers (stroke severity)
Step 2
T1 Psychological Variables (T1 stress)
Step 3
T2 Psychological Variables (T2 stress)
Step 4
T3 Psychological variables (T3 stress)

In Step 1 the control variables (age and stroke severity) were entered. In Step 2, T1 stress was entered to investigate H_6 *a*. In Step 2, T2 stress was entered, controlling for Step 1, to investigate the relationship between stress at T2 and QoL at T3 (H_6 *b*). In the final Step, T3 stress was entered to investigate H_6 *c*, once the previous variables were controlled for. Means and standard deviations for the model variables are shown in Table 7.10.

Table 7.10

Means and standard deviations for the model variables.

Variable	*M*	*SD*
T3 QoL	48.91	19.17
Age	67.56	14.46
Stroke Severity (Mild vs. Mod)	.43	.50
Stroke Severity (Mild vs. Severe)	.21	.41
T1 Stress	1.38	.70
T2 Stress	1.59	.74
T3 Stress	1.54	.71

Multicollinearity was checked and there were no violations as the tolerance values were all above 0.10, the Variance Inflation Factor (VIF) values were all below 10, and there were no correlations above r = 0.7 (see Table 7.11 for the correlations table).

To assess if outliers were causing a distortion the critical chi-square value using an alpha value of .001 (Pallent, 2013) was investigated. This value was 24.32. The Mahalanobis distance was slightly above this at 30.23. This was due to only one case. In data sets there will be a few cases that will exceed this criteria however, if it is minimal it is acceptable to keep this data (Pallent, 2013). Cooks distance was below 1 (0.14), therefore there were no strong violations of outliers. Normality, homoscedasticity and linearity were also satisfied.

Table 7.11

Correlations among T3 QoL, Age, Stroke Severity, T1, T2 & T3 Stress.

Variable	2.	3.	4.	5.	6.	7.
1. T3 QoL	-.32***	-.04	-.35***	-.16	-.45***	-.58***
2. Age	-	.03	.20*	-.38***	.06	.04
3. Stroke (Mild vs. Mod)		-	-.46***	-.07	.02	-.02
4. Stroke (Mild vs. Sev)			-	.09	.14	.22*
5. T1 Stress				-	.27**	.19*
6. T2 Stress					-	.45***
7. T3 Stress						-

Note. *** $p < .001$ ** $p < .01$ and * $p < .05$.

Table 7.11 illustrates the absence of multicollinearity. The psychological variables were the most highly correlated with T3 QoL, with T2 stress (r = -.45***) and T3 stress (r = -.58***) being more highly correlated than the control variables.

A hierarchical multiple regression analysis was conducted to investigate the predictive value of T1, T2 and T3 Stress on T3 QoL when age and stroke markers were controlled for. Age and stroke severity were entered in Step 1 explaining 22% of the variance in T3 QoL. T1 stress was entered in Step 2 and explained an additional 6% of the variance. T2 stress was entered in Step 3 which accounted for an additional 11% of the variance and T3 stress was entered in Step 4 with the total variance accounted for being 48.7% (adjusted R^2) (R^2 =51.8%), F (6, 92) = 16.50, p < 0.001, N= 99 (30.77% missing data), with each step being significant at p < 0.001.

Table 7.12

Regression coefficients for H_6 a, *b* and *c*.

Variable	*B*	*SE B*	β	Δ*R*
Step 1				0.22
Age	-.31	.12	-.23*	
Stroke Severity (Mild vs. Mod)	-8.23	3.96	-.21*	
Stroke Severity (Mild vs. Severe)	-18.63	4.89	-.40***	
Step 2				.06
Age	-.45	.13	-.34**	
Stroke Severity (Mild vs. Mod)	-7.92	3.83	-.21*	
Stroke Severity (Mild vs. Severity)	-16.28	4.80	-.35**	
T1 Stress	-7.27	2.63	-.27**	
Step 3				.11
Age	-.38	.12	-.29**	
Stroke Severity (Mild vs. Mod)	-6.72	3.57	-.18	
Stroke Severity (Mild vs. Severity)	-14.35	4.49	-.31**	
T1 Stress	-4.19	2.56	-.15	
T2 Stress	-8.92	2.25	-.35***	
Step 4				.14
Age	-.38	.11	-.29**	
Stroke Severity (Mild vs. Mod)	-5.59	3.18	-.15	
Stroke Severity (Mild vs. Severity)	-10.56	4.06	-.23**	
T1 Stress	-3.53	2.28	-.13	
T2 Stress	-4.46	2.18	-.17*	
T3 Stress	-11.34	2.24	-.42***	

Note. *** $p < .001$ ** $p < .01$ and * $p < .05$. Stroke severity (Mild vs. Mod) was dummy coded 0 and 1. Stroke severity (Mild vs. Sev) was dummy coded 0 and 1.

In the final model age and severe stroke severity were the significant control predictors of stroke T3 QoL (see Table 7.12). Severe stroke severity (β= -.23, t (97) = -2.60, $p < .001$) at stroke onset predicted a strong relationship with QoL at T3, with

higher severity at Time 1 predicting poorer QoL at T3. For example a one unit increase (from 0 to 1) in severe stroke severity scores predicted a -.23 decrease in QoL at the fixed T3 point. This illustrates severe strokes were associated with poorer recovery scores at T3.

Age in the final model demonstrated a negative predictive relationship with T3 QoL (β= -.29, t (99) = 3.49, $p < .01$), with a one unit (*SD*) increase (14.46) predicting a -.29 decrease in T3 QoL scores. This illustrates older participants reported lower QoL scores at T3.

In Step 2 ($H_6\,a$), T1 stress significantly predicted T3 QoL. T1 stress (β= -.13, t (99) = -1.55, $p < .01$) demonstrated a negative relationship with T3 QoL, where a one unit (*SD*) increase (.70) in stress scores predicted a -.13 decrease in T3 QoL scores.

In Step 3 ($H_6\,b$) T2 stress significantly predicted T3 QoL. T3 stress (β= -.17, t (99) = -2.04, $p < .05$) demonstrated a negative relationship with T3 QoL, where a one unit (*SD*) increase (.74) in stress scores predicted a -.17 decrease in T3 QoL scores. This illustrates the more stress this sample reported at T2, the worse their psychological recovery was at T3.

In the final model ($H_6\,c$) T3 stress significantly predicted T3 QoL. T3 stress (β= -.42, t (99) = -5.07, $p < .001$) demonstrated a negative relationship with T3 QoL, where a one unit (*SD*) increase (.71) in T3 stress scores predicted a -.42 decrease in T3 QoL scores. This illustrates the more stress this sample reported at T3, the worse their recovery was at T3.

In the final model, age made the biggest unique contribution (β= -.29**) from the control variables, with T3 stress making the biggest psychological contribution (β= -.42***). The results from this analysis indicate that stress at T1, T2 and T3 are predictive of poorer QoL at the fixed T3 point.

7.4.2 *H_6 d:* High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at a fixed time point of 0-6 weeks post stroke (T1) will predict poorer QoL at a fixed time point of 6 months post stroke (T3).

and

***H_6 f:* High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at a fixed time point of 6 months post stroke (T3) will predict poorer QoL at a fixed time point of 6 months post stroke (T3).**

This section covers both *H_6 d* and *f.* In the screening procedure T1 and T3 Bells were the cognitive variables that were significant in predicting T3 QoL. A hierarchical multiple regression analysis was conducted to investigate if cognitive variables at T1 (*H_6 d*) and T3 (*H_6 f)* could predict psychological recovery at T3 when demographic and stroke markers were controlled for. Table 7.13 illustrates the outline for the model.

Table 7.13

Outline of H_6 d and f model.

Variable
Step 1
Demographic (age) & Stroke markers (stroke severity)
Step 2
T1 Cognitive Variables (T1 Bells)
Step 3
T3 Cognitive Variables (T3 Bells)

In Step 1 control variables (age and stroke severity) were entered. In Step 2, T1 Bells was entered to be assessed in their prediction of T3 QoL and also to be

controlled for once T3 Bells was entered in Step 3. The variables chosen to be entered in this sequence are based on $H_{6.}$ Means and standard deviations for the model variables are shown in Table 7.14.

Table 7.14

Means and standard deviations for the model variables.

Variable	*M*	*SD*
T3 QoL	48.32	18.69
Age	66.76	14.95
Stroke Severity (Mild vs. Mod)	.43	.50
Stroke Severity (Mild vs. Severe)	.23	.43
T1 Bells	27.13	8.65
T3 Bells	29.82	6.68

Multicollinearity was checked and there were no violations as the tolerance values were all above 0.10, the Variance Inflation Factor (VIF) values were all below 10, and there were no correlations above r = 0.7 (see Table 7.14 for the correlations table). Normality, homoscedasticity and linearity were also satisfied.

To assess if outliers were causing a distortion, the critical chi-square value using an alpha value of .001 (Pallent, 2013) was investigated. This value was 20.52. The Mahalanobis distance was below this at 19.49. Cooks distance was below 1 (0.08), therefore there were no violations of outliers.

Table 7.15

Correlations among T3 QoL, Age, Stroke Severity, T1, T2 & T3 Stress.

Variable	2.	3.	4.	5.	6.
1. T3 QoL	-.34***	-.10	-.32**	.34***	.45***
2. Age	-	.07	.17	-.14	-.31**
3. Stroke (Mild vs. Mod)		-	-.48***	.05	.03
4. Stroke (Mild vs. Sev)			-	-.21*	-.27**
5. T1 Bells				-	.65***
6. T3 Bells					-

Note. *** $p < .001$ ** $p < .01$ and * $p < .05$.

Table 7.15 illustrates the absence of multicollinearity. Age was the strongest correlated control variable with T3 QoL (r = -.34***), whilst T3 Bells was the most significant cognitive variable (r = .45***).

A hierarchical multiple regression analysis was conducted to investigate the predictive value of T1 and T3 Bells on T3 QoL when age and stroke markers were controlled for. Age and stroke severity were entered in Step 1 explaining 25% of the variance in T3 QoL. T1 Bells was entered in Step 2 and explained an additional 6% of the variance. T3 Bells was entered in Step 3 which accounted for an additional 3% of the variance with the total variance being 30.2% (adjusted R^2) (R^2 =33.9%), F (5, 88) = 9.05, $p < 0.001$, N= 94 (34.27% missing data), with each step being significant at $p < 0.001$.

Table 7.16

Regression coefficients for H_6 d and f.

Variable	*B*	*SE B*	β	Δ*R*
Step 1				0.25
Age	-.32	.12	-.25**	
Stroke Severity (Mild vs. Mod)	-10.77	3.95	-.29**	
Stroke Severity (Mild vs. Sev)	-18.37	4.67	-.42***	
Step 2				.06
Age	-.29	.11	-.23**	
Stroke Severity (Mild vs. Mod)	-10.38	3.82	-.28**	
Stroke Severity (Mild vs. Sev)	-16.09	4.60	-.37**	
T1 Bells	.52	.20	.24**	
Step 3				.03
Age	-.22	.12	-.18	
Stroke Severity (Mild vs. Mod)	-9.94	3.76	-.27**	
Stroke Severity (Mild vs. Sev)	-14.68	4.57	-.33**	
T1 Bells	.20	.25	.09	
T3 Bells	.70	.34	.25*	

Note. *** $p < .001$ ** $p < .01$ and * $p < .05$. Stroke severity (Mild vs. Mod) was dummy coded 0 and 1. Stroke severity (Mild vs. Sev) was dummy coded 0 and 1.

In the final model stroke severity was the strongest predictor of stroke recovery in all Steps. Age also featured as a prominent predictor. In the final model, stroke severity explained the most significant contribution to T3 QoL (see Table 7.15) with moderate (β= -.27, t (92) = -.64, $p < .01$) and severe strokes (β= -.33, t (92) = -3.21, $p < .01$) at stroke onset predicting a strong relationship with psychological recovery at T3, with higher severity at Time 1 predicting poorer QoL at T3. For example a one unit increase (from 0 to 1) in severe stroke severity scores predicted a -.33 decrease in QoL at T3 and a one unit increase (from 0 to 1) in moderate stroke severity scores predicted a -.27 decrease in T3 QoL scores. This illustrates moderate and severe strokes were associated with poorer T3 QoL scores.

Age in the final model demonstrated a negative predictive relationship with T3 QoL (β= -.18, *t* (92) = -1.93, *p* < .05), with a one unit (*SD*) increase (14.95) predicting a -.18 decrease in T3 QoL scores. This illustrates older participants reported poorer T3 QoL scores.

In Step 2 (H_6 *d*), T1 Bells significantly predicted T3 QoL. T1 Bells (β= .24, *t* (92) = .81, *p* < .05) demonstrated a positive relationship with T3 QoL, where a one unit (*SD*) increase (8.65) in T1 Bells scores predicted a .24 increase in T3 QoL scores.

In the final model (H_6 *f*) T3 Bells significantly predicted T3 QoL. T3 Bells (β= .25, *t* (92) = 2.10, *p* < .05) demonstrated a positive relationship with T3 QoL, where a one unit (*SD*) increase (6.68) in T3 Bells scores predicted a .25 increase in T3 QoL scores. This illustrates that as visual neglect improves at T1 and T3, QoL improves at the fixed T3 point.

The Bonferroni correction was calculated as *p* = .03. T3 Bells exceeded this threshold but was significant at *p* = .04. At the expense of possibly committing a Type II error, T3 Bells was retained in this analysis and reported.

In the final model, severe stroke severity made the biggest unique contribution (β= .33**) with T3 Bells making the biggest cognitive contribution (β= .25*). The results from this analysis indicate that visual neglect at T1 and T3 are related to improvements in QoL at T3.

The next section summarises the results from this chapter.

7.5 Results Summary

In this section the results are summarised.

1. *H_4 a*: High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 0-6 weeks post stroke (Time 1) will predict poorer QoL at a fixed time point of 3 months post stroke (Time 2).

In this analysis T1 social support confirmed *H_4 a.* The control variables (age and stroke severity) accounted for 17% of the variance, with T1 social support accounting for an additional 8% of the variance.

i. Moderate strokes at baseline predicted poorer QoL at T2.
ii. Severe strokes at baseline predicted poorer QoL at T2.
iii. High social support scores at T1 predicted improvements in QoL at T2.

2. *H_5 a*: High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 3 months post stroke (Time 2) will predict poorer QoL at a fixed time point of 3 months post stroke (Time 2).

In this analysis T2 depression and T2 stress confirmed *H_5 a.* The control variables (age and stroke severity) accounted for 18% of the variance, with T2 depression and T2 stress accounting for an additional 33% of the variance (after previous T1 social support was controlled for).

i. Moderate strokes at baseline predicted poorer QoL at T2.
ii. Severe strokes at baseline predicted poorer QoL at T2.
iii. Older age was associated with poorer QoL at T2.
iv. Higher depression scores at T2 predicted poorer QoL at T2.
v. Higher stress scores at T2 predicted poorer QoL at T2.

3. H_6 *a:* High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 0-6 weeks post stroke (Time 1) will predict poorer QoL at a fixed time point of 6 months post stroke (Time 3).

   In this analysis T1 stress confirmed H_6 *a*. The control variables (age and stroke severity) accounted for 22% of the variance, with T1 stress accounting for an additional 6% of the variance.

i. Severe strokes at baseline predicted poorer QoL at T3.
ii. Older age was associated with poorer QoL at T3.
iii. Higher stress scores at T1 predicted poorer QoL at T3.

4. H_6 *b:* High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 3 months post stroke (Time 2) will predict poorer QoL at a fixed time point of 6 months post stroke (Time 3).

In this analysis T2 stress confirmed H_6 *b*. The control variables (age and stroke severity) accounted for 22% of the variance, with T2 stress accounting for an additional 11% of the variance (once T1 stress was controlled for).

i. Severe strokes at baseline predicted poorer QoL at T3.
ii. Older age was associated with poorer QoL at T3.
iii. Higher stress scores at T2 predicted poorer QoL at T3.

5. H_6 *c:* High depression, stress, Type D personality, repressive coping and low social support at a fixed time point of 6 months post stroke (Time 3) will predict poorer QoL at a fixed time point of 6 months post stroke (Time 3).

In this analysis T3 stress confirmed H_6 *c*. The control variables (age and stroke severity) accounted for 22% of the variance, with T3 stress accounting for an additional 14% of the variance (once T1 and T2 stress were controlled for).

i. Severe strokes at baseline predicted poorer QoL at T3.
ii. Older age was associated with poorer QoL at T3.
iii. Higher stress scores at T3 predicted poorer QoL at T3.

6. *H_6 d:* High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at a fixed time point of 0-6 weeks post stroke (T1) will predict poorer QoL at a fixed time point of 6 months post stroke (T3).

In this analysis the T1 Bells cancellation task confirmed *H_6 d*. The control variables (age and stroke severity) accounted for 25% of the variance, with T1 Bells accounting for an additional 6% of the variance.

i. Moderate strokes at baseline predicted poorer QoL at T3.
ii. Severe strokes at baseline predicted poorer QoL at T3.
iii. Older age was associated with poorer QoL at T3.
iv. Higher Bells scores at T1 predicted improvements in QoL at T3.

7. *H_6 f:* High cognitive impairment (visual memory, verbal memory, visual neglect and executive function) at a fixed time point of 6 months post stroke (T3) will predict poorer QoL at a fixed time point of 6 months post stroke (T3).

i. Moderate strokes at baseline predicted poorer QoL at T3.
ii. Severe strokes at baseline predicted poorer QoL at T3.
iii. Older age was associated with poorer QoL at T3.
iv. Higher Bells scores at T3 predicted improvements in QoL at T3. The Bonferroni correction calculated for H_6 was $p = .03$. This variable exceeded this threshold at $p = .04$.

Chapter 8

Discussion

8.1 Rationale and Summary

The aim of this thesis was to investigate the role of psychological and cognitive variables at fixed time points (Time 1: 0-6 weeks post stroke, Time 2: 3 months post stroke and Time 3: 6 months post stroke) in predicting physical recovery (at the 3 fixed time points) and psychological recovery (at the fixed time points of Time 2 and Time 3) from stroke. The rationale behind this chapter is to further explore the results of the analyses conducted.

In order to develop a research question a systematic review (Study One) was conducted which informed the research study (Study Two). Study Two was a quantitative, longitudinal study which investigated physical and psychological recovery from stroke, using mood (depression and stress), a social aspect (social support), coping styles (repressive coping and sense of coherence) and personality (Type D personality) factors, alongside cognitive neuropsychological factors (visuo-spatial impairment, visual and verbal short term memory and executive function), in addition to demographic factors and stroke markers. To the author's knowledge this is the first longitudinal study on acute stroke recovery combining these factors in relation to psychological and physical outcomes of stroke.

This chapter firstly outlines the findings of Study One. This is followed by a justification of the study design and a brief discussion on multiple testing. A discussion on the results of Study Two's Physical recovery Model and Psychological Recovery Model are presented. Methodological limitations, measurement issues and strengths are then acknowledged. The Chapter closes with a discussion of healthcare policy, implication for theory, clinical significance of the findings, avenues for future research and a final conclusion.

8.2 Study One: Do Psychological Factors Affect Stroke Risk And Recovery? A Systematic Review.

A comprehensive systematic review was conducted yielding 101,807 search results. Twenty five international studies were identified which examined design, method of data collection, predictors and conclusions.

The review papers were all of a longitudinal design. However in 9 of the studies the length of follow up was not clear, therefore it was difficult to conclude the affect of time points on recovery across review papers.

Six studies failed to report on attrition. Attrition can cause a cohort bias leaving patients who are more able to comply with the investigations inclusion criteria which in turn produces significant results (Bryman, 2008). Acknowledging this consequence of stroke research should give researchers greater impetus to describe attrition rates and what effect this has had on their findings and conclusions.

The main predictors of stroke recovery identified by the systematic review included depression (Parikh et al., 1990; Morris et al., 1992; Schubert et al., 1992a; Schubert et al., 1992c; Morris et al., 1993; Loong et al., 1995; Simonsick et al., 1995; Herrmann et al., 1998; van de Weg., 1999; Chemerinski et al., 2001; Lai et al., 2002; Saxena et al., 2007; Bos et al., 2008 & Bilge et al., 2008), positive emotion (which were the positively worded questions on the CESD depression scale) (Ostir et al., 2008), pre-stroke trait introversion (Morris et al., 1993), severely life threatening events (House et al., 1990), active coping, extrovert personality (Elmstahl et al., 1996), state self esteem (Chang et al., 1998), perceived control (Johnston et al., 1999; 2004) and the psychological and environmental domains of the WhoQoLBREF measure (Hamzet et al., 2009).

However, 5 studies have disagreed and have reported there is no association between depression and functional status (Morris et al., 1990; Schubert et al., 1992b; Johnston et al., 1999; 2004; Cassidy et al., 2004 & Nannetti et al., 2005) and there is no association between anxiety and functional status (Johnston et al., 1999; 2004).

Some concerns regarding the review papers have been noted which will affect the strength of their conclusions. Ostir et al., (2008) assessed the effect of positive emotion on functional recovery. They use 4 items of positive emotion from the CESD depression scale. There is no Cronbach alpha information on these 4 items

and no Cronbach alpha value was offered by the Authors. Simply using 4 items from this scale would not be enough to conclude the presence of positive emotion, therefore the conclusions of this study are spurious.

The implications for this review show that there is scope for further research in the area of psychological factors and stroke recovery. It is difficult to comment on the applicability of all the findings because of the differences in the psychological and clinical measures utilised, the differences in the measures of recovery, length of the study duration, lack of demographic data, differences and lack of stroke definition and differences in statistical analyses. A methodological quality assessment was devised in order to categorise studies into Good, Intermediate and Poor groups. Ten studies were categorised as being of Good quality, whilst 15 studies were categorised as being of Intermediate quality. No studies were categorised as Poor. The review papers do provide good research ideas and do give insight into the area of psychology and stroke recovery.

The strengths of this systematic review were that synthesised material specific to this topic were amalgamated together and the execution of a systematic review is critical and should limit bias (Wright, Brand, Dunn, & Spindler, 2007).

The main limitation of the systematic review was that only 1 person was searching the literature. Normally in systematic review creation there would be a team of people searching and cross checking colleagues. Also, the articles that were accumulated in the systematic review were the only ones available. This can be interpreted as publication and citation bias as published work can be indicative of selective reporting and not publishing unfavourable results. Unpublished and grey literature were not incorporated into this review as they were unobtainable. Additionally, unpublished literature has not gone through peer review and therfore the quality of the research cannot be guaranteed (Pannucci & Wilkins, 2010).

Systematic reviews are important in the generation of hypotheses (Khunti, 1999; Webb & Roe, 2007; Deb, Wijeysundera, Ko, Tsubota, Hill, & Fremes, 2013; Cheng et al., 2013; Campbell et al., 2014; Patel, Laffan, Waheed, & Brett, 2014). The research hypotheses were formulated based upon the findings of the systematic review and additionally, the literature review and the gaps it highlighted. The variables chosen to be investigated were depression, stress, social support, Type D

personality, repressive coping and sense of coherence with additional cognitive variables (visuo-spatial impairment, visual and verbal short term memory and executive function) and their effect on psychological and physical recovery from stroke.

The following section discusses hypothesis generation and a justification of the analysis used.

8.3 Hypothesis Generation and Justification of Analysis Used

The systematic review in Chapter 2 (p. 61) and the literature review in Chapter 3 (p. 150) were instrumental in the hypothesis generating phase. Systematic reviews can be used as hypothesis generating tools which form a logical progression between review results and the final hypotheses (Khunti, 1999; Webb & Roe, 2007; Deb et al., 2013; Cheng et al., 2013; Campbell et al., 2014; Patel, Laffan, Waheed, & Brett, 2014). The hypotheses generated concentrated on the predictive relationship between variables at fixed time points, (Parikh et al., 1990; Elmstahl et al., 1996; Chang et al., 1998; Cassidy et al., 2004; Johnston et al., 2004; Ostir et al., 2008) e.g., investigating the relationship of Time 1 depression with, for example, Time 2 stroke recovery.

Importance of systematic reviews have risen in Health Psychology with their conclusions informing Health Psychology practise and research (Marks, Murray, Evans & Estacio, 2011) although many Health Psychologists do not implement systematic reviews and do not use their research findings (Suls, Davidson & Kaplan, 2010).

Longitudinal research is defined as the same variable or variables measured at, at least two different time points (Menard, 1991). There are different methods in analysing longitudinal study designs with most approaches focusing on the current research methodology (Parikh et al., 1990; Elmstahl et al., 1996; Chang et al., 1998; Cassidy et al., 2004; Johnston et al., 2004; Ostir et al., 2008; Rydstedt, Cropley, & Devereux, 2011), which is in line with the hypotheses of this study.

Another method of analysing longitudinal data is to use difference or residual scores. Difference scores are calculated by subtracting Time 1 scores from Time 2 scores thereby producing a new variable, which is the difference *between* times. This

ultimately means replacing the original variable with a difference score. Residualized change scores are calculated by using the standardized residuals in linear or hierarchical regressions by regressing Time 2 scores onto Time 1 scores (Prochaska, Velicer, Nigg & Prochaska, 2008; Parschau et al., 2011). The Time 2 scores are regressed onto the Time 1 scores and then separated from the Time 2 scores, however this approach can result in incorrect conclusions. The measurement error in the Time 1 scores should be minimal otherwise the regressor is weak. Additionally the Time 2 scores measurement error is not acknowledged as predicted Time 2 scores are subtracted from observed Time 2 scores (Cochran, 1968). However, two outcome variables would not be in an analysis together in the current study based on the systematic review and the hypotheses.

Judd, Kenny & McClelland (2001) have written an article on mediation and moderation of treatment effects. In this article they additionally discuss difference scores and residuals. Residualized change scores are another method of analysing data and so both difference and residualized change scores will be discussed. However the use of difference or residualized change scores have been criticised for decades in favour of using component scores (the full variable, as has been adopted in the current study). All consequent analysis can be conducted on the new residual score not on the original variable. That is to keep the residuals (unexplained variance) as a new predictor variable however, this method has been criticised as it is the errors of prediction that remain in this residual variable (Wurm & Fisicaro, 2014).

Change scores may be used for disciplines in industry where absolute measures are used, however using change scores for the social sciences where measurements are made on subjective scales have been criticised (Bock, 1976). The Judd, Kenny & McClelland (2001) article investigates ordinary least square regression models but polynomial regressions which investigate curvilinear relationships may be better suited to addressing this issue (Edwards, 2001).

Cronbach & Furby (1970) claim these change scores focus on participants who failed to change the most or did change the most, without acknowledging the variable as a whole. They also assert these scores *"lead to fallacious conclusions, primarily because such scores are systematically related to any random error of measurement. Although the unsuitability of such scores has long been discussed, they*

are still employed by some otherwise sophisticated investigators." (p.68). Willett (1997) states using difference scores in multiple regression analysis is flawed as the difference score contains both measurement error and true change which can result in distorted statistics.

The reliability of residualized change scores has also been questioned (Traub 1967; Judd & Kenny, 1981), although there have been disagreements over this (Willett, 1994; Stewart, Carson, & Cardy, 1996). However if difference scores do have acceptable reliability, then component measures would display stronger reliabilities. This illustrates that the original variables should be utilised in place of a difference score variable where possible (Edwards, 1994). In the current study component variables were used.

Difference scores are also likely to inflate the Type I error rate by producing false positive conclusions (Wanous et al., 1992). Edwards (2001) takes this view further by asserting that difference scores affect both the Type I and Type II error rate by reducing the Type I error rate at the detriment of the Type II error rate. Additionally variables can also be prone to enlarged statistical significance when residualized scores are used because of the manufactured reduced standard error (Wurm & Fisicaro, 2014).

There have also been criticisms of using hierarchical regression as assessing changes over time by entering Time 1 in Step 1 and Time 2 in Step 2 and Time 3 in Step 3. *"Despite its appeal, controlling for component measures does not yield conservative tests of difference scores. Instead, this approach alters the relationships difference scores are intended to capture, such that a coefficient on a difference score that seems to support a congruence hypothesis may represent a relationship that is quite different, depending on the coefficients on the component measures." (Edwards, 2001, p. 272)*

Investigating if changes in depression are related to changes in outcome, is very similar to investigating if component measures are related to outcome. This will not necessarily strengthen analyses but it will repeat it (Cohen-Goldberg, 2012). Additionally, using these methods may not contribute to applied research in real terms: *"The limitations of the approach are that it may be difficult to interpret for media and policymakers; may lack meaning in terms of health benefits; treats each behavior equally; and is not widely used and documented. Additionally, residualized*

change scores are not suited to address the issue of whether there is a significant change across time overall (i.e., ignoring groups) as the mean of a residualized change score is zero. (Prochaska, Velicer, Nigg & Prochaska, 2007, p.3)"

Difference scores used as independent variables force the reduction of the explained variance in statistics which can cause a weakened analysis and the assumptions of difference scores analysis have been rejected in empirical work (Edwards, 1991, 1993, 1994, 1996; Edwards & Harrison, 1993).

More recently Wurm & Fisicaro (2014) identified numerous studies that use multiple regression in which researchers adopted this approach with erroneous beliefs, such as believing residualizing variables will dissociate one variable from another, it will manage multicollinearity and believing this can investigate if one predictor can explain more variance than another predictor (p.38). One study added residuals to an otherwise complete data set (Baayen, Feldman, & Schreuder, 2006), which is unnecessary (Wurm & Fisicaro, 2014).

Additionally, in very recent research on this by Cohen-Goldberg (2012) it has been strongly argued that unresidualized scores have the same results as residualized scores and also in hierarchical regression "*The fact that residualizing does not affect any aspect of the outcome for the residualized variable may come as a surprise to some researchers*" (Wurm & Fisicaro, 2014, p.40). This concludes that using the original variables and using change score variables should produce the same results. When the original component variable has been used, to then use a change score to analyse relationships between predictors and outcome, would be to essentially *repeat* the analysis which is a redundant cause. Moreover, altering the variables will reduce the comparability of this research with others in the same area.

Residualized scores can also be difficult to interpret, as the sign of the beta weights can change from plus to minus confusing the interpretation (Ambridge et al. 2010). Gottman & Krokoff (1990) question the viability of using change scores and question why researchers continue to use this method. These authors advocate the stance that over complicating statistics is not always the answer for communicating statistics. Anderson (1963) encourages us to see the data as it really is rather than searching for something else within the data. Willett (1997) states *"I strongly advise the researcher to avoid residual change scores as measures of within-person change"* (p.218).

Cronbach & Furby (1970) conclude with: *"It appears that investigators who ask questions regarding gain scores would ordinarily be better advised to frame their questions in other ways."* (p.80).

The risk of misinterpretation is a warning that this method should not be used in favour of component variables. To be close to "true" analysis the original variables should be used without the use of residualization or unnecessary altering of the variable. This allows for a clearer interpretation of the results (Wurm & Fisicaro, 2014).

Willett (1997) suggests collecting multiple time point data and using individual growth modelling to analyse this type of data. Adding more waves of data would improve reliability of these difference scores. Or executing analysis such as structural equation modelling (SEM) may be better suited to deal with the statistical issues outlined here (Mroczek & Little, 2006; Mun, von Eye, & White, 2009). SEM can be used with a model for assessing residualized change instead of attempting this via multiple regression analyses. This has been outlined in an article by Raykov (1993).

In the current study the method of longitudinal analysis used was to analyse component measures. The use of change scores was not included in the hypotheses of this study as this method was not a dominant method used from the studies drawn upon in the systematic and literature reviews. Also, this method does not answer the lengthy hypotheses outlined. These hypotheses *did not* include assessing change of variables between time points, the difference between times and it does not also assess changes in the dependent variables. Auxiliary hypotheses do not have a cogent grounding in the current study. Accruing different analyses and more hypotheses (when 69 hypotheses are in the study) will interfere with the integrity of the research analyses. Additionally, this would cause analysis to be repeated, which would be redundant and would contribute to the difficulty in managing and maintaining multiple testing in an already complex design.

Additionally, employing methodologies which have been historically criticised cannot be justified in this context. Therefore, the most appropriate analysis was employed and focus must be firmly kept upon the hypotheses that have been generated from the systematic review and the literature review.

The next section discusses multiple testing.

8.4 Multiple Testing

The multiple analysis of tests in statistics may cause a Type I error. A Type I error is a false positive result. The most frequently used method of controlling for Type I errors is the Bonferroni Correction. This correction reduces the level of α, therefore producing a stricter level of α. However, this may cause a Type II error. A Type II error is a false negative. This is to conclude a test is negative when it is not (Shaffer, 1995).

The current study used a Bonferroni correction because of the multiple testing performed. To conduct more tests would result in a stricter level of α, in which it would be very difficult to obtain any meaningful results. As there is debate in the literature over multiple testing (Shaffer, 1995; Perneger, 1998; Gelman, Hill, Yajema, 2012), results that exceeded the Bonferroni threshold but were significant were reported. This was to acknowledge the possibility of conducting a Type II error. The results reported as exceeding the Bonferroni threshold should be treated prudently.

The next section will outline the results of Study Two.

8.5 Study Two: Quantitative Study.

Study Two investigated psychological and cognitive variables in predicting physical and psychological recovery from stroke. The main analysis involved a series of hierarchical multiple regression analyses and mediation analyses (there were no significant moderating relationships). A short preliminary analysis was conducted using repeated measures ANOVAs to assess changes at different time points of the main study variables. This will be discussed in the next section.

8.5.1 Preliminary Analysis: Variables at fixed time points.

A repeated measures ANOVA was used to explore significant changes in variables at the 3 fixed time points. Psychological recovery did not change significantly from Time 2 and 3, whilst physical recovery did demonstrate a significant change. Time 1 physical recovery was significantly different to Times 2 and 3. This illustrates that physical recovery improved significantly at Time 2 and

Time 3 compared to baseline. Depression did have a significant change over time, demonstrating this is a modifiable factor. Depression significantly decreased at Time 2 and Time 3, which is an expected result. Although in other research depression has decreased then increased (Astrom, Adolfsson & Asplund, 1993). Type D personality has been argued to be a dispositional trait (Pedersen and Denollet, 2006). The results of this study do support this as there were no changes over time for this variable, however earlier in 2000, Denollet, Vaes, & Brutaert, asserted that Type D is not a static personality type as emotional processing can affect Type D personality. Therefore, more research should be conducted in this area.

Stress also did not change over time. Although, this could be a modifiable factor stress reduction techniques are not normally practised in stroke rehabilitation, instead focusing on the stress relief of caregivers (Servaes, Draper, Conroy, & Bowring, 1999; Hartke & King, 2003; Legg et al., 2011; King et al., 2012).

Depression and stress are controlled by different systems in the body, and therefore a reduction in one does not necessarily mean a reduction in the other. Depression is controlled by the hypothalamic pituitary adrenocortical system (HPA) (with the release of cortisol), whilst stress reactions are primarily controlled by the sympathetic adrenomedullary system (SAM) (with the release of epinephrine and norepinephrine) (Lundberg, 2005). Stress reactions may not change because the SAM system is still registering threats, whilst the HPA system may have less or more activation in the triggering of negative emotional stimuli. That is to say, negative emotions may change at different time points, as the participant adjust to life after stroke however, the threat of stressors are still registered by the SAM system. In this study depression increased at Time 3, whilst stress plateaued. Positive social support may also have a protective effect against depression, as the release of oxytocin, inhibits the release of cortisol (Uvnas-Moberg, 1998; Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003; Southwick, Vythilingam, & Charney, 2005). In this research however, social support did not change significantly over time. This could be due to social relationships not changing or participants may not admit to them changing.

The Bells cancellation task (visual neglect) improved at Time 2 and Time 3 indicating visual neglect improving which has also been reported by Malhotra, Mannan, Driver & Husain, 2004). The forward digit span (verbal short term

memory) demonstrated a significant change between Time 2 and Time 3, with a decline at Time 3 which indicates a reduction in verbal short term memory. However, the Stroke Association (2015c) report short term memory can improve with time.

Although the Stroop test was removed from the main study due to missing data, the repeated one way ANOVA demonstrated there was a significant reduction between Time 1 and Time 3 which illustrates an improvement in Stroop reaction times. These finding have been echoed by Nys et al., (2005) and Ballard, Rowan, Stephens, Kalaria & Kenny (2003).

The line bi-section (visual neglect) and RBMT (visual short term memory) do not change significantly at the 3 time points, although visual short term memory can decay with time (Donkin, Nosofsky, Gold & Shiffrin, 2014). Stroop reaction times improved significantly over time, whilst Stroop errors did not change, which demonstrates an improvement in executive function.

The next section will discuss results from the Physical Recovery Model.

8.6 Main Analysis

8.6.1 The Physical Recovery Model

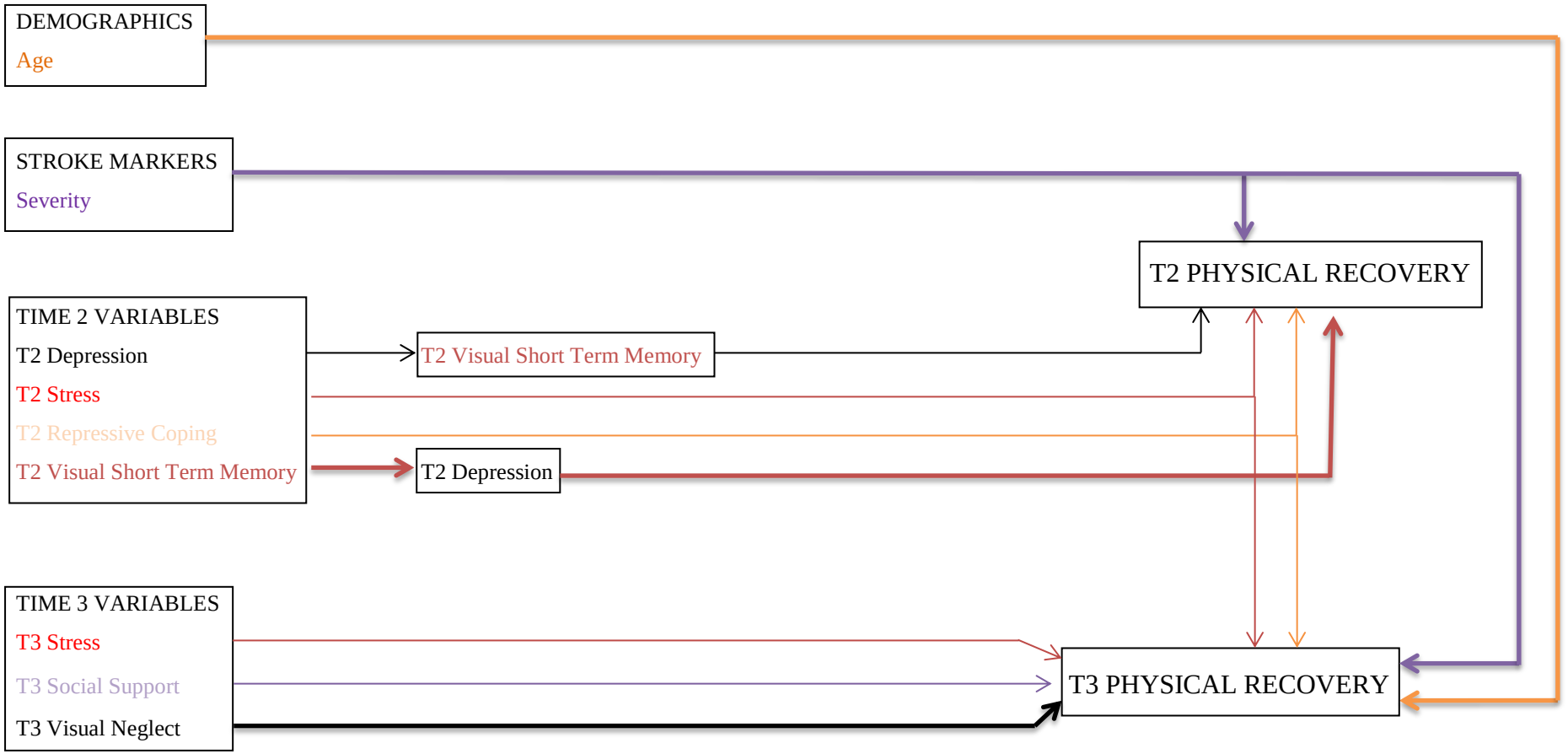
The main analysis in this research was conducted with a series of hierarchical multiple regressions with mediation analyses, the results of which are outlined below. Figure 8.2 illustrates the amended theoretical model based on the significant variables.

8.6.1.2 Main Variables non-significant at Time 1

Variables at Time 1 were not significant in predicting physical recovery. There are some explanations as to why this is the case. These include the impairment of higher cognitive functions after lesion occurrence, social desirability and rapport building.

Luria (1980) contributed heavily to the area of the lesioned brain and loss of higher cortical functions. Luria asserted the lesioning of the brain may have further affects in brain processing, over and above that of the function of the specific lesion location. Therefore, lesions may occur anywhere in the brain with the impairment of

Figure 8.1 Theoretical Model Results for Physical Recovery



higher cortical functions decreasing. The brain is a complex system, whereby impairment can cause deficits in higher intellectual function regardless of the location of the lesion. Higher mental functions are affected by lesions in different parts of the brain, as numerous functions interact together to form a higher function. This understanding of the higher cognitive functions becoming impaired after lesioning is still supported in recent times (Levenson, 2011). Therefore, participants in the acute phase of stroke recovery may lose higher cortical functions such as, the awareness of oneself, judgements about one's situation, and the ability to knit together different informational data to produce a coherent understanding of the effects of the life event which has occurred. This could be a major explanation as to why variables at Time 1 were not significant but they became significant at later time points when higher cortical functioning improves.

Another explanation as to why responses at Time 1 were not significant could be due to social desirability. Social desirability describes the behaviour of a participant answering a question favourably in order to maintain a positive image of themselves in front of the researcher to reduce feelings of embarrassment (Lee & Renzetti 1993; Johnson & van de Vijver 2002). This is achieved by distorting answers to adhere to social norms (Rauhut & Krumpal, 2008) and is cognitively believed to be a voluntary action (Holtgraves, 2004).

The presence of other people may also cause participants to distort their answers (Aquilino, 1997). The setting for the Time 1 data collection was within the hospital ward at the bedside of the participant. Although care was taken to promote privacy, participants can be aware of other patients in the neighbouring beds being able to hear them, as well as nurses and doctors and family members that are waiting for them to finish. At the Time 2 and 3 visits which were in the participants' homes, greater privacy could be accomplished and interestingly more significant results were reported at these time points. Additionally, this could also be due to the building of rapport.

Rapport is how easy the exchange is between the researcher and the participant (Given, 2008). This can be achieved by making sure the participant is at ease, empathising with them, maintaining good eye contact and building trust (Springwood & King, 2001; Hull, 2007).

The dropout rate for the current study was 11.2% at Time 2 and 4.9% at Time 3, which are low levels of attrition. This could be interpreted as a consequence of building good rapport. At Times 2 and 3, more significant findings were reported. This could be due to a combination effect of regaining higher cortical function, decreased social desirability and increased rapport.

The following sections will investigate the results for the main variables at Time 2 and Time 3 for the Physical Recovery Model. These will be outlined by variable. Firstly, demographic and stroke markers at baseline and their association with physical recovery will be discussed.

8.6.1.3 Demographic and Stroke Markers and the Physical Recovery Model

As has been identified by the systematic review psychological studies routinely overlook stroke severity. The majority of the review papers did not include stroke severity (Parikh et al., 1990; Morris et al 1990; House et al., 1990; Morris et al., 1992; Schubert et al., 1992a; 1992b; 1992c; Morris et al 1993.,Loong et al., 1995; Simonsick et al., 1995; Emstahl et al 1996; Herrmann et al., 1998; Chang et al., 1998; Johnston et al 1999; van de Weg et al., 1999; Chemerinski et al., 2001; Cassidy et al., 2004; Johnston 2004; Nannetti et al., 2005; Bos et al., 2008; Ostir et al., 2008; Bilge et al., 2008 & Hamzet et al., 2009). As a consequence of this, the current study can contribute to psychological literature in the area of stroke by reporting on this factor.

Moderate and severe stroke severity at Time 1 was a consistent predictor of physical recovery from stroke, significantly predicting poorer recovery at Times 2 ($\beta = .49$, $p < .001$ and $\beta = .56$, $p < .001$ respectively) and Time 3 ($\beta = .41$, $p < .001$ and $\beta = .53$, $p < .001$) respectively). This is an expected relationship. As the milder the initial stroke, the easier the recovery.

Age was a significant demographic predictor, with older age at Time 1 predicting poorer recovery at Time 3 mRS ($\beta = .28$, $p < .001$). This could be due to participants' of an older age recovering slower physically which was expected. Older age in stroke patients has been reported as predicting poor functional outcome at 3, 6 and 12 month follow up (Jehkonen et al., 2000), however age has additionally been reported to be weakly correlated with mRS in a 15 year longitudinal study (Teasdale & Engberg 2005). There is an absence of research which claims younger age is

predictive of poor stroke recovery therefore, it can be concluded that age can play a factor in physical recovery from stroke.

Depression and the Physical Recovery Model are discussed in the following section.

8.6.1.4. Depression and the Physical Recovery Model

As has been stated in the literature review, depression may be triggered by stressful events (Kessler, 1997). The findings of this research study have shown no evidence of this at the three separate time points (*H_1 g, H_2 i, H_3 k*). Depression is characterised by lethargic behaviours (Pruessner, Hellhammer, Pruessner, & Lupien, 2003) and stress is characterised by the fight or flight response (Lundberg, 2005). Therefore these two hormonal consequences are different (hopelessness versus action). Perhaps stress may initially occur followed by depression, however this is not supported by the data of the current study.

Depression has been well documented with exhibiting a relationship with stroke (Colantonio, Kasi, & Ostfeld, 1992; Larson, Owens, Ford, & Eaton, 2001; Ohira et al., 2001; Lawrence, & Grasby, 2001; Nilsson & Kessing, 2004; Krishnan, Mast, Ficker, Lawhorne, & Lichtenberg, 2005; Salaycik et al., 2007). However, studies have also reported depression having a non-significant relationship with stroke recovery (Morris et al., 1990; Schubert et al., 1992b; Johnston et al., 1999; 2004; Cassidy et al., 2004 & Nannetti et al., 2005). In the current study depression fails to make a significant impact upon stroke recovery (*H_1 a, H_2 a, b, H_3 a, b, c*). Depression plays an important role in the theoretical model by either directly predicting an outcome or by being in mediating relationship with other variables (*H_1 c, d, e, f, g, m, o, p, q, r, H_2 e, f, g, h, I, o, q, r, s, t, H_3 g, h, I, j, ,k, q, s, t, u, v*). But as this variable is not significant the majority of these mediating relationships fail to become viable.

The non-significant scores for depression were not expected and does not fulfil the prediction that depression would be a strong, consistent factor in recovery from stroke. The low scores for depression could be due to impairment of higher cortical functions (Luria, 1980), social desirability or a denial of what has happened. Conversely, low depression scores could be due to the participants' knowing they are receiving help or an acceptance of the situation. Or they could be due to the effects of

existing antidepressant drugs. Research has shown that antidepressant medication after a stroke can improve mood (Andersen, Vestergaard, & Lauritzen, 1994a; van de Weg et al., 1999; Gainotti, Antonucci, Marra & Paolucci, 2001; Aben et al., 2001; Arseniou, Arvaniti, & Samakouri, 2011; Chollet et al., 2013).

Additionally, if depression ratings were low due to low reporting of depressive symptomatology, the participants may not be offered antidepressant medication as it may be deemed not necessary to administer. However, therapy is not usually offered to patients despite the wealth of Psychological research regarding the positive influence of therapies. In a systematic review investigating the frequency of depression after stroke, Hackett, Yapa, Parag, & Anderson, (2005) conclude there is a lack of effective treatment of depression using psychological therapies and/or antidepressants.

Furthermore depression causes lethargy (Lundberg, 2005). This lethargy can have a negative impact on rehabilitation after stroke by not making full use of speech therapy and physiotherapy (Sinyour et al., 1986; Laidler, 1994; Dafer, Rao, Shareef & Sharma, 2008).

Stress and the Physical Recovery Model are discussed in the following section.

8.6.1.5. Stress and the Physical Recovery Model

One of the major shortfalls in the area of stress and stroke recovery is a lack of consensus within the literature. Studies have reported that stress has a negative influence on stroke recovery (Harmsen, Rosengren, Tsipogianni, & Wilhelmsen, 1990; SoRelle, 2001) whereas other research has found no relationship to report (Eckar, 1954; Macko et al., 1996; Nielsen et al., 2005).

Lazarus & Folkman (1984) asserted that stress is managed by coping (problem focused coping and emotion focused coping). This research cannot comment on the role of problem focused coping as this component was not measured however social support was measured, which is a component of emotion focused coping. Social support did not act as a mediator between stress and physical recovery at all 3 time points (H_1 *h*, H_2 *j*, H_3 *i*). Stress and social support have a historical relationship (see Section 8.5.3.5, on social support). Therefore, the non-significant

findings were unexpected. This could be due to social desirability, where participants' exaggerate their social support scores which therefore causes a misalignment.

Lazarus and Folkman's (1984) theory of stress has been widely accepted in the literature (Cooper, Dewe & O'Driscoll, 2001; Yu, Chiu, Lin, Wang & Chen, 2007). Although the current study was not designed to specifically test this model, it can partially acknowledge this model by having tested social support as a mediator between stress and physical recovery at all 3 time points (H_1 *h*, H_2 *j*, H_3 *l*) (although social support is only a component of emotion focussed coping). This study does not show evidence that in a post stroke sample social support acts as a mediator.

In a systematic review investigating Lazarus and Folkman's (1984) model with stroke (although other brain disorders were included which adds to the complexity of this area) only 14 papers were identified. The authors of this review explain this could be due to a publication bias in which papers with significant findings are favoured to be published. This review also highlighted the central tendency to focus on the coping element of this model rather than on stress itself, (Donnellan, Hevy, Hickey & O'Neill, 2006) which is the interest of the current study.

Lazarus and Folkman, (1984) also believe stress is influenced by the environment, and personality factors. The current study did not measure the environment however, Type D personality was measured. This variable was not significant as a mediator between stress and stroke recovery at all 3 time points (H_1 *j*, H_2 *l*, H_3 *n*). Therefore, there is no evidence from this study that can support Lazarus and Folkman (1984). It is reasonable to hypothesise that personality would be a mediator between stress and physical recovery therefore it is advisable for future research to replicate this aspect of the study. A possible reason to explain why this is not significant may be due to the specific sample as the prediction is viable.

Another model of stress is the Stress exacerbation model. This model explains that the more stressors one has to manage, the higher the stress level of the individual (Rook, 1998). This is a logical assumption to make. However, a search of the literature shows that this model has not been adopted by many researchers. This is due to the understanding that perceived stress may be more relevant than quantity of stress. Much like the Life Events Checklist (Cohen, Tyrrell, & Smith, 1991) which

was identified in the systematic review, this type of stress measurement is dependent upon the participant experiencing certain stressors and a certain number of stressors. A participant could be experiencing several small stressors versus a participant whom is experiencing one big stressor. However, that single stressor could outweigh several small stressors. The current research study cannot contribute on the support of this model as the quantity of stressors was not measured.

The current study adopted to measure perceived psychological stress as perceived stress is considered more revealing (Andreou et al., 2011), just as perceived social support is more revealing than quantity of social support (Zimet, Dahlem, Zimet, & Farley, 1988).

There is some debate in the literature regarding the effect of psychological stress on stroke (Macko et al., 1996) and research in this area is lacking. In the current study high stress at Time 2 was associated with poorer recovery at Time 2 *(H_2 b)* (β= .35, $p < .001$) and Time 3 (*H_3 b)* (β= .22, $p < .01$), and Time 3 stress was associated with poorer recovery at Time 3 (*H_3 c)* (β= .24, $p < .01$). This finding is valuable to stroke research as this can contribute to the existing literature on the continuing debate of the relationship between psychological stress and stroke. These findings suggest that stress in the acute phase (Time 1) does not have an impact upon physical recovery, however at later stages (Times 2 and 3) there is a significant impact on recovery. This could be explained by Luria's (1980) contribution to Cognitive Neuropsychology's understanding of impairment of higher cortical functions after lesioning in the brain. This may also be due to social desirability effects and the early stages of rapport building.

Perhaps also perceived psychological stress increased at the later stages because of adaption and realisation of the consequences of stroke. In these later stages of data collection the participants' were either at home or in nursing homes. Leaving the supportive environment of the hospital and adapting to stroke independently at home may cause stress levels to rise. Additionally, if participants were unable to care for themselves and were admitted to nursing homes this may have caused an increase in stress.

Social support and the Physical Recovery Model are discussed in the following section.

8.6.1.6. Social support and the Physical Recovery Model

Longitudinal research has provided evidence that social isolation can increase the risk of morbidity and mortality from all causes (Seeman et al., 1993). Therefore the role of social support in health recovery is very important.

Classic theories of social support include the Main Effect Hypothesis and the Stress Buffering Hypothesis. The Main Effect Hypothesis suggests social support mediates the stress-illness link which can have a direct effect on protecting the immune system (Cooper, 1984). The results of the current study however, do not support this theory as social support was not a mediator between stress and physical recovery at all 3 time points (*H_1 h, H_2 j, H_3 l*). A search of the literature reveals very little in the publications of the Main Effect Hypothesis in stroke.

The second theory is the Stress Buffering Hypothesis. This theory suggests social support buffers the individual from stress (Sarason, Sarason, Shearin, & Pierce, 1983; Cohen & Syme, 1985; Knapp & Hewison, 1998). In the current study social support was not significant as a moderator of this relationship at all 3 time points (*H_1 l, H_2 n, H_3 p*). This could be due to social desirability.

Other research studies such as the Morbidity and Interventions in General Practise study failed to find evidence for the Stress Buffering Hypothesis (Tijhuis, Flap, Foets, & Groenewegan, 1995) and a search of the literature of the Stress Buffering Hypothesis and stroke yields surprisingly absent results. Beckley (2006) tested this model, however she deviated away from the traditional theory and instead investigated social support as a moderator between functional outcome and community participation, thus omitting stress altogether whereas Friedland and McColl (1987) found evidence of the stress buffering model in stroke patients.

In an innovative study by Mezuk, Diex Roux & Seeman (2010) biological markers for social support (C-reactive protein (CRP), interleukin-6 (IL-6), and fibrinogen) were measured in the Multi-Ethnic Study of Atherosclerosis, specifically to test the Main Effect Hypothesis and the Stress Buffering Hypothesis. For the Main Effect Hypothesis only CRP was modestly significant in men for supporting this theory. There was no biological evidence for the Stress Buffering Hypothesis. Therefore there is not strong evidence for the biological grounding of this theory.

Improved functioning in stroke patients has been associated with reported higher levels of social support in the acute stroke phase, whilst poor social support was predictive of impaired functional improvement (Glass & Maddox, 1992; Glass, Matcher, Belyea, & Feussner, 1993) (*H_1 a, H_2 a, b, H_3 a, b, c*). In the current study, there was no evidence for this relationship. However, the current study reports that higher levels of social support reported at Time 3, predicted poorer recovery at Time 3 ($\beta = .18$, $p < .01$). This could be due to the type of social support received which did not allow stroke patients to be independent, therefore possibly allowing physical recovery to decline.

Too much social support can lead to lower levels of recovery. This could be due to inadvertently causing lower levels of motivation in a participant (Watzlawick & Coyne, 1980). Therefore, higher social support does not necessarily result in improved recovery but could result in poorer recovery.

Type D personality and the Physical Recovery Model are discussed in the following section.

8.6.1.7 Type D Personality and the Physical Recovery Model

This is the first study to the author's knowledge which investigates Type D personality with stroke. Type D personality has been reported to have a strong relationship with cardiovascular disease (Denollet & De Potter, 1992; Denellot, Sys, & Brutsaert, 1995; Denellot et al., 1996; Denellot, 1998a; Pedersen & Denollet, 2004; Schiffer et al., 2005; Smith et al., 2007; Schiffer, Pedersen, Broers, Widdershoven, & Denollet, 2008) and disability (Denollet, 2000). As cardiovascular disease and stroke are both conditions which affect the vascular system, it was reasonable to hypothesise that Type D personality would also play an important role in stroke recovery. Therefore, it was hypothesised that Type D personality would have a strong relationship with stroke recovery at all 3 time points (*H_1 a, H_2 a, b, H_3 a, b, c*). However, Type D personality failed to be a significant predictor of physical recovery. Additionally as has been mentioned in Section 8.5.3.4, Type D personality was hypothesised to be a mediator between stress and physical recovery at all 3 time points (*H_1 j, H_2 l, H_3 n*), which was not significant.

A minority of research studies have reported no association between Type D and coronary heart disease. In a recent study which investigated the incident risk of

coronary heart disease in a 10 year follow up no significant results were reported (Larson, Barger, & Sydeman, 2013).

Type D personality has also been associated with mortality from cardiovascular disease (Erdman, Duivenvoorden, Verhage, Kazemier, & Hugenholtz, 1986; Pedersen et al., 2004; Schiffer, Smith, Pedersen, Widdershoven, & Denollet, 2010). The mortality of patients in this study was low and therefore no evidence is available to support that Type D personality and mortality from stroke are related. There was a 7% mortality rate at Time 2 and a 0.7% mortality rate at Time 3.

The majority of the Type D personality experiments have been executed in the Netherlands by Denollet's team. Consequently there may be some methodological differences which may not be evident in the papers. There could also be differences in cultural factors. Research comparing Danish personality differences or similarities with British personality characteristics are scarce. Therefore, this area of Type D personality and stroke in a British cohort should be further examined.

Repressive coping and the Physical Recovery Model are discussed in the following section.

8.6.1.8. Repressive Coping and the Physical Recovery Model

To date there have been no published studies on repressive coping and stroke. As repressive coping is considered a dispositional variable most studies are cross sectional (see Myers 2010 for a review). There have been longitudinal studies on repressive coping and cardiovascular disease investigating mortality (Frasure-Smith et al., 2002; Denellot, Martens, Nyklicek, Conraads, & de Gelder, 2008) and how much information regarding heart disease was remembered by repressors (Shaw et al., 1985).

As can be seen there is an absence in the literature concerning longitudinal studies linking repressive coping with stroke. The results from the current study are able to contribute to this area. This study reported repressors at Time 2 experience poorer recovery at Time 2 (H_2 *b*) (β= .22, $p < .001$) and repressors at Time 2 experience poorer recovery at Time 3 (H_3 *b*) (β= .18, $p < .01$). (The Bonferroni correction calculated for H_3 was $p = .02$. This analysis exceeded this value at $p = .04$). It appears that the 3 month post stroke period in recovery from stroke is important for repressors, in predicting 3 and 6 month physical recovery. Time 1 may

be too acute and Time 3 is not a significant time point. Time 2 may symbolise a time where adaption to stroke recovery is realised.

There is evidence which indicates the repressive coping style may be associated with adverse physical health, for example with melanoma, cardiovascular (Kneier & Temoshok, 1984) and breast cancer patients (Kreitler et al., 1993; Jensen, 1987; Giese-Davis et al., 2004, 2006). For the first time in psychological literature, stroke can be added to the conditions which may be adversely affected by displaying the repressive coping style.

Sense of coherence and the Physical Recovery Model are discussed in the following section.

8.6.1.9. Sense of Coherence and the Physical Recovery Model

Sense of Coherence is how a person copes with stressful situations to avoid negative stress (Antonovsky, 1987). Much like social support, SoC has been hypothesised to be a moderator between stress and ill health (Richardson & Ratner, 2005). Antonovsky's theory asserts that SoC affects both physical and psychological aspects of disease (Benz, Angst, Lehmann, & Aeschlimann, 2013). Most studies focus on SoC and caregiver burden not on stroke patients (Van Puymbroeck, Hinojosa, & Rittman, 2008; Chumbler, Rittman, & Wu, 2008; Forsberg-Warleby, Moller, & Blomstrand, 2002), however there has been one major study which successfully linked SoC with stroke (Surtees et al., 2006).

In the current study SoC did not progress onto the final stage of the analysis, because of the low Cronbach's alpha obtained (Time 1: 0.31, Time 2: .57 and Time 3: 0.41). Therefore the role of SoC as a predictive psychological variable cannot be expanded upon in this study. The 3 item measure of SoC was used in order to place a low burden upon participants. However, future studies may be well advised to use the original 29 item scale or the shortened 13 item version (Antonovsky 1979; 1987).

Cognitive factors and the Physical Recovery Model are discussed in the following section.

8.6.1.10 Cognitive Factors and the Physical Recovery Model

This research study also acknowledged the impact of cognitive impairment in stroke patients. Within Health Psychology it is not common practise to adopt

cognitive neuropsychological methods within the research design. In doing this, the current research adds a depth to existing Health Psychological design. Additionally, cognitive neuropsychological studies generally have smaller samples and are cross sectional (Nickles, Howard, Best, 2011). The current study has a larger sample and is longitudinal in design therefore this study has the potential to make an impact in the literature.

Neuropsychological consequences of stroke are often ignored (Dennis, O'Rourke, Lewis, Sharpe, & Warlow, 2000), although cognitive impairment can predict functional outcome (Paolucci et al., 1996; Zinn et al., 2004; Oksala, Jokinen, & Melkas, 2009) which makes it very important to be included in studies which investigate illnesses in the brain.

Many studies use the Mini Mental State Examination (MMSE) (Folstein, Folstein, & McHugh, 1975) which is a short test for dementia (Nys et al., 2005), however this measure does not record specific cognitive impairment (Fatoye et al., 2007). A more specific way to measure cognitive impairment is to use a cognitive battery (Jaillard, Naegele, Trabucco-Miguel, LeBas, & Hommel, 2009). Therefore a strong methodological factor of this study was to use a specific cognitive battery.

The cognitive factors investigated in the current study were verbal and visual short term memory, visual neglect and executive function, which are discussed in the following sections.

8.6.1.11 Verbal Short Term Memory and the Physical Recovery Model

Short term memory problems after stroke can affect how long patients stay in hospital, rehabilitation and adhering to medications (Galski, Bruno, Zorowitz, & Walker, 1993). A search of the literature reveals an absence of studies investigating verbal short term memory with physical stroke recovery longitudinally however, poor visual memory has been related to reduced physical activity in a one year longitudinal study (Pahlman, Savborg, & Tarkowski, 2012). Other studies in this area are lacking.

In the current study short term verbal memory problems did not act as independent predictors of physical recovery from stroke at the 3 fixed time points (H_1 *b*, H_2 *c, d*, H_3 *d, e, f*). This could be due to the stroke lesion not occurring in areas

of the brain necessary for short term memory, such as the prefrontal dorsolateral cortex.

Memory and depression are cited as important consequences of stroke (Kauhanen et al., 1999; Nys et al., 2005; Passier, Visser-Meily, & van Zandvoot, 2010) however verbal memory was not mediated by depression at the 3 fixed time points (*H_1 q, H_2 s, H_3 u*).

Visual short term memory and the Physical Recovery Model are discussed in the following section.

8.6.1.12 Visual Short Term Memory and the Physical Recovery Model

The area of visual short term memory and physical recovery from stroke is an area which needs more attention (Barker-Collo, 2007). In the current study short term visual memory problems did not act as independent predictors of physical recovery from stroke at the 3 fixed time points (*H_1 b, H_2 c, d, H_3 d, e, f*). This could be because visual memory simply does not affect recovery from stroke.

In the current study depression was found to be a partial mediator of visual memory and physical recovery at Time 2 (*H_2 r*), however, Time 1 (*H_1 p*) and Time 3 (*H_3 t*) were not significant.

The *a* path reported higher scores in visual memory at Time 2 predicted lower scores at Time 2 depression (*B*= -.08, $p < .01$). This is an expected result, as improvements in vision leads to lower reported depression. The *b* path reported the expected trend of higher scores at Time 2 depression predicted higher scores at Time 2 physical recovery at the fixed time points (*B*= .46, $p < .01$). And the *c* (*B*= -.17, $p < .001$) and the *c'* path (*B*= -.13, $p < .01$) both report improved Time 2 visual memory scores predicted improvements in Time 2 physical recovery scores.

Additionally, visual memory at 3 months post stroke acted as a partial mediator between depression and physical recovery at 3 months post stroke (*H_2 f*). The *a* path reported worse Time 2 depression scores predicted poorer Time 2 visual memory scores (*B*= -.59, $p < .05$). (The Bonferroni correction for H_3 was calculated at $p = .02$. The *a* path exceeded this at $p = .03$). The *b* path reported better visual memory was associated with better physical recovery (*B*= -.13, $p < .01$). The *c* (*B*=

.53, $p < .001$) and the *c'* path ($B = .46$, $p < .001$) both reported poorer Time 2 depression scores predicted poorer Time 2 physical recovery scores.

However this was not found at Time 1 (H_1 *d*) and Time 3 (H_3 *h*). Depression has an effect on memory (Cipolli et al., 1996). This could explain why Time 1 and Time 3 were not significant as depression level would play an important role in these mediating relationships and depression did not have an impact at these time points.

The area of visual short term memory, depression and stroke outcome is in need of further research (Barker-Collo 2007). There is uncertainty if depression causes cognitive impairment or if cognitive impairment causes depression (Nussbaum 1994; Spalletta, Guida, & Caltagirone, 2003). In the current study, this cannot be answered. This could be investigated in a longitudinal study of healthy participants, before they develop cognitive impairment therefore pre-impairment depressive scores can be recorded. This type of epidemiological study design is normally conducted when researching disease onset, e.g., a disease free sample followed longitudinally, recording disease onset on the way (Surtees, Wainwright, & Khaw, 2006; Salaycik et al., 2007). However, this research design is not adopted by Cognitive Neuropsychology and would be difficult to manage.

Visual neglect and the Physical Recovery Model are discussed in the following section.

8.6.1.13 Visual Neglect and the Physical Recovery Model

The role of visual neglect on stroke recovery is inconclusive with more research being conducted in this area being advised from a systematic review (Pollock et al., 2011). However, visual neglect has been reported as being a predictor of poor functional outcome at 3, 6 and 12 month follow up in a study of stroke patients (Jehkonen et al., 2000). Visual neglect may hinder the effects of functional recovery (Sunderland, Wade, Langton, & Hewer, 1987; Bailey, Riddoch, & Crome, 2002; Jones & Shinton 2006; van Wyk, Eksteen, & Rheeder, 2014; Siong, Woo, & Chan, 2014) and rehabilitation (Barrett & Muzaffar, 2014).

The current study supports the view that better visual neglect at Time 3 predicted improvements in recovery at Time 3 (H_3 *f*) ($\beta = -.20$, $p < .01$). The other combinations of time points (H_1 *b*, H_2 *c, d*, H_3 *d, e*) were not significant. Jehkonen et

al., (2000) reported consistent relationships longitudinally between visual neglect and functional outcome. The current study investigated stroke in the acute phase, which Jehkonen et al., (2000) did not. However, the differences between the current study and Jehkonen et al., (2000) is that the 3 month post stroke time point was significant, however in the current study it was not. It is plausible that the recovery of visual function can take time to develop recovery of function.

Studies investigating visual neglect, depression and stroke are scarce. However some studies have reported an association between visual neglect and depression (Elliott et al.,1996; Tsai et al., 2003; Nys et al., 2006). However, these studies do not incorporate physical recovery after a stroke.

A predicted relationship in the current study was visual neglect would be a mediator between depression and physical outcome at all 3 time points (H_1 *c*, H_2 *e*, H_3 *g*), and depression would be a mediator between visual neglect and physical outcome at all 3 time points (H_1 *o*, H_2 *q*, H_3 *s*). However, this was not supported by this study. A combination of all 3 variables must be exhibited in order to illustrate a mediating relationship.

Executive function and the Physical Recovery Model are discussed in the following section.

8.6.1.14 Executive Function and the Physical Recovery Model

Executive function was not added to the final analysis due to the loss of data of this variable. However, an overview of the hypothesised function of executive function as proposed in the study will be given. Executive function was hypothesised to be affected by high stress and depression as proposed by Lawrence & Grasby, (2001). This was hypothesised to be present at all 3 time points (H_1 *f, k*, H_2 *h, m*, H_3 *j, o*). Direct effects of executive function on stroke recovery are scarce in the literature, although this impairment has been reported to be associated with impaired activities of daily living in stroke patients (Bour, Rasquin, Limburg, & Verhey, 2011; Chung, Pollock, Campbell, Durward, Hagen, 2013; Middleton, Lam, & Fahmi, 2014). These predictions were outlined in H_1 *b*, H_2 *c, d*, H_3 *d, e*, and *f* but could not be tested.

The Psychological Recovery Model is discussed in the following section.

8.6.2 The Psychological Recovery Model

The main analysis for this model was conducted with a series of hierarchical multiple regressions, the results of which are outlined below. In this section, Time 1, Time 2 and Time 3 variables were significant in predicting QoL at Time 2 and Time 3. Figure 8.2 illustrates the amended theoretical model based on the significant variables.

8.6.2.1 Demographic & Stroke Markers and the Psychological Recovery Model

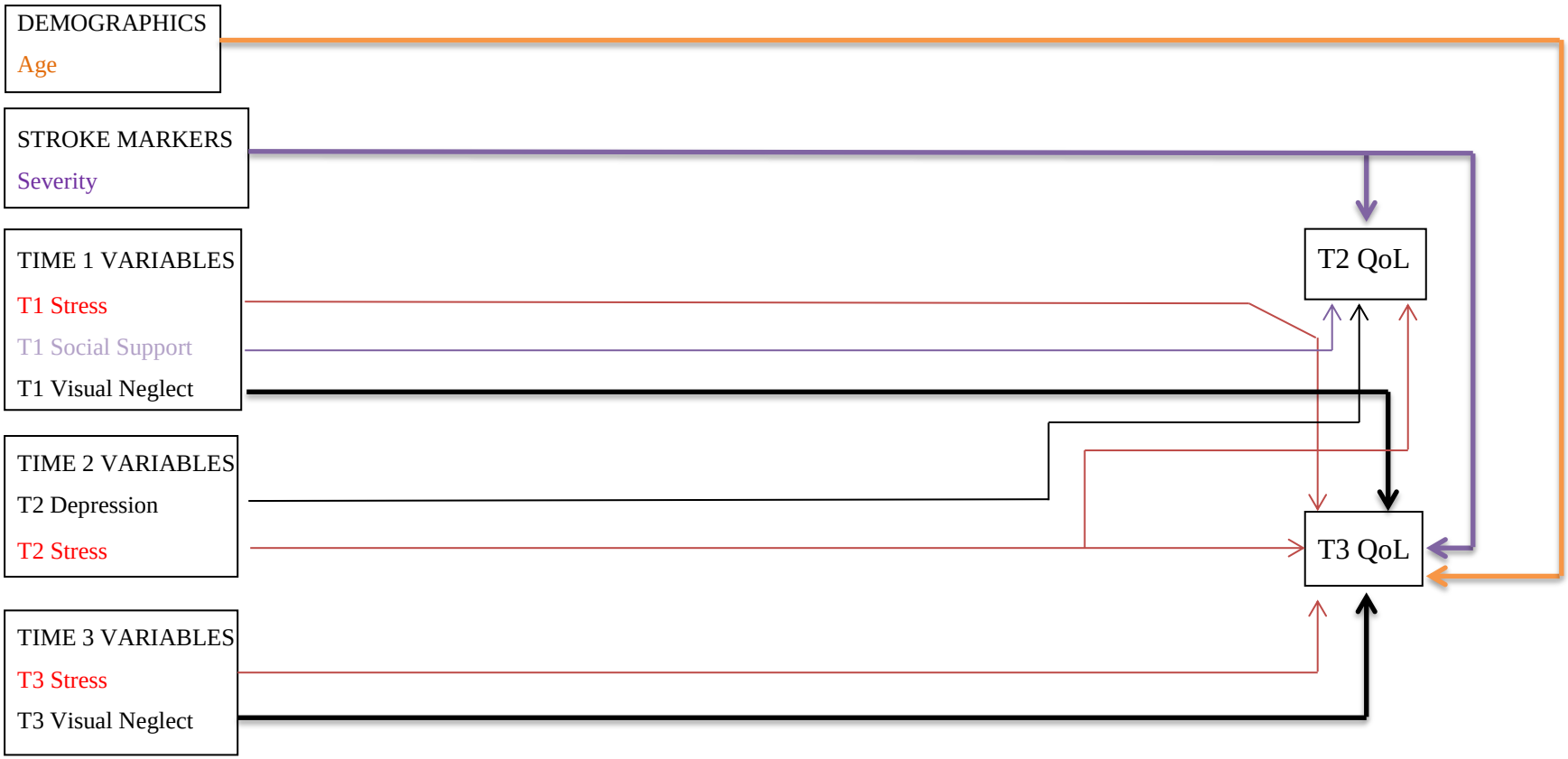
Stroke severity was a significant predictor throughout the analysis for QoL. Moderate and severe strokes at baseline predicted poorer Time 2 QoL ((β= -.31, p < .01 and β= -.31, p < .01 respectively) and severe strokes at baseline predicted Time 3 QoL (β= -.23, p < .001). These findings are expected, as it has been documented that stroke severity is related to QoL (Gosman-Hedstrom, Claesson & Blomstrand, 2008; Carod-Artal & Eguido, 2009**;** Owolabi, 2011; Lopez-Espuel, 2014).

Additionally, older age at baseline was associated with poorer QoL at Time 2 (β= -.21, p < .05) and Time 3 QoL (β= -.29, p < .01). Gurcay, Bal, & Cakci, (2009), in their cross sectional study, also found a significant relationship between age and QoL in regards to psychological stroke recovery, whereas Haacke, Althaus, Spottke, Siebert, Back & Dodel (2005) and Jipan et al. (2006) report no significant relationship between age and QoL.

As stroke severity has an impact on both Time 2 and 3 QoL, it seems reasonable to also conclude that older age would also have an impact at these time points.

The next section discusses depression and the Psychological Recovery Model.

Figure 8.2 Theoretical Model Results for Psychological Recovery



8.6.2.2 Depression and the Psychological Recovery Model

Depression has been reported to have an adverse effect on health related quality of life in recovering stroke patients (Kim, Warren, Madill, & Hadley, 1999; Lofgren, Gustafson, & Nyberg, 1999; Carod-Artal, Egido, Gonzalez, & de Seijas, 2000; Kwok et al., 2006; Naess, Waje-Andreassen, Thomassen, Nyland, & Mhyr, 2006; Patel, McKevitt, Lawerence, Rudd, & Wolfe, 2007; Teoh, Sims, & Milgrom, 2009).

The current study lends support for this. Time 2 depression scores predicted poorer QoL at Time 2 (H_5 *a*) (β= -.17, $p < .05$), however this relationship was not significant at other time points (H_4 *a*, H_6 *a*, *b*, *c*). The relationship is significant at the 3 month post stroke point only therefore in the acute phase of stroke depression was not significant. This could be due to the impairment of higher cortical functions or social desirability. Six months post stroke depression was also not significant. This could be due to fluctuating levels of emotion.

The next section discusses stress and the Psychological Recovery Model.

8.6.2.3 Stress and the Psychological Recovery Model

Studies investigating QoL, stress and stroke mainly focus on caregiver burdens (Op Reimer, de Haan, Rijinders, Limberg, & van den Bos, 1998; Gaugler, 2010; Jaracz, Grabowska-Fudala, & Kozubski, 2012; Kniepmann, 2012; Bhattacharjee, Vairale, Gawali, & Dala, 2012; Clay et al., 2013).

Limited studies have been conducted on QoL, psychological stress and stroke recovery. Baune & Aljeesh (2006) conducted a study based in the Gaza Strip. The results of this study were weak with only one domain of the WHOQoL-BREF (the Global domain) reported as significant, whilst the remaining domains (physical, psychological, social and environmental domains) were insignificant.

In the current study psychological stress featured prominently with QoL. Stress at Time 2, predicted QoL at Time 2 (H_5 *a*) (β= -.49, $p < .001$). QoL at Time 3 was significant at predicting Time 1 stress (H_6 *a*) (β= -.13, $p < .01$), Time 2 stress (H_6 *b*) (β= -.17, $p < .05$) and Time 3 stress (H_6 *c*) (β= -.42, $p < .001$). These findings illustrate that perceived psychological stress and QoL are strongly related, with all time points predicting Time 3 QoL, and Time 2 specifically being relevant in predicting Time 2 QoL. This could be because constant stress would ultimately lead

to not enjoying one's life. Additionally, psychological stress seems to affect QoL more so than depression. This could be due to the alertness of stress, whilst depression characterises lethargy.

Studies illustrating a link between stress and QoL longitudinally are scarce and therefore findings are very important to literature.

The next section discusses social support and the Psychological Recovery Model.

8.6.2.4 Social Support and the Psychological Recovery Model

Social support can have a positive effect on QoL in stroke patients (Gottlieb, Golander, & Bar-Tel, 2001; Tang et al., 2005) and conversely participants with low social support report lower QoL (Kim, Warren, Madill, & Hadley, 1999; Kwok et al., 2006).

In the current study high social support scores at Time 1 predicted improvements in QoL at Time 2 (H_4 *a*) ($\beta = .27$, $p < .001$). Here social support in the acute stroke phase plays the significant role, whereas the subsequent time points remain non-significant. This could be explained by as time progresses less social support is perceived, as the most social support was offered in the acute phase of recovery.

In the social support literature, the main focus is on how social support has beneficial effects. Lynch et al., (2008) found in a qualitative study that social support was a main theme identified among stroke survivors in relation to importance of QoL. Therefore, social support was expected to play a much bigger role. However, there are salient results reported in that Time 1 social support is predictive of Time 2 QoL.

The next section discusses Type D personality and the Psychological Recovery Model.

8.6.2.5 Type D Personality and the Psychological Recovery Model

This is the first study to the authors' knowledge which is investigating stroke, Type D personality and QoL. Type D personality has been reported to predict QoL in other studies (Pedersen & Denollet, 2003; Pedersen, Theuns, Muskens-Heemskerk, Erdman, & Jordaens, 2007; Pelle et al., 2008; Bartels et al., 2010; Schiffer, Pedersen,

Widdershoven, & Denollet, 2008; Dubayova, 2009; Saraoudi, 2011**;** Staniute, 2015**),** however, within the current study, there have been no significant results (*H_4 a, H_5 a, H_6 a, b, c*). As this is the first known study investigating Type D personality and stroke it is too early to make conclusions as to if Type D personality is important with stroke. Therefore it is advisable for future research to investigate this relationship further.

The next section discusses repressive coping and the Psychological Recovery Model.

8.6.2.6 Repressive Coping and the Psychological Recovery Model

To the authors knowledge this is the first study investigating the role of repressive coping with QoL in stroke patients. A search of the literature shows there is a scarcity of studies investigating repressive coping with QoL, as the focus remains on physical health. More studies should be conducted on the relationship between repressive coping, QoL and stroke.

In the current study there were no significant results (*H_4 a, H_5 a, H_6 a, b, c*). This could be explained as QoL is a self-report measure, therefore repressors avoid negative affect which can be explained by the non-significant result. However, with the physical recovery measure, there were significant results, this could be because it is an observer rated measure.

The next section discusses sense of coherence and the Psychological Recovery Model.

8.6.2.7 Sense of Coherence and the Psychological Recovery Model

No studies were found on QoL, SoC and stroke recovery. SoC has been reported to predict QoL in a sample of coronary heart disease patients (Motzer & Stewart, 1996; Wrzesniewski & Wlodarczyk 2012; Silarova et al., 2012), whilst no effect of SoC was found on QoL in a sample of women with coronary heart disease problems (Bergman, Malm, Bertero & Karlsson 2011; Piegza et al. 2014).

As Soc exhibited a low Cronbach's alpha value, this measure was not tested (although it was predicted in *H_4 a, H_5 a, H_6 a,b,c*).

The next section discusses cognitive factors and the Psychological Recovery Model.

8.6.2.8 Cognitive Factors and the Psychological Recovery Model

Quality of life is not routinely studied in cognitive studies, however a minority of studies have found an association showing lower QoL is related to cognitive impairment in cross sectional studies of stroke patients (Nys et al., 2006; Gurcay, Bal, & Cakci, 2009). Specific cognitive impairments shall be discussed in the following sections.

8.6.2.9 Visual and Verbal Memory and the Psychological Recovery Model

Verbal memory and QoL have been reported to have no relationship in aneurysmal subarachnoid hemorrhages (Al-Khindi, MacDonald & Schweizer, 2010). The current study did not find a relationship between verbal memory and QoL in stroke patients (H_4 *b*, H_5 *b*, H_6 *d*, *e*, *f*). However, studies in this area are lacking. However impairments in visual memory have been associated with disability and lower quality of life in a 5 year longitudinal study (Barker-Collo, Feigin, Parag, Lawes, & Senior, 2010), although the current study does not support this (H_4 *b*, H_5 *b*, H_6 *d*, *e*, *f*).

The next section discusses visual neglect and the Psychological Recovery Model.

8.6.2.10 Visual Neglect and the Psychological Recovery Model

Visual neglect has been reported as being a strong predictor of QoL 6 months post stroke in a first ever study on this subject (Nys et al., 2006) and visual neglect at discharge has been associated with lower QoL (Franceschini, La Porta, & Agosti, 2010). However, no other studies were found on QoL and visual neglect in stroke patients.

In a recent review investigating stroke related vision problems and quality of life, poorer QoL was reported from patients with visual field defects. However, although visual neglect was acknowledged no information on studies investigating visual neglect and QoL were reported (Sand et al., 2013).

The current study is able to contribute to this area. Higher Bells scores at Time 1 predicted improvements in QoL at Time 3 (H_6 *d*) ($\beta = .24$, $p < .05$), and higher Bells scores at Time 3 predicted improvements in QoL at Time 3 ($\beta = .25$, $p <$

.05). (The Bonferroni correction calculated for H_6 was p = .03. This variable exceeded this threshold at p = .04) (H_6 *f*).

What is apparent here is that Time 2 visual neglect fails to be significant, indicating that the acute phase and the end phase are the important time points, with the subacute phase (3 months post stroke) playing a lesser role. This could be due to simply statistical factors at Time 2 failing to reach the statistical threshold.

The next section discusses executive function and the Psychological Recovery Model.

8.6.2.11 Executive Function and the Psychological Recovery Model

Although executive function was not added to the final analysis it was predicted to have an effect on QoL (H_4 *b*, H_5 *b*, H_6 *d, e, f*). The relationship between executive function and QoL is in dispute in the literature. Executive function has been found to have a direct effect on QoL (Brookes et al., 2014) but also a non-significant relationship has also been reported (D'Aniello, Scarpina, & Mauro, 2014; Al-Khindi, MacDonald, & Schweizer, 2010). As there is a dispute and scarcity in the literature for cognitive studies including QoL, more can be achieved in this area.

Methodological limitations are discussed in the next section.

8.7 Methodological Limitations

It is salient to discuss the issue of bias in research to be able to acknowledge the weaknesses that are present in research designs. Reliability is important to consider because it is concerned with the repeatability of the study and the consistency of the test used to measure a concept or the consistency of different observer ratings. Internal reliability measures items on a scale to see if they are consistent, this is normally measured with a Cronbach's alpha statistic (Bryman 2008).

All of the measures had good Cronbach alpha values (see table 5.2), except for one. The 3 item SoC had a Cronbach alpha value of 0.31 at Time 1, 0.57 at Time 2 and 0.41 at Time 3. The 3 item SoC scale has been reported to yield similar ratings compared with the 29 item SoC scale (Antonovsky, 1993), therefore a higher Cronbach alpha value was expected.

The Cronbach's alpha coefficient can change depending on the number of items in a scale. The larger the number of items, the higher the alpha value may be. Shorter scales may score lower on alpha however, this may not be due to the fact that there is low internal consistency but because the items in the scale are below 10 items. (Pallent, 2013).

The 3 item SoC has been used successfully in stroke research assessing the risk of stroke in a longitudinal study from Cambridge researchers Surtees et al., (2006). In the European Prospective Investigation into Cancer (EPIC-Norfolk) the 3 item SoC had a Cronbach's alpha of 0.35, which could be due to the low number of items in the scale, however it was still used in a study of 20,921 participants and was found to be a significant predictor of mortality and stress adaptive coping. Therefore it was deemed as beneficial to stroke research to again assess the use of the 3 item SoC. Nevertheless, SoC was not included in the final analysis due to the low Cronbach's alpha obtained.

Biases can affect the quality of research studies. Recruitment can cause bias if the procedure is not standardised (Pannuci & Wilkens 2010). In this study participants were recruited consecutively. Consecutive patients are preferred compared to non-consecutive patients because there is reduced biasing in the recruitment stage as participants are approached in the order they are admitted to hospital. With non-consecutive patients, they may be purposefully chosen, which may cause a selection bias. Researcher bias is also important to recognise as the researcher knows which health condition is being investigated and is therefore more aware of information that fits in with risk factors and related variables. Confounding factors affects all research studies. This is where an unmeasured factor influences the outcome. The best way to address these unknown factors is to have true randomisation in a large sample (Pannuci & Wilkens 2010).

Social desirability bias can also be present which can cause participants to offer socially desirable answers (Bowling 1997). The settings of data collection included hospitals, the home environment, nursing homes and telephone interviews. All studies first time point measurement was taken in the hospital. In face to face interviews at home and telephone interviews, participants may demonstrate social

desirability bias as they may change their answers to be more positive if family members are present, or conversely, they may be more honest in their responses, as they are comfortable in their familiar environment.

Additionally, a modified White Coat Effect (WCE) can occur. This is when blood pressure readings are taken by a doctor or a nurse cause an increase in blood pressure in response to the test, because by being tested raises blood pressure (Saladini, Benetti, Malipiero, Casiglia, & Palatini, 2012; Garcia-Donaire et al., 2012). Therefore, if a participant is in a hospital and being questioned on negative mood they may respond more negatively to these questions due to being in hospital.

This can manifest as recall bias (Pannuci & Wilkens 2010), as a participant has been diagnosed with a stroke their recall about the events leading up to the stroke may be altered e.g., once diagnosed with a stroke, a participant may report higher levels of stress or depression when asked how they have been feeling before the stroke occurred.

Attrition bias is concerned with the drop out rate from the study which can lead to a biased outcome (Jüni & Egger, 2005) for example, healthy people may remain in the study which may bias the results. The attrition rate of this study was 29.37%, which is nearly a third of the study participants. 16.1% of this loss was due to refusals. In a study investigating attrition, patients whom scored high on depression at baseline, were 1.5 times more likely to be lost to follow up (Farmer & Locke, 1994), therefore, the loss to follow up could be due to negative affect.

Loss to follow up also results in a cohort bias, in which healthier participants remain enrolled in the study, thereby producing results which may support the hypothesis under investigation (Bryman, 2008).

It is difficult to know exactly to what extent these biases have occurred. However, the Researcher of this study attempted to treat each participant the same, thereby aiming to reduce effects of bias from the Researcher.

Biases can be random (participants may be careless when responding), or biases may be systematic (participants may be prone to offering socially desirable answers). For example, participants may report erroneous levels of social support,

because of either not wanting to admit they have insufficient social support or because of social desirability (Wilcox, Kasl, & Berkman 1994).

Additionally, the colour word Stroop which tests executive function, was removed from the final analysis, because of the loss of data due to participants not all being able to complete this test successfully. Also, there were discrepant findings with the emergence of reverse Stroop responses. As this is not measuring executive function, these reverse Stroop data were removed. As a consequence, if the Stroop was included in the regression analysis, the final numbers would have diminished down to 65 participants. In order to retain as much valuable data as possible, it was decided to remove the Stroop from the final analysis.

However, this is not to subtract from the importance of the relationship between executive function and stroke. In fact, it only strengthens it. Participants' failing at completing the Stroop successfully, does indicate that stroke patients could have executive dysfunction. Some participants were unable to follow instructions for the control task therefore, conclusions regarding executive dysfunction become more complicated.

Language impairments can cause difficulties with research designs and gaining informed consent, which lead stroke patients to be excluded. However, excluding these patients reduces the generalizability of results (Townsend, Brady & McLaughlan 2007). Therefore, other methods of data capture should be devised, which can include this group of stroke patients.

One area of improvement would be to also collect data on stroke lesion and location as brain lesions can affect many functional processes which can impact on a patient's behaviour in many ways (Lezak, 1995).

Methodological measurement issues are discussed in the next section.

8.8 Methodological Measurement Issues

Brief measures were used in order to maximise the amount of data collected in an acute stroke environment as administering tests in the acute phase of stroke is exhausting for the patient (Duits, Munnecom, van Heugten, & van Oostenbrugge, 2007) and can cause respondent fatigue (Anastasi, 1976).

For some constructs there are no standardised definitions such as, for QoL, social support and stress. Stress was measured using the Perceived Stress Scale (PSS) (Cohen, Karmack, & Mermelstein, 1983), which is the most frequently used measure of perceived stress therefore allowing for comparisons with a larger pool of studies. Social support was measured using the Multidimensional Perceived Social Support Scale (MPSS) (Zimet, Dahlem, Zimet, & Farley, 1988). As perceived social support has been shown to be more powerful than received social support, this measure was chosen as again, comparisons can be yielded if needed as this measure is well known. Depression was measured with the CESD-10 (Andresen, Malmgren, Carter, & Patrick, 1994). Although this is a short measure for depression it is well validated although depression tools are not based on neurologically impaired samples (American Psychiatric Association 1994). There can additionally be potential difficulties in assessing depression as symptoms of depression and the physical consequences of stroke may overlap e.g., fatigue and decreased appetite (Aben & Verhey, 2006).

There is only one current measure available to measure Type D personality which is the DS-14 (Denollet, 2005). It does have good reliability; however, this construct was not significant at all in this study. Repressive coping uses two measures to determine the repressive coping style (anxiety and defensiveness). Median splits were used to define repressors. However, this method also includes borderline repressors in with extreme repressors. The advantage of this is being able to use the complete data set which can aid in gaining power for analysis (Myers 2000). Repressors have an inclination for positive self report (Myers & Vetere 1997) therefore there may be some inaccuracy and distortion in self reporting from repressors (Myers 2000). Suggestions for combating this include conducting semi-structured interviews with a trained researcher (Myers, Brewin & Winter 1999) or self-report measures that take these factors into account (Myers 2010). Myers (2010) has suggested more longitudinal studies should be conducted on repressive coping as the majority have been cross sectional. This study has measured repressive coping 3 times post stroke, thereby collecting valuable longitudinal data.

Physical functioning in stroke is primarily assessed using, either the mRS (Bonita & Beaglehole 1988) or the Barthel Index (BI) (Mahoney & Barthel, 1965;

Feigin, Barker-Collo, McNauhton, Brown & Kerse 2008). However, these measures only acknowledge physical functioning and therefore a measure of QoL should also be used (Haacke et al., 2006). This suggestion has been taken on board with this study in which mRS and QoL are used as outcome measures.

The SF-36 (Ware Jr. & Sherbourne, 1992) is a general measure of health related quality of life and not a specific stroke measure (Anderson, Laubscher, & Burns, 1996). Stroke measures are lengthy and therefore difficult to use in an acute stroke setting. Quality of life was measured in stroke patients using the SF-36 and the Sickness Impact Profile. The results demonstrated that both measures were correlated (Carod-Artal, Egido, Gonzalez, & Varela de Seijas 2000). Due to this Suenkeler et al., (2002) rationalised using the SF-36 in a longitudinal study of stroke and QoL.

However, it is important to bear in mind that participants may change responses whilst in longitudinal studies because their criterion for determining responses may change which would include scale recalibration and reconceptualization of a construct. For instance, a participant may answer they have good recovery after stroke but revising that a few months later they would recalibrate to perceive their health was poor, and presently they have good health. Therefore, there have been hypotheses on whether changes in responses are due to a response shift, or if indeed they are due to a true change. There have been attempts at trying to decipher this by using factor analysis to examine changes in response structures however, there have been mixed findings (Ahmed, Mayo, Corbiere, Wood-Dauphinee, Hanley, & Cohen, 2005).

Methodological strengths are discussed in the next section.

8.9 Methodological Strengths

Stroke recovery can change over time. This is better acknowledged with repeated measures over time to determine any changes in psychological and physical factors. The length of follow up is important as stroke presentation combined with stroke severity will determine stroke recovery. A longer follow up period is more beneficial to concluding any related factors compared with a shorter follow up period. Longitudinal designs are difficult to execute due to the time involved and the

cost of the time involved. For this reason these study designs are utilised less in social science research (Bryman, 2008). This research study assessed an acute clinical stroke sample, recruited 0-6 weeks post stroke from NHS hospitals and followed up at 3 month and 6 month post stroke time intervals. This study also recruited consecutive stroke patients which reduces bias in the recruitment stage and should result in a general sample of stroke patients in regards to demographics, age and ethnicity.

Additionally, most health psychological studies do not report on stroke type and severity. This study did and also reported on stroke classification. Stroke type is important to record because of the differences in the stroke itself. This can give reasons as to the causes of the stroke and this can be related to stroke severity. Ischemic strokes are more common than haemorrhagic strokes, but haemorrhagic strokes are often more fatal. Ischemic strokes are caused by a blockage in an artery that leads to the brain which can be the result of an unhealthy lifestyle, such as poor diet, smoking, lack of exercise and drinking alcohol. Haemorrhagic strokes are caused by a ruptured vessel or artery in the brain from high blood pressure which causes pressure on the vessel walls. The risk of this type of stroke is often difficult to determine as opposed to the ischemic stroke which has more measureable risk factors (Barnett, Mohr, Stein, & Yatsu, 1998). It is useful to report the type of stroke as this can be compared with other factors such as demographic factors, ethnicity, age, risk factors and psychosocial variables, such as stress.

In research studies that are investigating recovery from stroke it is important to record stroke severity. Only including mild strokes will not yield data on the realistic nature of stroke and will produce a homogenous sample. Also, stroke severity will undoubtedly have an effect on stroke recovery and psychological wellbeing. This information should be available when considering studies that claim to investigate stroke recovery. Stroke also causes language impairments, which lead research studies to exclude this group which causes a cohort bias. In this study patients with mild, moderate and severe stroke severity have been recruited, additionally including patients with receptive dysphasia.

Often research does not include power calculations; therefore it is difficult to conclude the statistical viability of the research as insufficient power may lead to

Type II errors. Power calculations determine the sample size needed to reach statistical power (Levine, Stephan, Krehbiel, & Berenson, 2011). The number of participants estimated from this analysis was 119, which included a 20% attrition loss. However, actual attrition was just over this. The main study recruited 143 participants to allow for attrition. The response rate for this study was high at 70.79%, whilst the death rate was low, with 10 deaths at Time 2, and 1 death at Time 3.

Another strength of this study is proxy ratings were not used. A proxy rating is where a third party answers questions on behalf of the participant. Proxy measures are used extensively in stroke research (Pohjasvaara et al., 2001; Pohjasvaara et al., 2002; Desrosiers et al., 2002; Desrosiers et al., 2006; Wilz, 2007) because sufferers of stroke can experience problems with dysphasia (language impairment), dysphagia (swallowing problems) and dysarthria (problems with the muscles that help one to speak resulting in slurred speech) (Barnett, Mohr, Stein, & Yatsu, 1998) which can make communication difficult. These communicative problems can lead researchers to search for proxy measurements however, these measurements may be biased from the proxy respondent, therefore producing questionable results. Proxy ratings should not be analysed along with self rated measures (Hilari, Wiggins, Roy, Byng, & Smith, 2003) as this will further contaminate any conclusions made. Additionally, proxies rate the participant's health worse than the participant would rate it (Dorman, Waddell, Slattery, Dennis, & Sandercock, 1997; Sneeuw et al., 1997; Pierre, Wood-Dauphinee, Korner-Birensky, Gayton, & Hanley, 1998) which causes a subjective discrepancy.

Proxy ratings are used to prevent exclusion of patients (Sneeuw et al., 1997) however, research should not be dependent on proxy ratings. It may be more useful to stroke research to investigate alternative approaches in collecting data from participants with communicative impairment, for example, using touch screen technology and Dragon voice activated software which would minimise the researcher/proxy-participant interaction and reduce bias.

Furthermore, this research incorporates cognitive neuropsychological measures. Often within health psychology the inclusion of cognitive neuropsychological measures is scarce. The Mini Mental State Examination

(MMSE) (Folstein, Folstein, & McHugh, 1975) is frequently used in research studies to measure cognitive impairment. However, the MMSE was designed to assess dementia in patients but it is commonly used to assess general cognitive impairment which is a misuse of the measure (Nys, van Zandvoort, de Kort, Jansen, Kappelle, & de Haan, 2005). Research should not use tests such as the MMSE but should use a neuropsychological battery of tests for more specific data collection (Nys et al., 2005).

There are 4 main cognitive domains: visuo-spatial impairment, memory, executive function and language (Kolb & Wishaw, 2009). As stroke can cause language difficulties this may result in the exclusion of a section of potential participants. Because of this severe language impairment was used as a guideline to exclude participants as the issue of informed consent was salient. The remaining cognitive domains were all represented in the study with in particular, visual neglect and visual short term memory predicting outcome. In future stroke research, it would be beneficial to construct a framework which would encompass language difficulties as part of the study, rather than as a screening guideline.

Limited studies have investigated the role of cognitive impairment and QoL, where cognitive impairment is measured by a cognitive neuropsychological battery. One study used a cognitive battery including orientation, memory, attention, visuo-spatial factors, language and arithmetic. The Trail Making Test B, which is a measure of attention (a complex visual scanning task) was related to QoL, 9 months post stroke. However, many patients were removed from analysis for not being able to complete the cognitive tests. This may have led to an erroneous conclusion (Hochstenbach, Anderson, van Limbeek, & Mulder, 2001).

Healthcare policy is discussed in the next section.

8.10 Healthcare Policy

Psychological research although vast in stroke has not made a noticeable impact on health care policy. Speech therapists help patients with dysphasia, aphasia, and swallowing issues by facilitating the use of throat muscles and vocal chords. Physiotherapists help patients to use limbs which may have been affected by the stroke by teaching exercises to help strengthen muscle groups. Occupational therapists help patients to become independent in their daily living, by helping them

to learn how to cook and manage themselves on a daily basis (Kumar & Clarke, 2009). However, there are no current psychological health care policies to help patients to deal with the emotional consequences of suffering a stroke despite the wealth of psychological research undertaken.

In the National Clinical Guidelines for Stroke (UK) it is stipulated that all patients should be screened for depression and even if they have mild depression their needs should be met, they should be provided with information and interventions should be made available to them, for example, increased exercise or social interaction, goal setting, or other psychological interventions. Therapy should be contemplated for patients (Intercollegiate Stroke Working Party, 2012). However, these guidelines were not evident during the course of this study.

In a study by Hart & Morris (2008) investigating depression screening for stroke patients there was a lack of compliance from health professionals in this research leading to a conclusion of raising compliance within NHS staff by increasing their knowledge of the guidelines and enhancing their skills.

In a study of inhabitants of nursing homes, those that lived in nursing homes had four times higher rates of depression compared to elderly people living in the community. Factors that predicted depression were loneliness, negative life events, age, functional and visual impairments, pain, and stroke. However, depression is not treated in nursing homes (Jongenelis et al., 2004).

Implications for theory are discussed in the next section.

8.11 Implications for Theory

The components of this study include demographic, stroke markers, psychological and cognitive factors but genetic risk factors should also be considered. As behavioural changes may not be enough to combat genetic predispositions to illness. However, family history of stroke and heart disease were not a focal point in this study.

Additionally, the health psychology discipline does not normally report stroke characteristics and cognitive neuropsychological factors. The implications for

theory are to encourage future health psychological research to incorporate these factors more comprehensively in order to produce better quality research.

Clinical significance of the findings is discussed in the next section.

8.12 Clinical Significance of the Findings

In this section only those variables that were significant will be discussed. These are depression, stress, social support, repressive coping, visual short term memory and visual neglect.

8.12.1 Depression Findings

Depression ratings were low in the current study. This could either be due to social desirability issues or from depression truly being low in this sample. This would cause participants to not be offered antidepressant medication. Additionally, therapy is not usually offered to patients (Hart & Morris 2008) and there is an inadequacy of effective treatments using psychological therapies and/or antidepressants. This finding is unexpected but is evident of governing bodies not taking heed of decades worth of psychological research. Although General Practitioners are able to refer patients for CBT using the Improving Access To Psychological Therapies Service (IAPT) through the National Health Service (NHS) (www.iapt.nhs.uk). This website claims that by April 2015 the services for adults will be completed. In May 2015, this completion had not been confirmed (IAPT, 2015).

Recovery from stroke can improve if depression is treated (Aben et al., 2001). This could be achieved with the use of antidepressant medications (Andersen, Vestergaard, & Lauritzen, 1994a; Arseniou, Arvaniti, & Samakouri, 2011) and therapies, such as psychosocial-behavioural therapy (Mitchell et al., 2009) and cognitive behavioural therapy (CBT) (Lincoln & Flannaghan, 2003). Nonetheless from the experience of conducting the current study, therapies are not offered to participants. However depression has not been completely absent from the current study as Time 2 depression predicted poorer QoL at Time 2.

The clinical significance of stress is discussed in the next section.

8.12.2 Stress Findings

Stress has been found to be important in both physical and psychological recovery from stroke in the current study. Additionally, from the current study, it has been observed stress interventions are not routinely offered in U.K hospitals.

Interventions have been investigated on yoga and mindfulness techniques (Lawrence, Booth, Mercer, & Crawford, 2013; Lazaridou, Philbrook, & Tzika, 2013), however, most studies have focused on relieving stress on stroke caregivers (Servaes, Draper, Conroy, & Bowring, 1999; Hartke & King, 2003; Legg et al., 2011; King et al., 2012).

The clinical significance of these findings is that stress interventions should be considered as part of stroke rehabilitation. This could be achieved by trained staff teaching yoga and mindfulness techniques being practised alongside traditional rehabilitation areas, such as physiotherapy. These could be offered both as inpatients and outpatients. However, these intervention studies have not stipulated what level of stroke severity they included and how many yoga positions the participants could successfully complete. This area could be further explored in future research.

The clinical significance of social support is discussed in the next section.

8.12.3 Social Support Findings

The overwhelming amount of research shows social support interventions do not have a positive impact on post stroke recovery (Friedland & McColl, 1992; Mant, Carter, Wide, & Winner, 2000; Clark, Rubenach, & Winsor, 2003; Lincoln, Francis, Lilley, Sharma, & Summerfield, 2003; Corr, Phillips, & Walker, 2004; Boter & HESTIA Study Group, 2004; Tilling, 2005; Burton & Gibbon, 2005).

Stroke support groups are adept at providing instrumental knowledge of stroke (Weltermann et al., 2000) and emotional support (Pierce & Salter, 2012). Stroke support groups were partially advertised in the hospitals which were involved in the recruitment phase of the current study however more research should be conducted on the role these support groups can play in social support for recovering stroke patients.

The clinical significance of repressive coping is discussed in the next section.

8.12.4 Repressive Coping Findings

To date there are no published studies on repressive coping and stroke recovery. The current study has identified the 3 month post stroke time point as important in predicting stroke recovery at 3 and 6 months post stroke.

To date there are no interventions focussing on repressive coping. This should be investigated further in the future, especially focussing on therapy. As repressors avoid negative affect, therapy could help with managing this. A useful style of therapy should be identified as this has not been achieved to date.

The clinical significance of visual short term memory is discussed in the next section.

8.12.5 Visual Short Term Memory Findings

Interventions to improve cognitive impairment mainly focus on speech therapy. Memory recovery is normally viewed as a spontaneous phenomenon in stroke recovery. Medications are not prescribed for this (Novitzke, 2008). Memory is routinely not worked on with patients. Visual short term memory was found to be significant in mediating relationships but verbal memory was not.

In a systematic review (das Nair & Lincoln, 2008) investigating rehabilitation of memory impairments in stroke patients, only two studies were identified, however these studies reported no significant findings. The results of the current study do illustrate that memory is an important area for recovery from stroke, however designing rehabilitations for memory this has yet to be fine tuned.

The clinical significance of visual neglect is discussed in the next section.

8.12.6 Visual Neglect Findings

Visual neglect can be addressed with the use of an eye patch as it encourages the patient to be more aware of the neglected side of vision by turning their head towards the neglected side of vision (Cicerone et al., 2000; Jutai et al., 2003). Visual neglect may not fully improve (Banich 2004) but the majority of patients researched by Jutai et al., (2003) improved at 3 months post stroke (Jutai et al., 2003).

Future avenues of research are discussed in the next section.

8.13 Future research

There is potential for this study to be extended to follow up participants at 5 years post stroke. This would be beneficial to stroke research to investigate the longer term interaction of psychological and cognitive influence on psychological and physical recovery from stroke.

It is also possible to investigate the relationship between the main variables in a different combination, perhaps investigating depression or stress as an outcome variable, predicted by physical recovery.

Additionally, Type D personality did not present itself as an independent predictor as hypothesized, however more analysis can be achieved in this area, especially as this is the first known study investigating Type D personality with stroke. From investigating the correlation matrices in Chapter 5, Section 5.2, p. 247-259, there are indications that Type D personality may have been mediated by other variables. At Time 2, Time 2 depression, stress and visual memory may act as mediators between Time 2 Type D personality (or vice versa) and Time 2 QoL. At Time 3, Time 3 stress and Time 3 repressive coping may act as mediators between Time 3 Type D personality (or vice versa) and Time 3 QoL. This possibility can be examined further separate to this thesis.

Future research should consider the findings of the current study and interventions, especially in repressive coping should be designed with the aid of therapists.

8.14 Conclusion

The current study contributes to stroke research in many ways. Firstly a comprehensive systematic review (Study One) was undertaken. This systematic review detailed the relationship between psychological variables and outcome which formed the basis of this thesis and facilitated the design of Study Two.

Study Two was a longitudinal study, harnessing a clinical sample. Three time points were measured, the first time point being collected at 0-6 weeks post stroke. This time point was in the acute phase of stroke recovery and is difficult data to obtain. Time 2 was recorded at 3 month post stroke and Time 3 was recorded at 6 months post stroke. A broad dataset was collected on depression, stress, social support, Type D personality, repressive coping and SoC. Additionally a cognitive

neuropsychological battery was added to record specific cognitive impairments in verbal and visual short term memory, visual neglect and executive function. All responses were from the participant. No proxy ratings were used, which is beneficial to stroke research.

Only two variables (SoC and executive function) were removed from the final analysis due to exhibiting a low Cronbach's alpha value and a loss to cases respectively. All the remaining variables were eligible to be included in the final analysis.

Due to the longitudinal nature of the research multiple testing was present. However, Bonferroni corrections were calculated. Bonferroni corrections although the most frequent method used to control alpha, it is also a strict method (Perneger, 1998). Gelman, Hill & Yajima (2012) remind us that using the Bonferroni correction can control for Type I errors (false positives), but this could be at the cost of committing a Type II error (a false negative). Where Bonferroni corrections were exceeded this was reported. However, these corrections were only slightly infringed, therefore those results were reported. No value judgement was made regarding which error (Type I or II) was more salient. Therefore an acknowledgement of both error types are made and consequently minor transgressions of the Bonferroni correction are accepted.

Both the Physical Recovery Model and the Psychological Recovery Model both developed 3 main hypotheses each (therefore 6 main hypotheses in total). Each hypothesis was separated into sub-hypothesis, separating time points, main psychological study variables from cognitive variables, and distinguishing between independent predictive relationships and mediating and moderating relationships. The total number of hypotheses was 69.

Hierarchical multiple regression analysis was the primary statistical method used to analyse the longitudinal data. Mediation analysis using the PROCESS macro was also used to determine significant mediating relationships. Moderating relationships were also analysed with the process MACRO, however no moderation was found.

For the Physical Recovery Model, the main variable identified which impacts upon physical recovery was stress. Stress at Time 2, predicted poorer recovery at

Time 2 and 3, and stress at Time 3 predicted poorer recovery at Time 3. This finding is valuable to stroke research as there are discrepancies in the literature regarding the relationship between stress and stroke.

Another valuable finding reported the relationship between repressive coping and physical recovery. This is the first known study investigating repressive coping in a stroke sample. Repressors at Time 2 predicted poorer recovery at Times 2 and 3.

Social support played a smaller role. This factor was only significant at Time 3, and reported that high social support at Time 3 was related to poorer recovery at Time 3.

Depression was expected to play a strong role in recovery from stroke. However this study reported depression was not an independent predictor of stroke recovery. Depression however, did exhibit mediating relationships, by mediating the relationship between Time 2 visual short term memory and Time 2 physical recovery. This relationship was also mirrored as Time 2 visual short term memory mediated the relationship between Time 2 depression and Time 2 physical recovery.

The main cognitive variable that was an independent predictor of physical recovery was Time 3 visual neglect which predicted Time 3 physical recovery. However, the remaining cognitive variables (verbal and visual short term memory) were not independent predictors.

In the Psychological Recovery Model, stress was again the main variable that had the most impact. Time 2 stress predicted both Times 2 and 3 QoL outcome, and Times 1, 2 and 3 stress predicted Time 3 QoL outcome. Again, this is a very valuable finding as stress and stroke literature is inconclusive.

Although depression was not an independent predictor for physical recovery, depression was reported as such for QoL, with Time 2 depression independently predicting Time 2 QoL outcome. However, depression was expected to be more prevalent than was reported.

Social support featured as an independent predictor only at Time 1, predicting QoL outcome at Time 2. Again, this variable was expected to be more present in the analysis.

Repressive coping was absent from this model. This could be because physical stroke severity was an observer rated measure and QoL was a self rated measure. Repressors tend to avoid negative affect and therefore on this self rated measure repressors would answer differently. However, on the observer rated measure (which the participant does not contribute to), significant results were reported.

Cognitive variables are not usually investigated with QoL, which is another strong point of this study. Again, the main cognitive variable that was predictive of outcome was visual neglect. In particular Time 1 and 3 was salient for outcome, with Time 2 not reporting significant levels. However, the remaining cognitive variables (verbal and visual short term memory) did not act as independent predictors.

In both models stroke severity and age were consistent important stroke marker and demographic factors. Additionally in both models Type D personality was absent. This was a surprising result as Type D personality has been found to be consistently related to cardiovascular disease.

In conclusion the current research study contributes to stroke research on many levels theoretically and methodologically. This study also highlights the versatility of Health Psychology in being able to incorporate Cognitive Neuropsychological methods.

Large theoretical frameworks encompassing these different types of variables do not currently exist. Existing frameworks such as the Main Effect Hypothesis and the Buffering Hypothesis have not been supported by the current research and therefore these findings cannot be contextualised within these frameworks. This thesis can offer an insight into constructing a new framework including previously absent variables such as repressive coping, visual neglect and visual memory. Additionally, diagrams have been constructed for the theoretical model results for physical recovery (Figure 8.1, p.338) and psychological recovery (Figure 8.2, p. 353).

Additionally the need for stroke research identified by the National Stroke Strategy (DoH, 2007) has been met as a longitudinal stroke study has been conducted investigating outcome and identifying areas which could be important for intervention studies: *"Estimation of the longer-term needs of patients (impairment,*

activity, participation, quality of life) at different time points post-stroke to help direct intervention studies to improve outcomes" (DoH, 2007, p.66).

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Stroke Data extraction form characteristics

Study name:
Author:
Year:
Country:

Study objectives	
main objective stated and specific aims that are relevant to this review	
METHODS	
Study design (observational, interventional, case series etc)	
▪ Population (define) ▪ numbers of patients ▪ Stroke status ▪ how patients accessed ▪ gender ▪ ethnicty ▪ setting	
Are patients undergoing an intervention? What type of treatment?	
measure of Stroke (define)	
Measure of Predictor (define)	
measure of Recovery (define) self completed/interview/clinical measure	
Psychological variables measured	
statistics (how relationship between predictor and recovery carried out)	
RESULTS	
Patients included in analysis (follow up available for)	
Correlation between variables - report statistics/confidence intervals etc	
Conclusions/NOTES	

National Research Ethics Service
Hammersmith and Queen Charlotte's & Chelsea Research Ethics Committee
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Ms Parminder Sonia Kaur Dhiman
1 Manor Waye
Uxbridge
Middlesex UB8 2BD

26 November 2009

Dear Ms Dhiman

Study Title:	**What is the association between psychosocial and cognitive factors and recovery from stroke?**
REC reference number:	**09/H0707/78**
Protocol number:	**3**

Thank you for your letter of 05 November 2009, responding to the Committee's request for further information on the above research and submitting revised documentation.

The further information has been considered on behalf of the Committee by the Vice-Chair.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised, subject to the conditions specified below.

Ethical review of research sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion" below).

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

For NHS research sites only, management permission for research ("R&D approval") should be obtained from the relevant care organisation(s) in accordance with NHS research governance arrangements. Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at http://www.rdforum.nhs.uk.
Where the only involvement of the NHS organisation is as a Participant Identification

Centre, management permission for research is not required but the R&D office should be notified of the study. Guidance should be sought from the R&D office where necessary.

Sponsors are not required to notify the Committee of approvals from host organisations.

It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document	*Version*	*Date*
Covering Letter		07 August 2009
Protocol	3	17 August 2009
REC application		20 August 2009
Questionnaire: "How you feel now"		
Questionnaire: "sense of coherence"		
Questionnaire: Sheffield screening test for acquired language disorders		
Questionnaire: multidimensional scale of perceived social support		
Questionnaire: perceived stress scale		
Questionnaire: CES-D 10		
Questionnaire: Marlow-Crowne social desirability scale - form B		
Questionnaire: DS 14		
Questionnaire: Digit span		
Questionnaire: Rivermead behavioural memory task		
Questionnaire: when/over/when/hard....Yellow/blue/green...		
Questionnaire: modified Rankin scale (MRS)		
Questionnaire: Health status questionnaire - SF-36		
Questionnaire: Physical outcomes measure		
Investigator CV	Dr Andrew Parton	
Evidence of insurance or indemnity	AON/Zurich Municipal	
Letter from Sponsor	Brunel University	20 August 2009
CV: Miss P Dhiman		17 August 2009
Stroke Support Groups	1	25 March 2009
Participant Information Sheet	4	03 November 2009
Participant Information Sheet: Interview Study	4	03 November 2009
Participant Information Sheet: Debriefing Sheet	2	25 March 2009
Participant Consent Form	2	03 November 2009
Participant Consent Form: Interview Study	2	03 November 2009
GP/Consultant Information Sheets	2	03 November 2009
Nurses Information Sheet	2	03 November 2009
Response to Request for Further Information		05 November 2009

National Research Ethics Service

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Now that you have completed the application process please visit the National Research Ethics Service website > After Review

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

The attached document *"After ethical review – guidance for researchers"* gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

We would also like to inform you that we consult regularly with stakeholders to improve our service. If you would like to join our Reference Group please email referencegroup@nres.npsa.nhs.uk.

09/H0707/78	**Please quote this number on all correspondence**

Yours sincerely

cc

Professor A George
Chair

Email: clive.collett@imperial.nhs.uk

Enclosures: "After ethical review – guidance for researchers"

Copy to: *Dr Bridget Dibb*

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What Psychosocial and Cognitive Factors Predict Recovery From Stroke?

Patient Information Sheet

I am inviting you to take part in a research study. Whether or not you take part is entirely your choice. Please ask any questions you want to about the research and I will try my best to answer them.

- **Why have I been asked to take part?**

I am doing research to find out how psychology can influence recovery from stroke and need some volunteers.

- **What is the goal of the research?**

The goal of the research is to find out if there is a relationship between psychological factors (how we think and feel) and recovery from stroke.

- **How long will this take?**

If you are willing to help, there are 8 questionnaires in total but they are not done all at once. You can fill these out yourself or I can read them out to you. It will take between 20 - 40 minutes. It is not a test, there are no right or wrong answers. I will also give you some cognitive assessment, which will take no longer than 15 minutes. We can split these in two sections so you do not get tired.

I will ask you to fill in the same questionnaires and take part in the same simple tasks 6 months later and again 3 months later.

- **Can I stop taking part, after I start?**

You can stop taking part at any time. This is voluntary research. If you stop taking part I would still like to use the information you have already provided me with, with your permission.

If you lose the capacity to consent during the study, you will be withdrawn and the information collected will still be continued to be used.

- **Who will benefit from this?**

If we identify psychological factors (for example, thoughts and feelings) which influence patients recovery from stroke, these findings can develop interventions to try and improve peoples recovery after stroke.

- **Where will this take place?**

These meetings will take place at the hospital, later on in your home or at Brunel University. You will be reimbursed for any travel costs.

- **Who will see my answers?**

You will not put your name on the questionnaires. All the answers you give will be *confidential*. You will not be able to be identified as no personal details will be collected on the questionnaires and all questionnaires will be coded. Only I will know the code.

- **Will anyone look at my medical notes?**

Yes, I will look at your medical notes only to get some basic information.

- **What procedures will be in place to detect and compensate for any possible "researcher effects" and "researcher bias"?**

As I will not know anyone before the study and I am not involved in your treatment, it will be unlikely that there will be any researcher bias. I am not from the NHS, I am from Brunel University. The answers you give me will not effect your treatment in any way and will be kept confidential.

- **Are there any risks or burdens for me?**

There will be no risks or burdens for you.

- **What assessment has this research gone through?**

Brunel University's Ethics Committee has reviewed this research proposal and has given clearance to proceed. Also the NHS Ethics Committee have also thoroughly assessed this research and has given approval.

- **Who should I get in contact with, should I have any questions?**

Contact details for Parminder Sonia Dhiman are at the top of this information sheet.

- **Who should I get in contact with, should I have any complaints?**

You can contact Professor David Bunce on 01895 267242 or david.bunce@brunel.ac.uk.

Department of Psychology
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What psychosocial and cognitive factors predict recovery from stroke?

Consent Form

Researcher: Parminder Sonia Dhiman

Code……..

Please tick the appropriate boxes:

- The study organiser has invited me to take part in this research. ☐
- I understand what is in the Patient Information Sheet. ☐
- I have had the chance to ask questions about the study. ☐
- I know the questionnaires will last approximately 40 minutes and the simple tasks will last approximately 30 minutes. ☐
- I know that the NHS Ethics & Brunel University's Ethics Committee has agreed to this study. ☐
- I understand that my information is strictly confidential. ☐
- I know that my name will be kept separate from my questionnaire. ☐
- I freely consent to be a participant in the study. ☐
- I know that I can stop taking part at any time. ☐
- I know my signature is not a waiver of any legal rights. ☐
- I understand that I will be able to keep a copy of the informed consent form for my records. ☐

Signature .....

Date .....

……

The following should be signed by the Investigator responsible for obtaining consent

As the Investigator responsible for this research, I confirm that I have explained to the volunteer named above the nature and purpose of the research to be undertaken.

Researcher's Name: .....

Researcher's Signature: ..... Date:

1 copy for patient; 1 copy for researcher; 1 copy to be kept with hospital notes

Version 1. 25/3/09

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What Psychosocial and Cognitive Factors Predict Recovery From Stroke?

Debriefing Sheet

This study was done to investigate if how we think and feel e.g., things such as stress, social support, depression, coping, personality, memory, language and thinking can predict the rate of physical and psychological recovery from stroke.

The following is a brief description to explain what was measured:

Social Support is the support we get from our social relationships. You answered a questionnaire about social support in 3 areas (family, friends and significant others).

Stress was asked about to see if you have felt stressed in the previous month.

Coping was asked about to see how people generally respond when they are confronted with stressful or difficult events in their lives. You were also asked some questions on repressive coping which is a coping style in which people report low signs of distress when they are stressed.

You were also asked questions about a personality type called Type D Personality, which is a personality style in which people experience negative emotions and hide the expression of these emotions in social situations.

Depression was looked at to see if you have had any depressive feelings during the previous week.

You also did some simple tests. In the first test you were asked to circle on a piece of paper bells and in the second test, you were asked to mark the centre of a long line. These tests were done to see if your vision was affected by the stroke. Memory was tested, by asking you to remember some numbers and pictures. You also did a colour naming task which tests when your brain does two things at once.

I also looked at how well you physically function by observing you and looking at your clinical notes. You were also asked to answer a questionnaire, which asked about your physical and mental quality of life.

The results of this study will remain confidential. If you would like to know the outcome of this investigation, please do not hesitate to contact me; my details are at the top of the page.

I would like to thank you for taking part in my research. Your time, effort and contribution has been greatly received.

Perceived Stress Scale

Instructions:
The questions in this scale ask you about your feelings and thoughts during the last month. In each case you will be asked to indicate how often you felt or thought a certain way. Although some of the questions are similar there are differences between them and you should treat each one as a separate question. The best approach is to answer each question fairly quickly. That is, don't try to count up the number of times you felt a particular way, but rather indicate the alternative that seems like a reasonable estimate.

For each question choose from the following alternatives:

0 = never
1 = almost never
2 = sometimes
3 = fairly often
4 = very often

1. In the last month, how often have you been upset because of something that happened unexpectedly?	
2. In the last month, how often have you felt that you were unable to control the important things in your life?	
3. In the last month, how often have you felt nervous and stressed?	
4. In the last month, how often have you dealt with irritating life hassles?	
5. In the last month, how often have you felt like you were effectively coping with important changes that were occurring in your life?	
6. In the last month, how often have you felt confident about your ability to handle your personal problems?	
7. In the last month, how often have you felt that things were going your way?	
8. In the last month, how often have you found that you could not cope with all the things you had to do?	
9. In the last month, how often have you been able to control the irritations in your life?	
10. In the last month, how often have you felt that you were on top of things?	
11. In the last month, how often have you been angered because of things that happened that were outside of your control?	
12. In the last month, how often have you found yourself thinking about things you have to accomplish?	
13. In the last month, how often have you been able to control the way you spend you time?	
14. In the last month, how often have you felt difficulties were piling so high up that you could not overcome them?	

Thank you this is the end of the Perceived Stress Scale

Center for Epidemiologic Studies Short Depression Scale (CES-D 10)

Below is a list of some of the ways you may have felt or behaved. Please indicate how often you have felt this way during the **past week**: *(circle **one** number on each line)*

During the past week...	**Rarely or none of the time** (less than 1 day)	**Some or a little of the time** (1-2 days)	**Occasionally or a moderate amount of time** (3-4 days)	**All of the time** (5-7days)
1. I was bothered by things that usually don't bother me	0	1	2	3
2. I had trouble keeping my mind on what I was doing	0	1	2	3
3. I felt depressed	0	1	2	3
4. I felt that everything I did was an effort	0	1	2	3
5. I felt hopeful about the future	0	1	2	3
6. I felt fearful	0	1	2	3
7. My sleep was restless	0	1	2	3
8. I was happy	0	1	2	3
9. I felt lonely	0	1	2	3
10. I could not "get going"	0	1	2	3

Multidimensional Scale of Perceived Social Support (Zimet, Dahlem, Zimet & Farley, 1988)

Instructions: We are interested in how you feel about the following statements. Read each statei carefully. Indicate how you feel about each statement.

Circle the "1" if you **Very Strongly Disagree**
Circle the "2" if you **Strongly Disagree**
Circle the "3" if you **Mildly Disagree**
Circle the "4" if you are **Neutral**
Circle the "5" if you **Mildly Agree**
Circle the "6" if you **Strongly Agree**
Circle the "7" if you **Very Strongly Agree**

1.	There is a special person who is around when I am in need.	1	2	3	4	5	6	7
2.	There is a special person with whom I can share my joys and sorrows.	1	2	3	4	5	6	7
3.	My family really tries to help me.	1	2	3	4	5	6	7
4.	I get the emotional help and support I need from my family.	1	2	3	4	5	6	7
5.	I have a special person who is a real source of comfort to me.	1	2	3	4	5	6	7
6.	My friends really try to help me.	1	2	3	4	5	6	7
7.	I can count on my friends when things go wrong.	1	2	3	4	5	6	7
8.	I can talk about my problems with my family.	1	2	3	4	5	6	7
9.	I have friends with whom I can share my joys and sorrows.	1	2	3	4	5	6	7
10.	There is a special person in my life who cares about my feelings.	1	2	3	4	5	6	7
11.	My family is willing to help me make decisions.	1	2	3	4	5	6	7
12.	I can talk about my problems with my friends.	1	2	3	4	5	6	7

Marlowe-Crowne Social Desirability Scale (Form B)

Listed below are a number of statements concerning personal attitudes and traits. Please read each item and decide whether the statement is true or false as it applies to you. For each item, please circle TRUE or FALSE.

.....

1. It is sometimes hard for me to go on with my work if I am not encouraged. TRUE or FALSE

.....

2. I sometimes feel resentful when I don't get my way. TRUE or FALSE

.....

3. There have been times when I felt like rebelling against people in authority, even though I knew they were right. TRUE or FALSE

.....

4. No matter who I'm talking to, I'm always a good listener. TRUE or FALSE

.....

5. There have been occasions when I took advantage of someone. TRUE or FALSE

.....

6. I am always willing to admit when I made a mistake. TRUE or FALSE

.....

7. I sometimes try to get even rather than forgive and forget. TRUE or FALSE

.....

8. I am always courteous, even to people who are disagreeable. TRUE or FALSE

.....

9. I have never been irked when people expressed ideas very different from my own. TRUE or FALSE

.....

10. There have been times when I was quite jealous of the good fortune of others. TRUE or FALSE

.....

11. I am sometimes irritated by people who ask favours of me. TRUE or FALSE

.....

12. I have never deliberately said something that hurt someone's feelings. TRUE or FALSE

.....

HOW YOU FEEL NOW

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the most appropriate answer for each statement to indicate how you feel **right now**, at this moment. There are no right or wrong answers. Don't spend too much time on any one statement but give the answer which seems to describe your present feelings best.

1) I feel calm

not at all *somewhat* *moderately* *very much*

2) I am tense

not at all *somewhat* *moderately* *very much*

3) I feel upset

not at all *somewhat* *moderately* *very much*

4) I am relaxed

not at all *somewhat* *moderately* *very much*

5) I feel content

not at all *somewhat* *moderately* *very much*

6) I am worried

not at all *somewhat* *moderately* *very much*

DS 14

Code……

Below are a number of statements that people often use to describe themselves. Please read each statement and then circle the appropriate number next to that statement to indicate your answer. There are no right or wrong answers: Your own impression is the only thing that matters.

0 = FALSE
1 = RATHER FALSE
2 = NEUTRAL
3 = RATHER TRUE
4 = TRUE

1) I make contact easily when I meet people ……..…….0 1 2 3 4
2) I often make a fuss about unimportant things……..…….……..0 1 2 3 4
3) I often talk to strangers…….…….……..0 1 2 3 4
4) I often feel unhappy…….……0 1 2 3 4
5) I am often irritated…….……..0 1 2 3 4
6) I often feel inhibited in social interactions…….……0 1 2 3 4
7) I take a gloomy view of things…….……...0 1 2 3 4
8) I find it hard to start a conversation…….……0 1 2 3 4
9) I am often in a bad mood…….……0 1 2 3 4
10) I am a closed kind of person…….……...0 1 2 3 4
11) I would rather keep other people at a distance…….…...0 1 2 3 4
12) I often find myself worrying about something…….…..0 1 2 3 4
13) I am often down in the dumps…….…0 1 2 3 4
14) When socialising, I don't find the right things to talk about……0 1 2 3 4

Sense of Coherence

1) Do you usually feel that the things that happen to you in your daily life are hard to understand?

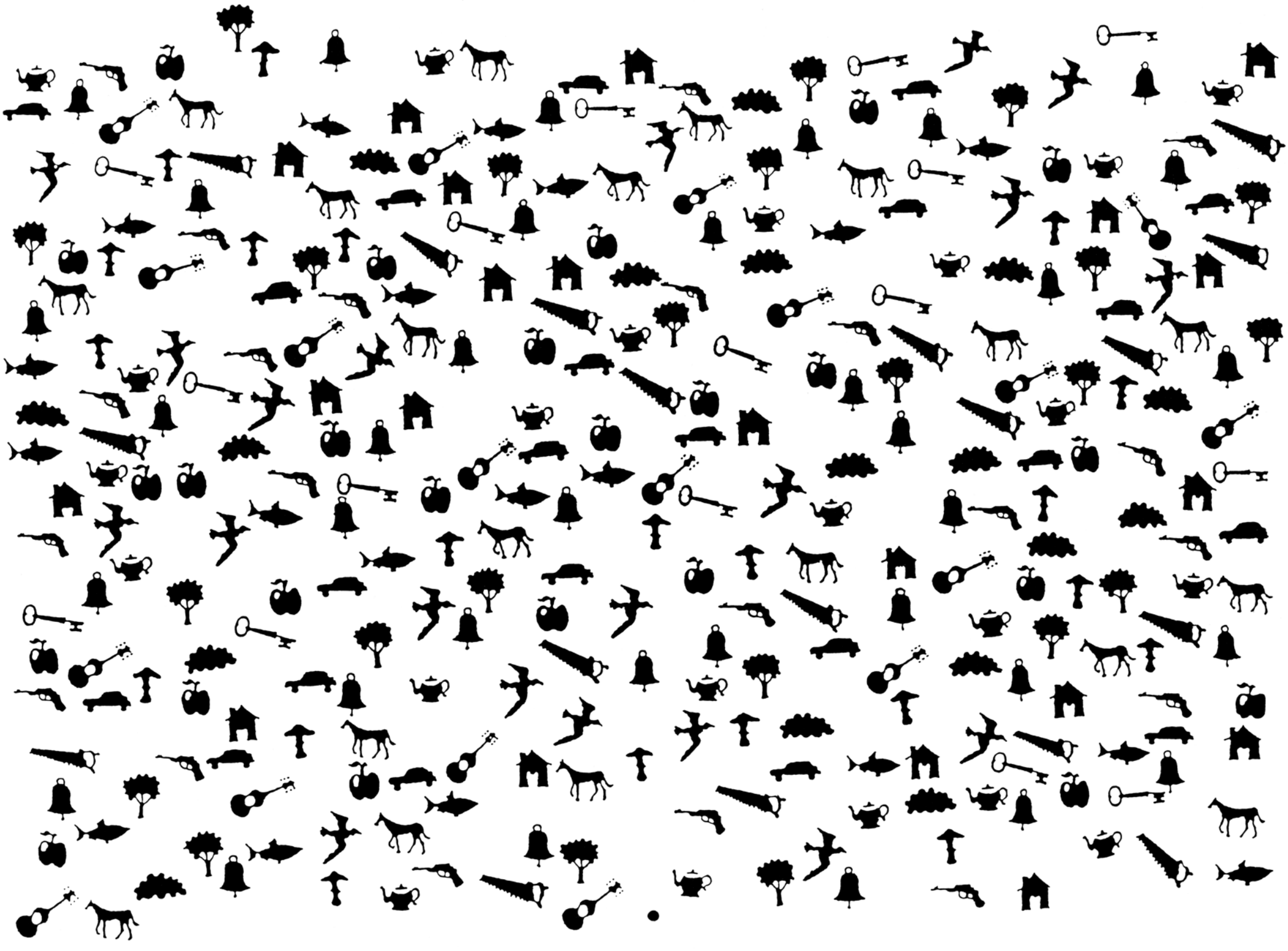
0	1	2
Yes	Sometimes	No

2) Do you usually see a solution to problems and difficulties that other people find hopeless?

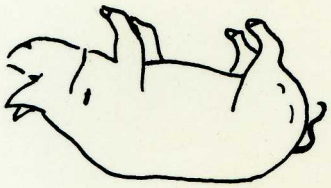
0	1	2
Yes	Sometimes	No

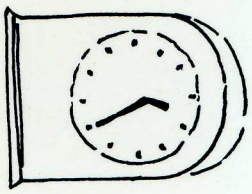
3) Do you usually feel that your daily life is a source of personal satisfaction?

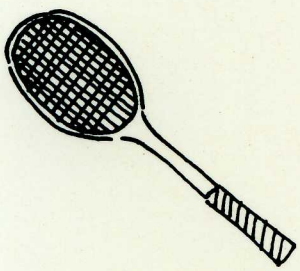
0	1	2
Yes	Sometimes	No

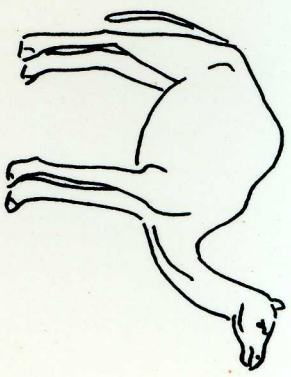


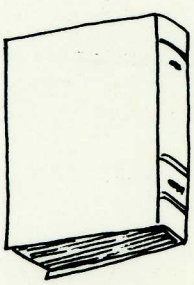
Item	Trial	
START 1.	Trial 1	1 – 7
	Trial 2	6 – 3
2.	Trial 1	5 – 8 – 2
	Trial 2	6 – 9 – 4
3.	Trial 1	6 – 4 – 3 – 9
	Trial 2	7 – 2 – 8 – 6
4.	Trial 1	4 – 2 – 7 – 3 – 1
	Trial 2	7 – 5 – 8 – 3 – 6
5.	Trial 1	6 – 1 – 9 – 4 – 7 – 3
	Trial 2	3 – 9 – 2 – 4 – 8 – 7
6.	Trial 1	5 – 9 – 1 – 7 – 4 – 2 – 8
	Trial 2	4 – 1 – 7 – 9 – 3 – 8 – 6
7.	Trial 1	5 – 8 – 1 – 9 – 2 – 6 – 4 – 7
	Trial 2	3 – 8 – 2 – 9 – 5 – 1 – 7 – 4
8.	Trial 1	2 – 7 – 5 – 8 – 6 – 2 – 5 – 8 – 4
	Trial 2	7 – 1 – 3 – 9 – 4 – 2 – 5 – 6 – 8

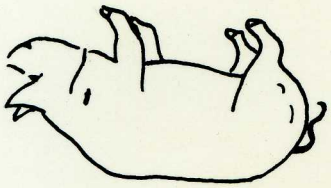


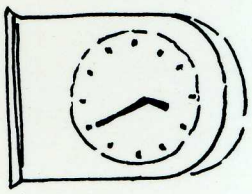


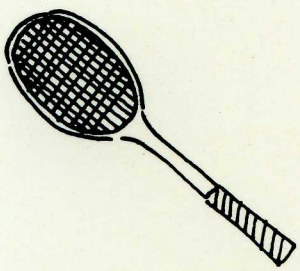


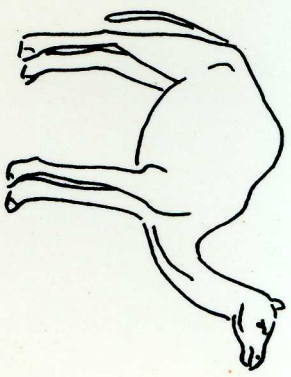


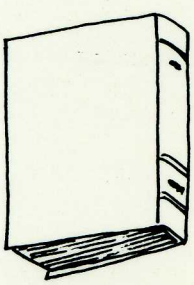


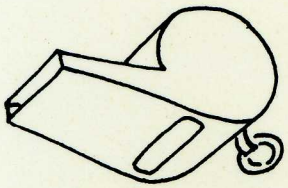


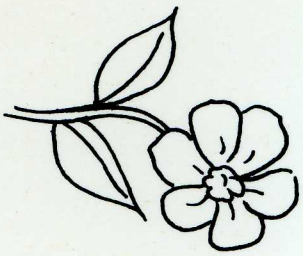


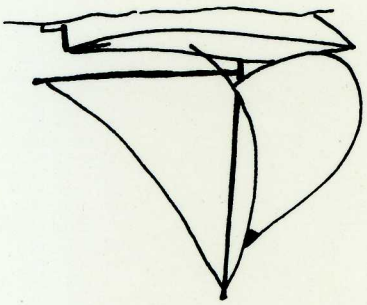


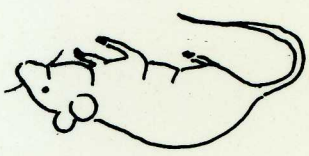


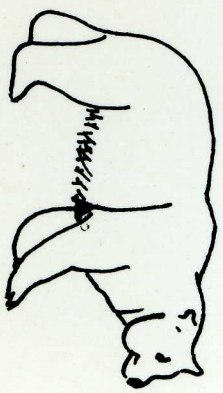


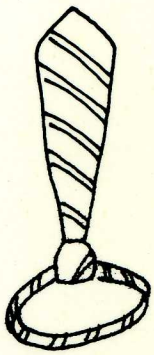


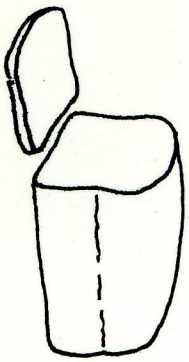


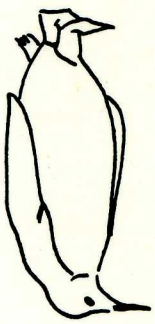


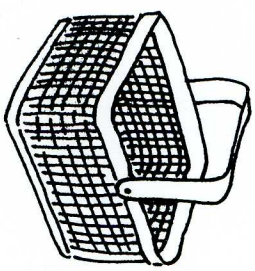


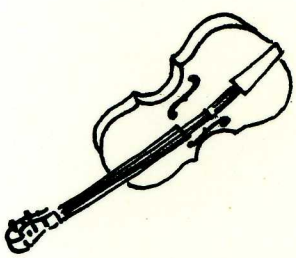












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HEALTH STATUS QUESTIONNAIRE (SF-36)

INSTRUCTIONS: This survey asks about your views about your health. This information will keep track of how you feel and how well you are able to do your usual activities. Answer every question by circling the number. 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?

Excellent.....	1
Very good.....	2
Good.....	3
Fair.....	4
Poor.....	5

---

2. **Compared to one year ago,** how would you rate your health in general **now**?

Much better than 1 year ago.....	1
Somewhat better than 1 year ago.....	2
About the same as 1 year ago	3
Somewhat worse now than 1 year ago.....	4
Much worse now than 1 year ago.....	5

---

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?
(Circle one number on each line)

	Yes, Limited A Lot	Yes, Limited A Little	No, Not Limited At All
a. Vigorous activities, such as running, lifting heavy objects, participating in strenuous sports....	1	2	3
b. Moderate activities, such as moving a table, pushing a vacuum cleaner, bowling or playing golf.....	1	2	3
c. Lifting or carrying groceries.....	1	2	3
d. Climbing several flights of stairs	1	2	3
e. Climbing one flight of stairs.....	1	2	3
f. Bending, kneeling or stooping.....	1	2	3

CONTINUED OVER PAGE

	Yes Limited A Lot	Yes, Limited A Little	No, Not Limited At All
g. Walking more than a mile.....	1	2	3
h. Walking half a mile.....	1	2	3
i. Walking one hundred yards.....	1	2	3
j. Bathing or dressing yourself.....	1	2	3

---

4. During the **past four weeks,** have you had any of the following problems in your work or other regular daily activities as a result of your physical health (Circle one number on each line)

	YES	NO
a. Cut down on the amount of time you spent on work or other activities.....	1	2
b. Accomplished less than you would like.....	1	2
c. Were limited in the kind of work and other activities.....	1	2
d. Had difficulty performing the work and other activities (for example it took extra effort).....	1	2

---

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)?

	YES	NO
a. Cut down the amount of time you spent on work or other activities.....	1	2
b. Accomplished less than you would like.....	1	2
c. Didn't do work or other activities as carefully as usual.....	1	2

---

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? (Circle one number)

1. Not at all 2. Slightly 3. Moderately

4. Quite a bit 5. Extremely

---

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

1. None 2. Very mild 3. Mild

4. Moderate 5. Severe 6. Very severe

---

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

1. Not at all 2. Slightly 3.Moderately

4. Quite a bit 5. Extremely

---

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

	1 All of the time	2 Most of the time	3 A good bit of the time	4 Some of the time	5 A little of the time	6 None of the time
a. Did you feel full of life?.....1		2	3	4	5	6
b. Have you been a very nervous person?.....1		2	3	4	5	6
c. Have you felt so down in the dumps nothing could cheer you up?.....1		2	3	4	5	6
d. Have you felt calm and peaceful?.....1		2	3	4	5	6
e. Did you have a lot of energy?.....1		2	3	4	5	6
f. Have you felt downhearted and low?.....1		2	3	4	5	6
g. Did you feel worn out?.....1		2	3	4	5	6
h. Have you been a happy person?.....1		2	3	4	5	6
i. Did you feel tired?.....1		2	3	4	5	6
j. Has your health limited your social activities?1		2	3	4	5	6

---

10. How TRUE or FALSE is each of the following statements of you?

	Definitely True	Mostly True	Not Sure	Mostly False	Definitely False
I seem to get ill more easily than other people	1	2	3	4	5
I am as healthy as anybody I know.....	1	2	3	4	5
I expect my health to get worse.....	1	2	3	4	5
My health is excellent.....	1	2	3	4	5

(U.K. Standard SF-36)

MODIFIED RANKIN SCALE (MRS)

Patient Name: _____

Rater Name: _____

Date: _____

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

TOTAL (0–6): _____

References

Rankin J. "Cerebral vascular accidents in patients over the age of 60."
Scott Med J 1957;2:200-15

Bonita R, Beaglehole R. "Modification of Rankin Scale: Recovery of motor function after stroke."
***Stroke* 1988 Dec;19(12):1497-1500**

Van Swieten JC, Koudstaal PJ, Visser MC, Schouten HJ, van Gijn J. "Interobserver agreement for the assessment of handicap in stroke patients."
***Stroke* 1988;19(5):604-7**

Table T:
Summary table, showing details of the 3 review studies from the update 2009-April 2013

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
22b. Seale et al. (2010) # USA.	840 participants (48.3% male, 51.7 % female). 70% ischemic strokes were recorded.	Longitudinal. Data collected within 24 hours of admission (T1), within 72 hours of discharge (T2) and 3 months post stroke T3).	Rehabilitation.	1. Four positive questions from the Centre For Epidemiologic Studies Depression Scale (CES D) – Positive emotion	An increase in positive emotion is associated with an increase in functional recovery. However, the authors also state change in positive emotion accounts for 2% of the variance in functional recovery.
24. Donnellan et al. (2010) Ireland.	153 participants (55% male). 102 Ischemic and 5 hemorrhagic strokes. 41 left hemisphere , 55 right hemisphere and 11	Longitudinal. Data collected at 1 month post stroke (T1) and 1 year post stroke (T2).	Rehabilitation.	1. Socio-demographic data 2. Clinical background 3. Hospital Anxiety & Depression Scale	Depression is an independent predictor of functional ability. Anxiety is not a predictor.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
	cerebellum/brain stem.			4. Stroke-Specific Quality of Life Scale (SSQoL) 5. Orpington Prognostic Score – stroke severity 6. Nottingham Extended Activities of Daily Living (NEADL)	
25. West et al. (2010) UK.	449 participants (253 male, 191 female). Stroke type not specified	Longitudinal. Data collected at 2-6 post stroke (T1), 6-10 weeks post stroke (T2), 12-14 weeks post stroke (T3), 24-26 weeks post stroke (T4)	Rehabilitation.	1. Mini Mental State Examination (MMSE) 2. Socio-demographic data 3. Clinical & Functioning data	Persistent psychological symptoms in the first 26 weeks after stroke are associated with a decrease in physical function at 52 weeks.

AUTHOR	NO. & TYPE OF PARTICIPANTS	STUDY DESIGN	INTERVENTION	MEASURES	OUTCOME
		and 52 weeks post stroke (T5).		4. Duke Severity Illness Scale – Comorbidity 5. General Health Questionnaire (GHQ) 6. Present State Examination – Depressed mood 7. Barthel Index (BI)	

Studies that share the same population: # Ostir et al. (2008) & Seale et al. (2010)

Table U:
Methodological Assessment of the Review Papers

STUDY	Sample Characteristics (Age, Gender, Ethnicity)	Stroke Diagnosis	Stroke Types	Length of Study	Time Points	Measurement of Psychological Variables	Measurement of Outcome Variables	Method of Analysis	Missing Data	Sample Size & Power	Overall Methodological Rating
GOOD											
1. Donnellan et al. (2010)	Poor	Good	Poor	Good	Good	Good	Good	Good	Good	Inter	15
INTERMEDIATE											
2. Seale et al. (2010) #	Inter	Good	Inter	Inter	Good	Inter	Inter	Good	Inter	Inter	13
3. West et al. (2010)	Inter	Poor	Poor	Inter	Good	Good	Good	Poor	Good	Inter	11

Studies that share the same population: * Schubert et al. (1992a & 1992b); ^ Johnston et al. (1999 & 2004); # Ostir et al. (2008) & Seale et al. (2010)

APPENIDIX U: Statistical Output

```
REGRESSION
  /DESCRIPTIVES MEAN STDDEV CORR SIG N
  /MISSING LISTWISE
  /STATISTICS COEFF OUTS R ANOVA COLLIN TOL CHANGE ZPP
  /CRITERIA=PIN(.05) POUT(.10)
  /NOORIGIN
  /DEPENDENT T2Rankin
  /METHOD=ENTER Age DV_StrokeSev_MildVSMod DV_StrokeSev_MildVSSev
  /METHOD=ENTER T2StressOverall T2RepCop
  /SCATTERPLOT=(*ZRESID ,*ZPRED)
  /RESIDUALS NORMPROB(ZRESID)
  /SAVE MAHAL COOK.
```

Regression

Notes

Output Created		24-FEB-2015 17:23:14
Comments		
Input	Data	H:\StrokeData_1.sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	143
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on cases with no missing values for any variable used.

Syntax		REGRESSION /DESCRIPTIVES MEAN STDDEV CORR SIG N /MISSING LISTWISE /STATISTICS COEFF OUTS R ANOVA COLLIN TOL CHANGE ZPP /CRITERIA=PIN(.05) POUT(.10) /NOORIGIN /DEPENDENT T2Rankin /METHOD=ENTER Age DV_StrokeSev_MildVSMod DV_StrokeSev_MildVSSev /METHOD=ENTER T2StressOverall T2RepCop /SCATTERPLOT=(*ZRESID ,*ZPRED) /RESIDUALS NORMPROB(ZRESID) /SAVE MAHAL COOK.
Resources	Processor Time	00:00:00.39
	Elapsed Time	00:00:03.69
	Memory Required	19220 bytes
	Additional Memory Required for Residual Plots	536 bytes
Variables Created or Modified	MAH_3	Mahalanobis Distance
	COO_3	Cook's Distance

[DataSet1] H:\StrokeData_1.sav

Descriptive Statistics

	Mean	Std. Deviation	N
T2Rankin	2.5327	1.04897	107
Age	67.9346	14.52147	107
DV_StrokeSev_MildVSMod	.4299	.49739	107
DV_StrokeSev_MildVSSev	.2056	.40605	107
T2StressOverall	1.5888	.74682	107

T2RepCop	.3925	.49061	107

Correlations

		T2Rankin	Age	DV_StrokeSev_MildVSMod	DV_StrokeSev_MildVSSev	T2StressOverall	T2RepCop
Pearson Correlation	T2Rankin	1.000	.212	.262	.405	.364	.085
	Age	.212	1.000	.027	.138	.053	.084
	DV_StrokeSev_MildVSMod	.262	.027	1.000	-.442	.067	-.002
	DV_StrokeSev_MildVSSev	.405	.138	-.442	1.000	.113	.017
	T2StressOverall	.364	.053	.067	.113	1.000	-.416
	T2RepCop	.085	.084	-.002	.017	-.416	1.000
Sig. (1-tailed)	T2Rankin	.	.014	.003	.000	.000	.193
	Age	.014	.	.390	.078	.295	.194
	DV_StrokeSev_MildVSMod	.003	.390	.	.000	.247	.491
	DV_StrokeSev_MildVSSev	.000	.078	.000	.	.124	.430
	T2StressOverall	.000	.295	.247	.124	.	.000
	T2RepCop	.193	.194	.491	.430	.000	.
N	T2Rankin	107	107	107	107	107	107
	Age	107	107	107	107	107	107
	DV_StrokeSev_MildVSMod	107	107	107	107	107	107
	DV_StrokeSev_MildVSSev	107	107	107	107	107	107
	T2StressOverall	107	107	107	107	107	107
	T2RepCop	107	107	107	107	107	107

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method

1	DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod[b]	.	Enter
2	T2RepCop, T2StressOverall[b]	.	Enter

a. Dependent Variable: T2Rankin

b. All requested variables entered.

Model Summary[c]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.646[a]	.418	.401	.81216	.418	24.609	3	103	.000
2	.722[b]	.521	.497	.74394	.103	10.877	2	101	.000

a. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

b. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T2RepCop, T2StressOverall

c. Dependent Variable: T2Rankin

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	48.697	3	16.232	24.609	.000[b]
	Residual	67.939	103	.660		
	Total	116.636	106			
2	Regression	60.737	5	12.147	21.948	.000[c]
	Residual	55.899	101	.553		
	Total	116.636	106			

a. Dependent Variable: T2Rankin

b. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

c. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T2RepCop, T2StressOverall

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Correlations			Collinearity Statistics	
		B	Std. Error	Beta			Zero-order	Partial	Part	Tolerance	VIF
1	(Constant)	1.170	.382		3.062	.003					
	Age	.008	.006	.111	1.454	.149	.212	.142	.109	.971	1.030
	DV_StrokeSev_MildVSMod	1.130	.178	.536	6.359	.000	.262	.531	.478	.797	1.255
	DV_StrokeSev_MildVSSev	1.618	.220	.626	7.365	.000	.405	.587	.554	.782	1.278
2	(Constant)	.410	.386		1.062	.291					
	Age	.006	.005	.084	1.195	.235	.212	.118	.082	.960	1.041
	DV_StrokeSev_MildVSMod	1.025	.164	.486	6.236	.000	.262	.527	.430	.781	1.280
	DV_StrokeSev_MildVSSev	1.458	.204	.564	7.142	.000	.405	.579	.492	.759	1.317
	T2StressOverall	.496	.109	.353	4.565	.000	.364	.414	.314	.792	1.263
	T2RepCop	.462	.164	.216	2.823	.006	.085	.270	.194	.810	1.234

a. Dependent Variable: T2Rankin

Excluded Variables[a]

Model	Beta In	t	Sig.	Partial Correlation	Collinearity Statistics		
					Tolerance	VIF	Minimum Tolerance

1	T2StressOverall	.260[b]	3.592	.001	.335	.970	1.031	.764
	T2RepCop	.066[b]	.877	.383	.087	.993	1.007	.782

a. Dependent Variable: T2Rankin

b. Predictors in the Model: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

Collinearity Diagnostics[a]

Model	Dimension	Eigenvalue	Condition Index	Variance Proportions					
				(Constant)	Age	DV_StrokeSev_MildVSMod	DV_StrokeSev_MildVSSev	T2StressOverall	T2RepCop
1	1	2.718	1.000	.01	.01	.03	.02		
	2	1.000	1.649	.00	.00	.15	.41		
	3	.260	3.236	.02	.02	.81	.56		
	4	.022	11.189	.97	.97	.00	.00		
2	1	3.977	1.000	.00	.00	.01	.01	.01	.01
	2	1.000	1.994	.00	.00	.15	.40	.00	.00
	3	.633	2.507	.00	.00	.01	.01	.03	.64
	4	.279	3.777	.01	.01	.82	.57	.05	.00
	5	.091	6.624	.05	.12	.00	.00	.85	.34
	6	.021	13.872	.94	.86	.00	.01	.06	.01

a. Dependent Variable: T2Rankin

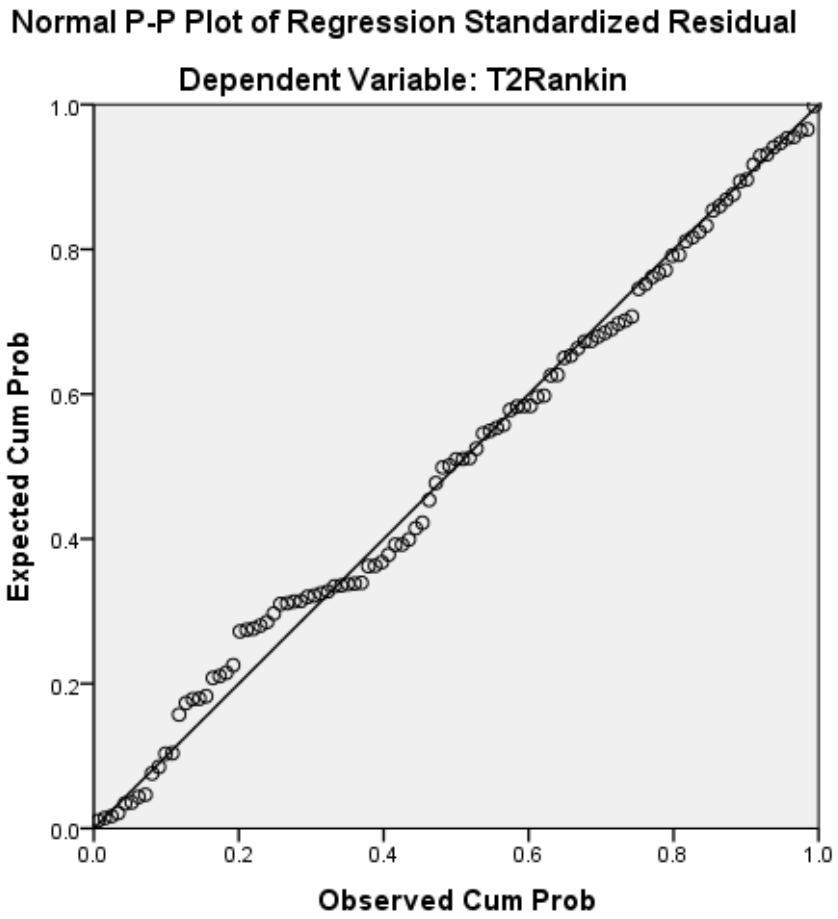
Residuals Statistics[a]

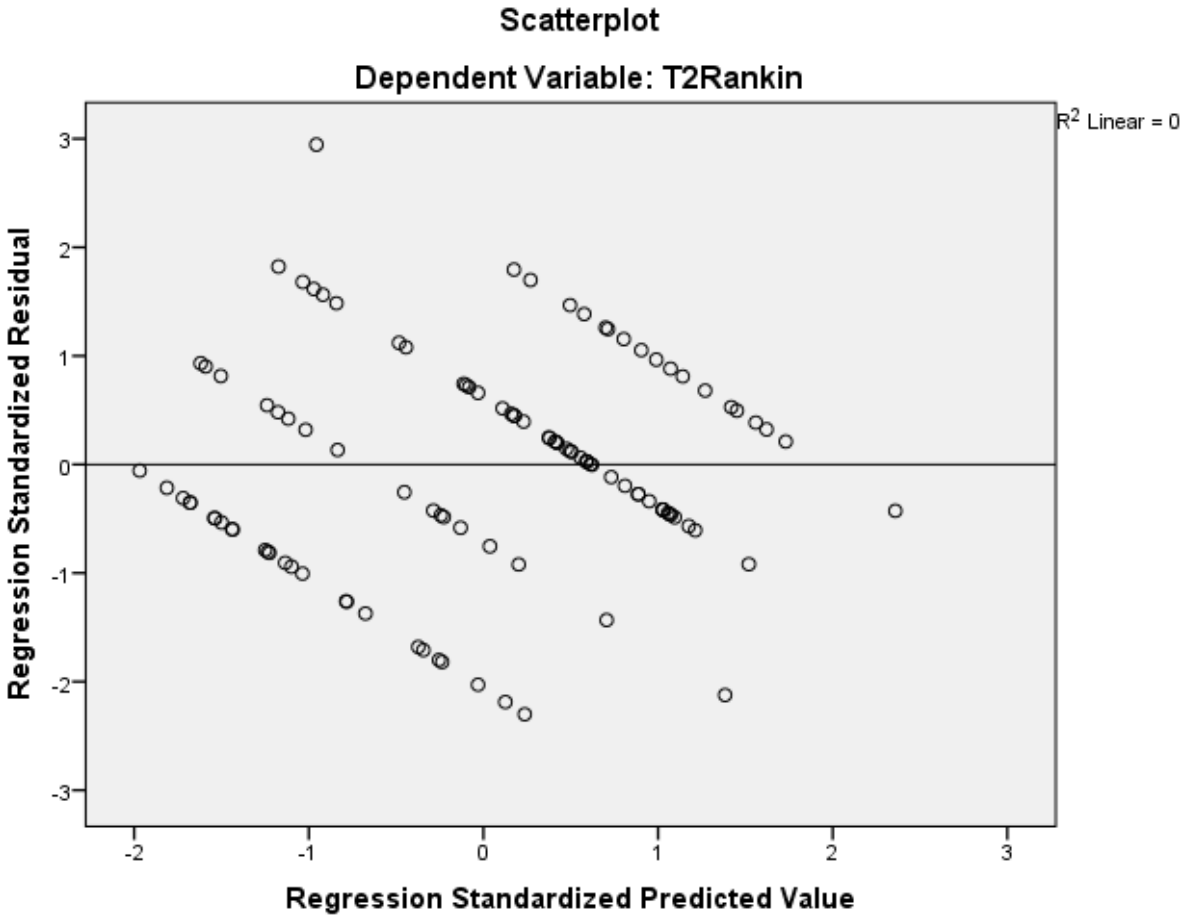
	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	1.0428	4.3180	2.5327	.75696	107
Std. Predicted Value	-1.968	2.358	.000	1.000	107
Standard Error of Predicted Value	.129	.326	.174	.029	107
Adjusted Predicted Value	1.0461	4.3604	2.5332	.75655	107
Residual	-1.71168	2.19154	.00000	.72619	107
Std. Residual	-2.301	2.946	.000	.976	107
Stud. Residual	-2.340	3.019	.000	1.004	107
Deleted Residual	-1.76973	2.30151	-.00046	.76776	107
Stud. Deleted Residual	-2.394	3.149	.000	1.015	107
Mahal. Distance	2.192	19.382	4.953	2.257	107
Cook's Distance	.000	.076	.010	.014	107

Centered Leverage Value	.021	.183	.047	.021	107

a. Dependent Variable: T2Rankin

Charts



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  /MISSING LISTWISE
  /STATISTICS COEFF OUTS R ANOVA COLLIN TOL CHANGE ZPP
  /CRITERIA=PIN(.05) POUT(.10)
  /NOORIGIN
  /DEPENDENT T3Rankin
  /METHOD=ENTER Age DV_StrokeSev_MildVSMod DV_StrokeSev_MildVSSev
  /METHOD=ENTER T2StressOverall T2RepCop
  /METHOD=ENTER T3StressOverall T3SocSupportOverall
  /SCATTERPLOT=(*ZRESID ,*ZPRED)
  /RESIDUALS NORMPROB(ZRESID)
  /SAVE MAHAL COOK.
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Regression

Notes

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Comments		
Input	Data	H:\StrokeData_1.sav
	Active Dataset	DataSet1
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	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	143
	Definition of Missing	User-defined missing values are treated as missing.
Missing Value Handling	Cases Used	Statistics are based on cases with no missing values for any variable used.

Syntax		REGRESSION /DESCRIPTIVES MEAN STDDEV CORR SIG N /MISSING LISTWISE /STATISTICS COEFF OUTS R ANOVA COLLIN TOL CHANGE ZPP /CRITERIA=PIN(.05) POUT(.10) /NOORIGIN /DEPENDENT T3Rankin /METHOD=ENTER Age DV_StrokeSev_MildVSMod DV_StrokeSev_MildVSSev /METHOD=ENTER T2StressOverall T2RepCop /METHOD=ENTER T3StressOverall T3SocSupportOverall /SCATTERPLOT=(*ZRESID,*ZPRED) /RESIDUALS NORMPROB(ZRESID) /SAVE MAHAL COOK.
Resources	Processor Time	00:00:00.22
	Elapsed Time	00:00:02.36
	Memory Required	20300 bytes
	Additional Memory Required for Residual Plots	520 bytes
Variables Created or Modified	MAH_8	Mahalanobis Distance
	COO_8	Cook's Distance

[DataSet1] H:\StrokeData_1.sav

Descriptive Statistics

	Mean	Std. Deviation	N
T3Rankin	2.4848	.94073	99
Age	67.5556	14.46193	99
DV_StrokeSev_MildVSMod	.4343	.49819	99
DV_StrokeSev_MildVSSev	.2121	.41089	99
T2StressOverall	1.5859	.74020	99
T2RepCop	.4040	.49320	99
T3StressOverall	1.5418	.71144	99
T3SocSupportOverall	5.3207	1.40660	99

Correlations

		T3Rankin	Age	DV_StrokeSev_MildVSMod	DV_StrokeSev_MildVSSev	T2StressOverall	T2RepCop	T3StressOverall	T3SocSupportOverall
Pearson Correlation	T3Rankin	1.000	.391	.177	.470	.249	.101	.380	.062
	Age	.391	1.000	.034	.198	.062	.057	.040	-.212
	DV_StrokeSev_MildVSMod	.177	.034	1.000	-.455	.018	-.016	-.015	.004
	DV_StrokeSev_MildVSSev	.470	.198	-.455	1.000	.141	.026	.224	-.037
	T2StressOverall	.249	.062	.018	.141	1.000	-.419	.454	-.229
	T2RepCop	.101	.057	-.016	.026	-.419	1.000	-.165	.148
	T3StressOverall	.380	.040	-.015	.224	.454	-.165	1.000	-.116
	T3SocSupportOverall	.062	-.212	.004	-.037	-.229	.148	-.116	1.000
Sig. (1-tailed)	T3Rankin	.	.000	.039	.000	.006	.159	.000	.271
	Age	.000	.	.369	.025	.271	.288	.348	.018
	DV_StrokeSev_MildVSMod	.039	.369	.	.000	.428	.439	.442	.483
	DV_StrokeSev_MildVSSev	.000	.025	.000	.	.082	.399	.013	.360
	T2StressOverall	.006	.271	.428	.082	.	.000	.000	.011
	T2RepCop	.159	.288	.439	.399	.000	.	.051	.071
	T3StressOverall	.000	.348	.442	.013	.000	.051	.	.127
	T3SocSupportOverall	.271	.018	.483	.360	.011	.071	.127	.

N	T3Rankin	99	99	99	99	99	99	99	99
	Age	99	99	99	99	99	99	99	99
	DV_StrokeSev_MildVSMod	99	99	99	99	99	99	99	99
	DV_StrokeSev_MildVSSev	99	99	99	99	99	99	99	99
	T2StressOverall	99	99	99	99	99	99	99	99
	T2RepCop	99	99	99	99	99	99	99	99
	T3StressOverall	99	99	99	99	99	99	99	99
	T3SocSupportOverall	99	99	99	99	99	99	99	99

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod[b]	.	Enter
2	T2RepCop, T2StressOverall[b]	.	Enter
3	T3SocSupportOverall, T3StressOverall[b]	.	Enter

a. Dependent Variable: T3Rankin

b. All requested variables entered.

Model Summary[d]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.688[a]	.474	.457	.69310	.474	28.512	3	95	.000
2	.719[b]	.517	.491	.67146	.043	4.112	2	93	.019
3	.765[c]	.586	.554	.62847	.069	7.580	2	91	.001

a. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

b. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T2RepCop, T2StressOverall

c. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T2RepCop, T2StressOverall, T3SocSupportOverall, T3StressOverall

d. Dependent Variable: T3Rankin

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	41.090	3	13.697	28.512	.000[b]
	Residual	45.637	95	.480		
	Total	86.727	98			
2	Regression	44.798	5	8.960	19.872	.000[c]
	Residual	41.930	93	.451		
	Total	86.727	98			
3	Regression	50.785	7	7.255	18.369	.000[d]
	Residual	35.942	91	.395		
	Total	86.727	98			

a. Dependent Variable: T3Rankin

b. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

c. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T2RepCop, T2StressOverall

d. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T2RepCop, T2StressOverall, T3SocSupportOverall, T3StressOverall

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Correlations			Collinearity Statistics	
		B	Std. Error	Beta			Zero-order	Partial	Part	Tolerance	VIF
1	(Constant)	.704	.338		2.080	.040					
	Age	.016	.005	.251	3.276	.001	.391	.319	.244	.941	1.062
	DV_StrokeSev_MildVSMod	.857	.159	.454	5.378	.000	.177	.483	.400	.777	1.287
	DV_StrokeSev_MildVSSev	1.436	.197	.627	7.284	.000	.470	.599	.542	.748	1.338
2	(Constant)	.237	.367		.647	.519					
	Age	.015	.005	.237	3.181	.002	.391	.313	.229	.936	1.068
	DV_StrokeSev_MildVSMod	.821	.155	.435	5.291	.000	.177	.481	.381	.770	1.298
	DV_StrokeSev_MildVSSev	1.342	.194	.586	6.919	.000	.470	.583	.499	.724	1.381
	T2StressOverall	.274	.103	.215	2.656	.009	.249	.266	.192	.791	1.265
	T2RepCop	.324	.153	.170	2.120	.037	.101	.215	.153	.811	1.232
3	(Constant)	-.877	.492		-1.784	.078					
	Age	.018	.005	.282	3.952	.000	.391	.383	.267	.892	1.121
	DV_StrokeSev_MildVSMod	.777	.146	.412	5.336	.000	.177	.488	.360	.766	1.306
	DV_StrokeSev_MildVSSev	1.215	.185	.531	6.562	.000	.470	.567	.443	.696	1.437
	T2StressOverall	.188	.107	.148	1.759	.082	.249	.181	.119	.647	1.545
	T2RepCop	.290	.143	.152	2.024	.046	.101	.208	.137	.806	1.240
	T3StressOverall	.310	.102	.235	3.039	.003	.380	.304	.205	.763	1.310
	T3SocSupportOverall	.119	.048	.178	2.499	.014	.062	.253	.169	.900	1.111

a. Dependent Variable: T3Rankin

Excluded Variables[a]

Model		Beta In	t	Sig.	Partial Correlation	Collinearity Statistics		
						Tolerance	VIF	Minimum Tolerance
1	T2StressOverall	.141[b]	1.896	.061	.192	.971	1.030	.729
	T2RepCop	.078[b]	1.048	.298	.107	.996	1.004	.748
	T3StressOverall	.252[b]	3.461	.001	.336	.940	1.064	.704
	T3SocSupportOverall	.143[b]	1.897	.061	.192	.955	1.047	.747
2	T3StressOverall	.231[c]	2.904	.005	.290	.764	1.310	.663
	T3SocSupportOverall	.173[c]	2.332	.022	.236	.900	1.111	.723

a. Dependent Variable: T3Rankin

b. Predictors in the Model: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

c. Predictors in the Model: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T2RepCop, T2StressOverall

Collinearity Diagnostics[a]

Model	Dimension	Eigenvalue	Condition Index	Variance Proportions							
				(Constant)	Age	DV_StrokeSev_MildVSMod	DV_StrokeSev_MildVSSev	T2StressOverall	T2RepCop	T3StressOverall	T3SocSupportOverall
1	1	2.732	1.000	.01	.01	.03	.02				
	2	1.000	1.653	.00	.00	.15	.39				
	3	.246	3.331	.03	.02	.82	.57				
	4	.021	11.282	.97	.97	.00	.02				
2	1	3.998	1.000	.00	.00	.01	.01	.01	.01		
	2	1.001	1.999	.00	.00	.15	.37	.00	.00		
	3	.622	2.535	.00	.00	.01	.01	.03	.64		
	4	.270	3.848	.01	.01	.80	.58	.06	.00		
	5	.089	6.717	.05	.13	.01	.00	.83	.33		
	6	.020	14.071	.94	.86	.00	.03	.07	.02		
3	1	5.757	1.000	.00	.00	.01	.00	.00	.01	.00	.00
	2	1.005	2.394	.00	.00	.15	.35	.00	.00	.00	.00
	3	.647	2.982	.00	.00	.01	.00	.02	.61	.01	.00
	4	.292	4.444	.00	.00	.81	.57	.02	.00	.01	.01
	5	.140	6.422	.01	.02	.00	.02	.09	.24	.36	.12
	6	.090	7.993	.00	.01	.00	.00	.63	.10	.58	.05
	7	.057	10.047	.01	.37	.02	.03	.18	.03	.01	.34
	8	.012	22.012	.98	.60	.00	.03	.07	.01	.02	.48

a. Dependent Variable: T3Rankin

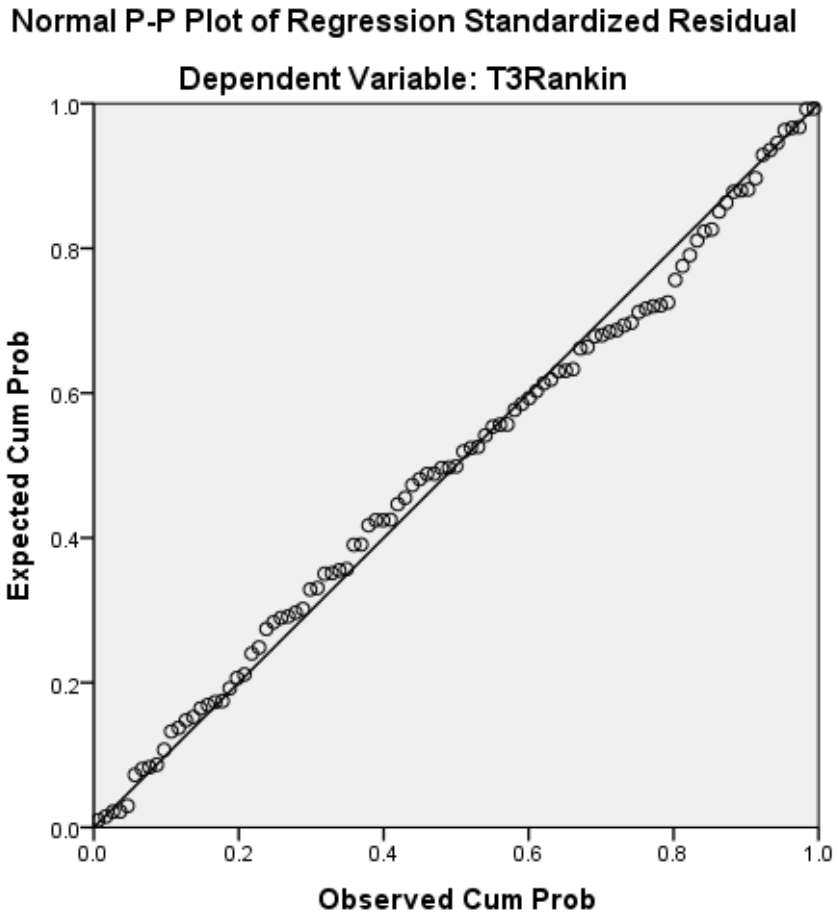
Residuals Statistics[a]

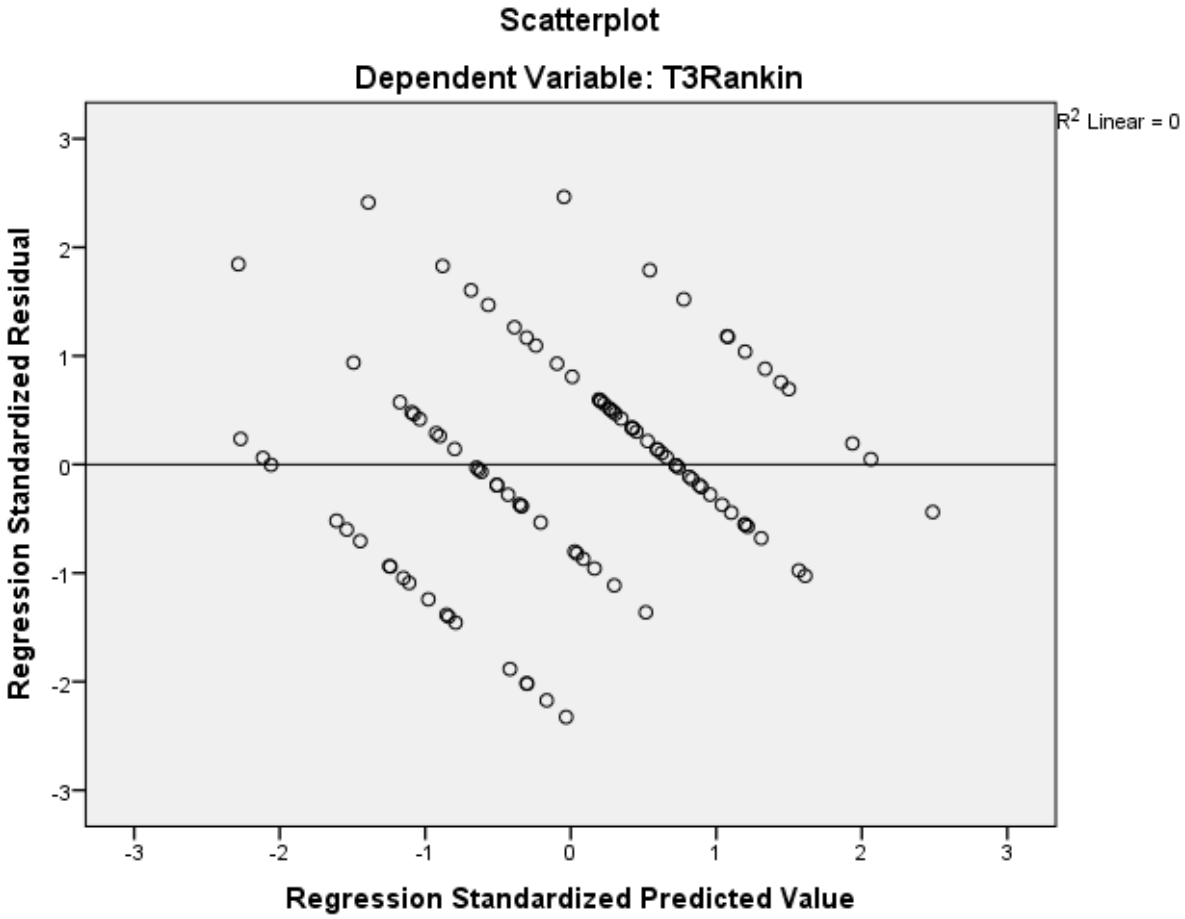
	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	.8407	4.2753	2.4848	.71987	99
Std. Predicted Value	-2.284	2.487	.000	1.000	99
Standard Error of Predicted Value	.114	.297	.176	.033	99
Adjusted Predicted Value	.7249	4.3170	2.4839	.72325	99
Residual	-1.46195	1.54886	.00000	.60560	99

Std. Residual	-2.326	2.465	.000	.964	99
Stud. Residual	-2.418	2.677	.001	1.006	99
Deleted Residual	-1.57952	1.82686	.00092	.66054	99
Stud. Deleted Residual	-2.486	2.773	.001	1.019	99
Mahal. Distance	2.229	20.879	6.929	3.236	99
Cook's Distance	.000	.161	.011	.020	99
Centered Leverage Value	.023	.213	.071	.033	99

a. Dependent Variable: T3Rankin

Charts






```
REGRESSION
  /DESCRIPTIVES MEAN STDDEV CORR SIG N
  /MISSING LISTWISE
  /STATISTICS COEFF OUTS R ANOVA COLLIN TOL CHANGE ZPP
  /CRITERIA=PIN(.05) POUT(.10)
  /NOORIGIN
  /DEPENDENT T3Rankin
  /METHOD=ENTER Age DV_StrokeSev_MildVSMod DV_StrokeSev_MildVSSev
  /METHOD=ENTER T3BellsTotal
  /SCATTERPLOT=(*ZRESID ,*ZPRED)
  /RESIDUALS NORMPROB(ZRESID)
  /SAVE MAHAL COOK.
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Regression

Notes

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Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on cases with no missing values for any variable used.
Syntax		REGRESSION /DESCRIPTIVES MEAN STDDEV CORR SIG N /MISSING LISTWISE /STATISTICS COEFF OUTS R ANOVA COLLIN TOL CHANGE ZPP /CRITERIA=PIN(.05) POUT(.10) /NOORIGIN /DEPENDENT T3Rankin /METHOD=ENTER Age DV_StrokeSev_MildVSMod DV_StrokeSev_MildVSSev /METHOD=ENTER T3BellsTotal /SCATTERPLOT=(*ZRESID ,*ZPRED) /RESIDUALS NORMPROB(ZRESID) /SAVE MAHAL COOK.

	Processor Time	00:00:00.28
	Elapsed Time	00:00:01.91
Resources	Memory Required	18780 bytes
	Additional Memory Required for Residual Plots	544 bytes
Variables Created or Modified	MAH_1	Mahalanobis Distance
	COO_1	Cook's Distance

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[DataSet1] H:\StrokeData_1.sav
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Descriptive Statistics

	Mean	Std. Deviation	N
T3Rankin	2.5053	.93255	95
Age	66.6842	14.88271	95
DV_StrokeSev_MildVSMod	.4316	.49792	95
DV_StrokeSev_MildVSSev	.2316	.42408	95
T3BellsTotal	29.8632	6.65664	95

Correlations

		T3Rankin	Age	DV_StrokeSev_MildVSMod	DV_StrokeSev_MildVSSev	T3BellsTotal
Pearson Correlation	T3Rankin	1.000	.376	.167	.481	-.403
	Age	.376	1.000	.059	.168	-.308
	DV_StrokeSev_MildVSMod	.167	.059	1.000	-.478	.040
	DV_StrokeSev_MildVSSev	.481	.168	-.478	1.000	-.268
	T3BellsTotal	-.403	-.308	.040	-.268	1.000
Sig. (1-tailed)	T3Rankin	.	.000	.053	.000	.000
	Age	.000	.	.286	.051	.001
	DV_StrokeSev_MildVSMod	.053	.286	.	.000	.348
	DV_StrokeSev_MildVSSev	.000	.051	.000	.	.004
	T3BellsTotal	.000	.001	.348	.004	.
N	T3Rankin	95	95	95	95	95
	Age	95	95	95	95	95
	DV_StrokeSev_MildVSMod	95	95	95	95	95
	DV_StrokeSev_MildVSSev	95	95	95	95	95
	T3BellsTotal	95	95	95	95	95

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod[b]	.	Enter
2	T3BellsTotal[b]	.	Enter

a. Dependent Variable: T3Rankin

b. All requested variables entered.

Model Summary[c]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.699[a]	.489	.472	.67773	.489	28.992	3	91	.000
2	.723[b]	.523	.502	.65818	.034	6.486	1	90	.013

a. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

b. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T3BellsTotal

c. Dependent Variable: T3Rankin

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	39.949	3	13.316	28.992	.000[b]
	Residual	41.798	91	.459		
	Total	81.747	94			
2	Regression	42.759	4	10.690	24.676	.000[c]
	Residual	38.988	90	.433		
	Total	81.747	94			

a. Dependent Variable: T3Rankin

b. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

c. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T3BellsTotal

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Correlations			Collinearity Statistics	
		B	Std. Error	Beta			Zero-order	Partial	Part	Tolerance	VIF
1	(Constant)	.796	.324		2.460	.016					
	Age	.015	.005	.236	3.067	.003	.376	.306	.230	.946	1.057

	DV_StrokeSev_MildVSMod	.884	.162	.472	5.459	.000	.167	.497	.409	.751	1.331
	DV_StrokeSev_MildVSSev	1.467	.193	.667	7.616	.000	.481	.624	.571	.732	1.365
	(Constant)	1.891	.533		3.551	.001					
	Age	.012	.005	.184	2.371	.020	.376	.242	.173	.880	1.136
2	DV_StrokeSev_MildVSMod	.859	.158	.459	5.452	.000	.167	.498	.397	.748	1.336
	DV_StrokeSev_MildVSSev	1.354	.192	.616	7.046	.000	.481	.596	.513	.694	1.442
	T3BellsTotal	-.028	.011	-.201	-2.547	.013	-.403	-.259	-.185	.854	1.171

a. Dependent Variable: T3Rankin

Excluded Variables[a]

Model		Beta In	t	Sig.	Partial Correlation	Collinearity Statistics		
						Tolerance	VIF	Minimum Tolerance
1	T3BellsTotal	-.201[b]	-2.547	.013	-.259	.854	1.171	.694

a. Dependent Variable: T3Rankin

b. Predictors in the Model: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

Collinearity Diagnostics[a]

Model	Dimension	Eigenvalue	Condition Index	Variance Proportions				
				(Constant)	Age	DV_StrokeSev_MildVSMod	DV_StrokeSev_MildVSSev	T3BellsTotal
1	1	2.744	1.000	.01	.01	.03	.02	
	2	1.000	1.656	.00	.00	.15	.36	
	3	.233	3.435	.03	.03	.81	.61	
	4	.023	10.814	.96	.97	.00	.01	
2	1	3.655	1.000	.00	.00	.02	.01	.00
	2	1.002	1.909	.00	.00	.14	.35	.00
	3	.277	3.629	.00	.00	.79	.49	.02
	4	.054	8.193	.00	.42	.05	.13	.31
	5	.011	18.375	1.00	.57	.01	.03	.67

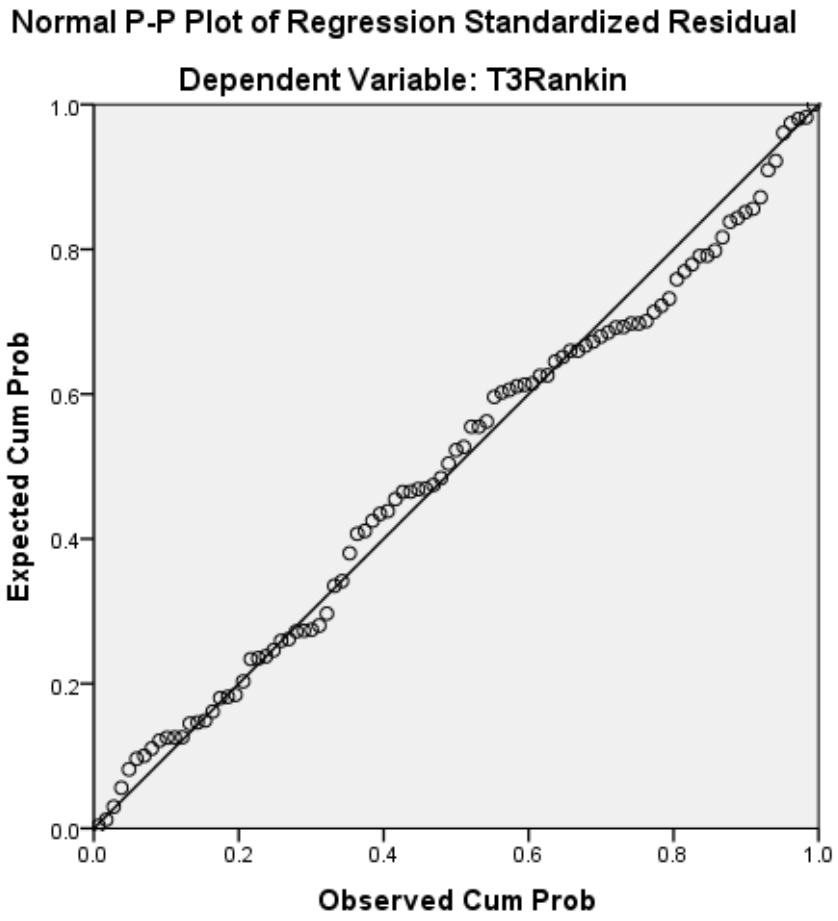
a. Dependent Variable: T3Rankin

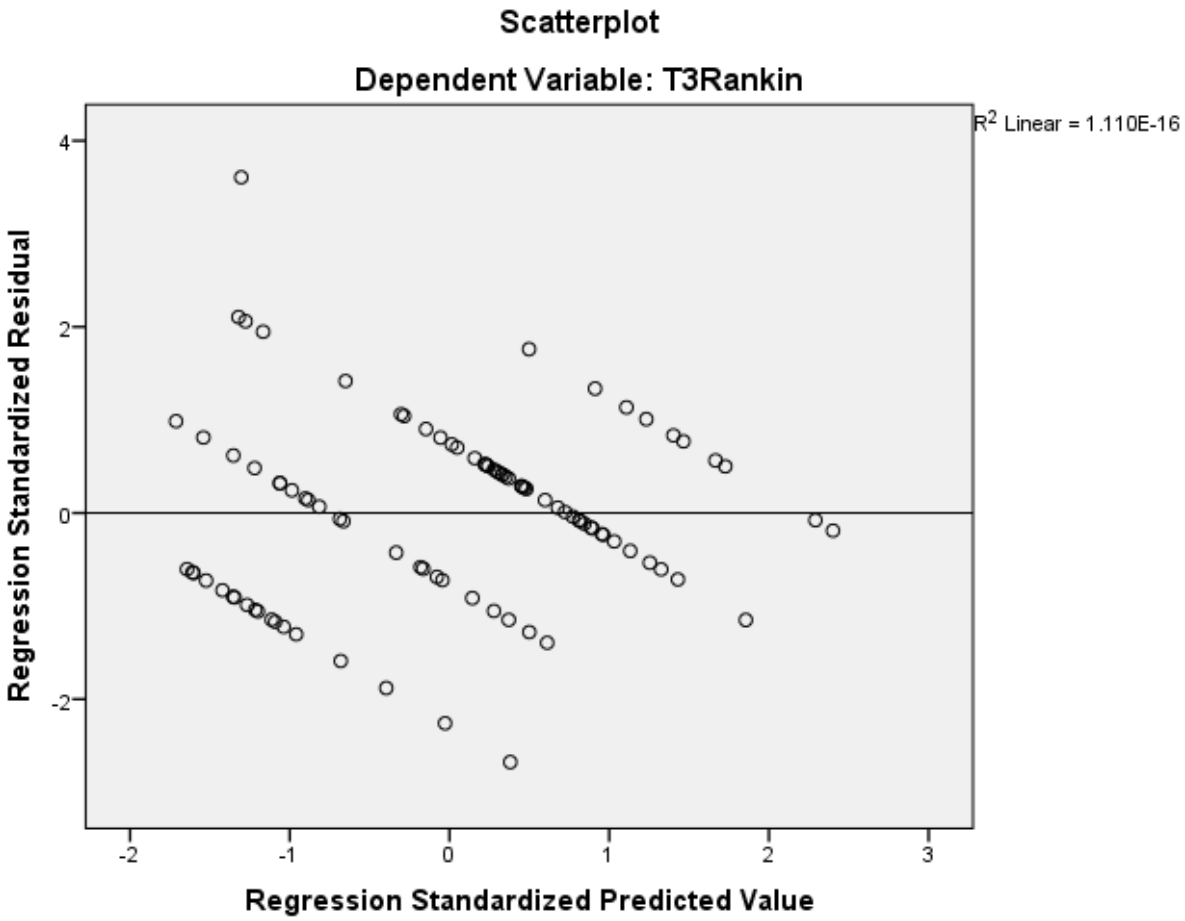
Residuals Statistics[a]

	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	1.3508	4.1247	2.5053	.67445	95
Std. Predicted Value	-1.712	2.401	.000	1.000	95
Standard Error of Predicted Value	.103	.279	.146	.037	95
Adjusted Predicted Value	1.3046	4.1519	2.5077	.67677	95
Residual	-1.76197	2.37397	.00000	.64403	95
Std. Residual	-2.677	3.607	.000	.978	95
Stud. Residual	-2.723	3.670	-.002	1.001	95
Deleted Residual	-1.82338	2.45758	-.00246	.67366	95
Stud. Deleted Residual	-2.827	3.957	.000	1.021	95
Mahal. Distance	1.320	15.880	3.958	2.939	95
Cook's Distance	.000	.095	.009	.015	95
Centered Leverage Value	.014	.169	.042	.031	95

a. Dependent Variable: T3Rankin

Charts


Normal P-P Plot of Regression Standardized Residual
Dependent Variable: T3Rankin
Expected Cum Prob
Observed Cum Prob
0.0
0.2
0.4
0.6
0.8
1.0


Scatterplot
Dependent Variable: T3Rankin
R2 Linear = 1.110E-16
Regression Standardized Residual
Regression Standardized Predicted Value
4
2
0
-2
-2
-1
0
1
2
3

```
Run MATRIX procedure:

***************** PROCESS Procedure for SPSS Release 2.11
****************

          Written by Andrew F. Hayes, Ph.D.       www.afhayes.com
    Documentation available in Hayes (2013).
www.guilford.com/p/hayes3

**************************************************************************
******
Model = 4
    Y = T2Rankin
    X = T2DepOve
    M = T2RBMT

Sample size
        104

**************************************************************************
******
Outcome: T2RBMT

Model Summary
          R       R-sq          F        df1        df2          p
      .2112      .0446     4.7614     1.0000   102.0000      .0314

Model
              coeff         se          t          p       LLCI
ULCI
constant     9.7331      .2947    33.0279      .0000     9.1486
10.3177
T2DepOve     -.5890      .2699    -2.1821      .0314    -1.1244
-.0536

**************************************************************************
******
Outcome: T2Rankin

Model Summary
          R       R-sq          F        df1        df2          p
      .3634      .1320     7.6829     2.0000   101.0000      .0008

Model
              coeff         se          t          p       LLCI
ULCI
constant     3.3420      .6129     5.4525      .0000     2.1261
4.5578
T2RBMT       -.1319      .0602    -2.1905      .0308     -.2514
-.0125
T2DepOve      .4560      .1680     2.7149      .0078      .1228
.7891

************************** TOTAL EFFECT MODEL
****************************
Outcome: T2Rankin

Model Summary
          R       R-sq          F        df1        df2          p
```

```
      .3014      .0908    10.1882     1.0000   102.0000      .0019

Model
              coeff         se          t          p       LLCI
ULCI
constant     2.0580      .1825    11.2745      .0000     1.6960
2.4201
T2DepOve      .5337      .1672     3.1919      .0019      .2020
.8653

***************** TOTAL, DIRECT, AND INDIRECT EFFECTS
********************

Total effect of X on Y
     Effect         SE          t          p       LLCI       ULCI
      .5337      .1672     3.1919      .0019      .2020      .8653

Direct effect of X on Y
     Effect         SE          t          p       LLCI       ULCI
      .4560      .1680     2.7149      .0078      .1228      .7891

Indirect effect of X on Y
          Effect    Boot SE   BootLLCI   BootULCI
T2RBMT      .0777      .0441      .0097      .1817

Partially standardized indirect effect of X on Y
          Effect    Boot SE   BootLLCI   BootULCI
T2RBMT      .0739      .0416      .0077      .1699

Completely standardized indirect effect of X on Y
          Effect    Boot SE   BootLLCI   BootULCI
T2RBMT      .0439      .0233      .0044      .0987

Ratio of indirect to total effect of X on Y
          Effect    Boot SE   BootLLCI   BootULCI
T2RBMT      .1456     1.3106      .0176      .5122

Ratio of indirect to direct effect of X on Y
          Effect    Boot SE   BootLLCI   BootULCI
T2RBMT      .1704      .3621      .0179     1.0459

R-squared mediation effect size (R-sq_med)
          Effect    Boot SE   BootLLCI   BootULCI
T2RBMT      .0275      .0156      .0048      .0698

Preacher and Kelley (2011) Kappa-squared
          Effect    Boot SE   BootLLCI   BootULCI
T2RBMT      .0450      .0232      .0049      .0994

Normal theory tests for indirect effect
     Effect         se          Z          p
      .0777      .0528     1.4709      .1413

******************** ANALYSIS NOTES AND WARNINGS
************************

Number of bootstrap samples for bias corrected bootstrap confidence
intervals:
     5000
```

```
Level of confidence for all confidence intervals in output:
    95.00

NOTE: Some cases were deleted due to missing data.  The number of
such cases was:
  39

------ END MATRIX -----
```

```
Run MATRIX procedure:

***************** PROCESS Procedure for SPSS Release 2.11
****************

          Written by Andrew F. Hayes, Ph.D.       www.afhayes.com
    Documentation available in Hayes (2013).
www.guilford.com/p/hayes3

**************************************************************************
******
Model = 4
    Y = T2Rankin
    X = T2RBMT
    M = T2DepOve

Sample size
        104

**************************************************************************
******
Outcome: T2DepOve

Model Summary
          R       R-sq          F        df1        df2          p
      .2112      .0446     4.7614     1.0000   102.0000      .0314

Model
              coeff         se          t          p       LLCI
ULCI
constant     1.6143      .3241     4.9814      .0000      .9715
2.2571
T2RBMT       -.0757      .0347    -2.1821      .0314     -.1446
-.0069

**************************************************************************
******
```

```
Outcome: T2Rankin

Model Summary
          R       R-sq          F        df1        df2          p
      .3634      .1320     7.6829     2.0000   101.0000      .0008

Model
              coeff         se          t          p       LLCI
ULCI
constant     3.3420      .6129     5.4525      .0000     2.1261
4.5578
T2DepOve      .4560      .1680     2.7149      .0078      .1228
.7891
T2RBMT       -.1319      .0602    -2.1905      .0308     -.2514
-.0125

************************* TOTAL EFFECT MODEL
****************************
Outcome: T2Rankin

Model Summary
          R       R-sq          F        df1        df2          p
      .2621      .0687     7.5253     1.0000   102.0000      .0072

Model
              coeff         se          t          p       LLCI
ULCI
constant     4.0780      .5666     7.1973      .0000     2.9542
5.2019
T2RBMT       -.1664      .0607    -2.7432      .0072     -.2868
-.0461

***************** TOTAL, DIRECT, AND INDIRECT EFFECTS
*******************

Total effect of X on Y
     Effect         SE          t          p       LLCI       ULCI
     -.1664      .0607    -2.7432      .0072     -.2868     -.0461

Direct effect of X on Y
     Effect         SE          t          p       LLCI       ULCI
     -.1319      .0602    -2.1905      .0308     -.2514     -.0125

Indirect effect of X on Y
            Effect    Boot SE   BootLLCI   BootULCI
T2DepOve     -.0345      .0165     -.0797     -.0124

Partially standardized indirect effect of X on Y
            Effect    Boot SE   BootLLCI   BootULCI
T2DepOve     -.0329      .0155     -.0754     -.0118

Completely standardized indirect effect of X on Y
            Effect    Boot SE   BootLLCI   BootULCI
T2DepOve     -.0544      .0233     -.1129     -.0184

Ratio of indirect to total effect of X on Y
            Effect    Boot SE   BootLLCI   BootULCI
T2DepOve      .2074      .1474      .0675      .6551

Ratio of indirect to direct effect of X on Y
```

```
            Effect    Boot SE   BootLLCI   BootULCI
T2DepOve      .2617     4.2357      .0718     1.8948

R-squared mediation effect size (R-sq_med)
            Effect    Boot SE   BootLLCI   BootULCI
T2DepOve      .0275      .0148      .0062      .0692

Preacher and Kelley (2011) Kappa-squared
            Effect    Boot SE   BootLLCI   BootULCI
T2DepOve      .0551      .0233      .0191      .1124

Normal theory tests for indirect effect
     Effect         se          Z          p
     -.0345      .0211    -1.6348      .1021

******************** ANALYSIS NOTES AND WARNINGS
************************

Number of bootstrap samples for bias corrected bootstrap confidence
intervals:
     5000

Level of confidence for all confidence intervals in output:
    95.00

NOTE: Some cases were deleted due to missing data.  The number of
such cases was:
  39

------ END MATRIX -----
```

```
Run MATRIX procedure:

***************** PROCESS Procedure for SPSS Release 2.11
***************

          Written by Andrew F. Hayes, Ph.D.       www.afhayes.com
    Documentation available in Hayes (2013).
www.guilford.com/p/hayes3

**************************************************************************
******
Model = 4
    Y = T2Rankin
    X = T2LineBi
    M = T2DepOve
```

```
Sample size
        104

*************************************************************************
******
Outcome: T2DepOve

Model Summary
          R       R-sq          F        df1        df2          p
      .1435      .0206     2.1439     1.0000   102.0000      .1462

Model
              coeff         se          t          p       LLCI
ULCI
constant      .9002      .0592    15.2140      .0000      .7829
1.0176
T2LineBi     -.0795      .0543    -1.4642      .1462     -.1873
.0282

*************************************************************************
******
Outcome: T2Rankin

Model Summary
          R       R-sq          F        df1        df2          p
      .3529      .1245     7.1826     2.0000   101.0000      .0012

Model
              coeff         se          t          p       LLCI
ULCI
constant     2.0600      .1800    11.4437      .0000     1.7029
2.4171
T2DepOve      .4865      .1666     2.9204      .0043      .1560
.8170
T2LineBi     -.1821      .0924    -1.9719      .0514     -.3653
.0011

************************** TOTAL EFFECT MODEL
****************************
Outcome: T2Rankin

Model Summary
          R       R-sq          F        df1        df2          p
      .2249      .0506     5.4355     1.0000   102.0000      .0217

Model
              coeff         se          t          p       LLCI
ULCI
constant     2.4980      .1032    24.2130      .0000     2.2933
2.7026
T2LineBi     -.2208      .0947    -2.3314      .0217     -.4087
-.0330

***************** TOTAL, DIRECT, AND INDIRECT EFFECTS
********************

Total effect of X on Y
     Effect         SE          t          p       LLCI       ULCI
     -.2208      .0947    -2.3314      .0217     -.4087     -.0330
```

```
Direct effect of X on Y
     Effect         SE          t          p       LLCI       ULCI
     -.1821      .0924    -1.9719      .0514     -.3653      .0011

Indirect effect of X on Y
             Effect    Boot SE   BootLLCI   BootULCI
T2DepOve     -.0387      .0307     -.1075     -.0056

Partially standardized indirect effect of X on Y
             Effect    Boot SE   BootLLCI   BootULCI
T2DepOve     -.0368      .0288     -.0961     -.0046

Completely standardized indirect effect of X on Y
             Effect    Boot SE   BootLLCI   BootULCI
T2DepOve     -.0394      .0210     -.0862     -.0057

Ratio of indirect to total effect of X on Y
             Effect    Boot SE   BootLLCI   BootULCI
T2DepOve      .1752      .2595      .0328      .5697

Ratio of indirect to direct effect of X on Y
             Effect    Boot SE   BootLLCI   BootULCI
T2DepOve      .2125     4.3998      .0310     1.2072

R-squared mediation effect size (R-sq_med)
             Effect    Boot SE   BootLLCI   BootULCI
T2DepOve      .0169      .0111      .0012      .0445

Preacher and Kelley (2011) Kappa-squared
             Effect    Boot SE   BootLLCI   BootULCI
T2DepOve      .0402      .0214      .0064      .0881

Normal theory tests for indirect effect
     Effect         se          Z          p
     -.0387      .0309    -1.2516      .2107

******************** ANALYSIS NOTES AND WARNINGS
************************

Number of bootstrap samples for bias corrected bootstrap confidence
intervals:
     5000

Level of confidence for all confidence intervals in output:
    95.00

NOTE: Some cases were deleted due to missing data.  The number of
such cases was:
  39

------ END MATRIX -----
```

```
REGRESSION
  /DESCRIPTIVES MEAN STDDEV CORR SIG N
  /MISSING LISTWISE
  /STATISTICS COEFF OUTS R ANOVA COLLIN TOL CHANGE ZPP
  /CRITERIA=PIN(.05) POUT(.10)
  /NOORIGIN
  /DEPENDENT T2QoLOverall
  /METHOD=ENTER Age DV_StrokeSev_MildVSMod DV_StrokeSev_MildVSSev
  /METHOD=ENTER T1SocialSupportOverall
  /SCATTERPLOT=(*ZRESID ,*ZPRED)
  /RESIDUALS NORMPROB(ZRESID)
  /SAVE MAHAL COOK.
```

Regression

Notes

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	Split File	<none>
	N of Rows in Working Data File	143
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on cases with no missing values for any variable used.

Syntax		REGRESSION /DESCRIPTIVES MEAN STDDEV CORR SIG N /MISSING LISTWISE /STATISTICS COEFF OUTS R ANOVA COLLIN TOL CHANGE ZPP /CRITERIA=PIN(.05) POUT(.10) /NOORIGIN /DEPENDENT T2QoLOverall /METHOD=ENTER Age DV_StrokeSev_MildVSMod DV_StrokeSev_MildVSSev /METHOD=ENTER T1SocialSupportOverall /SCATTERPLOT=(*ZRESID ,*ZPRED) /RESIDUALS NORMPROB(ZRESID) /SAVE MAHAL COOK.
Resources	Processor Time	00:00:00.47
	Elapsed Time	00:00:10.37
	Memory Required	18820 bytes
	Additional Memory Required for Residual Plots	544 bytes
Variables Created or Modified	MAH_2	Mahalanobis Distance
	COO_2	Cook's Distance

[DataSet1] H:\StrokeData_1.sav

Descriptive Statistics

	Mean	Std. Deviation	N
T2QoLOverall	46.4403	19.88717	108
Age	68.1759	14.66946	108
DV_StrokeSev_MildVSMod	.4259	.49679	108
DV_StrokeSev_MildVSSev	.2037	.40463	108

T1SocialSupportOverall	5.5278	1.24435	108

Correlations

		T2QoLOverall	Age	DV_StrokeSev_MildVSMod	DV_StrokeSev_MildVSSev	T1SocialSupportOverall
Pearson Correlation	T2QoLOverall	1.000	-.260	-.184	-.196	.283
	Age	-.260	1.000	.013	.128	-.038
	DV_StrokeSev_MildVSMod	-.184	.013	1.000	-.436	-.014
	DV_StrokeSev_MildVSSev	-.196	.128	-.436	1.000	.010
	T1SocialSupportOverall	.283	-.038	-.014	.010	1.000
Sig. (1-tailed)	T2QoLOverall	.	.003	.028	.021	.001
	Age	.003	.	.448	.094	.349
	DV_StrokeSev_MildVSMod	.028	.448	.	.000	.442
	DV_StrokeSev_MildVSSev	.021	.094	.000	.	.458
	T1SocialSupportOverall	.001	.349	.442	.458	.
N	T2QoLOverall	108	108	108	108	108
	Age	108	108	108	108	108
	DV_StrokeSev_MildVSMod	108	108	108	108	108
	DV_StrokeSev_MildVSSev	108	108	108	108	108
	T1SocialSupportOverall	108	108	108	108	108

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod[b]	.	Enter
2	T1SocialSupportOverall[b]	.	Enter

a. Dependent Variable: T2QoLOverall

b. All requested variables entered.

Model Summary[c]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.417[a]	.174	.150	18.33412	.174	7.298	3	104	.000
2	.499[b]	.249	.220	17.56478	.075	10.310	1	103	.002

a. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

b. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1SocialSupportOverall

c. Dependent Variable: T2QoLOverall

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	7359.904	3	2453.301	7.298	.000[b]
	Residual	34958.560	104	336.140		
	Total	42318.464	107			
2	Regression	10540.746	4	2635.187	8.541	.000[c]
	Residual	31777.718	103	308.522		
	Total	42318.464	107			

a. Dependent Variable: T2QoLOverall

b. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

c. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1SocialSupportOverall

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Correlations			Collinearity Statistics	
		B	Std. Error	Beta			Zero-order	Partial	Part	Tolerance	VIF
1	(Constant)	74.928	8.558		8.756	.000					
	Age	-.295	.122	-.217	-2.411	.018	-.260	-.230	-.215	.978	1.023
	DV_StrokeSev_MildVSMod	-12.567	3.975	-.314	-3.161	.002	-.184	-.296	-.282	.805	1.242
	DV_StrokeSev_MildVSSev	-14.975	4.921	-.305	-3.043	.003	-.196	-.286	-.271	.792	1.262
2	(Constant)	49.683	11.359		4.374	.000					
	Age	-.280	.117	-.207	-2.390	.019	-.260	-.229	-.204	.976	1.024
	DV_StrokeSev_MildVSMod	-12.470	3.809	-.312	-3.274	.001	-.184	-.307	-.280	.805	1.242
	DV_StrokeSev_MildVSSev	-15.131	4.714	-.308	-3.209	.002	-.196	-.302	-.274	.792	1.262
	T1SocialSupportOverall	4.385	1.366	.274	3.211	.002	.283	.302	.274	.998	1.002

a. Dependent Variable: T2QoLOverall

Excluded Variables[a]

Model		Beta In	t	Sig.	Partial Correlation	Collinearity Statistics		
						Tolerance	VIF	Minimum Tolerance
1	T1SocialSupportOverall	.274[b]	3.211	.002	.302	.998	1.002	.792

a. Dependent Variable: T2QoLOverall

b. Predictors in the Model: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

Collinearity Diagnostics[a]

Model	Dimension	Eigenvalue	Condition Index	Variance Proportions				
				(Constant)	Age	DV_StrokeSev_MildVSMod	DV_StrokeSev_MildVSSev	T1SocialSupportOverall
1	1	2.712	1.000	.01	.01	.03	.02	
	2	1.000	1.647	.00	.00	.15	.42	
	3	.266	3.191	.02	.02	.81	.55	
	4	.022	11.104	.97	.97	.00	.00	
2	1	3.640	1.000	.00	.00	.02	.01	.00
	2	1.000	1.908	.00	.00	.15	.42	.00
	3	.298	3.493	.00	.01	.82	.55	.01
	4	.047	8.799	.00	.45	.01	.01	.51
	5	.015	15.653	.99	.54	.01	.00	.47

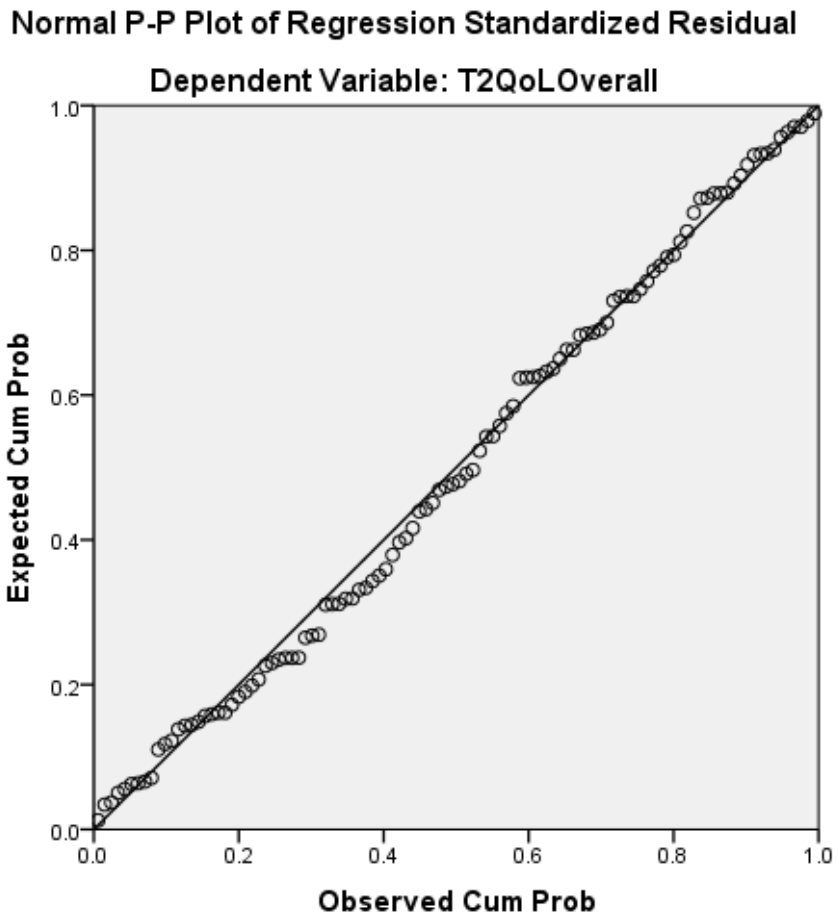
a. Dependent Variable: T2QoLOverall

Residuals Statistics[a]

	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	20.6819	69.1800	46.4403	9.92530	108
Std. Predicted Value	-2.595	2.291	.000	1.000	108
Standard Error of Predicted Value	2.613	7.940	3.687	.832	108
Adjusted Predicted Value	15.2181	68.9445	46.4579	10.05907	108
Residual	-39.41692	40.31613	.00000	17.23334	108
Std. Residual	-2.244	2.295	.000	.981	108
Stud. Residual	-2.300	2.337	.000	1.004	108
Deleted Residual	-41.40260	41.78078	-.01759	18.06955	108
Stud. Deleted Residual	-2.350	2.389	.000	1.011	108
Mahal. Distance	1.377	20.875	3.963	2.639	108
Cook's Distance	.000	.137	.010	.017	108
Centered Leverage Value	.013	.195	.037	.025	108

a. Dependent Variable: T2QoLOverall

Charts


Normal P-P Plot of Regression Standardized Residual
Dependent Variable: T2QoLOverall
Expected Cum Prob
Observed Cum Prob
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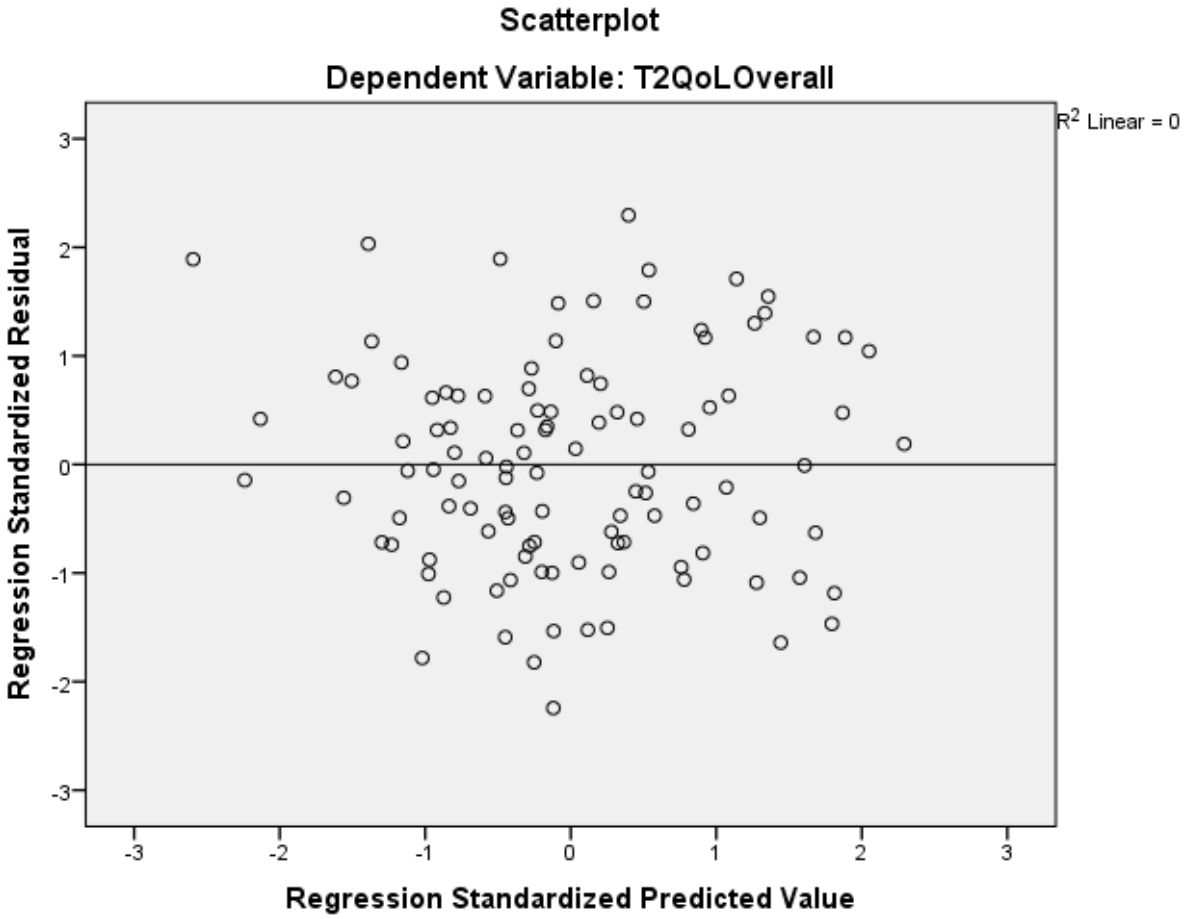
0.2
0.4
0.6
0.8
1.0

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REGRESSION
  /DESCRIPTIVES MEAN STDDEV CORR SIG N
  /MISSING LISTWISE
  /STATISTICS COEFF OUTS R ANOVA COLLIN TOL CHANGE ZPP
  /CRITERIA=PIN(.05) POUT(.10)
  /NOORIGIN
  /DEPENDENT T2QoLOverall
  /METHOD=ENTER Age DV_StrokeSev_MildVSMod DV_StrokeSev_MildVSSev
  /METHOD=ENTER T1SocialSupportOverall
  /METHOD=ENTER T2DepYesNo T2StressOverall
  /SCATTERPLOT=(*ZRESID ,*ZPRED)
  /RESIDUALS NORMPROB(ZRESID)
  /SAVE MAHAL COOK.
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Regression

Notes

Output Created		24-FEB-2015 21:47:00
Comments		
Input	Data	H:\StrokeData_1.sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	143
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on cases with no missing values for any variable used.

Syntax		REGRESSION /DESCRIPTIVES MEAN STDDEV CORR SIG N /MISSING LISTWISE /STATISTICS COEFF OUTS R ANOVA COLLIN TOL CHANGE ZPP /CRITERIA=PIN(.05) POUT(.10) /NOORIGIN /DEPENDENT T2QoLOverall /METHOD=ENTER Age DV_StrokeSev_MildVSMod DV_StrokeSev_MildVSSev /METHOD=ENTER T1SocialSupportOverall /METHOD=ENTER T2DepYesNo T2StressOverall /SCATTERPLOT=(*ZRESID ,*ZPRED) /RESIDUALS NORMPROB(ZRESID) /SAVE MAHAL COOK.
Resources	Processor Time	00:00:00.25
	Elapsed Time	00:00:01.84
	Memory Required	19716 bytes
	Additional Memory Required for Residual Plots	528 bytes
Variables Created or Modified	MAH_4	Mahalanobis Distance
	COO_4	Cook's Distance

[DataSet1] H:\StrokeData_1.sav

Descriptive Statistics

	Mean	Std. Deviation	N
T2QoLOverall	46.6641	19.84372	107
Age	67.9346	14.52147	107
DV_StrokeSev_MildVSMod	.4299	.49739	107
DV_StrokeSev_MildVSSev	.2056	.40605	107
T1SocialSupportOverall	5.5249	1.24985	107
T2, Dep diagnosis Yes (10 and above) /No (below 10)	.4112	.49437	107
T2StressOverall	1.5888	.74682	107

Correlations

		T2QoLOverall	Age	DV_StrokeSev_MildVSMod	DV_StrokeSev_MildVSSev	T1SocialSupportOverall	T2, Dep diagnosis Yes (10 and above) /No (below 10)	T2StressOverall
Pearson Correlation	T2QoLOverall	1.000	-.246	-.196	-.203	.288	-.569	-.675
	Age	-.246	1.000	.027	.138	-.043	.084	.053
	DV_StrokeSev_MildVSMod	-.196	.027	1.000	-.442	-.012	.157	.067
	DV_StrokeSev_MildVSSev	-.203	.138	-.442	1.000	.011	.092	.113
	T1SocialSupportOverall	.288	-.043	-.012	.011	1.000	-.213	-.282
	T2, Dep diagnosis Yes (10 and above) /No (below 10)	-.569	.084	.157	.092	-.213	1.000	.623
	T2StressOverall	-.675	.053	.067	.113	-.282	.623	1.000
Sig. (1-tailed)	T2QoLOverall	.	.005	.022	.018	.001	.000	.000
	Age	.005	.	.390	.078	.332	.195	.295
	DV_StrokeSev_MildVSMod	.022	.390	.	.000	.450	.054	.247

	DV_StrokeSev_MildVSSev	.018	.078	.000	.	.453	.174	.124
	T1SocialSupportOverall	.001	.332	.450	.453	.	.014	.002
	T2, Dep diagnosis Yes (10 and above) /No (below 10)	.000	.195	.054	.174	.014	.	.000
	T2StressOverall	.000	.295	.247	.124	.002	.000	.
N	T2QoLOverall	107	107	107	107	107	107	107
	Age	107	107	107	107	107	107	107
	DV_StrokeSev_MildVSMod	107	107	107	107	107	107	107
	DV_StrokeSev_MildVSSev	107	107	107	107	107	107	107
	T1SocialSupportOverall	107	107	107	107	107	107	107
	T2, Dep diagnosis Yes (10 and above) /No (below 10)	107	107	107	107	107	107	107
	T2StressOverall	107	107	107	107	107	107	107

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod[b]	.	Enter
2	T1SocialSupportOverall[b]	.	Enter
3	T2, Dep diagnosis Yes (10 and above) /No (below 10), T2StressOverall[b]	.	Enter

a. Dependent Variable: T2QoLOverall

b. All requested variables entered.

Model Summary[d]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.422[a]	.178	.154	18.25058	.178	7.438	3	103	.000
2	.506[b]	.257	.227	17.44265	.078	10.763	1	102	.001
3	.766[c]	.586	.561	13.14507	.330	39.799	2	100	.000

a. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

b. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1SocialSupportOverall

c. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1SocialSupportOverall, T2, Dep diagnosis Yes (10 and above) /No (below 10), T2StressOverall

d. Dependent Variable: T2QoLOverall

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	7432.353	3	2477.451	7.438	.000[b]
	Residual	34307.615	103	333.084		
	Total	41739.968	106			
2	Regression	10706.881	4	2676.720	8.798	.000[c]
	Residual	31033.087	102	304.246		
	Total	41739.968	106			
3	Regression	24460.685	6	4076.781	23.593	.000[d]
	Residual	17279.283	100	172.793		
	Total	41739.968	106			

a. Dependent Variable: T2QoLOverall

b. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

c. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1SocialSupportOverall

d. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1SocialSupportOverall, T2, Dep diagnosis Yes (10 and above) /No (below 10), T2StressOverall

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Correlations			Collinearity Statistics	
		B	Std. Error	Beta			Zero-order	Partial	Part	Tolerance	VIF
1	(Constant)	73.422	8.587		8.551	.000					
	Age	-.262	.124	-.192	-2.113	.037	-.246	-.204	-.189	.971	1.030
	DV_StrokeSev_MildVSMod	-13.306	3.992	-.334	-3.333	.001	-.196	-.312	-.298	.797	1.255
	DV_StrokeSev_MildVSSev	-15.829	4.936	-.324	-3.207	.002	-.203	-.301	-.286	.782	1.278
2	(Constant)	47.689	11.352		4.201	.000					
	Age	-.245	.119	-.179	-2.065	.042	-.246	-.200	-.176	.969	1.032
	DV_StrokeSev_MildVSMod	-13.261	3.816	-.332	-3.475	.001	-.196	-.325	-.297	.797	1.255
	DV_StrokeSev_MildVSSev	-16.046	4.718	-.328	-3.401	.001	-.203	-.319	-.290	.782	1.279
	T1SocialSupportOverall	4.452	1.357	.280	3.281	.001	.288	.309	.280	.998	1.002
3	(Constant)	82.219	9.578		8.585	.000					
	Age	-.226	.089	-.166	-2.533	.013	-.246	-.246	-.163	.967	1.034
	DV_StrokeSev_MildVSMod	-8.858	2.947	-.222	-3.005	.003	-.196	-.288	-.193	.758	1.318
	DV_StrokeSev_MildVSSev	-10.184	3.623	-.208	-2.811	.006	-.203	-.271	-.181	.753	1.327
	T1SocialSupportOverall	1.678	1.069	.106	1.570	.120	.288	.155	.101	.913	1.095
	T2, Dep diagnosis Yes (10 and above) /No (below 10)	-6.953	3.366	-.173	-2.066	.041	-.569	-.202	-.133	.589	1.699
	T2StressOverall	-13.018	2.236	-.490	-5.822	.000	-.675	-.503	-.375	.585	1.711

a. Dependent Variable: T2QoLOverall

Excluded Variables[a]

Model		Beta In	t	Sig.	Partial Correlation	Collinearity Statistics		
						Tolerance	VIF	Minimum Tolerance
1	T1SocialSupportOverall	.280[b]	3.281	.001	.309	.998	1.002	.782
	T2, Dep diagnosis Yes (10 and above) /No (below 10)	-.500[b]	-6.400	.000	-.535	.941	1.063	.759
	T2StressOverall	-.625[b]	-9.331	.000	-.679	.970	1.031	.764
2	T2, Dep diagnosis Yes (10 and above) /No (below 10)	-.459[c]	-5.871	.000	-.504	.897	1.115	.757
	T2StressOverall	-.592[c]	-8.542	.000	-.648	.890	1.123	.761

a. Dependent Variable: T2QoLOverall

b. Predictors in the Model: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

c. Predictors in the Model: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1SocialSupportOverall

Collinearity Diagnostics[a]

Model	Dimension	Eigenvalue	Condition Index	Variance Proportions						
				(Constant)	Age	DV_StrokeSev_MildVSMod	DV_StrokeSev_MildVSSev	T1SocialSupportOverall	T2, Dep diagnosis Yes (10 and above) /No (below 10)	T2Stress Overall
1	1	2.718	1.000	.01	.01	.03	.02			
	2	1.000	1.649	.00	.00	.15	.41			
	3	.260	3.236	.02	.02	.81	.56			
	4	.022	11.189	.97	.97	.00	.00			
2	1	3.646	1.000	.00	.00	.02	.01	.00		
	2	1.000	1.909	.00	.00	.15	.41	.00		
	3	.292	3.536	.01	.01	.82	.56	.02		
	4	.047	8.807	.00	.44	.01	.02	.52		
	5	.015	15.718	.99	.55	.00	.00	.46		
3	1	5.020	1.000	.00	.00	.01	.01	.00	.01	.00
	2	1.000	2.240	.00	.00	.14	.40	.00	.00	.00
	3	.536	3.061	.00	.00	.00	.00	.01	.48	.01
	4	.292	4.144	.00	.00	.83	.58	.01	.01	.02
	5	.095	7.279	.00	.01	.01	.00	.06	.50	.78
	6	.044	10.699	.00	.56	.00	.01	.40	.00	.04
	7	.013	20.031	.99	.42	.00	.01	.53	.01	.15

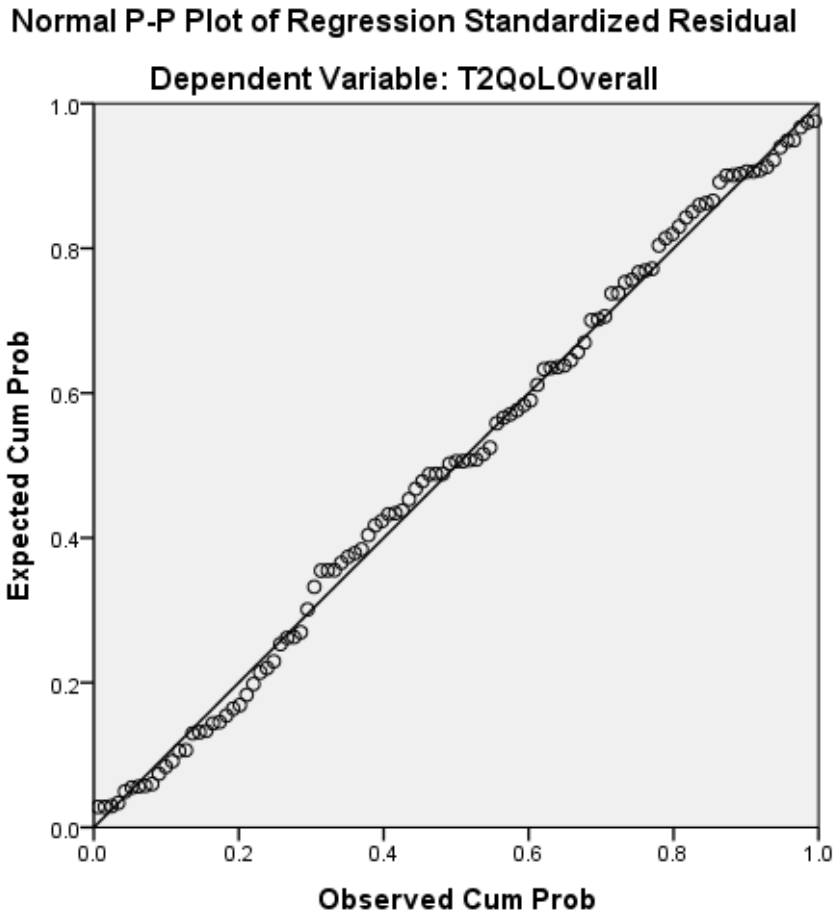
a. Dependent Variable: T2QoLOverall

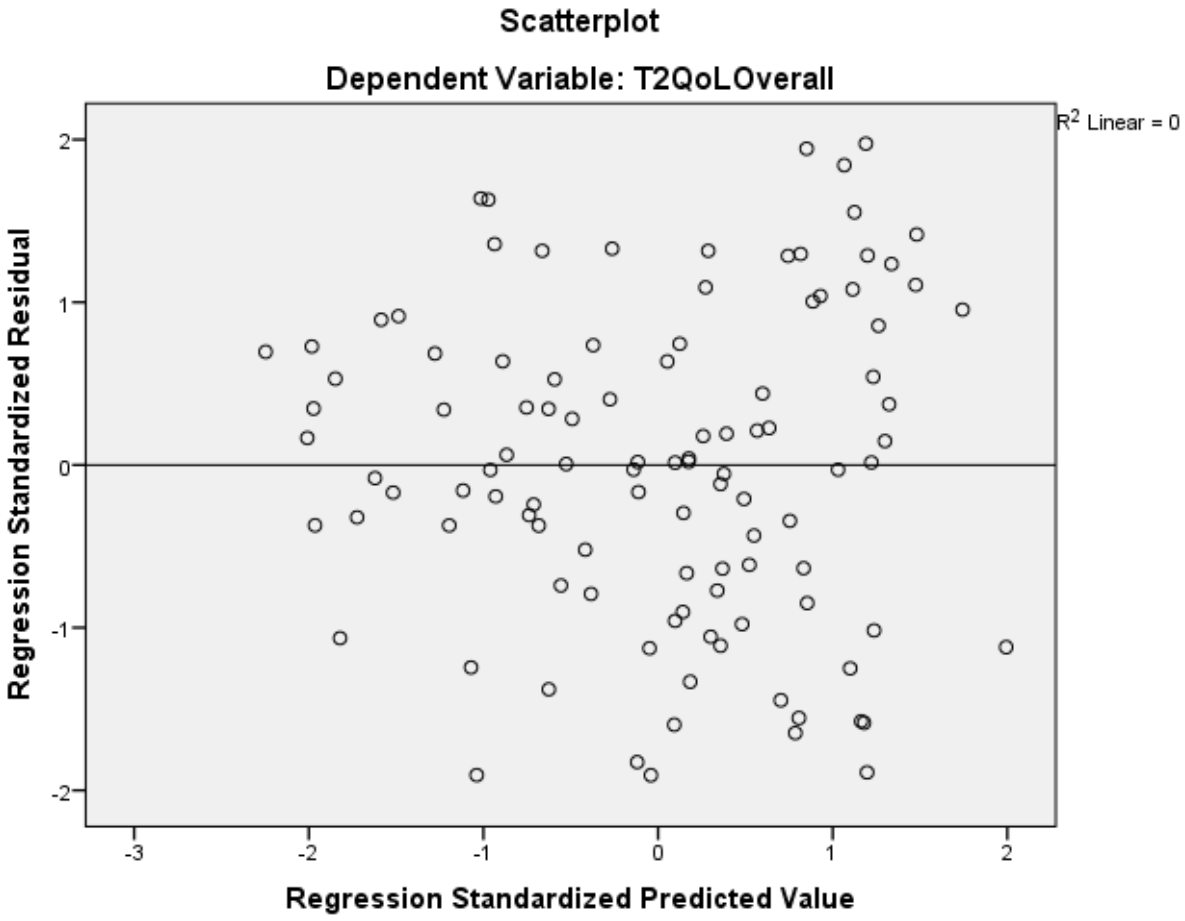
Residuals Statistics[a]

	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	12.5272	76.9439	46.6641	15.19083	107
Std. Predicted Value	-2.247	1.993	.000	1.000	107
Standard Error of Predicted Value	2.246	6.410	3.293	.682	107
Adjusted Predicted Value	11.4394	77.9595	46.6545	15.26355	107
Residual	-25.06767	25.96033	.00000	12.76762	107
Std. Residual	-1.907	1.975	.000	.971	107
Stud. Residual	-1.976	2.027	.000	1.001	107
Deleted Residual	-26.93591	27.56174	.00955	13.57752	107
Stud. Deleted Residual	-2.006	2.060	.000	1.008	107
Mahal. Distance	2.104	24.217	5.944	3.221	107
Cook's Distance	.000	.063	.009	.012	107
Centered Leverage Value	.020	.228	.056	.030	107

a. Dependent Variable: T2QoLOverall

Charts



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REGRESSION
  /DESCRIPTIVES MEAN STDDEV CORR SIG N
  /MISSING LISTWISE
  /STATISTICS COEFF OUTS R ANOVA COLLIN TOL CHANGE ZPP
  /CRITERIA=PIN(.05) POUT(.10)
  /NOORIGIN
  /DEPENDENT T3QoLOverall
  /METHOD=ENTER Age DV_StrokeSev_MildVSMod DV_StrokeSev_MildVSSev
  /METHOD=ENTER T1StressOverall
  /METHOD=ENTER T2StressOverall
  /METHOD=ENTER T3StressOverall
  /SCATTERPLOT=(*ZRESID ,*ZPRED)
  /RESIDUALS NORMPROB(ZRESID)
  /SAVE MAHAL COOK.
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Regression

Notes

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	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	143
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on cases with no missing values for any variable used.

Syntax		REGRESSION /DESCRIPTIVES MEAN STDDEV CORR SIG N /MISSING LISTWISE /STATISTICS COEFF OUTS R ANOVA COLLIN TOL CHANGE ZPP /CRITERIA=PIN(.05) POUT(.10) /NOORIGIN /DEPENDENT T3QoLOverall /METHOD=ENTER Age DV_StrokeSev_MildVSMod DV_StrokeSev_MildVSSev /METHOD=ENTER T1StressOverall /METHOD=ENTER T2StressOverall /METHOD=ENTER T3StressOverall /SCATTERPLOT=(*ZRESID,*ZPRED) /RESIDUALS NORMPROB(ZRESID) /SAVE MAHAL COOK.
Resources	Processor Time	00:00:00.37
	Elapsed Time	00:00:03.04
	Memory Required	19892 bytes
	Additional Memory Required for Residual Plots	528 bytes
Variables Created or Modified	MAH_7	Mahalanobis Distance
	COO_7	Cook's Distance

[DataSet1] H:\StrokeData_1.sav

Descriptive Statistics

	Mean	Std. Deviation	N
T3QoLOverall	48.9099	19.17206	99
Age	67.5556	14.46193	99
DV_StrokeSev_MildVSMod	.4343	.49819	99
DV_StrokeSev_MildVSSev	.2121	.41089	99
T1StressOverall	1.3802	.70229	99
T2StressOverall	1.5859	.74020	99
T3StressOverall	1.5418	.71144	99

Correlations

		T3QoLOverall	Age	DV_StrokeSev_MildVSMod	DV_StrokeSev_MildVSSev	T1StressOverall	T2StressOverall	T3StressOverall
Pearson Correlation	T3QoLOverall	1.000	-.316	-.040	-.348	-.157	-.451	-.582
	Age	-.316	1.000	.034	.198	-.376	.062	.040
	DV_StrokeSev_MildVSMod	-.040	.034	1.000	-.455	-.071	.018	-.015
	DV_StrokeSev_MildVSSev	-.348	.198	-.455	1.000	.094	.141	.224
	T1StressOverall	-.157	-.376	-.071	.094	1.000	.273	.185
	T2StressOverall	-.451	.062	.018	.141	.273	1.000	.454
	T3StressOverall	-.582	.040	-.015	.224	.185	.454	1.000
Sig. (1-tailed)	T3QoLOverall	.	.001	.347	.000	.061	.000	.000
	Age	.001	.	.369	.025	.000	.271	.348
	DV_StrokeSev_MildVSMod	.347	.369	.	.000	.244	.428	.442
	DV_StrokeSev_MildVSSev	.000	.025	.000	.	.177	.082	.013
	T1StressOverall	.061	.000	.244	.177	.	.003	.034
	T2StressOverall	.000	.271	.428	.082	.003	.	.000
	T3StressOverall	.000	.348	.442	.013	.034	.000	.
N	T3QoLOverall	99	99	99	99	99	99	99
	Age	99	99	99	99	99	99	99
	DV_StrokeSev_MildVSMod	99	99	99	99	99	99	99
	DV_StrokeSev_MildVSSev	99	99	99	99	99	99	99
	T1StressOverall	99	99	99	99	99	99	99
	T2StressOverall	99	99	99	99	99	99	99

T3StressOverall	99	99	99	99	99	99	99

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod[b]	.	Enter
2	T1StressOverall[b]	.	Enter
3	T2StressOverall[b]	.	Enter
4	T3StressOverall[b]	.	Enter

a. Dependent Variable: T3QoLOverall

b. All requested variables entered.

Model Summary[e]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.469[a]	.220	.196	17.19542	.220	8.942	3	95	.000
2	.528[b]	.279	.248	16.62283	.059	7.657	1	94	.007
3	.619[c]	.384	.351	15.45055	.105	15.805	1	93	.000
4	.720[d]	.518	.487	13.73248	.135	25.726	1	92	.000

a. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

b. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1StressOverall

c. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1StressOverall, T2StressOverall

d. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1StressOverall, T2StressOverall, T3StressOverall

e. Dependent Variable: T3QoLOverall

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	7931.823	3	2643.941	8.942	.000[b]
	Residual	28089.834	95	295.682		
	Total	36021.658	98			
2	Regression	10047.722	4	2511.930	9.091	.000[c]
	Residual	25973.936	94	276.318		
	Total	36021.658	98			
3	Regression	13820.745	5	2764.149	11.579	.000[d]
	Residual	22200.912	93	238.719		
	Total	36021.658	98			
4	Regression	18672.200	6	3112.033	16.502	.000[e]
	Residual	17349.457	92	188.581		
	Total	36021.658	98			

a. Dependent Variable: T3QoLOverall

b. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

c. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1StressOverall

d. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1StressOverall, T2StressOverall

e. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1StressOverall, T2StressOverall, T3StressOverall

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Correlations			Collinearity Statistics	
		B	Std. Error	Beta			Zero-order	Partial	Part	Tolerance	VIF
1	(Constant)	77.044	8.389		9.184	.000					
	Age	-.305	.124	-.230	-2.464	.016	-.316	-.245	-.223	.941	1.062
	DV_StrokeSev_MildVSMod	-8.228	3.955	-.214	-2.081	.040	-.040	-.209	-.188	.777	1.287
	DV_StrokeSev_MildVSSev	-18.629	4.889	-.399	-3.810	.000	-.348	-.364	-.345	.748	1.338
2	(Constant)	96.321	10.691		9.009	.000					
	Age	-.451	.131	-.340	-3.450	.001	-.316	-.335	-.302	.788	1.269
	DV_StrokeSev_MildVSMod	-7.924	3.825	-.206	-2.072	.041	-.040	-.209	-.181	.777	1.288
	DV_StrokeSev_MildVSSev	-16.275	4.802	-.349	-3.389	.001	-.348	-.330	-.297	.724	1.381
	T1StressOverall	-7.268	2.627	-.266	-2.767	.007	-.157	-.274	-.242	.829	1.207
3	(Constant)	100.420	9.990		10.052	.000					
	Age	-.379	.123	-.286	-3.084	.003	-.316	-.305	-.251	.771	1.298
	DV_StrokeSev_MildVSMod	-6.722	3.568	-.175	-1.884	.063	-.040	-.192	-.153	.771	1.297
	DV_StrokeSev_MildVSSev	-14.345	4.490	-.307	-3.195	.002	-.348	-.314	-.260	.716	1.397
	T1StressOverall	-4.191	2.561	-.154	-1.637	.105	-.157	-.167	-.133	.753	1.328
	T2StressOverall	-8.924	2.245	-.345	-3.976	.000	-.451	-.381	-.324	.882	1.133
4	(Constant)	108.771	9.031		12.044	.000					
	Age	-.382	.109	-.288	-3.493	.001	-.316	-.342	-.253	.771	1.298

	DV_StrokeSev_MildVSMod	-5.593	3.179	-.145	-1.760	.082	-.040	-.180	-.127	.767	1.303
	DV_StrokeSev_MildVSSev	-10.556	4.060	-.226	-2.600	.011	-.348	-.262	-.188	.691	1.446
	T1StressOverall	-3.525	2.280	-.129	-1.546	.126	-.157	-.159	-.112	.751	1.332
	T2StressOverall	-4.457	2.181	-.172	-2.044	.044	-.451	-.208	-.148	.739	1.354
	T3StressOverall	-11.336	2.235	-.421	-5.072	.000	-.582	-.467	-.367	.761	1.314

a. Dependent Variable: T3QoLOverall

Excluded Variables[a]

Model		Beta In	t	Sig.	Partial Correlation	Collinearity Statistics		
						Tolerance	VIF	Minimum Tolerance
1	T1StressOverall	-.266[b]	-2.767	.007	-.274	.829	1.207	.724
	T2StressOverall	-.387[b]	-4.648	.000	-.432	.971	1.030	.729
	T3StressOverall	-.518[b]	-6.709	.000	-.569	.940	1.064	.704
2	T2StressOverall	-.345[c]	-3.976	.000	-.381	.882	1.133	.716
	T3StressOverall	-.489[c]	-6.339	.000	-.549	.910	1.099	.692
3	T3StressOverall	-.421[d]	-5.072	.000	-.467	.761	1.314	.691

a. Dependent Variable: T3QoLOverall

b. Predictors in the Model: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

c. Predictors in the Model: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1StressOverall

d. Predictors in the Model: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1StressOverall, T2StressOverall

Collinearity Diagnostics[a]

Model	Dimension	Eigenvalue	Condition Index	Variance Proportions						
				(Constant)	Age	DV_StrokeSev_MildVSMod	DV_StrokeSev_MildVSSev	T1Stress Overall	T2Stress Overall	T3Stress Overall
1	1	2.732	1.000	.01	.01	.03	.02			
	2	1.000	1.653	.00	.00	.15	.39			
	3	.246	3.331	.03	.02	.82	.57			
	4	.021	11.282	.97	.97	.00	.02			
2	1	3.527	1.000	.00	.00	.02	.01	.01		
	2	1.001	1.877	.00	.00	.16	.37	.00		
	3	.294	3.465	.00	.00	.65	.49	.15		
	4	.163	4.650	.02	.07	.17	.09	.48		
	5	.014	15.638	.98	.93	.00	.04	.36		
3	1	4.395	1.000	.00	.00	.01	.01	.01	.01	
	2	1.001	2.095	.00	.00	.16	.36	.00	.00	
	3	.305	3.795	.00	.00	.67	.51	.10	.02	
	4	.164	5.177	.01	.06	.16	.08	.49	.01	
	5	.120	6.042	.02	.03	.01	.00	.07	.95	
	6	.014	17.457	.96	.91	.00	.04	.33	.00	
4	1	5.274	1.000	.00	.00	.01	.01	.00	.00	.00
	2	1.003	2.293	.00	.00	.16	.34	.00	.00	.00
	3	.309	4.132	.00	.00	.68	.52	.07	.02	.01
	4	.169	5.578	.00	.03	.12	.07	.58	.02	.07
	5	.136	6.221	.03	.07	.04	.00	.01	.23	.25
	6	.094	7.477	.00	.00	.00	.01	.01	.72	.66
	7	.014	19.255	.96	.90	.00	.05	.32	.00	.02

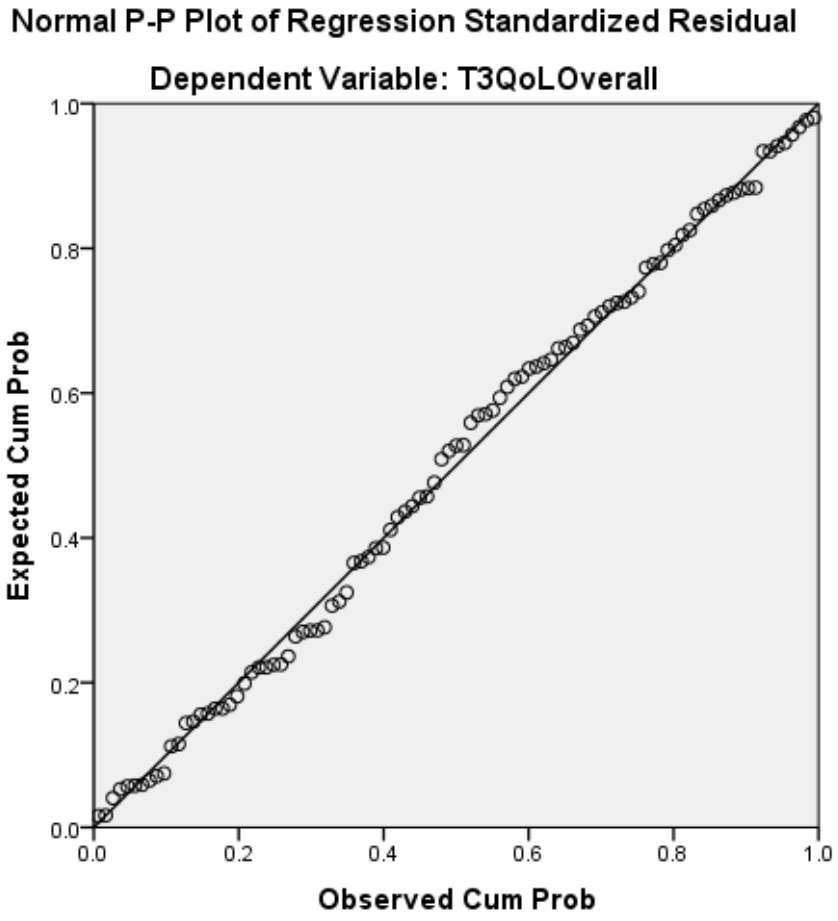
a. Dependent Variable: T3QoLOverall

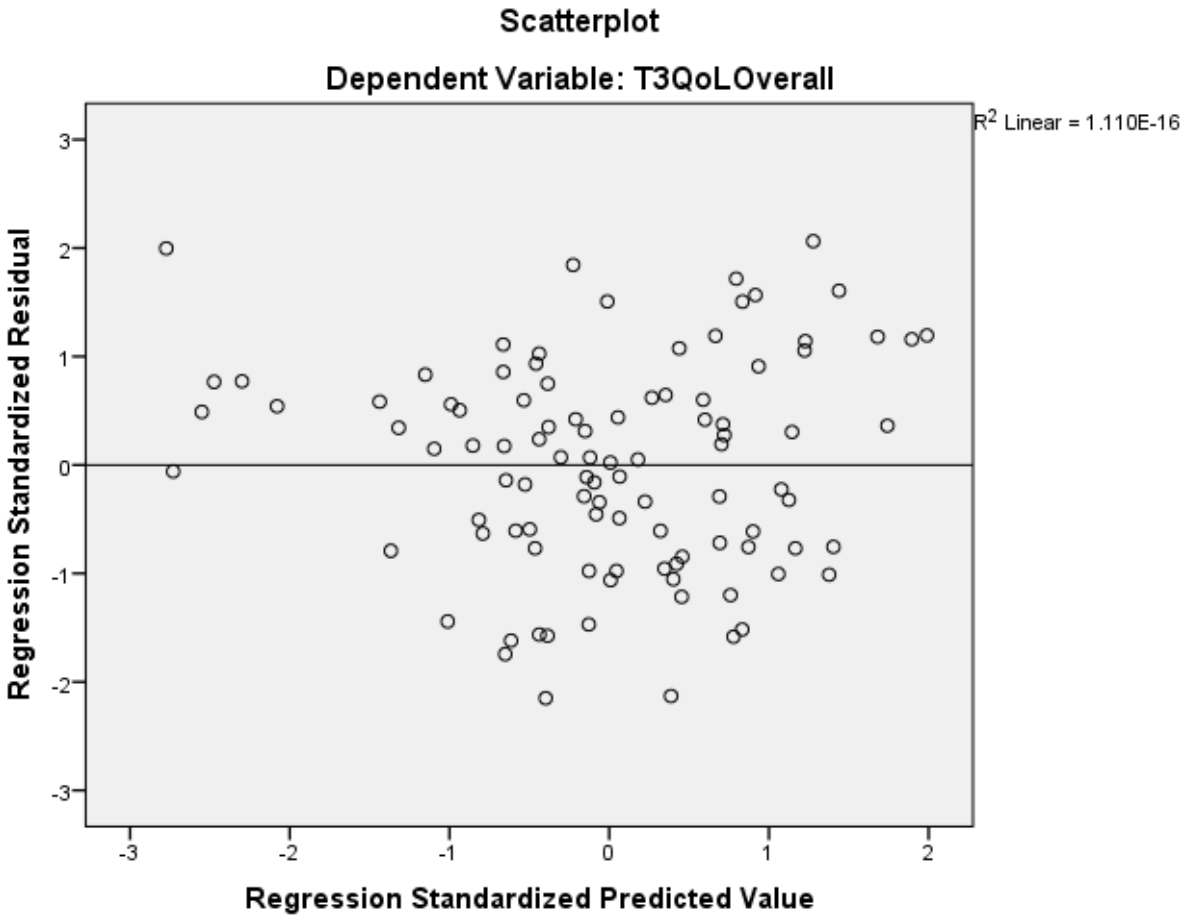
Residuals Statistics[a]

	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	10.6305	76.3694	48.9099	13.80336	99
Std. Predicted Value	-2.773	1.989	.000	1.000	99
Standard Error of Predicted Value	2.239	7.751	3.554	.843	99
Adjusted Predicted Value	7.4564	74.9658	48.9096	13.91414	99
Residual	-29.52672	28.32323	.00000	13.30546	99
Std. Residual	-2.150	2.062	.000	.969	99
Stud. Residual	-2.336	2.126	.000	1.005	99
Deleted Residual	-34.83780	30.59912	.00033	14.33311	99
Stud. Deleted Residual	-2.395	2.168	.000	1.013	99
Mahal. Distance	1.614	30.234	5.939	3.818	99
Cook's Distance	.000	.140	.011	.019	99
Centered Leverage Value	.016	.309	.061	.039	99

a. Dependent Variable: T3QoLOverall

Charts






```
REGRESSION
  /DESCRIPTIVES MEAN STDDEV CORR SIG N
  /MISSING LISTWISE
  /STATISTICS COEFF OUTS R ANOVA COLLIN TOL CHANGE ZPP
  /CRITERIA=PIN(.05) POUT(.10)
  /NOORIGIN
  /DEPENDENT T3QoLOverall
  /METHOD=ENTER Age DV_StrokeSev_MildVSMod DV_StrokeSev_MildVSSev
  /METHOD=ENTER T1BellsTotal
  /METHOD=ENTER T3BellsTotal
  /SCATTERPLOT=(*ZRESID ,*ZPRED)
  /RESIDUALS NORMPROB(ZRESID)
  /SAVE MAHAL COOK.
```

Regression

Notes

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Comments		
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	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	143
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on cases with no missing values for any variable used.

Syntax		REGRESSION /DESCRIPTIVES MEAN STDDEV CORR SIG N /MISSING LISTWISE /STATISTICS COEFF OUTS R ANOVA COLLIN TOL CHANGE ZPP /CRITERIA=PIN(.05) POUT(.10) /NOORIGIN /DEPENDENT T3QoLOverall /METHOD=ENTER Age DV_StrokeSev_MildVSMod DV_StrokeSev_MildVSSev /METHOD=ENTER T1BellsTotal /METHOD=ENTER T3BellsTotal /SCATTERPLOT=(*ZRESID,*ZPRED) /RESIDUALS NORMPROB(ZRESID) /SAVE MAHAL COOK.
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	Elapsed Time	00:00:01.84
	Memory Required	19604 bytes
	Additional Memory Required for Residual Plots	536 bytes
Variables Created or Modified	MAH_11	Mahalanobis Distance
	COO_11	Cook's Distance

[DataSet1] H:\StrokeData_1.sav

Descriptive Statistics

	Mean	Std. Deviation	N
T3QoLOverall	48.3200	18.68540	94
Age	66.7553	14.94627	94
DV_StrokeSev_MildVSMod	.4255	.49707	94
DV_StrokeSev_MildVSSev	.2340	.42567	94
T1BellsTotal	27.1277	8.64750	94
T3BellsTotal	29.8191	6.67842	94

Correlations

		T3QoLOverall	Age	DV_StrokeSev_MildVSMod	DV_StrokeSev_MildVSSev	T1BellsTotal	T3BellsTotal
Pearson Correlation	T3QoLOverall	1.000	-.341	-.104	-.324	.335	.445
	Age	-.341	1.000	.065	.166	-.135	-.306
	DV_StrokeSev_MildVSMod	-.104	.065	1.000	-.476	.052	.033
	DV_StrokeSev_MildVSSev	-.324	.166	-.476	1.000	-.210	-.265
	T1BellsTotal	.335	-.135	.052	-.210	1.000	.646
	T3BellsTotal	.445	-.306	.033	-.265	.646	1.000
Sig. (1-tailed)	T3QoLOverall	.	.000	.159	.001	.000	.000
	Age	.000	.	.267	.055	.097	.001
	DV_StrokeSev_MildVSMod	.159	.267	.	.000	.308	.376
	DV_StrokeSev_MildVSSev	.001	.055	.000	.	.021	.005
	T1BellsTotal	.000	.097	.308	.021	.	.000
	T3BellsTotal	.000	.001	.376	.005	.000	.
N	T3QoLOverall	94	94	94	94	94	94
	Age	94	94	94	94	94	94
	DV_StrokeSev_MildVSMod	94	94	94	94	94	94
	DV_StrokeSev_MildVSSev	94	94	94	94	94	94
	T1BellsTotal	94	94	94	94	94	94
	T3BellsTotal	94	94	94	94	94	94

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod[b]	.	Enter
2	T1BellsTotal[b]	.	Enter
3	T3BellsTotal[b]	.	Enter

a. Dependent Variable: T3QoLOverall

b. All requested variables entered.

Model Summary[d]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.501[a]	.251	.226	16.43371	.251	10.077	3	90	.000
2	.554[b]	.307	.275	15.90638	.055	7.066	1	89	.009
3	.583[c]	.339	.302	15.61187	.033	4.390	1	88	.039

a. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

b. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1BellsTotal

c. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1BellsTotal, T3BellsTotal

d. Dependent Variable: T3QoLOverall

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	8164.394	3	2721.465	10.077	.000[b]
	Residual	24306.017	90	270.067		
	Total	32470.412	93			
2	Regression	9952.263	4	2488.066	9.834	.000[c]
	Residual	22518.148	89	253.013		
	Total	32470.412	93			
3	Regression	11022.133	5	2204.427	9.045	.000[d]
	Residual	21448.279	88	243.730		
	Total	32470.412	93			

a. Dependent Variable: T3QoLOverall

b. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

c. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1BellsTotal

d. Predictors: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1BellsTotal, T3BellsTotal

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Correlations			Collinearity Statistics	
		B	Std. Error	Beta			Zero-order	Partial	Part	Tolerance	VIF
1	(Constant)	78.285	7.859		9.961	.000					
	Age	-.316	.117	-.253	-2.694	.008	-.341	-.273	-.246	.946	1.058
	DV_StrokeSev_MildVSMod	-10.772	3.952	-.287	-2.726	.008	-.104	-.276	-.249	.752	1.329
	DV_StrokeSev_MildVSSev	-18.366	4.671	-.418	-3.932	.000	-.324	-.383	-.359	.735	1.361
2	(Constant)	61.486	9.889		6.217	.000					
	Age	-.287	.114	-.229	-2.515	.014	-.341	-.258	-.222	.937	1.067
	DV_StrokeSev_MildVSMod	-10.378	3.828	-.276	-2.711	.008	-.104	-.276	-.239	.751	1.331
	DV_StrokeSev_MildVSSev	-16.093	4.601	-.367	-3.498	.001	-.324	-.348	-.309	.709	1.410
	T1BellsTotal	.522	.196	.241	2.658	.009	.335	.271	.235	.944	1.059
3	(Constant)	44.537	12.636		3.525	.001					
	Age	-.223	.116	-.179	-1.928	.057	-.341	-.201	-.167	.873	1.145
	DV_StrokeSev_MildVSMod	-9.943	3.763	-.265	-2.642	.010	-.104	-.271	-.229	.749	1.335
	DV_StrokeSev_MildVSSev	-14.675	4.566	-.334	-3.214	.002	-.324	-.324	-.278	.694	1.442
	T1BellsTotal	.199	.247	.092	.808	.421	.335	.086	.070	.576	1.735
	T3BellsTotal	.703	.335	.251	2.095	.039	.445	.218	.182	.522	1.916

a. Dependent Variable: T3QoLOverall

Excluded Variables[a]

Model		Beta In	t	Sig.	Partial Correlation	Collinearity Statistics		
						Tolerance	VIF	Minimum Tolerance
1	T1BellsTotal	.241[b]	2.658	.009	.271	.944	1.059	.709
	T3BellsTotal	.312[b]	3.334	.001	.333	.855	1.169	.695
2	T3BellsTotal	.251[c]	2.095	.039	.218	.522	1.916	.522

a. Dependent Variable: T3QoLOverall

b. Predictors in the Model: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod

c. Predictors in the Model: (Constant), DV_StrokeSev_MildVSSev, Age, DV_StrokeSev_MildVSMod, T1BellsTotal

Collinearity Diagnostics[a]

Model	Dimension	Eigenvalue	Condition Index	Variance Proportions					
				(Constant)	Age	DV_StrokeSev_MildVSMod	DV_StrokeSev_MildVSSev	T1BellsTotal	T3BellsTotal
1	1	2.741	1.000	.01	.01	.03	.02		
	2	1.000	1.656	.00	.00	.16	.36		
	3	.235	3.416	.03	.03	.81	.61		
	4	.024	10.774	.96	.97	.00	.01		
2	1	3.623	1.000	.00	.00	.02	.01	.01	
	2	1.003	1.901	.00	.00	.15	.36	.00	
	3	.286	3.561	.01	.00	.77	.47	.05	
	4	.070	7.173	.02	.26	.07	.16	.63	
	5	.018	14.205	.98	.73	.00	.00	.32	
3	1	4.566	1.000	.00	.00	.01	.01	.00	.00
	2	1.004	2.132	.00	.00	.14	.35	.00	.00
	3	.316	3.802	.00	.00	.75	.42	.02	.01
	4	.075	7.778	.01	.29	.10	.20	.21	.02
	5	.029	12.591	.07	.19	.00	.00	.69	.32
	6	.010	21.063	.92	.52	.01	.02	.09	.65

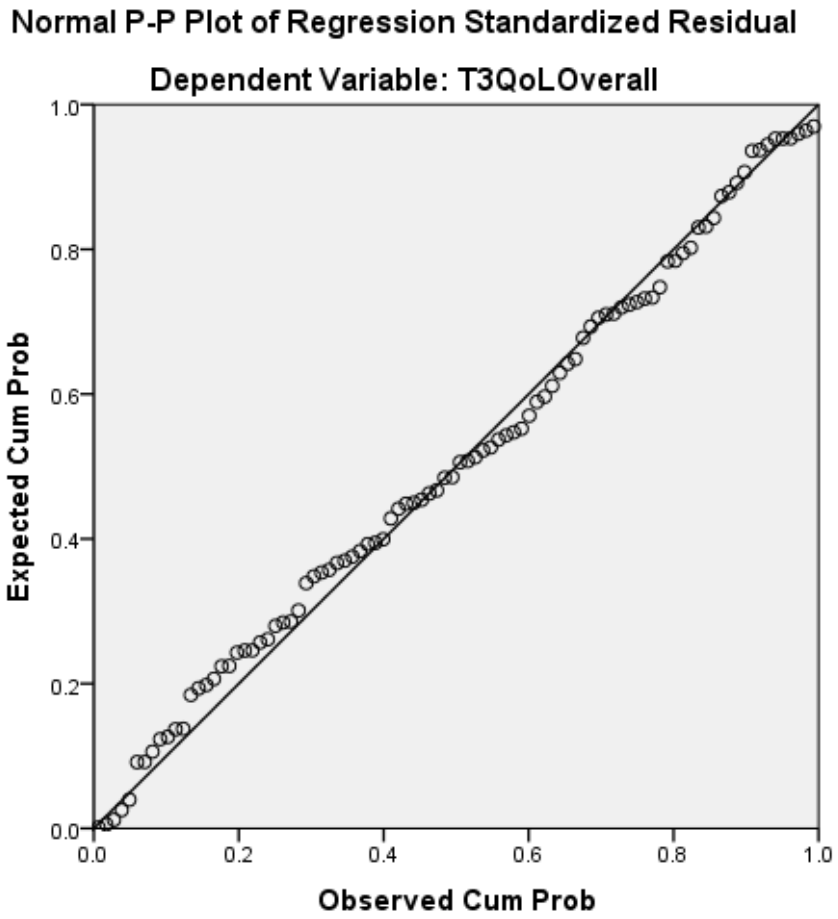
a. Dependent Variable: T3QoLOverall

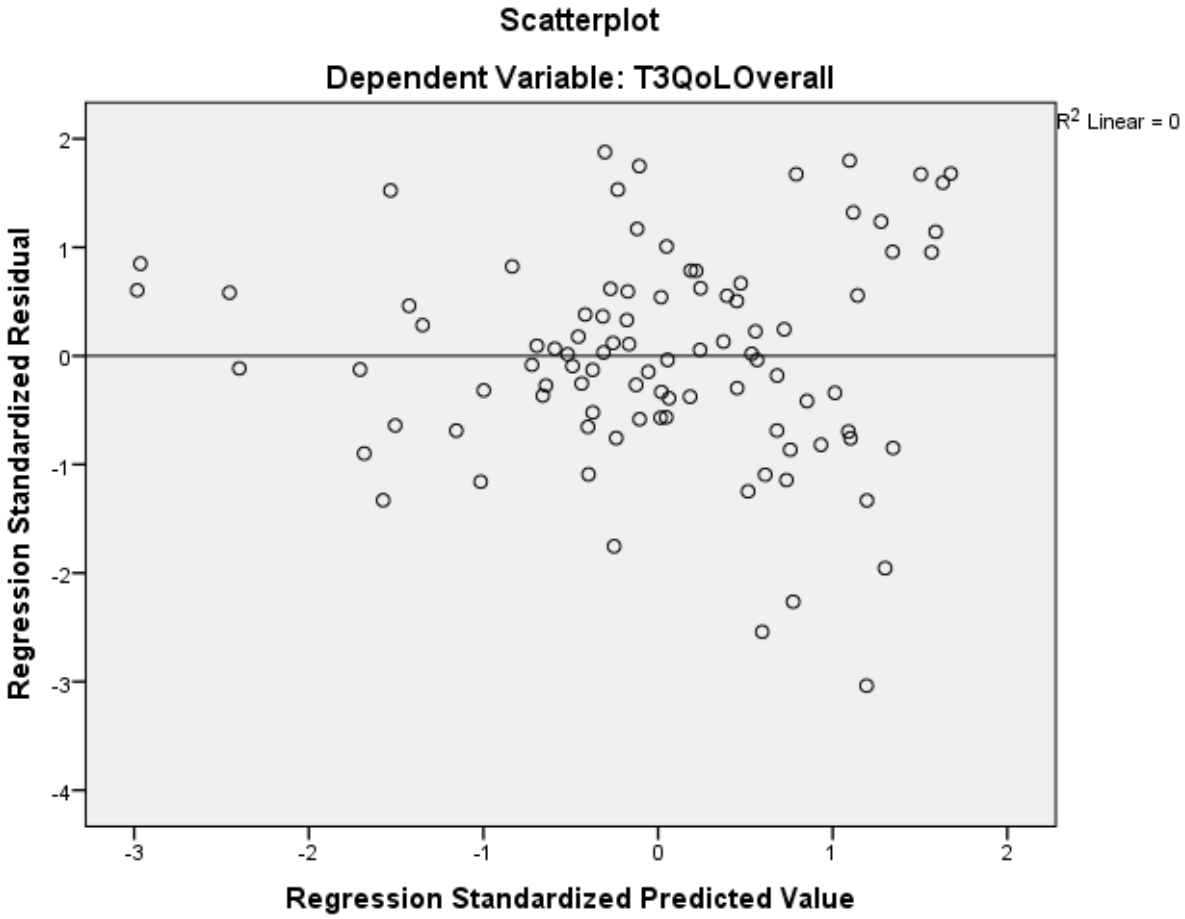
Residuals Statistics[a]

	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	15.8487	66.5677	48.3200	10.88658	94
Std. Predicted Value	-2.983	1.676	.000	1.000	94
Standard Error of Predicted Value	2.481	7.326	3.796	1.077	94
Adjusted Predicted Value	13.3595	64.6834	48.2547	11.12308	94
Residual	-47.42613	29.30050	.00000	15.18640	94
Std. Residual	-3.038	1.877	.000	.973	94
Stud. Residual	-3.098	1.905	.002	1.001	94
Deleted Residual	-49.33508	30.18178	.06531	16.08832	94
Stud. Deleted Residual	-3.264	1.934	.000	1.015	94
Mahal. Distance	1.360	19.487	4.947	3.787	94
Cook's Distance	.000	.075	.010	.015	94
Centered Leverage Value	.015	.210	.053	.041	94

a. Dependent Variable: T3QoLOverall

Charts


Normal P-P Plot of Regression Standardized Residual
Dependent Variable: T3QoLOverall
Expected Cum Prob
Observed Cum Prob
0.0
0.2
0.4
0.6
0.8
1.0


Scatterplot
Dependent Variable: T3QoLOverall
R2 Linear = 0
Regression Standardized Residual
Regression Standardized Predicted Value
2
1
0
-1
-2
-3
-4
-3
-2
-1
0
1
2